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DC Dutta's Textbook of OBSTETRICS

including Perinatology and Contraception

Eighth Edition

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Dedicated to

The students of obstetrics
past and present

Preface to the Eighth Edition

DC Dutta's Textbook of Obstetrics is in service to the medical fraternity for the last 31 years. It primarily aims at the medical students, trainee residents, practicing doctors and the midwives. It has evolved to provide comprehensive and updated information in a concise and easy-to-read format. The eighth edition has come out with an international standard to meet the overwhelming demand in many parts of the world. All the chapters have been extensively revised and strategically reorganized. Medical advances up to the time of publication have been incorporated. Contemporaneous guidelines from different professional and academic organizations like RCOG, ACOG, WHO, FIGO, NICHD, CDC, NICE, ICOG and DIPSI are provided. Management options based on experience, derived from large obstetric services are also mentioned. This is particularly so in a situation where no evidence exists or it is difficult to follow due to limitation of resources. Objective of this edition is to provide current cutting edge information, to enable the candidates to qualify the examination in India and its equivalents internationally.

Dutta's eighth edition in its fully coloured format is profusely illustrated with a total number of 320 line drawings, sketches and photographs. In recognition of the advanced technology, the book provides sonograms, including Doppler studies, M R Images, microphotographs, data graphs and laparoscopic images. All these are in most vivid colors. Practical obstetrics (Chapter 42) with a total number of 52 high-quality photographs of instruments, specimens, sonograms, M R Images and drugs is of immense value, specifically for the practical part of the examination. The total information in Chapter 42 amounts to a *mini textbook-cum-colour atlas* in obstetrics.

For easy text reference and reading, contents and index have also been expanded. A list of abbreviations and a few updated reviews with websites are provided. The uniqueness of this text lies in its presentations, which are simple, lucid and unambiguous. Presentation of summary tables, algorithms and key-points for each chapter are a special attraction. These are for quick revision and recapitulation before the examination.

This book is available in various e-Book formats. Dutta's has a long-standing association with its sister books: Textbook of Gynecology (6th Edition–2013), Bedside Clinics and Viva-voce (1st Edition–2015), Master Pass in Obstetrics and Gynaecology (2nd Edition–2015)

I had the opportunity to visit many of the medical institutions in this country and abroad. The feedback that I received from the teachers and students was invaluable. Many of these suggestions have been addressed to in this edition.

I do hope this comprehensive textbook will continue to be of immense educational resource to the readers as ever.

I am grateful to all who have taught me, most of all the patients and my beloved students.

According to the author's desire, the book is dedicated to the ***students of obstetrics—past and present***, who strive continuously to improve maternal and newborn's health, wherever they work.

P-13, New CIT Road
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HIRALAL KONAR

Preface to the First Edition

Over the years, there was an absolute dearth of a single comprehensive textbook of obstetrics, worth to be prescribed to the students. Moreover, of the textbooks currently available, most have been written with an orientation for the developed countries.

Being constantly insisted and hard-pressed by my beloved students, I ultimately decided to write a compact, comprehensive and practically oriented textbook of obstetrics. It is an attempt to encourage the students to learn obstetrics in a comparatively easy way. The aim was to emphasize the simplicities rather than complexities of knowledge. The book is written in a clear and concise language and in author's own style, which holds the reader's interest. Controversies are avoided and the management of the obstetrical problems is being highlighted with the facilities available to most of the third world countries. Extensive illustrations and flow charts (schemes) have been used as and when needed to add lucidity and clarity to the subject and to emphasize the practical nature of the book.

Although the book has been written primarily for the undergraduates, it should also prove to be useful to nurses (midwives), those aspiring for diploma and postgraduate degrees in obstetrics and also to the practicing obstetricians. I, however, do not consider this book to be an ideal one but a humble attempt has been made to remove the bottlenecks, as far as possible, of the books available to the students at present.

Acknowledgments: Very little of what is worthwhile in this book could not have been brought to publication without the generous cooperation, advice and assistance of many of my colleagues, seniors and juniors.

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I have much pleasure in expressing my cordial appreciation to the house-surgeons, internees and students of Nilratan Sircar Medical College, Calcutta for all the help they have rendered in the preparation of the final drafts of the manuscripts, checking the proofs and in compiling the Index. Without their constant encouragement and active assistance, this book could never have been published. I express my sincere thanks to my publisher 'Central Educational Enterprises' for their sincere efforts in publishing the book within the stipulated period in spite of the adverse circumstances.

In preparing a textbook like this, I have utilized the knowledge of a number of stalwarts in my profession and consulted many books and publications. I wish to express my appreciation and gratitude to all of them, including the related authors and publishers.

As a teacher, I have learnt a lot from the students and more so while writing this book and, as such, I could not think of dedicating the book to anyone else but the *students of obstetrics* for which I express my gratitude.

DC DUTTA

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Acknowledgments

The job of editing such a comprehensive text is stupendous. I have consulted many of my esteemed colleagues in the profession in this country and abroad, multitude of eminent authors, many current evidence-based studies, guidelines and recommendations. I do gratefully acknowledge my legacy to all the teachers, related authors and the publishers.

I express my sincere thanks to all the teachers and students of different medical institutes, midwifery institutes, nursing colleges in India and abroad for their valued suggestions, new ideas and contribution of photographs. The editor always welcomes the views of the students and the teachers through online access of the Student-Teacher Platform in our websites hiralalkonar.com and dcdutta.com, and e-mail ID h.kondr@gmail.com.

The eighth edition gratefully acknowledges the insightful wisdom of the following teachers, who are associated since the first edition of the book.

Dr KM Gun MD, FRCOG, FRCS, FACS, Professor (Rtd), Dr BN Chakravorty MD, FRCOG, DSc, Director, Institute of Reproductive Medicine, Kolkata, Dr B Hore MD, Professor (Rtd), Consultant Anesthetist, Dr Subir Kumar Dutta MD, Professor (Rtd), Consultant Pathologist for their contribution, continued guidance and valuable suggestions. The manuscript of the chapter on perinatology (Chapter 33) has been thoroughly read and authoritatively revised by Dr Shyamal Banerjee, MD, Professor, Department of Pediatrics, NRS Medical College Hospital, Kolkata, for which he deserves special appreciations. The author expresses his sincere thanks to Professor S Banerjee, DME, Dept. of H&FW, Govt. of West Bengal, Professor (Mrs) M Roy, Principal, Professor PB Chakraborty, MSVP and Professor (Mrs) A Biswas, Head of the Dept., Obs. & Gynae. for their support and encouragement. Mrs Madhusri Konar, MA, BEd deserves full credit for her sincere and patient secretarial job as ever. General Electrical Pvt Ltd is appreciated for the good quality of ultrasonograms. During the production of this textbook, I had the continued assistance and support of a good number of skilled personnel from the publishing house. The author gratefully acknowledges Shri Jitendar P Vij, Group Chairman and Mr Ankit Vij, Group President, Jaypee Brothers Medical Publishers (P) Ltd for their generous support. The author expresses his sincere thanks to Dr Sakshi Arora, Dr Mrinalini Bakshi, Ms Nitasha Arora, along with the entire team of Jaypee Brothers Medical Publishers (P) Ltd, New Delhi for their professional guidance, suggestions and support in bringing out this thoroughly revised eighth edition.

I would like to extend my sincere thanks to all the respected teachers and beloved students, who contacted me with their suggestions for the improvement of this book. Their input has been invaluable and is much appreciated. I wish I could acknowledge each one of them in writing.

Hiralal Konar

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Abbreviations

AC : Abdominal Circumference
aCL : anti-Cardiolipin Antibodies
ACOG : American College of Obstetricians and Gynecologists
AFI : Amniotic Fluid Index
AFP : Alpha Fetoprotein
AFV : Amniotic Fluid Volume
AGS : Antigas Gangrene Serum
ALT : Alanine Amniotransferase
AMTSL : Active Management of Third Stage of Labor
AN : Atrial Natriuretic Factor
APH : Antepartum Hemorrhage
aPLs : anti-Phospholipid Antibodies
APTT : Activated Partial Thromboplastin Time
ARDS : Acute Respiratory Distress Syndrome
ARM : Artificial Rupture of Membranes
ART : Assisted Reproductive Technology
ASD : Atrial Septal Defect
AST : Aspartate Amniotransferase
ATP : Adenosine Triphosphate
ATS : Anti-Tetanus Serum
AUA : Abnormal Uterine Action
β2GP-1 : β2 Glycoprotein-1
BMI : Body Mass Index
BPD : Biparietal Diameter
BPD : Broncho-pulmonary Dysplasia
BPP : Biophysical Profile
CBG : Corticosteroid-binding Globulin
CCF : Congestive Cardiac Failure
CDC : Center for Disease Control
CH : Crown Heel
CIN : Cervical Intraepithelial Neoplasia
CIRCI : Critical Illness-related Corticosteroid Insufficiency
CMQCC : California Maternal Quality Care Collaboration
CO : Cardiac Output
COCs : Combined Oral Contraceptives
COX : Cyclo-oxygenase
CPAP : Continuous Positive Airway Pressure
CPD : Cephalopelvic Disproportion
CPK : Creatinine Phosphokinase
CPT : Complete Perineal Tear
CRH : Corticotrophin-releasing Hormone
CRL : Crown Rump Length
CRP : C-Reactive Protein
CRS : Congenital Rubella Syndrome
CS : Cesarean Section
CSE : Combined Spinal Epidural
CST : Contraction Stress Test
CT : Computed Tomography
CTG : Cardiotocography
CTPA : Computed Tomographic Pulmonary Angiography
CVP : Central Venous Pressure
CVS : Chorionic Villus Sampling
CVS : Congenital Vericella Syndrome
CXR : Chest X-ray
D/D : Diamniotic-Dichorionic
D/M : Diamniotic-Monochorionic

D&E : Dilatation and Evacuation
DBP : Diastolic Blood Pressure
DFMC : Daily Fetal Movement Counting
DIC : Disseminated Intravascular Coagulopathy
DIPSI : Diabetes in Pregnancy Study Group India
DMPA : Depot Medroxy Progesterone Acetate
DTA : Deep Transverse Arrest
DV : Ductus Venosus
DVT : Deep Vein Thrombosis
EFM : Electronic Fetal Monitoring
EACA : Epsilon Amino Caproic Acid
EAS : External Anal Sphincter
ECG : Electro Cardiography
ECV : External Cephalic Version
EDD : Expected Date of Delivery
EFM : Electronic Fetal Monitoring
EGN : Etonogestrel
EmOC : Emergency Obstetric Care
EmONC : Emergency Obstetric and Newborn Care
EPF : Early Pregnancy Factor
ERPC : Evacuation of Retained Products of Conception
ES : Embryonic Stem
FBS : Fetal Blood Sampling
FDP : Fibrin Degradation Products
FFA : Free Fatty Acid
ffDNA : free fetal DNA
FFP : Fresh Frozen Plasma
FGR : Fetal Growth Restriction
FHR : Fetal Heart Rate
FHS : Fetal Heart Sound
FIGO : International Federation of Gynecology and Obstetrics
FISH : Fluorescence in situ Hybridization
FL : Femur Length
FMH : Fetomaternal Hemorrhage
FRU : First Referral Unit
FSH : Follicle-stimulating Hormone
GA : Gestational Age
GBS : Group B Streptococcus
GDM : Gestational Diabetes Mellitus
GLUT : Glucose Transporter
GLUT-1 : Glucose Transporter-1
GnRH : Gonadotropin-releasing Hormone
GTN : Gestational Trophoblastic Neoplasia
GTN : Glyceryl Trinitrate
GTT : Glucose Tolerance Test
HAART : Highly Active Anti-retroviral Therapy
Hb : Hemoglobin
HC : Head Circumference
hCG : Human Chorionic Gonadotropin
HCS : Hemopoietic Stem Cells
hCT : Human Chorionic Thyrotropin
HDFN : Hemolytic Disease of the Fetus and Newborn
HDL : High Density Lipoprotein
HELLP : Hemolysis, Elevated Liver Enzymes, Low Platelet
HIE : Hypoxic Ischemic Encephalopathy
HIV : Human Immunodeficiency Virus
HLA : Human Leucocyte Antigen
HMD : Hyaline Membrane Disease
HPL : Human Placental Lactogen
HR : Heart Rate
IAS : Internal Anal Sphincter
ICA : Incordinate Uterine Action
ICH : Intracranial Hemorrhage
ICOG : Indian College of Obstetricians & Gynaecologists
IFM : Intrapartum Fetal Monitoring
IGF-1 : Insulin Growth Factor-1
IgG : Immunoglobulin G
IOL : Induction of Labour
IPT : Intraperitoneal Transfusion
ITU : Intensive Care Unit
IUD : Intrauterine Device
IUCD : Intrauterine Contraceptive Device
IUFD : Intrauterine Fetal Death
IUGR : Intrauterine Growth Restriction
IUT : Intrauterine Transfusion
IVC : Inferior Vena Cava
IVH : Intraventricular Hemorrhage
IVIg : Intravenous Immunoglobulin
IVT : Intravascular Transfusion
JSY : Janani Suraksha Yojana

KB : Kleihauer-Betke
L : S Ratio : Lecithin-Sphingomyelin Ratio
L/S : Lecithin/Sphingomyelin
LA : Lupus Anticoagulant
LAM : Lactational Amenorrhoea
LBW : Low Birth Weight
LDH : Lactic Dehydrogenase
LDL : Low Density Lipoprotein
LGA : Large for Gestational Age
LH : Luteinizing Hormone
LMA : Left Mentoanterior
LMP : Last Menstrual Period
LMWH : Low Molecular Weight Heparin
LNG-IUS : Levonorgestrel-Intrauterine System
LOA : Left Occiput Anterior
LOP : Left Occiput Posterior
LOT : Left Occiput Transverse
LPD : Luteal Phase Defect
LSA : Left Sacrum Anterior
LSCS : Lower Segment Cesarean Section
LVP : Largest Vertical Pocket
M/M : Monoamniotic-Monochorionic
MAP : Mean Arterial Pressure
MCA : Middle Cerebral Artery
MCU : Microcirculatory Unit
MDGs : Millennium Development Goals
MFMU : Maternal Fetal Medicine Unit
MLCK : Myocin Light Chain Kinase
MMR : Maternal Mortality Ratio
MOMs : Multiples of the Medians
MRA : Magnetic Resonance Angiography
MRI : Magnetic Resonance Imaging
MSAFP : Maternal Serum Alphafeto Protein
MVA : Manual Vacuum Aspiration
NEC : Necrotising Enterocolitis
NICE : National Institute of Clinical Excellence
NICHD : National Institute of Child Health and Development
NICU : Neonatal Intensive Care Unit
NK : Natural Killer Cells
NO : Nitric Oxide
NPN : Non-Protein Nitrogen
NRFS : Non-reassuring Fetal Status
NST : Non-stress Test
NSV : No-Scalpel Vasectomy
NT : Nuchal Translucency
NTD : Neural Tube Defect
OA : Occiput Anterior
OGN : Oestrogen
OP : Occiput Posterior
OT : Operation Theatre
PAPP-A : Pregnancy-associated Plasma Protein-A
PBI : Protein-bound Iodine
PCA : Patient-controlled Analgesia
PCOS : Polycystic Ovarian Syndrome
PCR : Polymerase Chain Reaction
PCWP : Pulmonary Capillary Wedge Pressure
PDA : Patent Ductus Arteriosus
PDS : Poly Dioxanone
PE : Pulmonary Embolism
PG : Phosphatidyglycerol
PGD : Preimplantation Genetic Diagnosis
PGN : Progestin
PGs : Prostaglandins
PI : Pulsality Index
PID : Pelvic Inflammatory Disease
PIH : Pregnancy Induced Hypertension
PO : Per Oral
POD : Pouch of Douglas
POP : Persistent Occiput Posterior
PPBs : Postprandial Blood Sugar
PPH : Postpartum Haemorrhage
PRES : Posterior Reversible Encephalopathy Syndrome
PROM : Prelabour Rupture of Membranes
PSV : Peak Systolic Velocity
PT : Prothrombin Time
PTB : Preterm Birth
PTH : Parathyroid Hormone
PVL : Periventricular Leukomalacia
PVP : Pulmonary Vascular Resistance
RAAS : Renin Angiotensin Aldosterone System
RBC : Red Blood Cells
RCOG : Royal College of Obstetricians and Gynaecologists
RDS : Respiratory Distress Syndrome

RhIg : Rhesus Immunoglobulin
RI : Resistance Index
RNTCR : Revised National Tuberculosis Control Programme
ROA : Right Occiput Anterior
ROP : Right Occiput Posterior
ROS : Reactive Oxygen Species
ROT : Right Occiput Transverse
RR : Respiratory Rate
RSP : Right Sacro Posterior
S C : Sub Cutaneous
SBP : Systolic Blood Pressure
SCJ : Squamocolumnar Junction
SFH : Symphysis Fundal Height
SGA : Small for gestational Age
SIRS : Systemic Inflammatory Response Syndrome
SLE : Systemic Lupus Erythematosus
SMI : Safe Motherhood Initiative
SV : Stroke Volume
SVD : Systemic Vascular Resistance
TA : Transabdominal
TAS : Transabdominal Sonography
TBG : Thyroxin Binding Globulin
TC : Transcervical
TDI : Total Dose Infusion
TDO : Transverse Diameter of the Outlet
TGFβ : Transforming Growth Factor β
TNFα : Tumour Necrosis Factor-α
TOLAC : Trial of Labour After Caesarean
TPS : Trans perineal Sonography
TRAP : Twin Reverse Arterial Perfusion
TTTS : Twin to Twin Transfusion Syndrome
TVS : Trans Vaginal Sonography
UE3 : Unconjugated Estriol
USG : Ultrasonogrpahy
UTI : Urinary Tract Infection
UV : Umbilical Vein
V/Q : Ventilation/Perfusion Ratio
VBAC : Vaginal Birth After Cesarean
VBAC-TOL : VBAC-Trial of Labor
VEGF : Vascular Endothelial Growth Factor
VSD : Ventricular Septal Defect
VUS : Venous Ultrasonography
VVF : Vesico Vaginal Fistula
WHO : World Health Organization

Chapter 1

Anatomy of Female Reproductive Organs

The reproductive organs in female are those which are concerned with copulation, fertilization, growth and development of the fetus and its subsequent exit to the outer world. The organs are broadly divided into:

- **External genitalia**
- **Internal genitalia**
- **Accessory reproductive organs**

EXTERNAL GENITALIA

(Synonyms: Vulva, Pudendum)

The vulva or pudendum includes all the visible external genital organs in the perineum. Vulva consists of the following: the mons pubis, labia majora, labia minora, hymen, clitoris, vestibule, urethra and Skene's glands, Bartholin's glands and vestibular bulbs (Fig. 1.1). It is therefore bounded anteriorly by mons pubis, posteriorly by the rectum, laterally by the genitocrural fold. The vulvar area is covered by keratinized stratified squamous epithelium.

MONS VENERIS (MONS PUBIS): It is the pad of subcutaneous adipose connective tissue lying in front of the pubis and in the adult female is covered by hair. The hair pattern (escutcheon) of most women is triangular with the base directed upwards.

LABIA MAJORA: The vulva is bounded on each side by the elevation of skin and subcutaneous tissue which form the labia majora. They are continuous where they join medially to form the **posterior commissure** in front of the anus. The skin on the outer convex surface is pigmented and covered with hair follicle. The thin skin on the inner surface has sebaceous glands but no hair follicle. The labia majora are covered with squamous epithelium and contain sweat glands. Beneath the skin, there is dense connective tissue and adipose tissue. The adipose tissue is richly supplied with venous plexus which may produce hematoma, if injured during childbirth. **The labia majora are homologous to the scrotum in the male.** The round ligament terminates at its upper border.

LABIA MINORA: They are two thin folds of skin, devoid of fat, on either side just within the labia majora. Except in the parous women, they are exposed only when the labia majora are separated. Anteriorly, they divide to enclose the clitoris and unite with each other in front and behind the clitoris to form the prepuce and frenulum respectively. The lower portion of the labia minora fuses across the midline to form a fold of skin known as **fourchette**. It is usually lacerated during childbirth. Between the fourchette and the vaginal orifice is the **fossa navicularis**. The labia minora contain no hair follicles or sweat glands. The folds contain connective tissues, numerous sebaceous glands, erectile muscle fibers and numerous vessels and nerve endings. **The labia minora are homologous to the penile urethra and part of the skin of penis in males**.

CLITORIS: It is a small cylindrical erectile body, measuring about 1.5–2 cm situated in the most anterior part of the vulva. **It consists of a glans, a body and two crura**. The clitoris consists of two cylindrical corpora cavernosa (erectile tissue). The glans is covered by squamous epithelium and is richly supplied with nerves. The vessels of the clitoris are connected with the vestibular bulb and are liable to be injured during childbirth. **Clitoris is homologous to the penis in the male** but it differs in being entirely separate from the urethra. It is attached to the under surface of the symphysis pubis by the suspensory ligament.

VESTIBULE: It is a triangular space bounded anteriorly by the clitoris, posteriorly by the fourchette and on either side by labia minora. **There are four openings into the vestibule**.

(a) *Urethral opening*: The opening is situated in the midline just in front of the vaginal orifice about 1–1.5 cm below the pubic arch. The paraurethral ducts open either on the posterior wall of the urethral orifice or directly into the vestibule.

(b) *Vaginal orifice and hymen*: The vaginal orifice lies in the posterior end of the vestibule and is of varying size and shape. In virgins and nulliparae, the opening is closed by the labia minora, but in parous, it may be exposed. It is incompletely closed by a septum of mucous membrane, called hymen. The membrane varies in shape but is usually circular or crescentic in virgins. The hymen is usually ruptured at the consummation of marriage. During childbirth, the hymen is extremely lacerated and is later represented by cicatrized nodules of varying size, called the carunculae myrtiformes. **On both sides it is lined by stratified squamous epithelium**.

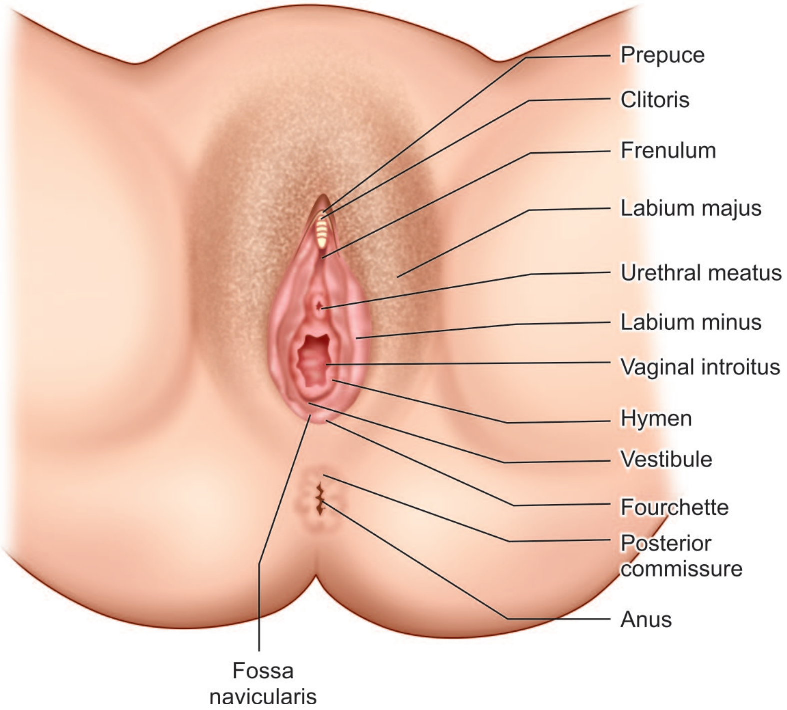


Fig. 1.1: The virginal vulva

(c) *Opening of Bartholin's ducts*: There are two Bartholin glands (greater vestibular gland), one on each side. **They are situated in the superficial perineal pouch**, close to the posterior end of the vestibular bulb. They are pea-sized and yellowish white in color. During sexual excitement, it secretes abundant alkaline mucus which helps in lubrication. The glands are of compound racemose variety and are lined by cuboidal epithelium. Each gland has got a duct which measures about 2 cm and opens into the vestibule outside the hymen at the junction of the anterior two-third and posterior one-third in the groove between the hymen and the labium minus. **The duct is lined by columnar epithelium but near its opening by stratified squamous epithelium. Bartholin's glands are homologous to the bulb of the penis in male.**

(d) Skene's glands are the largest paraurethral glands. **Skene's glands are homologous to the prostate in the male. The two Skene's ducts** may open in the vestibule on either side of the external urethral meatus.

VESTIBULAR BULB: These are bilateral elongated masses of erectile tissues situated beneath the mucous membrane of the vestibule. Each bulb lies on either side of the vaginal orifice in front of the Bartholin's gland and is incorporated with the bulbocavernosus muscle. **They are homologous to the bulb of the penis and corpus spongiosum in the male**. They are likely to be injured during childbirth with brisk hemorrhage (Fig. 1.2).

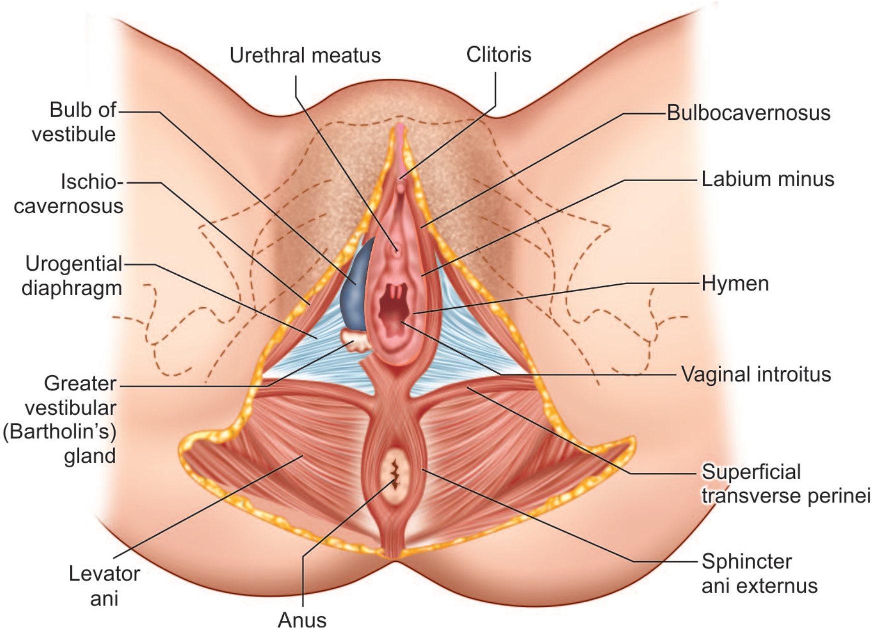

Urethral meatus
Clitoris
Bulb of vestibule
Bulbocavernosus
Ischio-cavernosus
Labium minus
Urogential diaphragm
Hymen
Vaginal introitus
Greater vestibular (Bartholin's) gland
Superficial transverse perinei
Levator ani
Sphincter ani externus
Anus

Fig. 1.2: Exposition of superficial perineal pouch with vestibular bulb and Bartholin's gland

PERINEUM: The details of its anatomy are described later in the chapter.

BLOOD SUPPLY: ***Arteries***—(a) Branches of internal pudendal artery—the chief being labial, transverse perineal, artery to the vestibular bulb and deep and dorsal arteries to the clitoris. (b) Branches of femoral artery—superficial and deep external pudendal.

Veins—The veins form plexuses and drain into: (a) Internal pudendal vein, (b) vesical or vaginal venous plexus and (c) Long saphenous vein. Varicosities during pregnancy are not uncommon and may rupture spontaneously causing visible bleeding or hematoma formation.

NERVE SUPPLY: The supply is through bilateral spinal somatic nerves— (a) anterosuperior part is supplied by the cutaneous branches from the ilioinguinal and genital branch of genitofemoral nerve

(L_1 and L_2) and the posteroinferior part by the pudendal branches from the posterior cutaneous nerve of thigh ($S_{1,2,3}$). Between these two groups, the vulva is supplied by the labial and perineal branches of the pudendal nerve ($S_{2,3,4}$).

LYMPHATICS: Vulval lymphatics have bilateral drainage. Lymphatics drain into—(a) superficial inguinal nodes, (b) intermediate groups of inguinal lymph nodes—**gland of Cloquet** and (c) external and internal iliac lymph nodes.

DEVELOPMENT: **External genitalia** is developed in the region of the cranial aspect of ectodermal cloacal fossa; **clitoris** from the genital tubercle; **labia minora** from the genital folds; **labia majora** from the labioscrotal swelling and the **vestibule** from the urogenital sinus.

INTERNAL GENITAL ORGANS

The internal genital organs in female include vagina, uterus, fallopian tubes and the ovaries. These organs are placed internally and require special instruments for inspection.

VAGINA

The vagina is a fibromusculomembranous sheath communicating the uterine cavity with the exterior at the vulva. It constitutes the excretory channel for the uterine secretion and menstrual blood. It is the organ of copulation and forms the birth canal of parturition. The canal is directed upwards and backwards forming an angle of 45° with the horizontal in erect posture. The long axis of the vagina almost lies parallel to the plane of the pelvic inlet and at right angles to that of the uterus. The diameter of the canal is about 2.5 cm, being **widest in the upper part and narrowest at its introitus**. It has got enough power of distensibility as evident during childbirth.

WALLS: Vagina has got an anterior, a posterior and two lateral walls. The anterior and posterior walls are opposed together but the lateral walls are comparatively stiffer especially at its middle, as such, it looks "H" shaped on transverse section. The length of the anterior wall is about 7 cm and that of the posterior wall is about 9 cm.

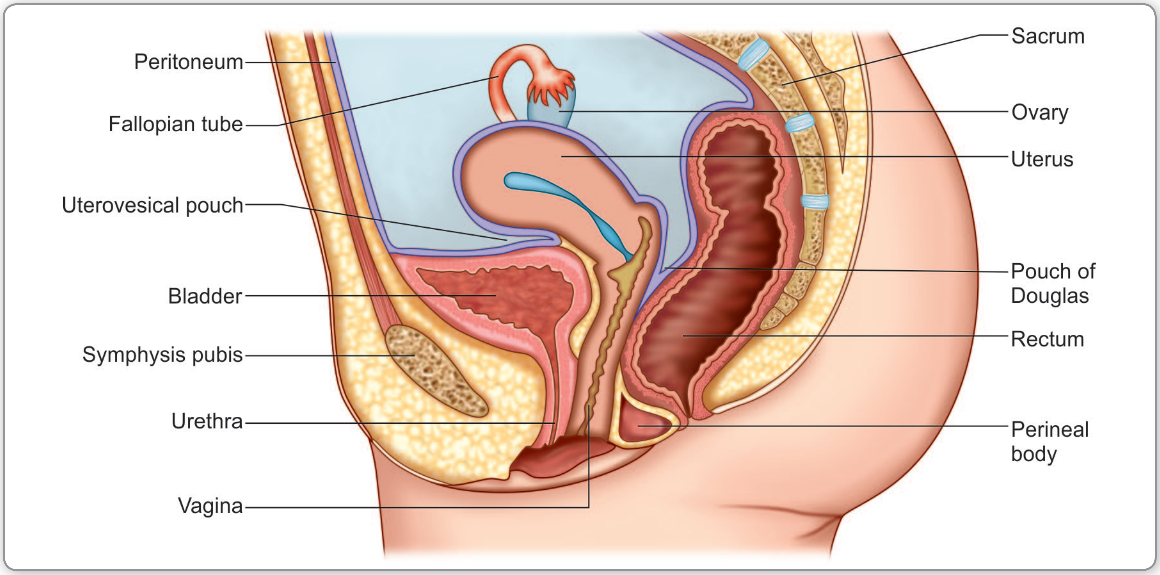


Fig. 1.3: Midsagittal section of the female pelvis showing relative position of the pelvic organs

FORNICES: The fornices are the clefts formed at the top of vagina (vault) due to the projection of the uterine cervix through the anterior vaginal wall where it is blended inseparably with its wall. There are four fornices—one anterior, one posterior and two lateral; the posterior one being deeper and the anterior, most shallow one.

RELATIONS:

Anterior—**The upper one-third** is related with base of the bladder and the lower two-thirds are with the urethra, the lower half of which is firmly embedded with its wall.

Posterior—**The upper one-third** is related with the pouch of Douglas, **the middle-third** with the anterior rectal wall separated by rectovaginal septum and **the lower-third** is separated from the anal canal by the perineal body (Fig. 1.3).

Lateral walls—**The upper one-third** is related with the pelvic cellular tissue at the base of broad ligament in which the ureter and the uterine artery lie approximately 2 cm from the lateral fornices. **The middle third** is blended with the levator ani and **the lower-third** is related with the bulbocavernosus muscles, vestibular bulbs and Bartholin's glands (Fig. 1.11).

STRUCTURES: Layers from within outwards are—(1) **mucous coat** which is lined by stratified squamous epithelium without any secreting glands (2) **submucous layer** of loose areolar vascular tissues (3) **muscular layer** consisting of indistinct inner circular and outer longitudinal muscles and (4) fibrous coat derived from the endopelvic fascia and is highly vascular.

VAGINAL SECRETION: The vaginal pH, from puberty to menopause, is acidic because of the presence of Döderlein's bacilli which produce lactic acid from the glycogen present in the exfoliated cells. **The pH varies with the estrogenic activity and ranges between 4 and 5**.

BLOOD SUPPLY: The arteries involved are—(1) Cervicovaginal branch of the uterine artery, (2) vaginal artery—a branch of anterior division of internal iliac or in common origin with the uterine, (3) middle rectal and (4) internal pudendal. These anastomose with one another and form two azygos arteries—anterior and posterior.

Veins drain into internal iliac veins and internal pudendal veins.

LYMPHATICS: On each side, the lymphatics drain into—(1) Upper one-third—internal iliac group, (2) middle one-third up to hymen—internal iliac group, (3) below the hymen—superficial inguinal group.

NERVE SUPPLY: The vagina is supplied by sympathetic and parasympathetic from the pelvic plexus. The lower part is supplied by the pudendal nerve.

DEVELOPMENT: The vagina is developed from the following sources: (a) Upper 4/5th, above the hymen—the mucous membrane is derived from endoderm of the canalized sinovaginal bulbs. The musculature is developed from the mesoderm of two fused Müllerian ducts. (b) Lower 1/5th, below the hymen is developed from the endoderm of the urogenital sinus. (c) External vaginal orifice is formed from the genital fold ectoderm after rupture of the urogenital membrane.

THE UTERUS

The uterus is a hollow pyriform muscular organ situated in the pelvis between the bladder in front and the rectum behind.

POSITION: Its normal position is one of the anteversion and anteflexion. The uterus usually inclines to the right (dextrorotation) so that the cervix is directed to the left (levorotation) and comes in close relation with the left ureter.

MEASUREMENTS AND PARTS: The uterus measures about 8 cm long, 5 cm wide at the fundus and its walls are about 1.25 cm thick. Its weight varies from 50 gm to 80 gm. It has got the following parts:

- **Body or corpus**
- **Isthmus**
- **Cervix**

(1) **Body or corpus**: The body is further divided into fundus—the part which lies above the openings of the uterine tubes. The body proper is triangular and lies between the openings of the tubes and the isthmus. The superolateral angles of the body of the uterus project outwards from the junction of the fundus and body and is called the **cornua of the uterus. The uterine tube, round ligament and ligament of the ovary are attached to it**. (2) **Isthmus** is a constricted part measuring about 0.5 cm, situated between the body and the cervix. **It is limited above by the anatomical internal os and below by the histological internal os (Aschoff).** Some consider isthmus as a part of the lower portion of the body of the uterus. (3) **Cervix** is cylindrical in shape and measures about 2.5 cm. It extends from the isthmus and ends at the external os which opens into the vagina after perforating its anterior wall. The part lying above the vagina is called supravaginal and that which lies within the vagina is called the vaginal part (Fig. 1.4).

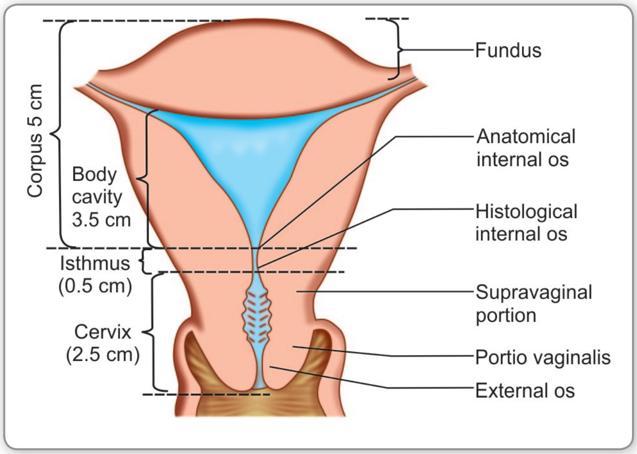

Corpus 5 cm
Fundus
Anatomical internal os
Body cavity 3.5 cm
Histological internal os
Isthmus (0.5 cm)
Supravaginal portion
Cervix (2.5 cm)
Portio vaginalis
External os

Fig. 1.4: Coronal section showing different parts of uterus

CAVITY: The cavity of the uterine body is triangular on coronal section with the base above and the apex below. It measures about 3.5 cm. There is no cavity in the fundus. The cervical canal is fusiform and measures about 2.5 cm. **Thus, the normal length of the uterine cavity is usually 6.5–7 cm.**

RELATIONS

Anteriorly—Above the internal os, the body forms the posterior wall of the uterovesical pouch. Below the internal os, it is separated from the base of the bladder by loose areolar tissue.

Posteriorly—It is covered with peritoneum and forms the anterior wall of the pouch of Douglas containing coils of intestine.

Laterally—The double fold of peritoneum of the broad ligament are attached between which the uterine artery ascends up. Attachment of the Mackenrodt's ligament extends from the internal os down to the supravaginal cervix and lateral vaginal wall. About 1.5 cm away at the level of internal os, a little nearer on the left side is the crossing of the uterine artery and the ureter. **The uterine artery crosses from above and in front of the ureter, soon before the ureter enters the ureteric tunnel (Fig. 1.5).**

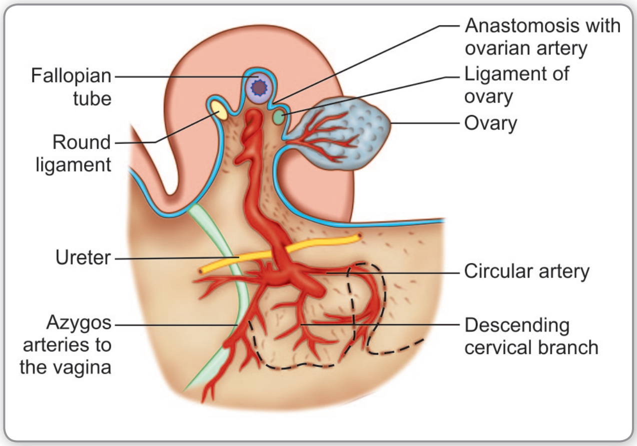

Fallopian tube
Anastomosis with ovarian artery
Ligament of ovary
Ovary
Round ligament
Ureter
Circular artery
Azygos arteries to the vagina
Descending cervical branch

Fig. 1.5: The relation of the ureter to the uterine artery

STRUCTURES

Body — The wall consists of three layers from outside inwards:

— ***Parametrium:*** It is the serous coat which invests the entire organ except on the lateral borders. The peritoneum is intimately adherent to the underlying muscles.

— ***Myometrium:*** It consists of thick bundles of smooth muscle fibers held by connective tissues and are arranged in various directions. During pregnancy, however, three distinct layers can be identified—outer longitudinal, middle interlacing and the inner circular.

— ***Endometrium:*** The mucous lining of the cavity is called endometrium. **As there is no submucous layer, the endometrium is directly opposed to the muscle coat**. It consists of lamina propria and surface epithelium. The surface epithelium is a single layer of ciliated columnar epithelium. The lamina propria contains stromal cells, endometrial glands, vessels and nerves. The glands are simple tubular and lined by mucus secreting non–ciliated columnar epithelium which penetrate the stroma and sometimes even enter the muscle coat. The endometrium is changed to decidua during pregnancy.

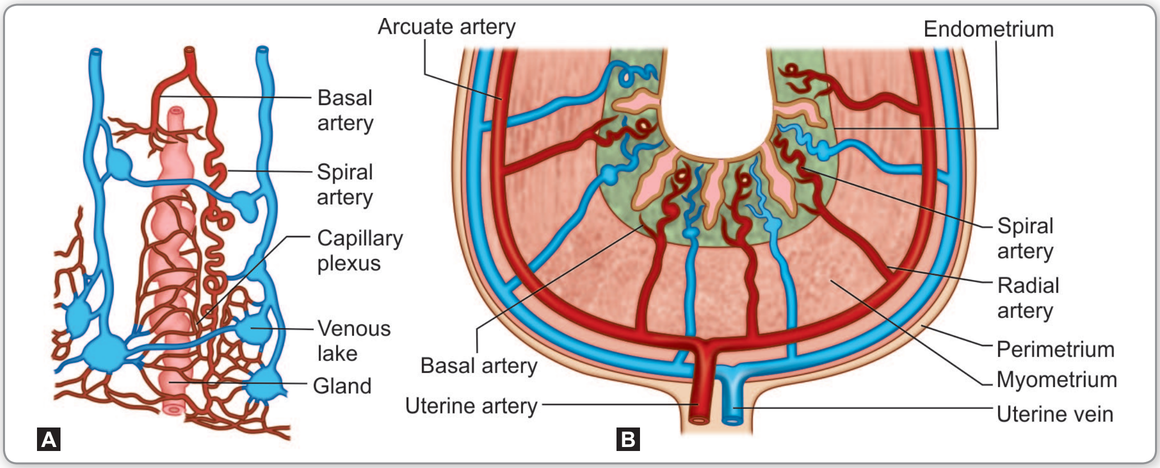
Cervix—The cervix is composed mainly of fibrous connective tissues. The smooth muscle fibers average 10–15%. Only the posterior surface has got peritoneal coat. Mucous coat lining the endocervix is simple columnar with basal nuclei and that lining the gland is non-ciliated secretory columnar cells. The vaginal part of the cervix is lined by stratified squamous epithelium. **The squamocolumnar junction is situated at the external os**.

SECRETION: The endometrial secretion is scanty and watery. Secretion of the cervical glands is alkaline and thick, rich in mucoprotein, fructose and sodium chloride.

PERITONEUM IN RELATION TO THE UTERUS: ***Traced anteriorly***—The peritoneum covering the superior surface of the bladder reflects over the anterior surface of the uterus at the level of the internal os. The pouch, so formed, is called **uterovesical pouch**.The peritoneum thereafter, is firmly attached to the anterior and posterior walls of the uterus and upper one-third of the posterior vaginal wall where from where it is reflected over the rectum. The pouch, so formed, is called **pouch of Douglas** (Fig. 1.3).

Traced laterally—The adherent peritoneum of the anterior and posterior walls of the uterus is continuous laterally forming the **broad ligament**. Laterally, it extends to the lateral pelvic walls where the layers reflect to cover the anterior and posterior aspects of the pelvic cavity. On its superior free border, lies the fallopian tube and on the posterior layer, the ovary is attached by mesovarium. The lateral one fourth of the free border is called **Infundibulopelvic ligament**.

BLOOD SUPPLY: ***Arterial supply*** — The blood supply is from the uterine arteries one on each side. The artery arises directly from the anterior division of the internal iliac or in common with superior vesical artery. The other sources are ovarian and vaginal arteries with which the uterine arteries anastomose. The internal supply of the uterus is shown in the Figures 1.6A and B.



Figs 1.6A and B: (A) Showing pattern of basal and spiral arteries in the endometrium; (B) Internal blood supply of uterus

Veins: The venous channels correspond to the arterial course and drain into internal iliac veins.

LYMPHATICS: ***Body***—(1) From the fundus and upper part of the body of the uterus, the lymphatics drain into preaortic and lateral aortic groups of glands. (2) Cornu drains to superficial inguinal gland along the round ligament. (3) Lower part of the body drains into external iliac groups.

Cervix—On each side, the lymphatics drain into: (1) external iliac, obturator lymph nodes either directly or through paracervical lymph nodes, (2) internal iliac groups and (3) sacral groups.

NERVES: The nerve supply of the uterus is derived principally from the sympathetic system and partly from the parasympathetic system. Sympathetic components are from T_5 and T_6 (motor) and T_{10} to L_1 spinal segments (sensory). The somatic distribution of uterine pain is that area of the abdomen supplied by T_{10} to L_8. The parasympathetic system is represented on either side by the pelvic nerve which consists of both motor and sensory fibers from S_2, S_3, S_4 and ends in the ganglia of Frankenhauser. The details are described in Chapter 33.

The cervix is insensitive to touch, heat and also when it is grasped by any instrument. **The uterus, too, is insensitive to** handling and even to incision over its wall.

DEVELOPMENT: The uterus is developed from the fused vertical part of the two Müllerian ducts.

FALLOPIAN TUBE (*Synonyms: Uterine tube, oviduct*)

The uterine tubes are paired structures, measuring about 10 cm and are situated in the medial three-fourth of the upper free margin of the broad ligament. Each tube has got two openings, one communicating with the lateral angle of the uterine cavity called uterine opening and measures 1 mm in diameter, the other is on the lateral end of the tube, called pelvic opening or abdominal ostium and measures about 2 mm in diameter.

PARTS: There are four parts. From medial to lateral are—(1) **intramural or interstitial** lying in the uterine wall and measures 1.25 cm in length and 1 mm in diameter, (2) **isthmus**—almost straight and measures about 3–4 cm in length and 2 mm in diameter, (3) **ampulla**—tortuous part and measures about 5 cm in length which ends in, (4) wide **infundibulum** measuring about 1.25 cm long with a maximum diameter of 6 mm. The abdominal ostium is surrounded by a number of radiating fimbriae (20–25), one of these is longer than the rest and is attached to the outer pole of the ovary called **ovarian fimbria** (Fig. 1.7).

STRUCTURES: It consists of three layers—(1) **Serous:** consists of peritoneum on all sides except along the line of attachment of mesosalpinx, (2) **Muscular:** arranged in two layers outer longitudinal and inner circular, (3) **Mucous membrane** has three different cell types and is thrown into longitudinal folds. The epithelium rests on a delicate vascular reticulum of connective tissue. **Mucous membrane is lined by**:

(i) **Columnar ciliated epithelial cells** that are most predominant near the ovarian end of the tube. These cells compose 25% of the mucosal cells, (ii) **Secretory columnar cells** are present at the isthmic segment and compose 60% of epithelial cells, (iii) **Peg cells** are found in between the above two cells. They are the variant of secretory cells.

FUNCTIONS: The important functions of the tubes are—(1) Transport of the gametes, (2) To facilitate fertilization and survival of zygote through its secretion.

BLOOD SUPPLY: Arterial supply is from the uterine and ovarian. **Venous drainage** is through the pampiniform plexus into the ovarian veins.

LYMPHATICS: The lymphatics run along the ovarian vessels to para-aortic nodes.

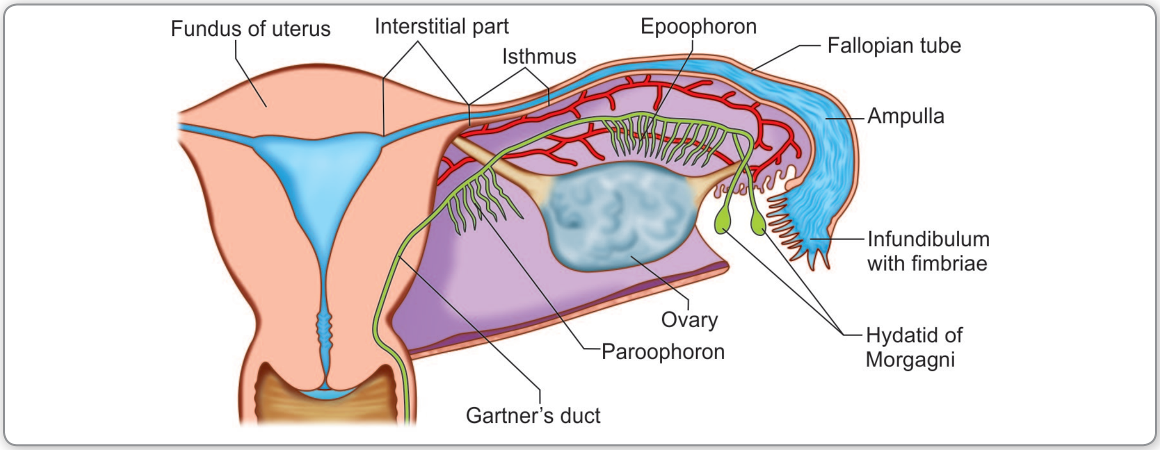


Fig. 1.7: Half of uterine cavity and fallopian tube of one side are cut open to show different parts of the tube. The vestigial structures in the broad ligament are shown

NERVE SUPPLY: The nerve supply is derived from the uterine and ovarian nerves. **The tube is very much sensitive to handling**.

DEVELOPMENT: The tube is developed from the upper vertical part of the corresponding **Müllerian duct** at about 6–10th week.

THE OVARY

The ovaries are paired sex glands or gonads in female which **are concerned for** (i) germ cell maturation, storage and its release and (ii) steroidogenesis. Each gland is oval in shape and pinkish gray in color and the surface is scarred during reproductive period. It measures about 3 cm in length, 2 cm in breadth and 1 cm in thickness. Each ovary presents two ends—tubal and uterine, two borders—mesovarium and free posterior and two surfaces—medial and lateral.

The ovaries are intraperitoneal structures. In nullipara, the ovary lies in the ovarian fossa on the lateral pelvic wall. **The ovary is attached to** the posterior layer of the broad ligament by the mesovarium, to the lateral pelvic wall by the infundibulopelvic ligament and to the uterus by the ovarian ligament.

RELATIONS: ***Mesovarium or anterior border***—A fold of peritoneum from the posterior leaf of the broad ligament is attached to the anterior border through which the ovarian vessels and nerves enter the hilum of the gland.

Posterior border is free and is related to the tubal ampulla. It is separated by the peritoneum from the ureter and the internal iliac artery.

Medial surface is related to fimbrial part of the tube.

Lateral surface is in contact with the ovarian fossa on the lateral pelvic wall.

The fossa is related *superiorly* to the external iliac vein, posteriorly to the ureter and internal iliac vessels and *laterally* to the peritoneum separating the obturator vessels and nerves (Fig. 1.8).

STRUCTURES: The ovary is covered by a single layer of cubical cell known as germinal epithelium. The substance of the gland consists of outer cortex and inner medulla.

Cortex—It consists of stromal cells which are thickened beneath the germinal epithelium to form **tunica albuginea**. During reproductive period (i.e., from puberty to menopause) the cortex is studded with numerous follicular structures, called the functional units of the ovary, in various phases of their development. These are related to sex hormone production and ovulation. **The structures include**—

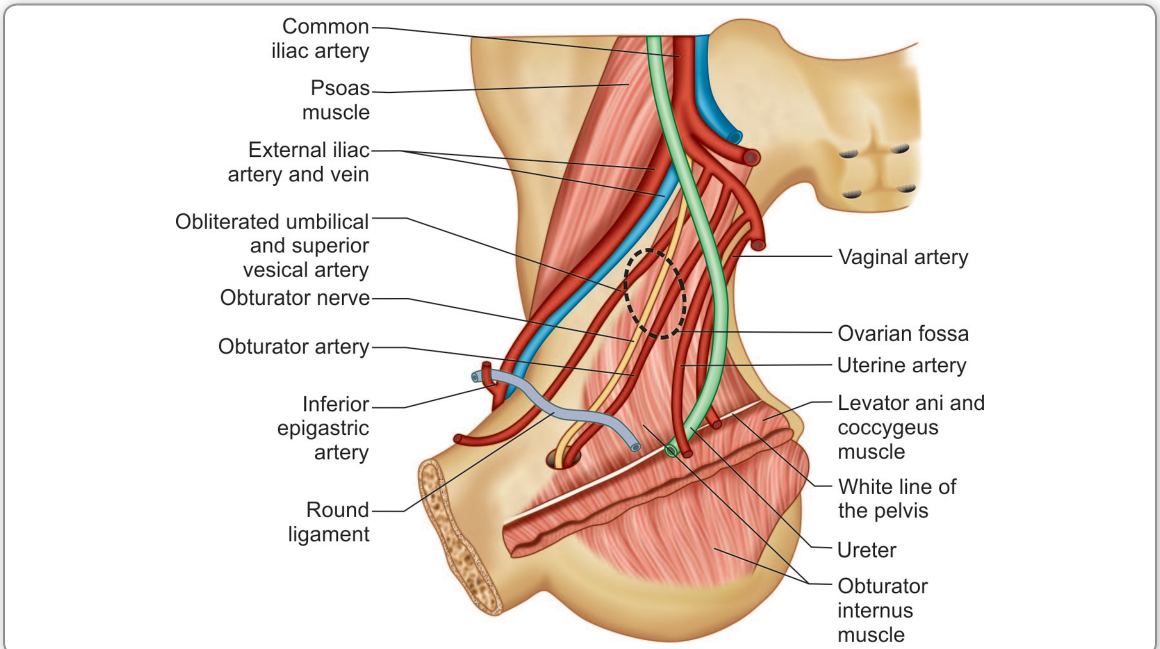

Common iliac artery
Psoas muscle
External iliac artery and vein
Obliterated umbilical and superior vesical artery
Obturator nerve
Obturator artery
Inferior epigastric artery
Round ligament
Vaginal artery
Ovarian fossa
Uterine artery
Levator ani and coccygeus muscle
White line of the pelvis
Ureter
Obturator internus muscle

Fig. 1.8: The ovarian fossa with the structures in the lateral pelvic wall

primordial follicles, maturing follicles, Graafian follicles and corpus luteum. Atresia of the structures results in formation of atretic follicles or corpus albicans (Fig. 1.9).

Medulla—It consists of loose connective tissues, few unstriped muscles, blood vessels and nerves. There is a small collection of cells called **"hilus cells" which are homologous to the interstitial cells of the testes**.

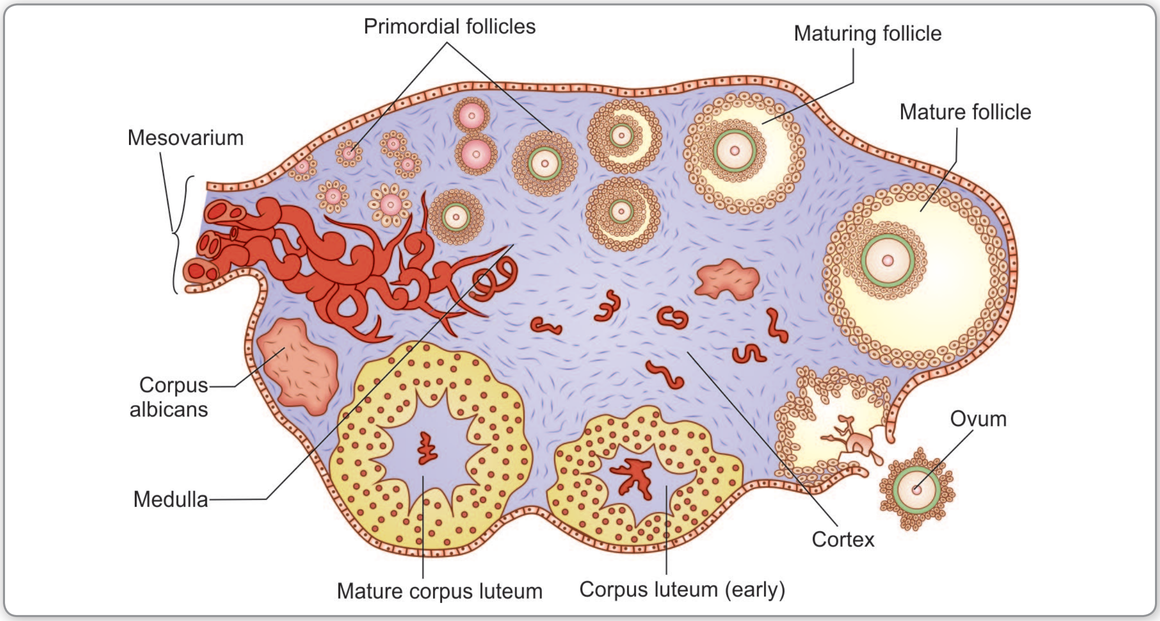

Primordial follicles
Maturing follicle
Mature follicle
Mesovarium
Corpus albicans
Medulla
Mature corpus luteum
Corpus luteum (early)
Cortex
Ovum

Fig. 1.9: Histological structure of the ovary

BLOOD SUPPLY: ***Arterial supply*** is from the ovarian artery, a branch of the abdominal aorta. ***Venous drainage*** is through pampiniform plexus, to form the **ovarian veins which drain into inferior vena cava on the right side and left renal vein on the left side**. Part of the venous blood from the placental site drains into the ovarian veins and thus may become the site of thrombophlebitis in puerperium.

LYMPHATICS: Through the ovarian vessels drain to the para-aortic lymph nodes.

NERVE SUPPLY: Sympathetic supply comes down along the ovarian artery from T_{10} segment. **Ovaries are sensitive to manual squeezing**.

DEVELOPMENT: The ovary is developed from the cortex of the undifferentiated genital ridges by about 9th week; the primary germ cells reaching the site migrating from the dorsal end of yolk sac.

MUSCLES AND FASCIA IN RELATION TO THE PELVIC ORGANS

The most important muscle supporting the pelvic organs is the levator ani which forms the pelvic floor. The small muscles of the perineum also have got some contribution.

PELVIC FLOOR

(Synonym: Pelvic diaphragm)

Pelvic floor is a muscular partition which separates the pelvic cavity from the anatomical perineum. It consists of three sets of muscles on either side—pubococcygeus, iliococcygeus and ischiococcygeus and these are collectively called levator ani. Its upper surface is concave and slopes downwards, backwards and medially and is covered by parietal layer of pelvic fascia. The inferior surface is convex and is covered by anal fascia. **The muscle with the covering fascia is called the pelvic diaphragm**.

ORIGIN: Each levator ani arises from the back of the pubic rami, from the condensed fascia covering the obturator internus (white line) and from the inner surface of the ischial spine.

INSERTION: From this extensive origin, **the fibers pass**, **backwards and medially** to be inserted in the midline from before backwards to the vagina (lateral and posterior walls), perineal body and anococcygeal raphe, lateral borders of the coccyx and lower part of the sacrum (Fig. 1.10).

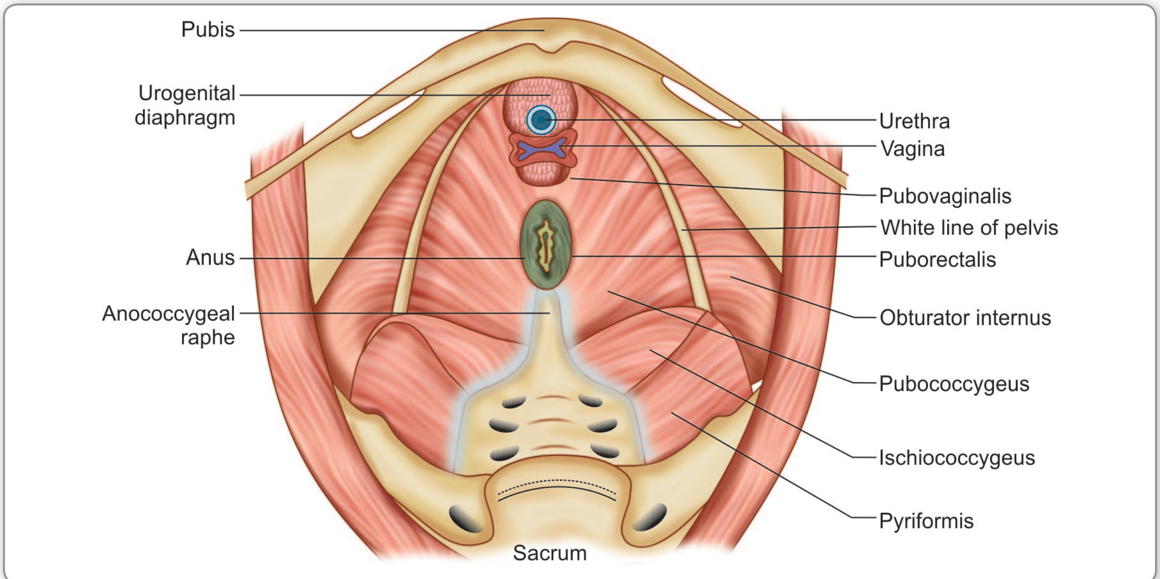


Fig. 1.10: Levator ani muscles viewed from above

GAPS: There are **two gaps** in the midline—(1) The anterior one is called **hiatus urogenitalis** which is bridged by the muscles and fascia of urogenital triangle and pierced by the urethra and vagina. (2) The posterior one is called **hiatus rectalis**, transmitting the rectum.

STRUCTURES IN RELATION TO PELVIC FLOOR

The superior surface is related with the following:

(1) **Pelvic organs** from anterior to posterior are bladder, vagina, uterus and rectum. (2) **Pelvic cellular tissues** between the pelvic peritoneum and upper surface of the levator ani which fill all the available spaces. (3) **Ureter** lies on the floor in relation to the lateral vaginal fornix. The uterine artery lies above and the vaginal artery lies below it. (4) **Pelvic nerves**.

The inferior surface is related to the anatomical perineum.

NERVE SUPPLY: It is supplied by the 4th sacral nerve, inferior rectal nerve and a perineal branch of pudendal nerve $S_{2,3,4}$.

FUNCTIONS: (1) To support the pelvic organs—The pubovaginalis which forms a "U" shaped sling, supports the vagina which in turn supports the other pelvic organs— bladder and uterus. Weakness or tear of this sling during parturition is responsible for prolapse of the organs concerned. (2) To maintain intra-abdominal pressure by reflexly responding to its changes. (3) Facilitates anterior internal rotation of the presenting part when it presses on the pelvic floor. (4) Puborectalis plays an ancillary role to the action of the external anal sphincter. (5) Ischiococcygeus helps to stabilize the sacroiliac and sacrococcygeal joints. (6) To steady the perineal body.

PELVIC FLOOR DURING PREGNANCY AND PARTURITION: **During pregnancy** levator muscles undergo hypertrophy, become less rigid and more distensible. Due to water retention, it swells up and sags down. **In the second stage**, the pubovaginalis and puborectalis relax and the levator ani is drawn up over the advancing presenting part in the second stage. Failure of the levator ani to relax at the crucial moment may lead to extensive damage of the pelvic structures. The effect of such a displacement is to elongate the birth canal which is composed solely of soft parts below the bony outlet. The soft canal has got deep lateral and posterior walls and its axis is in continuation with the axis of the bony pelvis.

PERINEUM

ANATOMICAL PERINEUM: Anatomically, **the perineum is bounded above** by the inferior surface of the pelvic floor, **below** by the skin between the buttocks and thighs. **Laterally**, it is bounded by the ischiopubic ramus, ischial tuberosities and sacrotuberous ligaments and **posteriorly**, by the coccyx. The diamond shaped space of the bony pelvic outlet is divided into two triangular spaces with the common base formed by the free border of the urogenital diaphragm. The anterior triangle is called **the urogenital triangle** which fills up the gap of the hiatus urogenitalis and is important from the obstetric point of view. The posterior one is called **the anal triangle**.

Urogenital triangle: It is pierced by the terminal part of the vagina and the urethra. The small perineal muscles are situated in two compartments formed by the ill-defined fascia. The compartments are superficial and deep perineal pouch. **The superficial pouch is formed by** the deep layer of the superficial perineal fascia (Colles fascia) and inferior layer of the urogenital diaphragm (perineal membrane). **The contents are** (Fig. 1.2) superficial transverse perinei (paired), bulbospongiosus covering the bulb of the vestibule, ischiocavernosus (paired) covering the crura of the clitoris and the Bartholin's gland (paired). **The deep perineal pouch** is formed by the inferior and superior layer of the urogenital diaphragm—together called urogenital diaphragm or triangular ligament. Between

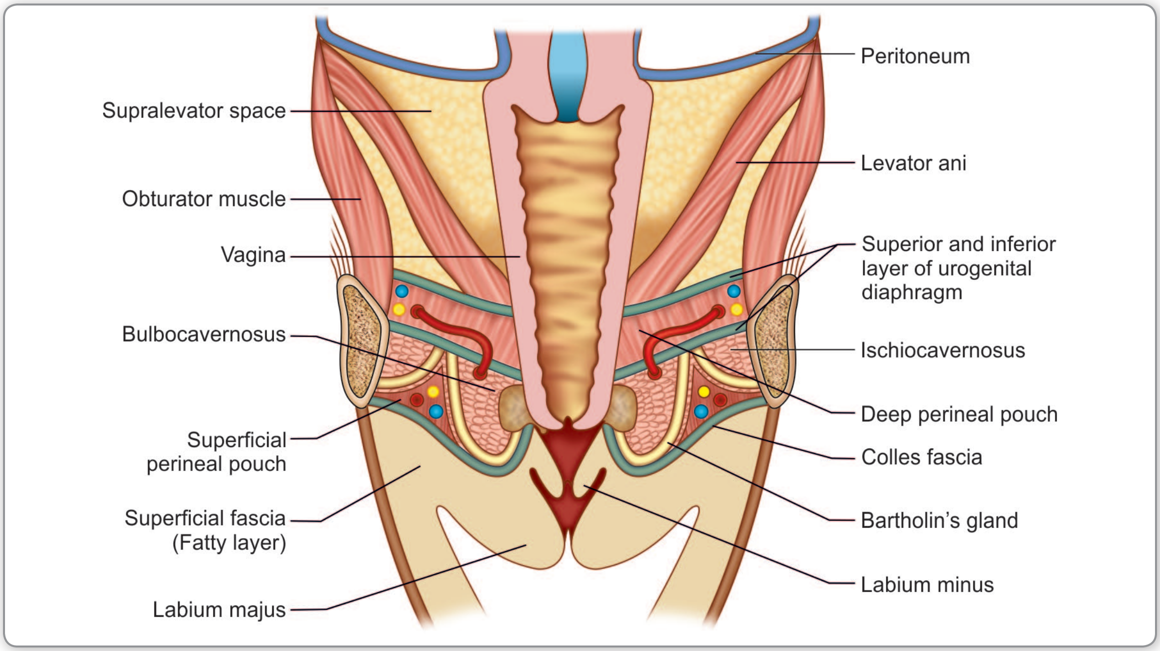

Peritoneum
Supralevator space
Levator ani
Obturator muscle
Vagina
Superior and inferior layer of urogenital diaphragm
Bulbocavernosus
Ischiocavernosus
Deep perineal pouch
Superficial perineal pouch
Colles fascia
Superficial fascia (Fatty layer)
Bartholin's gland
Labium minus
Labium majus

Fig. 1.11: Schematic diagram showing the pelvic muscles, fascia and cellular tissue as seen from the front

the layers there is a potential space of about 1.25 cm. **The contents are the following muscles**—deep transverse perinei (paired) and sphincter urethrae membranaceae. Both the pouches contain vessels and nerves (Fig. 1.11).

Anal triangle: It has got no obstetric importance. It contains the terminal part of the anal canal with sphincter ani externus, anococcygeal body, ischiorectal fossa, blood vessels, nerves and lymphatics.

OBSTETRICAL PERINEUM: *(Synonyms: Perineal body, central point of the perineum).* The pyramidal shaped tissue where the pelvic floor and the perineal muscles and fascia meet in between the vagina and the anal canal is called the obstetrical perineum. **It measures about 4 cm** × **4 cm** with the base covered by the perineal skin and the apex is pointed and is continuous with the rectovaginal septum.

The musculofascial structures involved are:

- *Fasciae*—(1) Two layers of superficial perineal fascia-superficial fatty layer and deeper layer called Colles fascia. (2) Inferior and superior layer of urogenital diaphragm, together called triangular ligament.
- *Muscles*—(1) Superficial and deep transverse perinei (paired). (2) Bulbospongiosus. (3) Levator ani-pubococcygeus part (paired), situated at the junction of the upper two-third and lower one-third of the vagina. (4) Sphincter ani externus (few fibers).

Importance: (1) It helps to support the levator ani which is placed above it. (2) By supporting the posterior vaginal wall, it indirectly supports the anterior vaginal wall, bladder and the uterus. (3) It is vulnerable to injury during childbirth. (4) Deliberate cutting of the structures during delivery is called episiotomy.

PELVIC FASCIA

For descriptive purpose, the pelvic fascia is grouped under the heading that covers the pelvic wall, the pelvic floor and the pelvic viscera.

Fascia on the pelvic wall: It is very tough and membranous. It covers the obturator internus and pyriformis and gets attached to the margins of the bone. **The pelvic nerves lie external to the fascia but the vessels lie internal to it**.

Fascia on the pelvic floor: It is not tough but loose. The superior and the inferior surfaces are covered by the parietal layer of the pelvic fascia which runs down from the white line to merge with the visceral layer of the pelvic fascia covering the anal canal (Fig. 1.11).

Fascia covering the pelvic viscera: The fascia is not condensed and often contains loose areolar tissue to allow distension of the organs.

PELVIC CELLULAR TISSUE

It lies between the pelvic peritoneum and the pelvic floor and fills up all the available empty spaces. It contains fatty and connective tissues and unstriated muscle fibers. Its distribution around the vaginal vault, supravaginal part of the cervix and into the layers of the broad ligament is called **parametrium**. Condensation occurs especially near the cervicovaginal junction to form ligaments which extend from the viscera to the pelvic walls on either side. These are Mackenrodt's ligaments, uterosacral ligaments and vesicocervical ligaments (fascia). All these constitute important supports of the uterus to keep it in position (Fig. 1.12).

Importance: (1) To support the pelvic organs. (2) To form protective sheath for the blood vessels and the terminal part of the ureter. (3) Infection spreads along the track, so formed, outside the pelvis

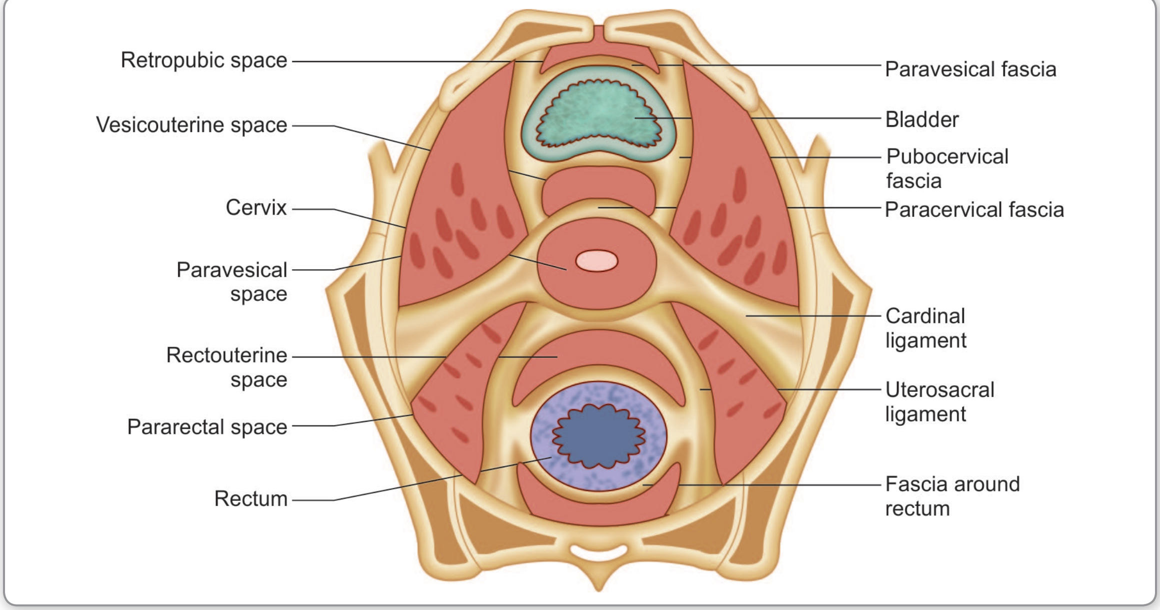

Retropubic space
Vesicouterine space
Cervix
Paravesical space
Rectouterine space
Pararectal space
Rectum
Paravesical fascia
Bladder
Pubocervical fascia
Paracervical fascia
Cardinal ligament
Uterosacral ligament
Fascia around rectum

Fig. 1.12: The main supporting ligaments of the uterus viewed from above

to the perinephric region along the ureter, to the buttock along the gluteal vessels, to the thigh along the external iliac vessels and to the groin along the round ligament. (4) Marked hypertrophy occurs during pregnancy to widen up the spaces.

FEMALE URETHRA

The female urethra extends from the neck of the bladder to the external urethral meatus which opens into the vestibule about 2.5 cm below the clitoris. **It measures about 4 cm and has a diameter of 6 mm**. Its upper half is separated from the anterior vaginal wall by loose areolar tissue and the lower half is firmly embedded in its wall. Numerous tubular glands called paraurethral glands open into the lumen through ducts. Of these, two are larger called Skene's ducts which open either on the posterior wall just inside the external meatus or into the vestibule. These glands are the sites for harboring infection and occasional development of benign adenoma or malignant changes. While piercing the deep perineal pouch it is surrounded by sphincter urethrae membranaceae which acts as an external sphincter.

STRUCTURES: **Mucous membrane** in the distal one-third is lined by stratified squamous epithelium but in the proximal two-third it becomes stratified transitional epithelium. **Submucous coat** is vascular. **Muscle coat** is arranged as inner longitudinal and outer circular.

BLOOD SUPPLY: ***Arterial supply***—Proximal parts are supplied by the inferior vesical branch and the distal part by a branch of internal pudendal artery. ***The veins*** drain into vesical plexus and into internal pudendal veins.

LYMPHATICS: Ear the meatus, the lymphatics drain into superficial inguinal glands and the rest drain into internal and external iliac group of glands.

NERVE SUPPLY: It is supplied by the pudendal nerve.

DEVELOPMENT: The urethra is developed from the vesicourethral portion of the cloaca.

THE URINARY BLADDER

The bladder is a hollow muscular organ with considerable power of distension. Its capacity is about 450 mL (15 oz) but can retain as much as 3–4 liters of urine. When distended it is ovoid in shape. It has got—(1) an apex (2) superior surface (3) base (4) two inferolateral surfaces and (5) neck, which is continuous with the urethra. **The base and the neck remain fixed even when the bladder is distended**.

RELATIONS: **The superior surface** is related with the peritoneum of the uterovesical pouch. **The base** is related with the supravaginal cervix and the anterior fornix. The ureters, after crossing the pelvic floor at the sides of the cervix, enter the bladder on its lateral angles. In the interior of bladder, the triangular area marked by three openings — two ureteric and one urethral, is called the trigone. **The inferolateral surfaces** are related with the space of Retzius. **The neck** rests on the superior layer of the urogenital diaphragm.

STRUCTURES: From outside inwards—(1) **Outer-visceral** layer of the pelvic fascia. (2) **Muscle layer** composed of muscles running in various directions. Near the internal urethral opening the circular muscle fibers provide involuntary sphincter. (3) **Mucous coat** is lined by transitional epithelium with no gland. There is no submucous coat.

BLOOD SUPPLY: ***The blood supply*** is through superior and inferior vesical arteries. ***The veins*** drain into vesical and vaginal plexus and thence to internal iliac veins.

LYMPHATICS: Lymphatics drain into external and internal iliac lymph nodes.

NERVE SUPPLY: The sympathetic supply is from the pelvic plexus and the parasympathetic via the pelvic plexus from the nervi erigentes ($S_{2,3,4}$). The parasympathetic produces contraction of the detrusor muscles and relaxation of the internal sphincter (nerve of evacuation). Sympathetic conveys afferent painful stimuli of overdistension.

DEVELOPMENT: The urinary bladder is developed from the upper part of the urogenital sinus.

PELVIC URETER

It extends from the crossing of the ureter over the pelvic brim up to its opening into the bladder. It measures about 13 cm in length and has a diameter of 5 mm. Ureter is retroperitoneal in course.

COURSE AND RELATIONS: The ureter enters the pelvis in front of the bifurcation of the common iliac artery over the sacroiliac joint behind the root of the mesentery on the right side and the apex of the mesosigmoid on the left side. As it courses downwards in contact with the peritoneum, it lies anterior to the internal iliac artery and behind the ovary and forms the posterior boundary of ovarian fossa (Fig. 1.8). On reaching the ischial spine, it lies over the pelvic floor and as it courses forwards and medially on the base of the broad ligament, **it is crossed by the uterine artery anteriorly** (Fig. 1.5). Soon, it enters into the ureteric tunnel and **lies close to the supravaginal part of the cervix, about 1.5 cm lateral to it**. After traversing a short distance on the anterior fornix of the vagina, it courses into the wall of the bladder obliquely for about 2 cm by piercing the lateral angle before it opens into the base of the trigone. **In the pelvic portion, the ureter is comparatively constricted** (a) where it crosses the pelvic brim (b) where crossed by the uterine artery and (c) in the intravesical part.

STRUCTURES: From outside inwards—(1) **Fibers derived from the visceral layer** of the pelvic fascia (2) **Muscle coat** consisting of three layers—outer and inner longitudinal and intermediate circular. (3) **Mucous layer** lined by transitional epithelium.

BLOOD SUPPLY: It has got segmental supply from nearly all the visceral branches of the anterior division of the internal iliac (uterine, vaginal, vesical, middle rectal) and superior gluteal arteries. The venous drainage corresponds to the arteries.

LYMPHATICS: The lymphatics from the lower part drain into the external and internal iliac lymph nodes and the upper part into the lumbar lymph nodes.

NERVE SUPPLY: Sympathetic supply is from the hypogastric and pelvic plexus; parasympathetic from the sacral plexus.

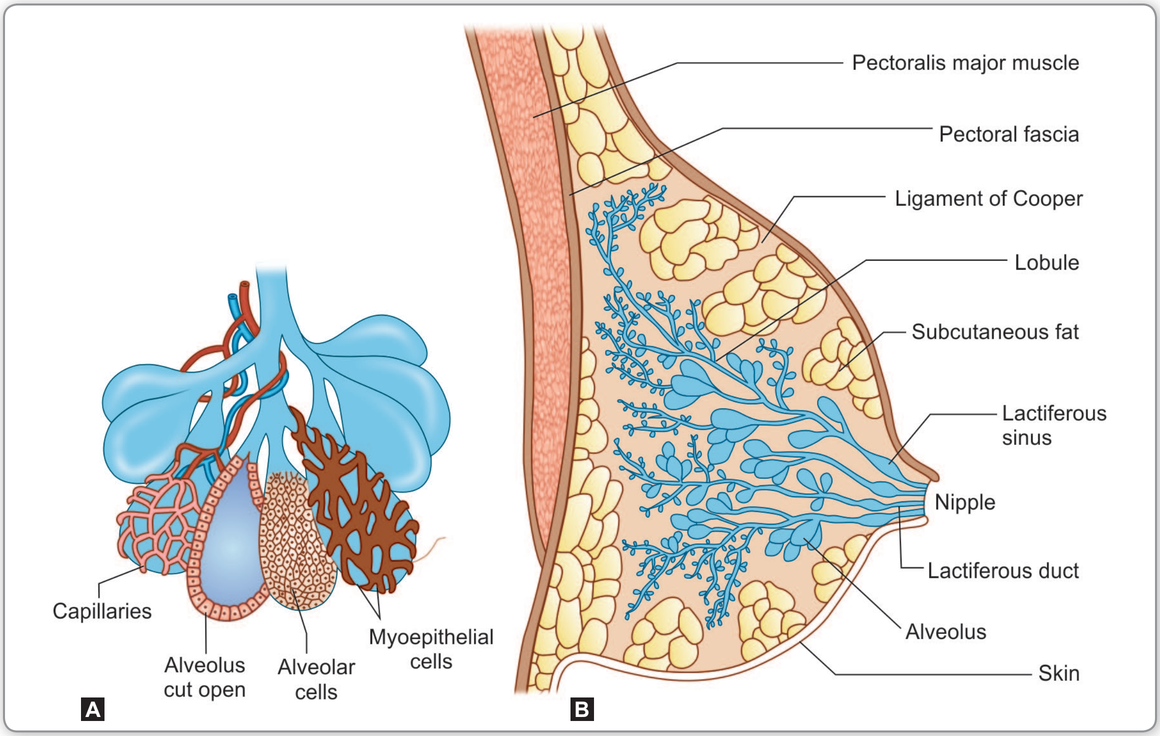
DEVELOPMENT: It is developed as an ureteric bud from the caudal end of the mesonephric duct.

THE BREAST

The breasts are large, modified sebaceous glands. The breasts are bilateral and in female constitute accessory reproductive organs as the glands are concerned with lactation following childbirth.

The shape of the breast varies in women and also in different periods of life. **But the size of the base of the breast is fairly constant**. It usually extends from the second to sixth rib in the midclavicular line. It lies in the subcutaneous tissue over the fascia covering the pectoralis major or even beyond that to lie over the serratus anterior and external oblique. A lateral projection of the breast towards the axilla is known as **axillary tail of Spence**. It lies in the axillary fossa, sometimes deep to the deep fascia. The breast weighs 200–300 gm during the childbearing age.

STRUCTURES ***(Non-lactating breasts):*** **The areola** is placed about the center of the breast and is pigmented. It is about 2.5 cm in diameter. **Montgomery glands** are accessory glands located around the



Figs 1.13A and B: (A) Structure of the basic unit of the mammary gland;. (B) Structure of adult female breast

periphery of the areola. They can secrete milk. **The nipple** is a muscular projection covered by pigmented skin. It is vascular and surrounded by unstriated muscles which make it erectile. It accommodates about 15–20 lactiferous ducts and their openings. Each milk duct (lactiferous duct) dilates to form lactiferous sinus at about 5–10 mm away from its opening in the nipple. When these sinuses are pulled in to the teat during nursing, the infants tongue, facial muscles and mouth squeeze the milk from the sinuses into the infant's oropharynx. The whole breast is embedded in the subcutaneous fat. **The fat is, however, absent beneath the nipple and areola**.

The mature breast consists of about 20% glandular tissue and 80% fat and the rest connective tissue (Figs 1.13A and B). The breast is composed of 12–20 lobes. Each lobe has one excretory duct (lactiferous duct) that opens at the nipple. Each lobe has about 10–100 lobules. **Cooper's ligaments** are the fibrous septa, that extend from the skin to the underlying pectoral fascia. These ligaments provide support to the breast. One lactiferous duct drains a lobe. The lining epithelium of the duct is cubical, becomes stratified squamous near the openings. Each alveolus is lined by columnar epithelium where milk secretion occurs. A network of branching longitudinal striated cells called **myoepithelial cells** surround the alveoli and the smaller ducts. There is a dense network of capillaries surrounding the alveoli. These are situated between the basement membrane and epithelial lining. Contraction of these cells squeezes the alveoli and ejects the milk into the larger duct. Behind the nipple, the main duct (lactiferous) dilates to form **ampulla** where the milk is stored.

Breast tissue is sensitive to the cyclic changes of hormones estrogen and progesterone. Women often feel breast tenderness and fullness during the luteal phase of the cycle. During the follicular phase, there is proliferation of the ductal system whereas during the luteal phase there is dilatation of the ductal system and differentiation of the alveolar cells into secretory cells. In postmenopausal women, the breast lobules and ducts atrophy. **Accessory breasts or nipples** can occur along the breast or milk line which extends from the axilla to the groin. **Supernumerary nipples** (**polythelia**) are associated with renal abnormalities (10%). **Asymmetry** of breasts is a normal variation. **Massive hypertrophy** of the breasts is a rare problem.

BLOOD SUPPLY: ***Arterial supply:*** (1) Lateral thoracic — branches of the axillary artery, (2) Internal mammary, (3) Inter costal arteries. ***Veins***—The veins follow the courses of the arteries.

LYMPHATICS: (1) **Lateral hemisphere**—anterior axillary nodes (75%). (2) **Upper convexity**—infraclavicular group. (3) **Medial convexity—**internal mammary chain of nodes (cross connection between the two breasts). There is no contralateral drainage of lymph, until and unless there is ipsilateral obstruction. (4) **Inferior convexity** — mediastinal glands.

NERVE SUPPLY: The nerve supply is from fourth, fifth and sixth intercostal nerves.

DEVELOPMENT: **The parenchyma** of the breasts is developed from the ectoderm. The **connective tissue stroma** is from the mesoderm.

QUESTIONS

Write Short Notes on:

1. Female Urethra (p. 15)
2. Isthmus of the uterus and its obstetric significance (p. 6)
3. Uterovesical pouch and its obstetric importance (p. 7)
4. Obstetric perineum (p. 13)

Chapter 2

Fundamentals of Reproduction

GAMETOGENESIS

The process involved in the maturation of the two highly specialized cells, spermatozoon in male and ovum in female before they unite to form zygote, is called gametogenesis.

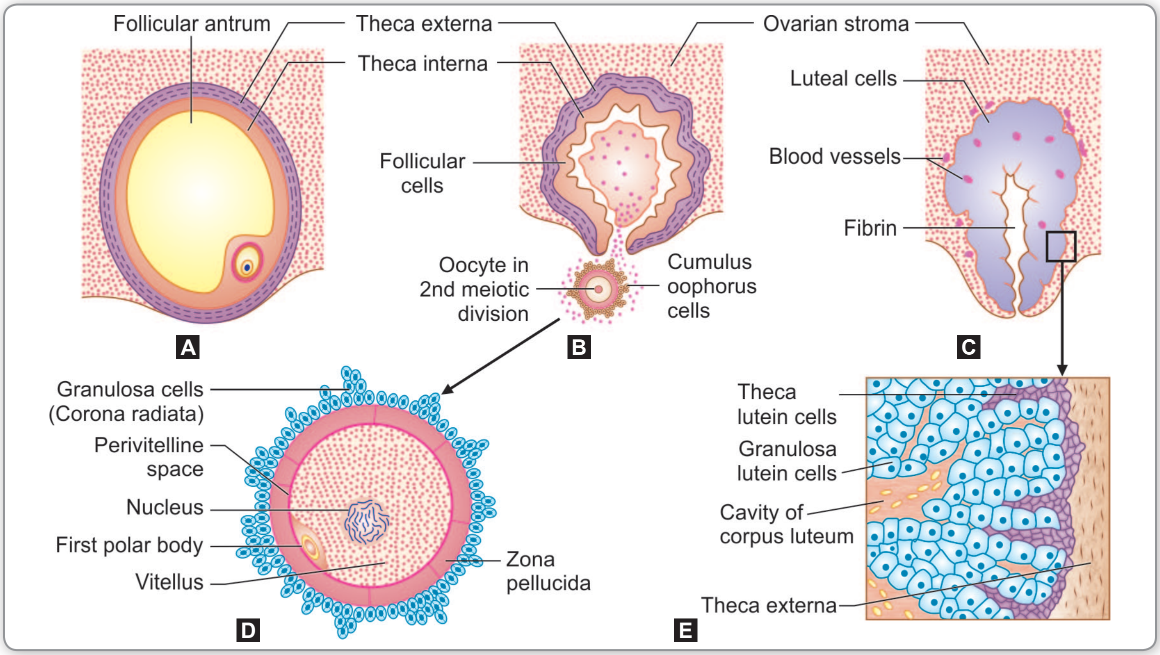
OOGENESIS

The process involved in the development of a mature ovum is called oogenesis. The primitive germ cells take their origin from the yolk sac at about the end of 3rd week and their migration to the developing gonadal ridge is completed round about the end of 4th week.

In the female gonads, the germ cells undergo a number of rapid mitotic divisions and differentiate into oogonia. The number of oogonia reaches its **maximum at 20th week, numbering about 7 million**. While the majority of the oogonia continue to divide, some enter into the prophase of the first meiotic division and are called primary oocytes. These are surrounded by flat cells and are called primordial follicles and are present in the cortex of the ovary. **At birth, there is no more mitotic division** and all the oogonia are replaced by primary oocytes which have finished the prophase of the first meiotic division and remain in resting phase (dictyotene stage) between prophase and metaphase. Total number of primary oocytes **at birth is estimated to be about 2 million**. The primary oocytes do not finish the first meiotic division until puberty is reached. **At puberty, some 400,000 primary oocytes are left behind**, the rest being atretic. Out of these, **some 400 are likely to ovulate during the entire reproductive period**.

Maturation of the oocytes: The essence of maturation is reduction of the number of chromosomes to half. Before the onset of first meiotic division, the primary oocytes double its DNA by replication, so they contain double the amount of normal protein content. There are 22 pairs of autosomes which determine the body characteristics and 1 pair of sex chromosomes, named "XX". The first stage of maturation occurs with full maturation of the ovarian follicle just prior to ovulation but **the final maturation occurs only after fertilization**.

The primary oocyte undergoes first meiotic division giving rise to secondary oocyte and one polar body. The two are of unequal size, the secondary oocyte contains haploid number of chromosomes (23, X), but nearly all the cytoplasm and the small polar body also contains half of the chromosomes (23, X) but with scanty cytoplasm. **Ovulation occurs soon after the formation of the secondary oocyte**.


Follicular antrum
Theca externa
Theca interna
Ovarian stroma
Luteal cells
Blood vessels
Fibrin
Follicular cells
Oocyte in 2nd meiotic division
Cumulus oophorus cells
A
B
C
Granulosa cells (Corona radiata)
Perivitelline space
Nucleus
First polar body
Vitellus
Zona pellucida
D
Theca lutein cells
Granulosa lutein cells
Cavity of corpus luteum
Theca externa
E

Figs 2.1A to E: Schematic diagram showing: (A) Mature Graafian follicle on the verge of ovulation (B) Ovulation with discharge of secondary oocyte surrounded by cumulus oophorus (C) Formation of corpus luteum (D) Secondary oocyte after first maturation division with formation of first polar body and (E) Microscopic structure of corpus luteum

The secondary oocyte completes the second meiotic division (homotypical) only after fertilization by the sperm in the Fallopian tube and results in the formation of two unequal daughter cells, each possessing 23 chromosomes (23, X), the larger one is called the mature ovum and the smaller one is the second polar body containing the same number of chromosomes. The first polar body may also undergo the second meiotic division. In the absence of fertilization, the secondary oocyte does not complete the second meiotic division and degenerates as such.

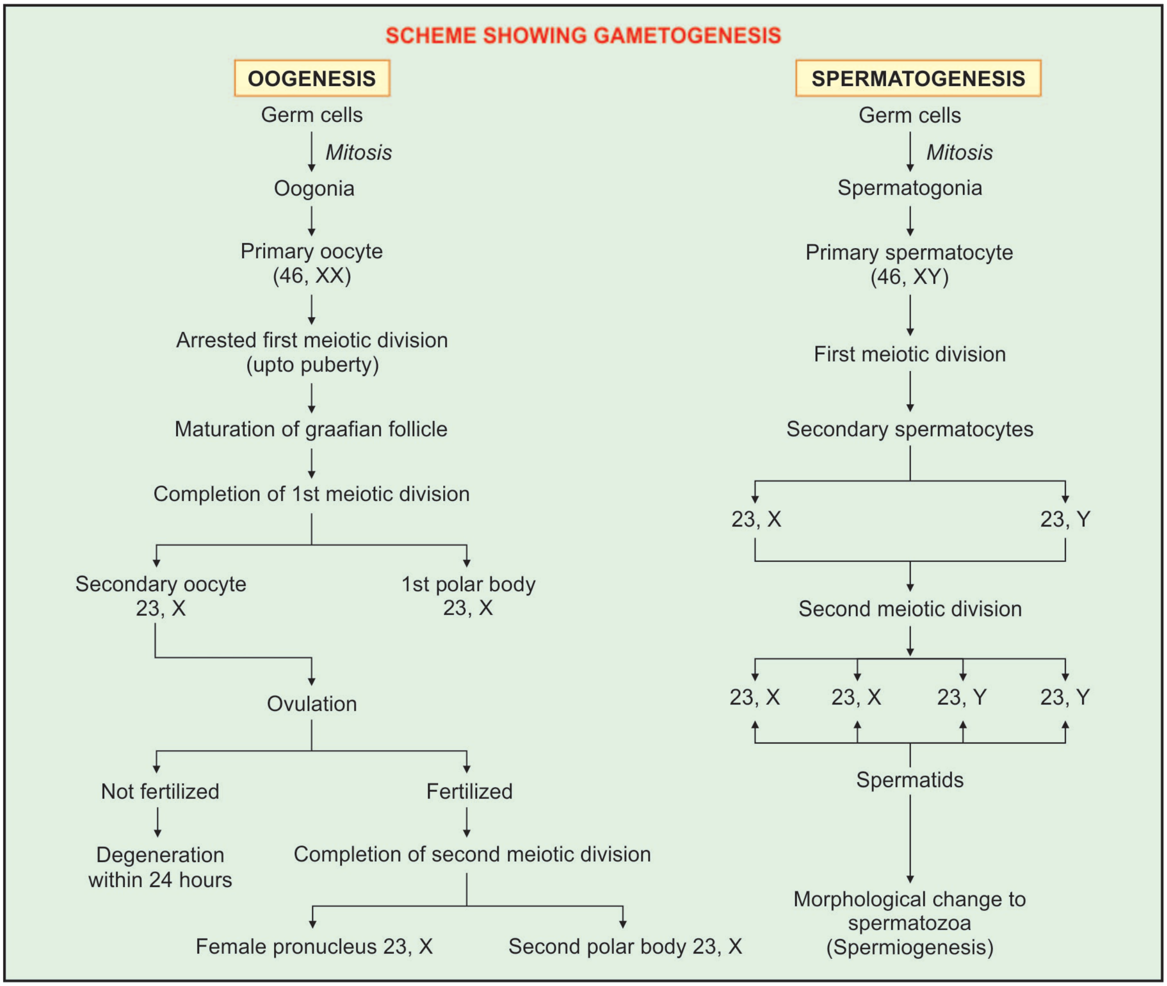
Chromosome nomenclature: The number designates the total number of chromosomes (in numerals) followed by the sex chromosome constitution after the comma.

Structure of a mature ovum: A fully mature ovum is the largest cell in the body and is about **130 microns in diameter**. It consists of cytoplasm and a nucleus with its nucleolus which is eccentric in position and **contains 23 chromosomes (23, X)**. During fertilization, the nucleus is converted into a female pronucleus. The ovum is surrounded by a cell membrane called **vitelline membrane**.

There is an outer transparent mucoprotein envelope, **the zona pellucida**. The zona pellucida is penetrated by tiny channels which are thought to be important for the transport of the materials from the granulosa cells to the oocyte. In between the vitelline membrane and the zona pellucida, there is a narrow space called **perivitelline space which accommodates the polar bodies**. The human oocyte, after its escape from the follicle, retains a covering of granulosa cells known as the **corona radiata** derived from the cumulus oophorus (Fig. 2.1).

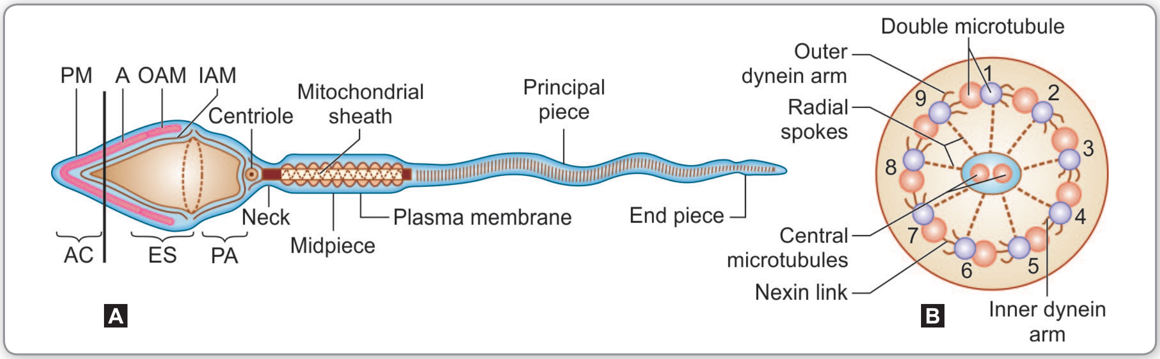
SPERMATOGENESIS

The process involved in the development of spermatids from the primordial male germ cells and their differentiation into spermatozoa is called spermatogenesis. Shortly before puberty, the primordial germ cells develop into spermatogonia and remain in the wall of seminiferous tubules. The


SCHEME SHOWING GAMETOGENESIS
OOGENESIS
Germ cells
Mitosis
Oogonia
Primary oocyte (46, XX)
Arrested first meiotic division (upto puberty)
Maturation of graafian follicle
Completion of 1st meiotic division
Secondary oocyte 23, X
1st polar body 23, X
Ovulation
Not fertilized
Fertilized
Degeneration within 24 hours
Completion of second meiotic division
Female pronucleus 23, X
Second polar body 23, X
SPERMATOGENESIS
Germ cells
Mitosis
Spermatogonia
Primary spermatocyte (46, XY)
First meiotic division
Secondary spermatocytes
23, X
23, Y
Second meiotic division
23, X
23, X
23, Y
23, Y
Spermatids
Morphological change to spermatozoa (Spermiogenesis)

spermatogonia, in turn, differentiate into primary spermatocytes which remain in the stage of prophase of the first meiotic division for a long time (about 16 days). Each spermatocyte contains 22 pairs of autosomes and 1 pair of sex chromosomes, named "XY". With the completion of the first meiotic division, two secondary spermatocytes are formed having equal share of cytoplasm and haploid number of chromosomes either 23, X or 23, Y. Then immediately follows the second meiotic division (homotypical) with the formation of four spermatids, each containing haploid number of chromosomes, two with 23, X and two with 23, Y. Immediately after their formation, extensive morphological differentiation of the spermatids occurs without further cell division to convert them into spermatozoa. The process is called **spermiogenesis.** In man, **the time required for a spermatogonium to develop into a mature spermatozoon is about 61 days**.

Sperm capacitation and acrosome reaction: Capacitation is the physiochemical change in the sperm by which it becomes hypermotile and is able to bind and fertilize a secondary oocyte. Capacitation takes place in the genital tract and **takes about 2–6 hours**. The changes involve cyclic AMP dependent phosphorylation with increase in intracellular pH (influx of Ca^{++} and efflux of H^{+}). **Activation of acrosomal membranes** causes release of hyaluronidase, hydrolytic enzymes, proacrosin, acrosin, that help the sperm to digest the zona pellucida and to enter into the oocyte. During **acrosomal reaction** the sperm plasma membrane fuses with the outer acrosomal membrane (Fig. 2.2). The sperm with acrosomal membrane bind the



Figs 2.2A and B: Structure of a mature spermatozoon: (A) PM = Plasma membrane; A = Acrosome; OAM = Outer acrosomal membrane; IAM = Inner acrosomal membrane; AC = Acrosomal cap; ES = Equatorial segment; PA = Post-acrosomal region. (B) The sperm tail with the axonemal complex which is essential for sperm motility. Cross sectional view shows nine pairs of peripheral doublets, joined each other by nexin links via two dynein protein arms. Energy for sperm motility is derived from mitochondria in the midpiece

Zona Protein (ZP_3), after passing between the corona radiata cells (Fig. 2.3). After acrosome reaction, the sperm binds to Zona Protein ZP_2. Then there is zona reaction to prevent polyspermy. Acrosome sperm penetrate the zona pellucida → reaches the perivitelline space → fuses with the oocyte plasma membrane. The sperm head swells and the fused membrane vesiculates. The whole sperm head, midpiece and tail are drawn into the cytoplasm. Gamete fusion is an integrin mediated process. About 3–6 hours after insemination, one polar and two pronuclear bodies are visible and they migrate to the center of the oocyte (Fig. 2.3).

Fertilization in vitro: Capacitation and acrosome reaction occur within few hours in simple media. Washed and motile sperm (2×10^5/mL) are added to the oocyte. In ICSI, microinjection of a single sperm into the oocyte is done.

Structure of a mature spermatozoon: It has got two parts, a head and a tail. The head consists principally of the condensed nucleus and acrosomal cap (Fig. 2.2). Acrosome is rich in enzymes. The tail is divided into four zones — the neck, the middle piece, the principal piece and the end piece.

OVULATION

Ovulation is a process whereby a secondary oocyte is released from the ovary following rupture of a mature Graafian follicle and becomes available for conception. Only one secondary oocyte is likely to rupture in each ovarian cycle which starts at puberty and ends in menopause. In relation to the menstrual period, the event occurs about 14 days prior to the expected period. However, menstruation can occur without ovulation and ovulation remains suspended during pregnancy and lactation.

MECHANISM: The process of ovulation is a complex one. Preovulatory changes occur both in the follicle and the oocyte.

Changes in the follicle: There is preovulatory enlargement of the Graafian follicle due to accumulation of follicular fluid and measures about 20 mm in diameter. The cumulus oophorus separates from the rest of the granulosa cells and floats freely in the antrum. The inner layer of the cells surrounding the oocyte is arranged radially and is termed corona radiata (Fig. 2.1). The follicular wall near the ovarian surface becomes thinner. The stigma develops as a conical projection which penetrates the outer surface layer of the ovary and persists for a while (½ – 2 minutes) as a thin membrane. **The cumulus escapes out of the follicle as a slow oozing process, taking about 1–2 minutes along with varying amount of follicular fluid (Fig. 2.1)**. The stigma is soon closed by a plug of plasma.

Changes in the oocyte: Significant changes in the oocyte occur just prior to ovulation (few hours). Cytoplasmic volume is increased along with changes in the number, distribution of mitochondria and in the Golgi apparatus. **Completion of the arrested first meiotic division occurs** with extrusion of first polar body, each containing haploid number of chromosomes (23, X).

CAUSES: The following are the possible explanations which may operate singly or in combination:

— **Endocrinal** • ***LH surge:*** Sustained peak level of estrogen for 24–36 hours in the late follicular phase → LH surge occurs from the anterior pituitary. Ovulation approximately occurs 16–24 hours after the LH surge. LH peak persists for about 24 hours. The LH surge stimulates completion of reduction division of the oocyte and initiates luteinization of the granulosa cells, synthesis of progesterone and prostaglandins.

- ***FSH rise:*** Preovulatory rise of progesterone facilitates the positive feedback action of estrogen to induce FSH surge → increase in plasminogen activator → plasminogen → plasmin → helps lysis of the wall of the follicle.

Thus, the combined LH/FSH midcycle surge is responsible for the final stage of maturation, rupture of the follicle and expulsion of the oocyte.

— **Stretching factor:** It is more a **passive stretching** than rise in intrafollicular pressure which remains static at about 15 mm Hg.

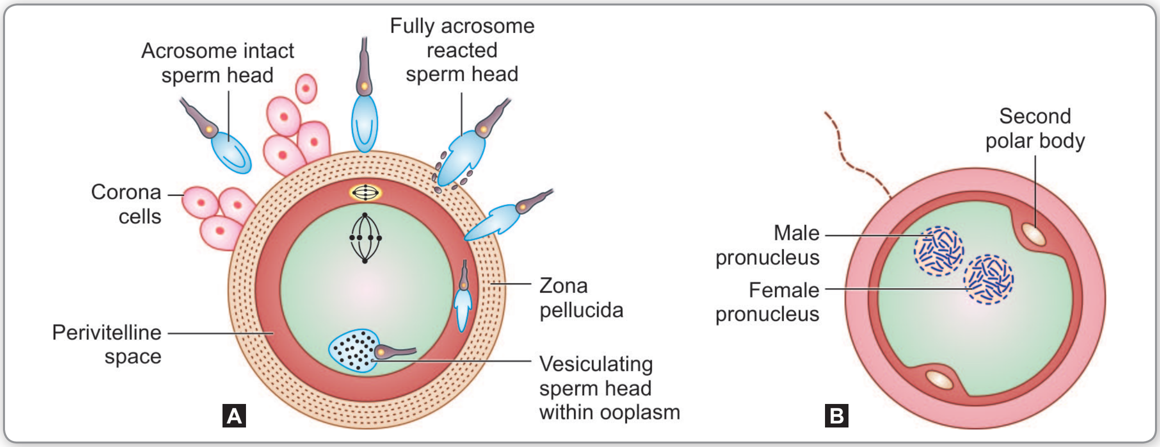
— **Contraction of the micromuscles** in the theca externa and ovarian stroma due to increased prostaglandin secretion.

EFFECT OF OVULATION: Following ovulation, the follicle is changed into corpus luteum (Fig. 2.1).

The ovum is picked up into the Fallopian tube and undergoes either degeneration or further maturation, if fertilization is to occur. **Menstruation is unrelated with ovulation** and anovular menstruation is quite common during adolescence, following childbirth and in women approaching menopause.

FERTILIZATION

Fertilization is the process of fusion of the spermatozoon with the mature ovum. It begins with sperm egg collision and ends with production of a mononucleated single cell called the zygote. Its objectives are: (1) To initiate the embryonic development of the egg and (2) To restore the chromosome number of the species. **Almost always, fertilization occurs in the ampullary part of the uterine tube**.


Acrosome intact sperm head
Fully acrosome reacted sperm head
Corona cells
Zona pellucida
Perivitelline space
Vesiculating sperm head within ooplasm
A
Second polar body
Male pronucleus
Female pronucleus
B

Figs 2.3A and B: Schematic diagram showing sequence of changes during fertilization : (A) Sperm (acrosome intact) in between the corona radiata cells → attachment with zona → acrosome reacted sperm penetrating the zona → acrosome reacted sperm in the perivitelline space → incorporated sperm with vesiculating head. (B) Formation of male and female pronuclei with completion of second polar body

APPROXIMATION OF THE GAMETES: The ovum, immediately following ovulation is picked up by the tubal fimbriae which partly envelope the ovary, especially at the time of ovulation. **The pick up action might be** muscular or by a kind of suction or by ciliary action or by a positive chemotaxis exerted by the tubal secretion. The ovum is rapidly transported to the ampullary part. **Fertilizable life span of oocyte ranges from 12 to 24 hours whereas that of sperm is 48 to 72 hours**.

Out of hundreds of millions of sperms deposited in the vagina at single ejaculation, only thousands capacitated spermatozoa enter the uterine tube while only 300–500 reach the ovum. The tubal transport is facilitated by muscular contraction and aspiration action of the uterine tube. **It takes only few minutes for the sperm to reach the Fallopian tube**.

CONTACT AND FUSION OF THE GAMETES (Fig. 2.3): Complete dissolution of the cells of the corona radiata occurs by the chemical action of the hyaluronidase liberated from the acrosomal cap of the hundreds of sperm present at the site (see p. 21).

— Penetration of the zona pellucida is facilitated by the release of hyaluronidase from the acrosomal cap. More than one sperm may penetrate the zona pellucida.

— Out of the many sperms, one touches the oolemma. Soon after the sperm fusion, penetration of other sperm is prevented by **zona reaction (hardening) and oolemma block**. This is due to release of cortical granules by exocytosis from the oocyte.

— Completion of the second meiotic division of the oocyte immediately follows, each containing haploid number of chromosomes (23, X). The bigger one is called the female pronucleus and the smaller one is called second polar body which is pushed to the perivitelline space.

— In the human, both the head and tail of the spermatozoon enter the cytoplasm of the oocyte but the plasma membrane is left behind on the oocyte surface. Head and the neck of the spermatozoon become male pronucleus containing haploid number of chromosomes (23, X) or (23, Y).

— The male and the female pronuclei unite at the center with restoration of the diploid number of chromosomes (46) which is constant for the species. The **zygote**, thus formed, contains both the paternal and maternal genetic materials. In some instances, an antigen called fertilizin present on the cortex and its coat of the ovum, reacts with the antibody called antifertilizin liberated at the plasma membrane of the sperm head. Thus the union between the two gametes may be an immunological reaction (chemotaxis).

— Sex of the child is determined by the pattern of the sex chromosome supplied by the spermatozoon. If the spermatozoon contains 'X' chromosome, a female embryo (46, XX) is formed; if it contains a 'Y' chromosome, a male embryo (46, XY) is formed.

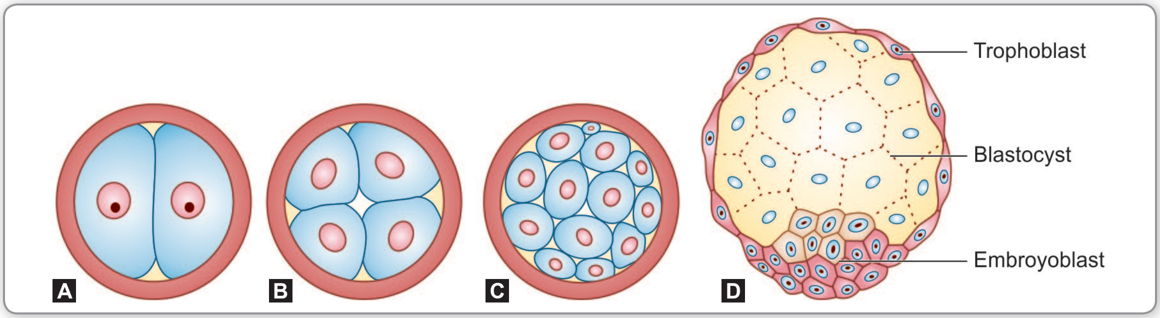
MORULA

After the zygote formation, typical mitotic division of the nucleus occurs by producing two blastomeres.

The two cell stage is reached approximately 30 hours after fertilization. Each contains equal cytoplasmic volume and chromosome numbers. The blastomeres continue to divide by binary division through 4, 8, 16 cell stage until a cluster of cells is formed and is called **morula**, resembling a mulberry. As the total volume of the cell mass is not increased and the zona pellucida remains intact, the **morula after spending about 3 days in the uterine tube enters the uterine cavity through the narrow uterine ostium (1 mm) on the 4th day in the 16-64 cell stage**. The transport is a slow process and is controlled by muscular contraction and movement of the cilia. The central cell of the morula is known as inner cell mass which forms the embryo proper and the peripheral cells are called outer cell mass which will form protective and nutritive membranes of the embryo.

BLASTOCYST

While the morula remains free in the uterine cavity **on the 4th and 5th day**, it is covered by a film of mucus. The fluid passes through the canaliculi of the zona pellucida which separates the cells of the morula and is now termed blastocyst (Fig. 2.4). **Zona hatching** is the next step so that trophectoderm cells interact with endometrial cells and implantation occurs.



Figs 2.4A to D: Schematic representation of the mitotic division of the zygote resulting in formation of: (A) Two-cell stage; (B) Four-cell stage; (C) Morula (see p. 24) and (D) Blastocyst

Due to blastocyst enlargement the zona pellucida becomes stretched, thinned and gradually disappears. **Lysis of zona and escape of embryo is called zona hatching**. The cells on the outer side of the morula (polar) become **trophectoderm** and the inner cells (apolar) become **inner cell mass** by the mediation of epithelial cadherin (E-cadherin) (protein). **Trophectoderm differentiates into chorion** (placenta) and the **inner cell mass into the embryo**. Completely undifferentiated cells are called the pluripotent **embryonic stem (ES) cells**. **ES cells are able to produce mature somatic cells of any germ layer (ectoderm, mesoderm and endoderm)**.

IMPLANTATION *(Syn: Nidation)*

Implantation occurs in the endometrium of the anterior or posterior wall of the body near the fundus **on the 6th day which corresponds to the 20th day of a regular menstrual cycle**. **Implantation occurs through four stages e.g**. apposition, adhesion, penetration and invasion.

CHANGES IN THE BLASTOCYST: The polar trophoblast cells adjacent to the inner cell mass are primarily involved in adhesion to the endometrial cells. **The factors responsible for blastocyst attachment are**: *P. selectin, heparin sulfate, proteoglycan, EGF, integrins, trophinin and others.* The signals for trophoblast multiplication arise from the inner cell mass.

ENDOMETRIUM AT THE IMPLANTATION SITE: (1) The endometrium is in the secretory phase corresponding to 20–21 days of cycle. (2) The microvilli on the surface of the trophectoderm interdigitate with the decidual cells to form the junctional complexes. **Endometrial receptivity and molecular signaling** during implantation is induced by progesterone, LIF (leukemia inhibitory factor), prostaglandins and COX-2.

APPOSITION: Occurs through pinopod formation. **Pinopods** are long finger like projections (microvilli) from the endometrial cell surface. These pinopods absorb the endometrial fluid which is secreted by the endometrial gland cells. This fluid, rich in glycogen and mucin provides nutrition to the blastocyst initially. Unless this fluid is absorbed, **adhesion phase** cannot occur. **Adhesion** of blastocyst to the endometrium occurs through the adhesion molecules like integrin, selectin and cadherin (glycoproteins).

PENETRATION: Actual penetration and invasion occur through the stromal cells in between the glands and is facilitated by the histolytic action of the blastocyst. With increasing lysis of the stromal cells, **the blastocyst**

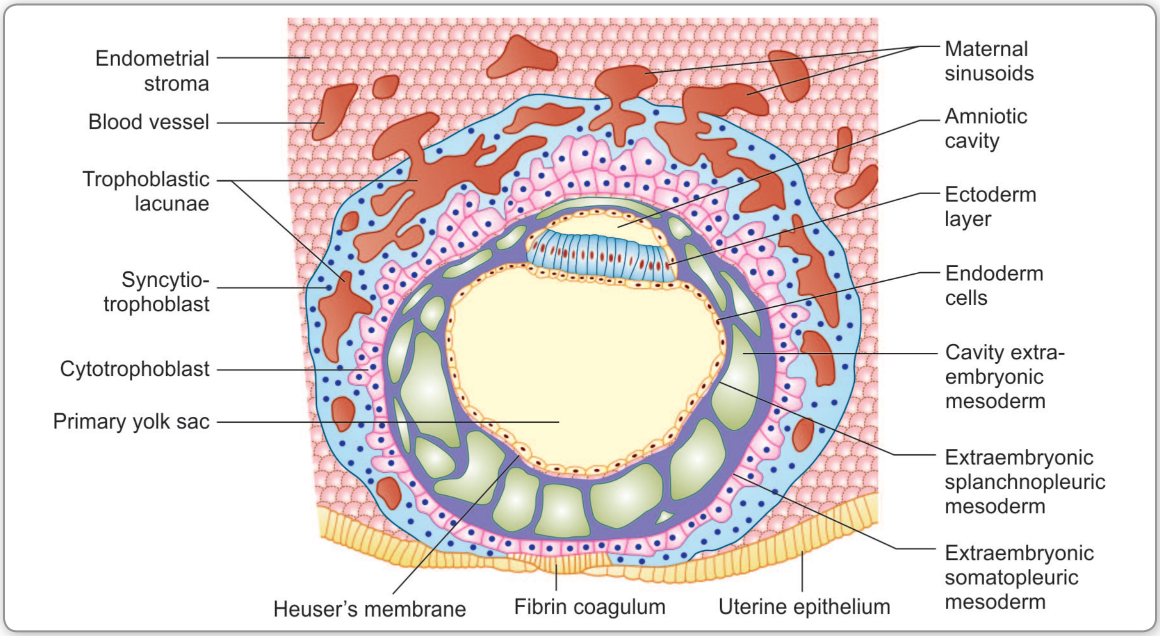

Endometrial stroma
Blood vessel
Trophoblastic lacunae
Syncytio-trophoblast
Cytotrophoblast
Primary yolk sac
Heuser's membrane
Fibrin coagulum
Uterine epithelium
Maternal sinusoids
Amniotic cavity
Ectoderm layer
Endoderm cells
Cavity extra-embryonic mesoderm
Extraembryonic splanchnopleuric mesoderm
Extraembryonic somatopleuric mesoderm

Fig. 2.5: Schematic representation showing interstitial implantation of the blastocyst in stratum compactum of the decidua

is burrowed more and more inside the stratum compactum of the decidua (Fig. 2.5). Vacuoles appear in the advancing syncytium which fuse to form large lacunae. These are more evident at the embryonic pole. Concurrently, the syncytial cells penetrate deeper into the stroma and erode the endothelium of the maternal capillaries. The syncytium by penetrating the vessels, not only becomes continuous with the endothelial lining but permits the maternal blood to enter into the lacunar system. Ultimately erosion of few maternal arteries with formation of blood space (lacunae) occurs. Nutrition is now obtained by aerobic metabolic pathway from the maternal blood. **Further penetration is stopped probably by the maternal immunological factor** and the original point of entry is sealed by fibrin clot and later by epithelium. **The process is completed by 10th or 11th day which corresponds to D 24-25 from LMP (Fig. 2.5)**.

This type of deeper penetration of the human blastocyst is called interstitial implantation and the blastocyst is covered on all sides by the endometrium (decidua). Occasionally, there may be increased blood flow into the lacunar spaces at the abembryonic pole. This results in disruption of the lacunae and extravasation of blood into the endometrial cavity. **This corresponds approximately to 13th day after fertilization (at about the expected day of the following period)**. This may produce confusion in determination of the expected date of delivery. *The process of implantation is controlled by the immunomodulatory role of various cytokines (interleukins 3, 4, 5, 6, 10, 13), many local peptides like epidermal growth factor (EGF), insulin like growth factor (IGF) and prostaglandins. Both the decidua and the embryo synthesize these molecules.*

TROPHOBLAST

As previously mentioned, the cells of the blastocyst differentiate into **an outer trophectoderm and an inner cell mass**. Just before implantation, the trophectoderm is further differentiated into an inner mononuclear cellular layer called **cytotrophoblast or Langhans' layer** and an outer layer of multinucleated syncytium called **syncytiotrophoblast**. The cytotrophoblasts that line the villous stems are the **villous cytotrophoblasts** (see Fig. 3.5). The cytotrophoblast cells that invade the decidua are known as '**interstitial extravillous cytotrophoblast**' (Fig. 3.5) and those that invade the lumens of the maternal spiral arteries (see Fig. 3.7) are known as '**intravascular extravillous cytotrophoblast**'.

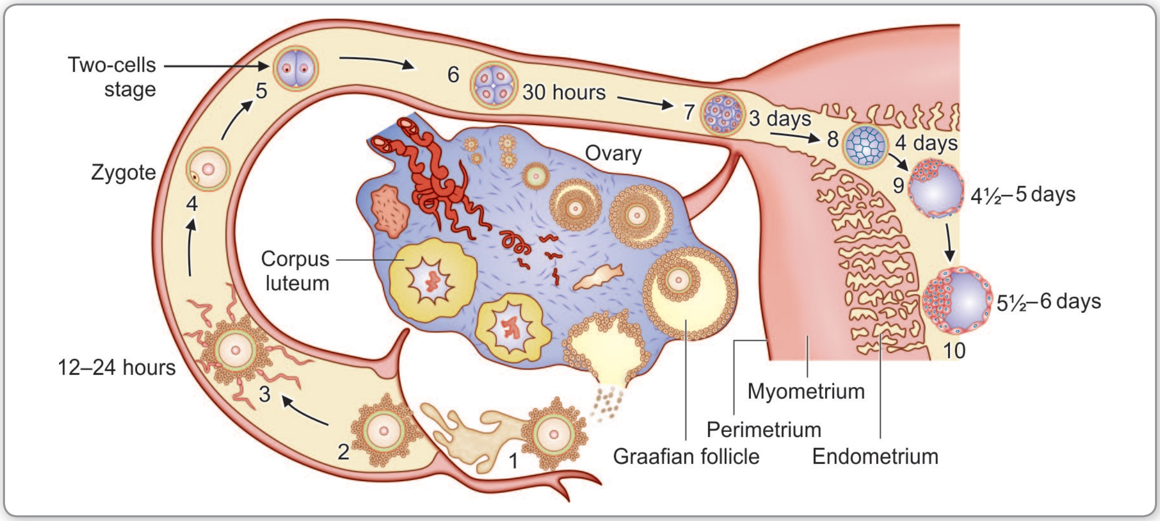


Fig. 2.6: Diagrammatic representation of the events — ovulation, fertilization and implantation: (1) Secondary oocyte extruded at ovulation (day 14 from LMP) (2) Secondary oocyte in tube (3) Fertilization with extrusion of second polar body (day 14–16) (4) Formation of zygote (5) Two-cell stage (6) Four-cell stage (7) Early morula (day 17) (8) Late morula (day 18) (9) Early blastocyst stage with disappearance of zona pellucida (day 19–20) (10) Early phase of implantation (day 20–21)

Throughout pregnancy, syncytiotrophoblast is derived from the cytotrophoblast. **Placenta and the fetal membranes are developed from the trophoblast**. It is involved in most of the functions ascribed to the placenta as a whole. **Thus, it serves at least 3 important functions — invasion, nutrition and production of hormones for the maintenance of pregnancy**. **Local cytokines regulate the invasion of the cytotrophoblasts in the decidua**.

THE DECIDUA

The decidua is the endometrium of the pregnant uterus. It is so named because much of it is shed following delivery.

Decidual reaction: **The increased structural and secretory activity of the endometrium that is brought about in response to progesterone following implantation is known as decidual reaction**.

Changes occur in all the components of the endometrium but most marked at the implantation site and first commence around maternal blood vessels. The fibrous connective tissues of the stroma become changed into epithelioid cells called decidual cells. The glands show marked dilatation and increased tortuosity with its lining epithelium showing evidences of active cell proliferation with increased secretory activity. There are areas of small interstitial hemorrhage and leukocytic infiltration specially at the implantation site.

The well developed decidua differentiates into three layers (Fig. 2.7): (1) **Superficial compact layer** consists

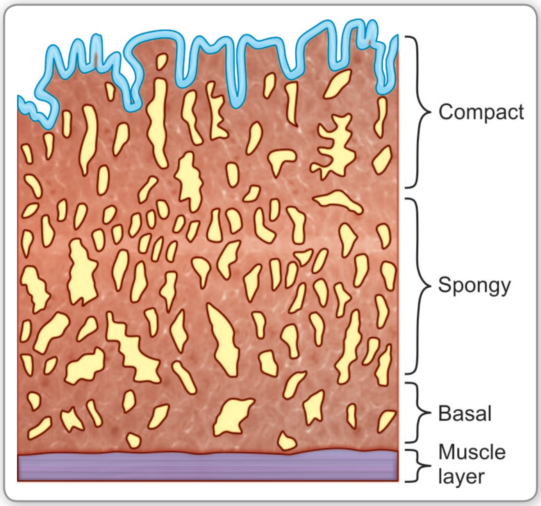


Fig. 2.7: Structure of decidua

of compact mass of decidual cells, gland ducts and dilated capillaries. The greater part of the surface epithelium is either thinned out or lost. (2) **Intermediate spongy layer** (cavernous layer) contains dilated uterine glands, decidual cells and blood vessels. **It is through this layer that the cleavage of placental separation occurs**. (3) **Thin basal layer** containing the basal portion of the glands and is opposed to the uterine muscle. **Regeneration of the mucous coat occurs from this layer following parturition**.

After the **interstitial implantation** of the blastocyst into the compact layer of the decidua, the different portions of the decidua are renamed as — (1) **Decidua basalis or serotina** — the portion of the decidua in contact with the base of the blastocyst (2) **Decidua capsularis or reflexa** — the thin superficial compact layer covering the blastocyst and (3) **Decidua vera or parietalis** — the rest of the decidua lining the uterine cavity outside the site of implantation. Its thickness progressively increases to maximum of 5–10 mm at the end of the second month and thereafter regression occurs with advancing pregnancy so that beyond 20th week, it measures not more than 1 mm.

As the growing ovum bulges towards the uterine cavity, the space between the decidua capsularis and the decidua vera, called the decidual space is gradually narrowed down and by 4th month, it is completely obliterated by the fusion of the decidua capsularis with the decidua vera. At term, they become atrophied due to pressure and the two cannot be defined as a double layer. **The decidua basalis, however, retains its characteristic appearance till term and becomes the maternal portion of the placenta (see Fig. 3.1)**.

Functions : (1) It provides a good nidus for the implantation of the blastocyst. (2) It supplies nutrition to the early stage of the growing ovum by its rich sources of glycogen and fat. (3) Deeper penetration of the trophoblast is controlled by **local peptides**, **cytokines and integrins**. (4) Decidua basalis takes part in the formation of basal plate of the placenta.

CHORION AND CHORIONIC VILLI

The chorion is the outermost layer of the two fetal membranes (chorion and amnion). It consists of two embryonic layers — outer trophoblast and inner primitive mesenchyme which appears on 9th day. At the beginning of the 3rd week, the syncytiotrophoblast produces irregular finger like projections which are lined internally by the cytotrophoblast. **These finger like buds are called primary stem villi — surrounded by lacunar spaces which will later form into intervillous spaces**.

After the appearance of the primitive mesenchyme and the development of the chorion, the primary stem villi are named **chorionic villi**. With the insinuation of the primary mesoderm into the central core of the villi structures, **secondary villi are formed on 16th day**. Later on mesodermal cells in the villi begin to differentiate into blood cells and blood vessels, thus forming villous capillary system. **These vascularized villi are called tertiary villi which are completed on 21st day**. Later on, this extra embryonic circulatory system establishes connection with the intraembryonic circulatory system through the body stalk (see Fig. 3.5).

Meanwhile, the cytotrophoblastic cells beyond the tips of the villus system penetrate into the overlying syncytium adjacent to the decidua. The cells become continuous with those of the neighboring villus system traversing through the syncytium. Thus, a thin outer cytotrophoblastic shell is formed which surrounds the entire blastocyst. The zone of the decidua immediately adjacent to the trophoblastic shell is called trophosphere which comprises of the compact layer of the decidua. Fibrinoid deposit appears on the syncytiotrophoblast outside the trophoblastic shell and is called **Nitabuch's membrane**. Maternal blood vessels pass through all the layers to reach the intervillous space (see Fig. 3.5).

The villi overlying the decidua basalis continue to grow and expand and are called **chorion frondosum** which subsequently forms the discoid placenta. The chorionic villi on the decidua capsularis gradually undergoes atrophy from pressure and become converted into **chorion laeve by the 3rd month** and lies intervening between the amnion and decidua on its outer surface. Remnant of decidual cells and of the trophoblast can however be distinguished microscopically (see Fig. 3.4).

DEVELOPMENT OF INNER CELL MASS

Along with the changes in the trophoblast, **on the 8th day**, the embryoblast differentiates into **bilaminar germ disc** which consists of **dorsal ectodermal layer** of tall columnar cells and ventral endodermal layer of flattened polyhedral cells. **The bilaminar germ disc is connected with the trophoblast by mesenchymal condensation, called connecting stalk or body stalk which later on forms the umbilical cord (Fig. 2.9)**.

Two cavities appear one on each side of the germ disc. (1) On 12th postovulatory day, a fluid filled space appears between the ectodermal layer and the cytotrophoblast which is called **amniotic cavity**. Its floor is formed by the **ectoderm** and the rest of its wall by **primitive mesenchyme**. (2) The **yolk sac** appear on the ventral aspect of the bilaminar disk and is lined externally by the primitive mesenchyme and internally by the migrating **endodermal cells from the endodermal layer of the germ disc (Fig. 2.9A)**.

Formation of trilaminar embryonic disk: Fourteen days after fertilization, proliferation of ectodermal cells in the midline, leads to formation of **primitive streak** (Fig. 2.8). Cells within the streak spread laterally between the ectoderm and endoderm as intraembryonic mesoderm. This intraembryonic mesoderm becomes continuous with the extraembryonic mesoderm at the lateral border of the embryonic disk.

Extraembryonic coelom (Fig. 2.9): Extraembryonic mesenchyme, derived from the trophoblast appears to separate the yolk sac from the blastocyst wall and also the amniotic cavity from the trophoblast of the chorion. Small cystic spaces (lacuna) now appear within the extraembryonic mesenchyme. These spaces gradually enlarge and fuse to form **extraembryonic coelom**. Progressive enlargement of the extraembryonic coelom, separates the amnion from the inner aspect of the chorion except at the caudal end of the embryo. There, the mesenchymal attachment persists to form **body stalk** (Fig. 2.9A). **Umbilical cord** develops from this body stalk.

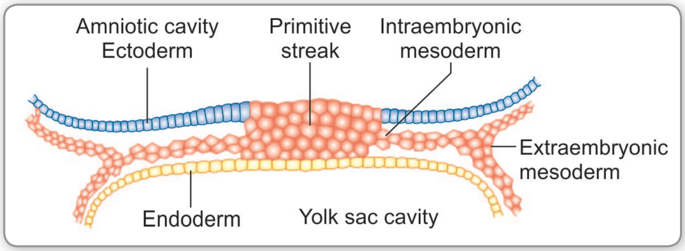


Fig. 2.8: Transverse section through the primitive streak region, showing invagination and lateral migration of the mesodermal cells, converting the bilaminar disk into a trilaminar one

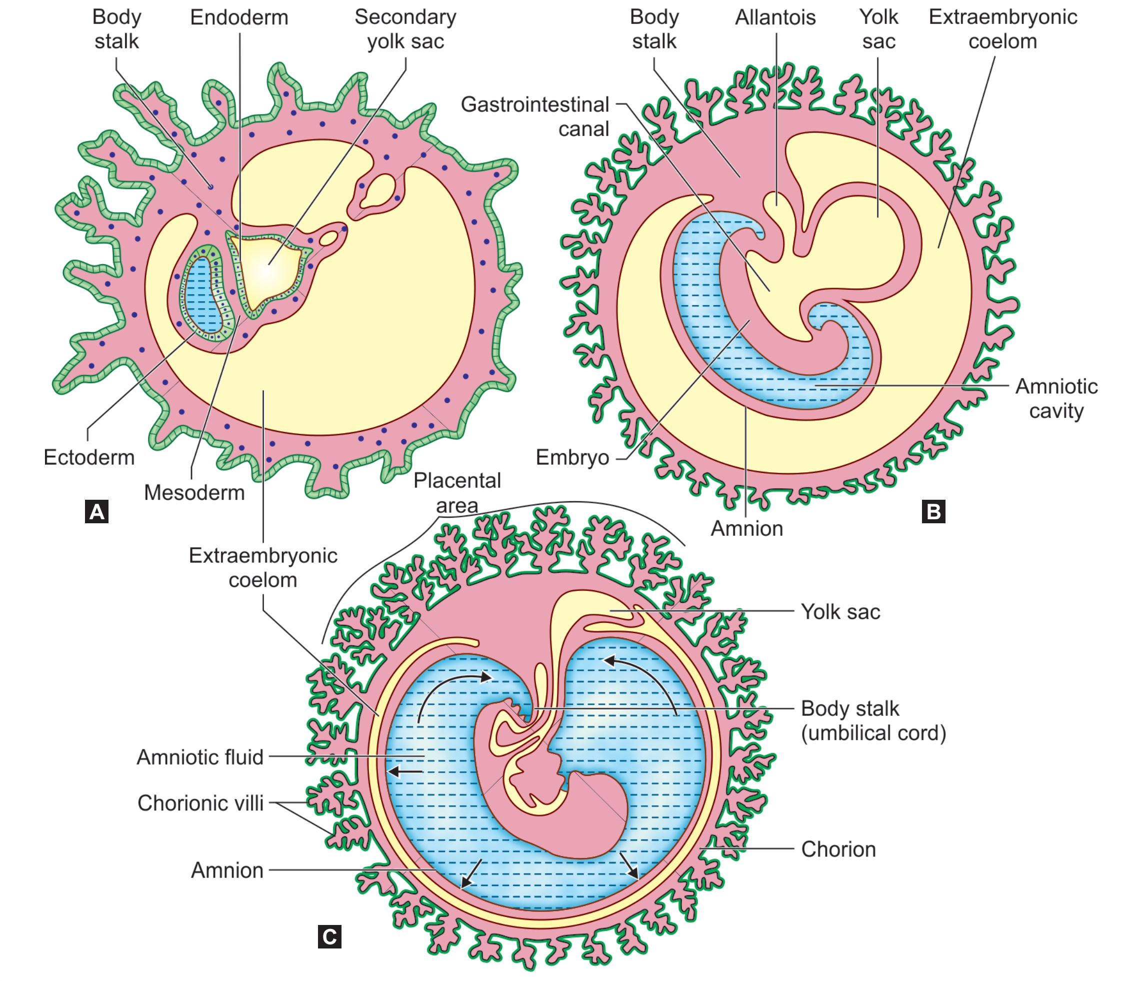
Subsequently the **amniotic cavity** enlarges at the expense of the extraembryonic coelom. The developing embryo bulges into the enlarged amniotic cavity. The **yolk sac** becomes partly incorporated into the embryo to form the gut. The part that remains outside is incorporated into the body stalk (Fig. 2.9B, C). Gradually the extraembryonic coelom is totally obliterated. The extraembryonic mesenchyme covering the amnion now fuses with the lining of the chorion. The single layer of fused amniochorion is now formed (Fig. 2.9C).

During the embryonic stage which extends from the fourth to eighth week, individual differentiation of the germ layers and formation of the folds of the embryo occur. Most of the tissues and organs are developed during this period, the details of which are beyond the description of this book. However, the major structures which are developed from the three germinal layers are mentioned below. The embryo can be differentiated as human at 8th week.

ECTODERMAL LAYER: Central and peripheral nervous system, epidermis of skin with its appendages, pituitary gland, chromaffin organs, salivary glands; mucous lining of the nasal cavity, paranasal sinus, roof of the mouth etc.

MESODERMAL LAYER: Bones, cartilage, muscles, cardiovascular system, kidney, gonads, suprarenals, spleen, most of the genital tract; mesothelial lining of pericardial, pleural and peritoneal cavity etc.

ENDODERMAL LAYER: Epithelial lining of the gastrointestinal tract, liver, gallbladder, pancreas; epithelial lining of respiratory tract and most of the mucous membrane of urinary bladder and urethra; bulbourethral and greater vestibular glands etc.


Body stalk
Endoderm
Secondary yolk sac
Body stalk
Allantois
Yolk sac
Extraembryonic coelom
Gastrointestinal canal
Amniotic cavity
Ectoderm
Mesoderm
A
Placental area
Embryo
Amnion
B
Extraembryonic coelom
Yolk sac
Body stalk (umbilical cord)
Amniotic fluid
Chorionic villi
Amnion
Chorion
C

Figs 2.9A to C: Schematic representation of formation of amniotic cavity, secondary yolk sac, extraembryonic coelom and body stalk: (A) Enlargement of extraembryonic coelomic cavity (B) The amniotic sac enlarges and begins to occupy the extraembryonic coelom (C) The amniotic sac has surrounded the embryo with almost completely obliterating the extraembryonic coelom; formation of body stalk completed

Table 2.1: Important Events Following Fertilization

'0' hour	— Fertilization (day-15 from LMP)
30 hours	— 2 cell stage (blastomeres)
40–50 hours	— 4 cell stage
72 hours	— 12 cell stage
96 hours	— 16 cell stage. Morula enters the uterine cavity
5th day	— Blastocyst
4–5th day	— Zona pellucida disappears
5–6th day	— Blastocyst attachment to endometrial surface
6–7th day	— Differentiation of cyto and syncytiotrophoblastic layers
10th day	— Synthesis of hCG by syncytiotrophoblast
9–10th day	— Lacunar network forms
10–11th day	— • Trophoblasts invade endometrial sinusoids establishing uteroplacental circulation • Interstitial implantation completed with entire decidual coverage
13th day	— Primary villi
16th day	— Secondary villi
21st day	— Tertiary villi
21st–22nd day	— Fetal heart. Fetoplacental circulation

QUESTIONS

1. What is decidua? What are the different types of decidua? (p. 27-28)
2. Mention the functions of decidua? (p. 28)
3. What is chorion? What is chorion frondosum? (p. 28, 32)
4. What are the different types of choronic villi? (p. 28)

Chapter 3

The Placenta and Fetal Membranes

THE PLACENTA

Only eutherian mammals possess placenta. **The human placenta is discoid**, because of its shape; **hemochorial**, because of direct contact of the chorion with the maternal blood and **deciduate**, because some maternal tissue is shed at parturition. The placenta is attached to the uterine wall and establishes connection between the mother and fetus through the umbilical cord. The fact that maternal and fetal tissues come in direct contact without rejection suggests immunological acceptance of the fetal graft by the mother.

DEVELOPMENT

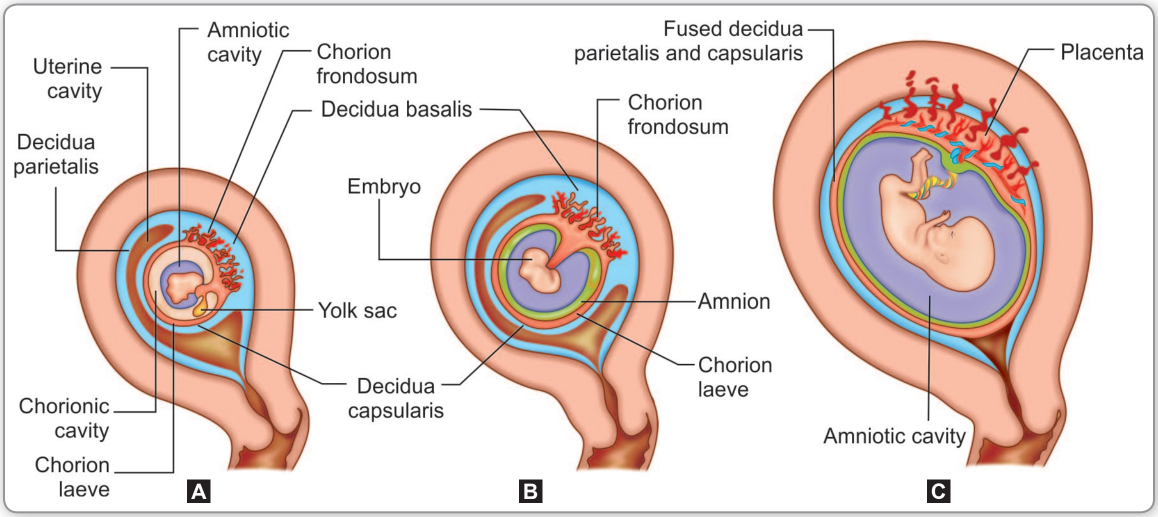
tThe placenta is developed from two sources. The principal component is fetal which develops from the chorion frondosum and the maternal component consists of decidua basalis.

When the **interstitial implantation is completed on 11th day**, the blastocyst is surrounded on all sides by lacunar spaces around cords of syncytial cells, called trabeculae. From the trabeculae develops the stem villi on 13th day which connect the chorionic plate with the basal plate. Primary, secondary and tertiary villi are successively developed from the stem villi. **Arterio-capillary-venous system in the mesenchymal core of each villus is completed on 21st day**. This ultimately makes connection with the intraembryonic vascular system through the body stalk (Fig. 2.9).

Simultaneously, lacunar spaces become confluent with one another and by 3rd–4th week, form a multilocular receptacle lined by syncytium and filled with maternal blood. This space becomes the future intervillous space. As the growth of the embryo proceeds, decidua capsularis becomes thinner beginning at 6th week and both the villi and the lacunar spaces in the abembryonic area get obliterated, converting the chorion into chorion laeve. This is, however, compensated by (a) exuberant growth and proliferation of the decidua basalis and (b) enormous and exuberant division and subdivision of the chorionic villi in the embryonic pole (chorion frondosum). **These two, i.e., chorion frondosum and the decidua basalis form the discrete placenta. It begins at 6th week and is completed by 12th week (Fig. 3.1)**.

Until the end of the 16th week, the placenta grows both in thickness and circumference due to growth of the chorionic villi with accompanying expansion of the intervillous space. Subsequently, there is little increase in thickness but it increases circumferentially till term.

The human hemochorial placenta derived its name from hemo- (blood) that is in contact with the syncytiotrophoblasts of chorionic tissue (Fig. 3.4).


Uterine cavity
Decidua parietalis
Amniotic cavity
Chorion frondosum
Decidua basalis
Yolk sac
Decidua capsularis
Chorionic cavity
Chorion laeve
A
Embryo
Chorion frondosum
Amnion
Chorion laeve
B
Fused decidua parietalis and capsularis
Placenta
Amniotic cavity
C

Figs 3.1A to C: Relation of the amniotic cavity, chorionic cavity and uterine cavity of successive stages — (A) end of the 8th week; (B) 10 weeks after the last period; (C) end of the 12th week

THE PLACENTA AT TERM

GROSS ANATOMY: The placenta, at term, is almost a circular disk with a diameter of 15–20 cm and thickness of about 3 cm at its center. It thins off toward the edge. It feels spongy and weighs about 500 gm, the proportion to the weight of the baby being roughly 1 : 6 at term and occupies about 30% of the uterine wall. **It presents two surfaces, fetal and maternal, and a peripheral margin**.

Fetal surface: The fetal surface is covered by the smooth and glistening amnion with the umbilical cord attached at or near its center. Branches of the umbilical vessels are visible beneath the amnion as they radiate from the insertion of the cord (Fig. 3.2). The amnion can be peeled off from the underlying chorion except at the insertion of the cord. **At term, about four-fifths of the placenta is of fetal origin**.

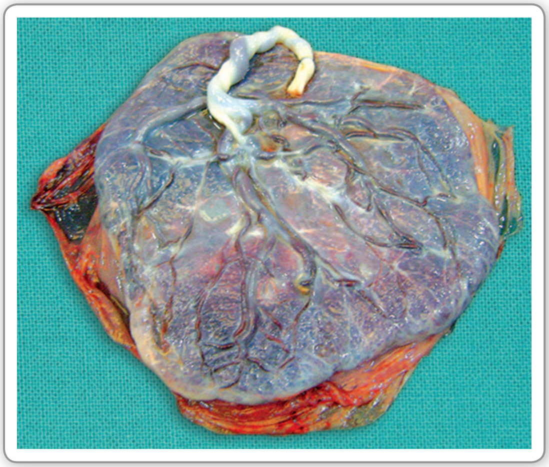


Fig. 3.2: Fetal surface of the placenta showing attachment of the umbilical cord with ramification of the umbilical vessels

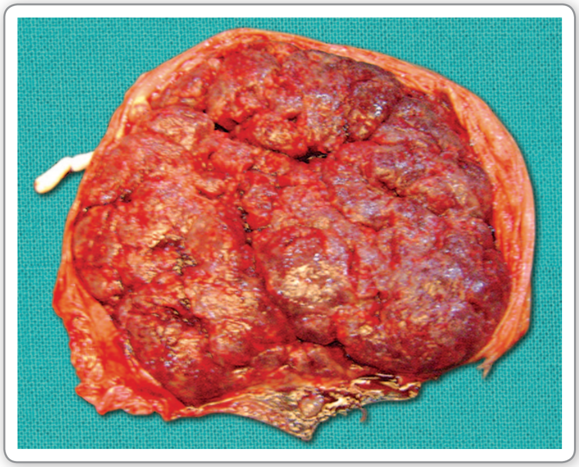


Fig. 3.3: Maternal surface of the placenta showing shaggy look with cotyledons limited by fissures

Maternal surface: The maternal surface is rough and spongy (Fig. 3.3). Maternal blood gives it a dull red color. A thin grayish, somewhat shaggy layer which is the **remnant of the decidua basalis (compact and spongy layer)** and has come away with the placenta, may be visible. The maternal surface is mapped out into 15–20 somewhat convex polygonal areas known as **lobes or cotyledons** which are limited by fissures. Each fissure is occupied by the **decidual septum which is derived from the basal plate**. Numerous small grayish spots are visible. These are due to deposition of calcium in the degenerated areas and are of no clinical significance. The maternal portion of the placenta amounts to less than one-fifth of the total placenta. **Only the decidua basalis and the blood in the intervillous space are of maternal origin**.

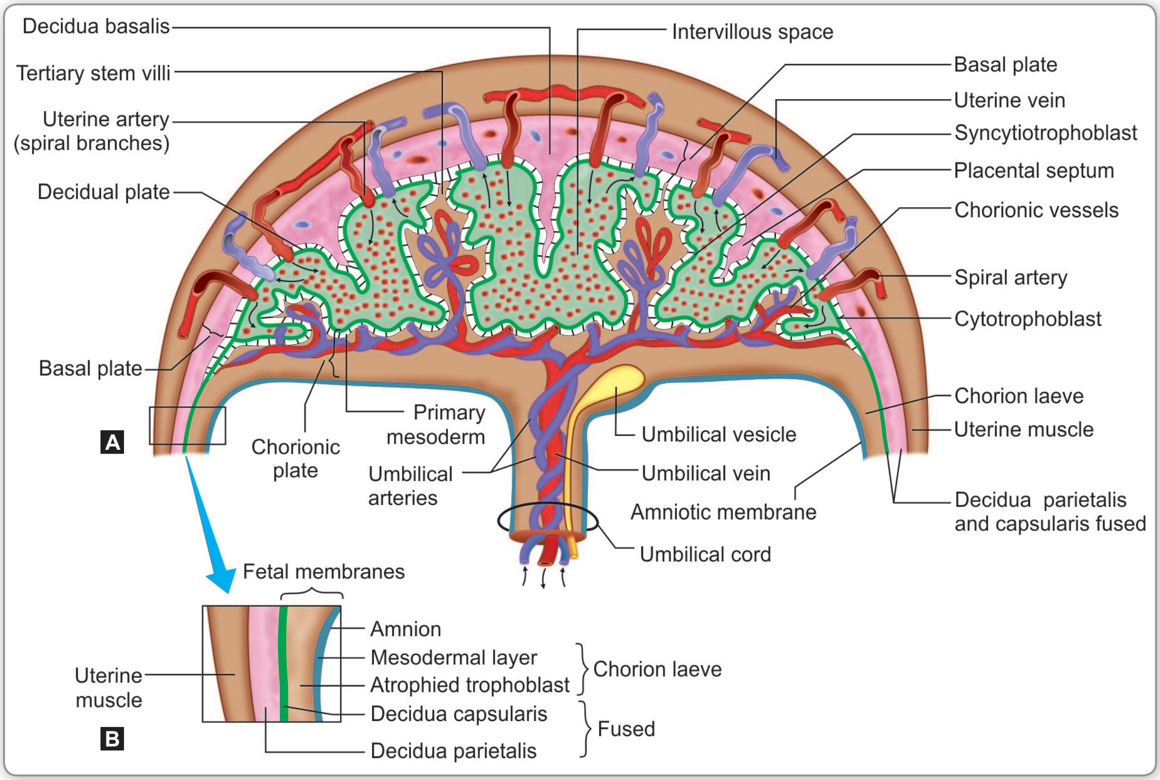
Margin: Peripheral margin of the placenta is limited by the fused basal and chorionic plates and is continuous with the chorion laeve and amnion. Essentially, the chorion and the placenta are one structure but **the placenta is a specialized part of the chorion**.

Attachment: The placenta is usually attached to the upper part of the body of the uterus encroaching to the fundus adjacent to the anterior or posterior wall with equal frequency. **The attachment to the uterine wall is effective due** to anchoring villi connecting the chorionic plate with the basal plate and also by the fused decidua capsularis and vera with the chorion laeve at the margin.

Separation: Placenta separates after the birth of the baby and **the line of separation is through the decidua spongiosum**.

STRUCTURES

The placenta consists of two plates. **The chorionic plate** lies internally. It is lined by the amniotic membrane. The umbilical cord is attached to this plate. **The basal plate** lies to the maternal aspect. Between the two plates lies the intervillous space containing the stem villi with their branches, the space being filled with maternal blood (Fig. 3.4).


Decidua basalis
Tertiary stem villi
Uterine artery (spiral branches)
Decidual plate
Basal plate
A
Chorionic plate
Primary mesoderm
Umbilical arteries
Intervillous space
Basal plate
Uterine vein
Syncytiotrophoblast
Placental septum
Chorionic vessels
Spiral artery
Cytotrophoblast
Chorion laeve
Uterine muscle
Umbilical vesicle
Umbilical vein
Amniotic membrane
Umbilical cord
Decidua parietalis and capsularis fused
Fetal membranes
Uterine muscle
B
Amnion
Mesodermal layer
Atrophied trophoblast
Chorion laeve
Decidua capsularis
Decidua parietalis
Fused

Figs 3.4A and B: Schematic view of (A) Structure of placenta at term; (B) Structure of the membranes in relation to decidua

AMNIOTIC MEMBRANE: It consists of single layer of cubical epithelium loosely attached to the adjacent chorionic plate. It takes no part in formation of the placenta.

CHORIONIC PLATE: From within outward, it consists of (i) primitive mesenchymal tissue containing branches of umbilical vessels, (ii) a layer of cytotrophoblast and (iii) syncytiotrophoblast. The stem villi arise from the plate. **It forms the inner boundary of the choriodecidual space**.

BASAL PLATE: It consists of the following structures from outside inwards. (1) Part of the compact and spongy layer of the decidua basalis; (2) Nitabuch's layer of fibrinoid degeneration of the outer syncytiotrophoblast at the junction of the cytotrophoblastic shell and decidua; (3) Cytotrophoblastic shell; (4) Syncytiotrophoblast (Figs 3.4 and 3.5).

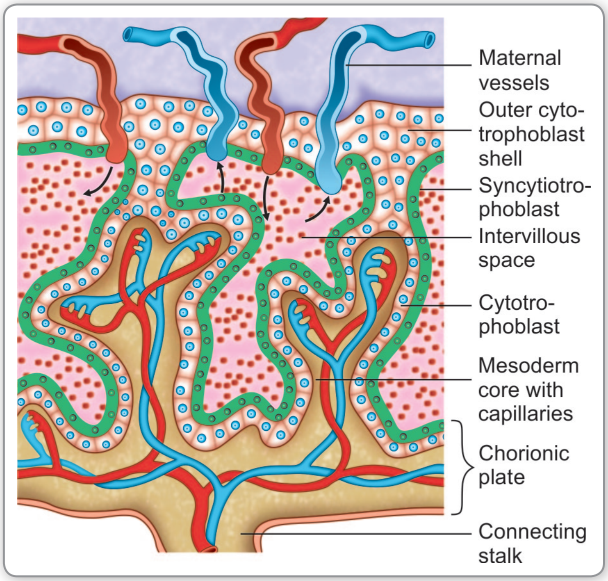
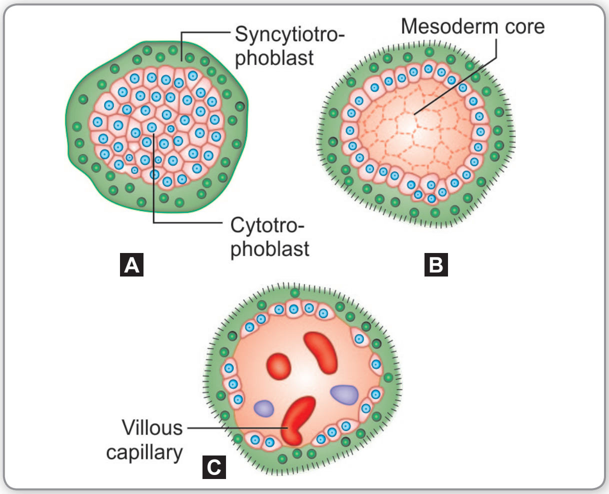


Fig. 3.5: Schematic diagram of stem villi, showing connection of the chorionic plate with the basal plate

The basal plate is perforated by the spiral branches of the uterine vessels through which the maternal blood flows into the intervillous space. At places, placental or **decidual septa** project from the basal plate into the intervillous space but fail to reach the chorionic plate. The septum consists of decidual elements covered by trophoblastic cells. The areas between the septa are known as **cotyledons (lobes)**, which are observed from the maternal surface, numbering 15–20.

INTERVILLOUS SPACE: It is bounded on the inner side by the chorionic plate and the outer side by the basal plate, limited on the periphery by the fusion of the two plates. **It is lined internally on all sides by the syncytiotrophoblast and is filled with slow flowing maternal blood**. Numerous branching villi which arise from the stem villi project into the space and constitute chief content of the intervillous space (Figs 3.4A and 3.5).

STEM VILLI: These arise from the chorionic plate and extends to the basal plate. With the progressive development — primary, secondary and tertiary villi are formed (Fig. 3.6). Functional unit of the placenta is called a **fetal cotyledon** or **placentome**, which is derived from a **major primary stem villus**. These major stem villi pass down through the intervillous space to anchor onto the basal plate (see Fig. 3.5). Functional subunit is called a **lobule**, which is derived from a **tertiary stem villi**. About 60 stem villi persist in human placenta. **Thus, each cotyledon (total 15–29) contains 3–4 major stem villi**. The villi are the functional unit of the placenta. The total villi surface, for exchange, approximately varies between 10 square meters and 14 square meters. The fetal



Figs 3.6A to C: Schematic diagram to show the development of the villus. (A) Primary; (B) Secondary; (C) Tertiary

capillary system within the villi is almost 50 km long. Thus while some of the villi are **anchoring the placenta** to the decidua, the majority are free within the intervillous space and are called **nutritive villi**. Blood vessels within the branching villi do not anastomose with the neighboring one.

STRUCTURE OF A TERMINAL VILLUS: In the early placenta, each terminal villus has got the following structures from outside inward: (1) Outer syncytiotrophoblast; (2) cytotrophoblast; (3) basement membrane; (4) Central stroma containing fetal capillaries, primitive mesenchymal cells, connective tissue and a few phagocytic (Hofbauer) cells.

In placenta at term, syncytiotrophoblast becomes relatively thin at places overlying the fetal capillaries and thicker at other areas containing extensive endoplasmic reticulum. The former is probably the site for transfer and the latter, the site for synthesis. The cytotrophoblast is relatively sparse. Basement membrane becomes thicker. Stroma contains dilated vessels along with all the constituents and few Hofbauer cells. **Hofbauer cells** are round cells that are capable of phagocytosis and can trap maternal antibodies crossing through the placenta (immune suppressive). These cells can express class II major histocompatibility complex (MHC) molecules.

PLACENTAL CIRCULATION

Placental circulation consists of independent circulation of blood in two systems:

- **Uteroplacental circulation**
- **Fetoplacental circulation**

UTEROPLACENTAL CIRCULATION (maternal circulation): **It is concerned with the circulation of the maternal blood through the intervillous space**. A mature placenta has a volume of about 500 mL of blood; 350 mL being occupied in the villi system and 150 mL lying in the intervillous space. As the intervillous blood flow at term is estimated to be 500–600 mL per minute, **the blood in the intervillous space is completely replaced about 3–4 times per minute**. **The villi depend on the maternal blood for their nutrition**, thus it is possible for the chorionic villi to survive for a varying period even after the fetus is dead. The pressure within the intervillous space is about 10–15 mm Hg during uterine relaxation and 30–50 mm Hg during uterine contraction. In contrast, the fetal capillary pressure in the villi is 20–40 mm Hg.

Arterial circulation: About 120–200 spiral arteries open into the intervillous space by piercing the basal plate randomly at numerous sites. Normally, there is cytotrophoblastic invasion into the spiral arteries initially up to the intradecidual portion within 12 weeks of pregnancy. Not only the endothelial

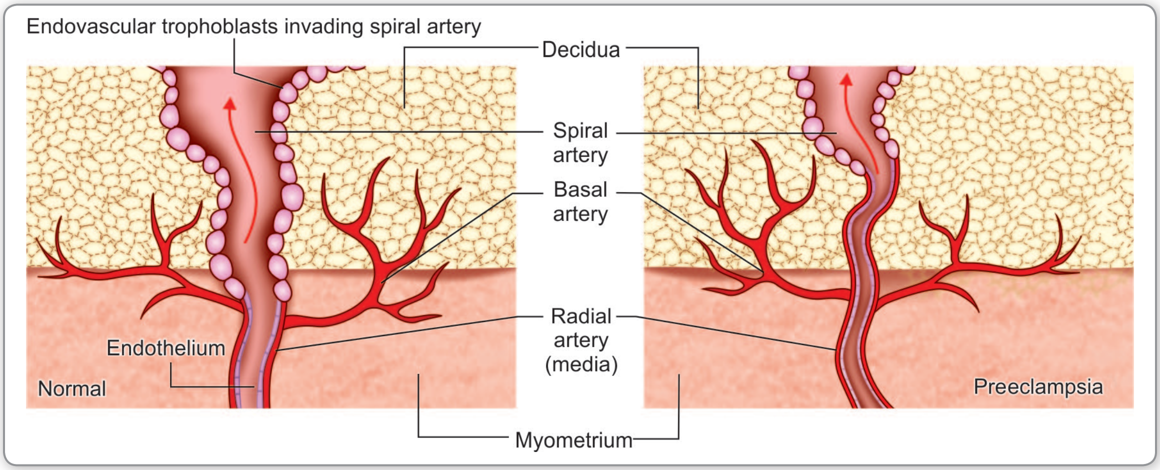


Fig. 3.7: Spiral arterioles at the placental site in normal and pre-eclamptic pregnancies

lining is replaced but also the musculoelastic media is destroyed and replaced by fibrinoid material. There is a secondary invasion of trophoblast between 12 weeks and 16 weeks extending up to radial arteries within the myometrium. Thus, spiral arteries are converted to large bore **uteroplacental arteries**. The net effect is funneling of the arteries which reduces the pressure of the blood to 70–80 mm Hg before it reaches the intervillous space. It thus increases the blood flow.

Trophoblast cells that do not take part in villous structure are known as extravillous trophoblast (EVT). EVT are of two types: (i) **endovascular** that migrates down the lumen of the spiral arteries and replaces the endothelium (Fig. 3.7) and (ii) **interstitial** that invades as far as the inner third of the myometrium. Further invasion is limited by the NK cells to prevent morbid adhesion of placenta (placenta accreta). Defects in trophoblast invasion and failure to establish maternal circulation correctly leads to complications of pregnancy (PIH, IUGR).

Venous drainage: The venous blood of the intervillous space drains through the uterine veins which pierce the basal plate randomly like the arteries. This concept of uteroplacental circulation is based on the studies of Ramsey and coworkers (1963, 1966). Intervillous hemodynamics is mentioned in Table 3.1.

Table 3.1: Summary of Intervillous Hemodynamics

▪ Volume of blood in mature placenta	500 mL
▪ Volume of blood in intervillous space	150 mL
▪ Blood flow in intervillous space	500–600 mL/min
▪ Pressure in intervillous space:	
• During uterine contraction	30–50 mm Hg
• During uterine relaxation	10–15 mm Hg
▪ Pressure in the supplying uterine artery	70–80 mm Hg
▪ Pressure in the draining uterine vein	8 mm Hg

Circulation in the intervillous space: The arterial blood enters the space under pressure (Fig. 3.8). Lateral dispersion occurs, after it reaches the chorionic plate. Villi help in mixing and slowing of the blood flow. Mild stirring effect by the villi pulsation aided by uterine contraction help migration of the blood toward the basal plate and thence to the uterine veins. Sometimes syncytial sprouts are set free in the intravillous circulation and are carried through the maternal circulation to the lungs where they disappear by lysis. About 100,000 syncytial sprouts circulate in maternal blood in 24 hours.

Short circuit of the arterial blood into the neighboring venous channels is prevented by the increased pressure of the endometrial arteries driving the blood in jets towards the chorionic plate. During uterine contraction, the veins are occluded but the arterial blood is forced into the intervillous space; while uterine relaxation facilitates venous drainage. This is brought about by the fact that the spiral arteries are perpendicular and the veins are parallel to the uterine wall. **Thus during contraction, larger volume of blood is available for exchange even though the rate of flow is decreased**. The blood in the intervillous space is protected from clotting by some fibrinolytic enzyme activity of the trophoblast.

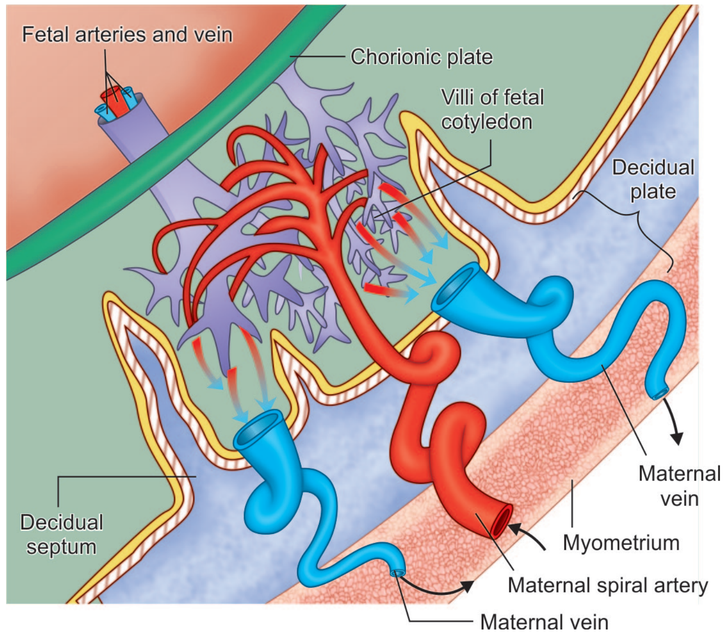


Fig. 3.8: Diagram of an intervillous space showing intervillous circulation

FETOPLACENTAL CIRCULATION: The two umbilical arteries carry the impure blood from the fetus. They enter the chorionic plate underneath the amnion, each supplying one half of the placenta. The arteries break up into small branches which enter the stems of the chorionic villi. Each in turn divides into primary, secondary and tertiary vessels of the corresponding villi. The blood flows into the corresponding venous channels either through the terminal capillary networks or through the shunts (Fig. 3.9). Maternal and fetal bloodstreams flow side by side, but in opposite direction. This countercurrent flow facilitates material exchange between the mother and fetus. The villous capillary pressure varies from 20–40 mm Hg. **The fetal blood flow through the placenta is about 400 mL/min**. This is mainly facilitated by the pumping action of the fetal heart.

PLACENTAL BARRIER (placental membrane): It is a partition between fetal and maternal circulation. However, this barrier is not a perfect barrier as fetal blood cells are found in maternal circulation so also the maternal blood cells are found in fetal circulation. The above two are separated by tissues called placental membrane or barrier, consisting of the following. **In early pregnancy, it consists of** (1) syncytiotrophoblast, (2) cytotrophoblast, (3) basement membrane, (4) stromal tissue, and (5) endothelium of the fetal capillary wall with its basement membrane. **It is about 0.025 mm thick (Fig. 3.10)**.

Near term, there is attenuation of the syncytial layer. Sparse cytotrophoblast and distended fetal capillaries almost fill the villus. The specialized zones of the villi where the syncytiotrophoblast is thin and anuclear is known as **vasculosyncytial membrane** (Fig. 3.11). These thin zones (0.002 mm) of terminal villi **alpha zones** are for gas exchange. The thick "**beta zones**" of the terminal villi with the layers remaining thick in patches are for hormone synthesis. An increase in thickness of the villous membrane is seen in cases with IUGR and cigarette smokers.

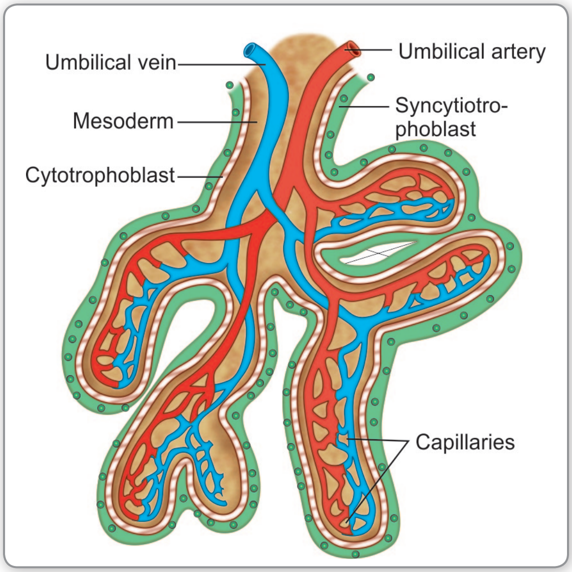

Umbilical vein
Umbilical artery
Mesoderm
Syncytiotro-
phoblast
Cytotrophoblast
Capillaries

Fig. 3.9: Schematic diagram showing terminal capillary networks in the villi

Table 3.2: Summary of Fetal Hemodynamics

Fetal blood flow through the placenta	400 mL/min	
Pressure in the umbilical artery	60 mm Hg	
Pressure in the umbilical vein	10 mm Hg	
Fetal capillary pressure in villi	20–40 mm Hg	
	Umbilical artery	**Umbilical vein**
O_2 saturation	50–60%	70–80%
PO_2	20–25 mm Hg	30–40 mm Hg

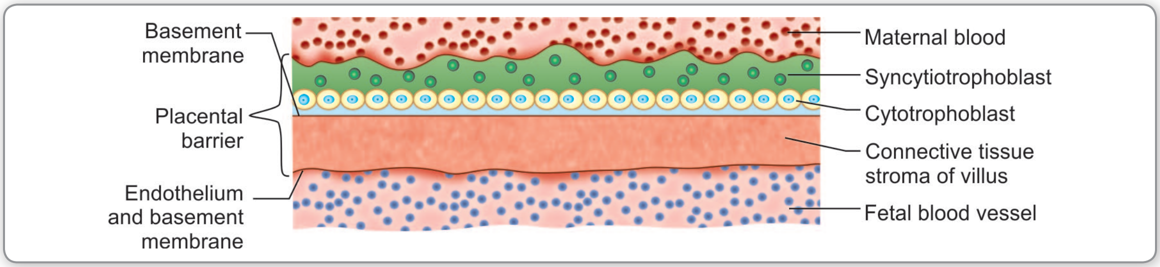

Basement
membrane
Placental
barrier
Endothelium
and basement
membrane
Maternal blood
Syncytiotrophoblast
Cytotrophoblast
Connective tissue
stroma of villus
Fetal blood vessel

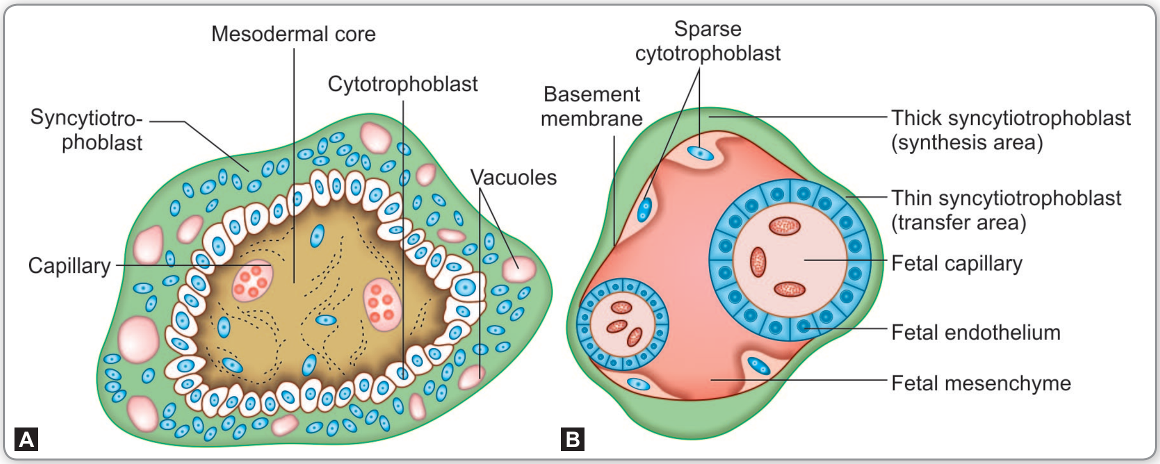
Fig. 3.10: Schematic diagram showing layers of placental barrier

PLACENTAL AGING

As the placenta has got a limited life span, it is likely to undergo degenerative changes as a mark of senescence. The aging process varies in degree and should be differentiated from the morbid process likely to affect the organ in some pathological states. **The aging process involves both the fetal and maternal components**.

VILLI CHANGES: The following changes are observed as pregnancy advances toward term (Fig. 3.11).

(1) Decreasing thickness of the syncytium and appearances of **syncytial knots** (aggregation of the syncytium in small areas on the sides of the villus); (2) Partial disappearance of trophoblast cells; (3) Decrease in the stromal tissue including Hofbauer cells (fetal macrophages); (4) Obliteration of some vessels and marked dilatation of the capillaries; (5) Thickening of the basement layer of the fetal endothelium and the cytotrophoblast; (6) Deposition of fibrin on the surface of the villi.


Mesodermal core
Cytotrophoblast
Syncytiotro-phoblast
Vacuoles
Capillary
A
Sparse cytotrophoblast
Basement membrane
Thick syncytiotrophoblast (synthesis area)
Thin syncytiotrophoblast (transfer area)
Fetal capillary
Fetal endothelium
Fetal mesenchyme
B

Figs 3.11A and B: Schematic diagram showing transverse section of a villus in (A) early month and (B) near term

DECIDUAL CHANGES: There is an area of fibrinoid degeneration where trophoblast cells (covered with syncytium) meet the decidua. This zone is known as **Nitabuch's layer**. This layer limits further invasion of the decidua by the trophoblast. **The membrane is absent in placenta accreta**.

INTERVILLOUS SPACE: The syncytium covering the villi and extending into the decidua of intervillous space undergoes fibrinoid degeneration and form a mass entangling variable number of villi. These are called white infarcts which vary in size from few millimeters to a centimeter or more. Calcification or even cyst formation may occur on it. Such type of degeneration is usually near the placental margin. There may be inconsistent deposition of fibrin called **Rohr's stria** at the bottom of the intervillous space and surrounding the fastening villi.

PLACENTAL FUNCTION

The main functions of the placenta are:

1. Transfer of nutrients and waste products between the mother and fetus. In this respect, it attributes to the following functions: • **Respiratory;** • **Excretory;** • **Nutritive**
2. **Endocrine function:** Placenta is an endocrine gland. It produces both steroid and peptide hormones to maintain pregnancy (p. 66).
3. **Barrier function.**
4. **Immunological function.**

Table 3.3: Factors for Placental Transfer from Mother to Fetus

A. Substance properties

- **Molecular weight:** Lower the molecular weight, more is the transfer
- **Lipid solubility:** Lipophilic substances diffuse readily
- **Ionization:** Nonionized form crosses lipid membrane freely
- **pH of blood:** Lower pH favors ionization of many drugs
- **Protein binding**
- **Spatial configuration**

B. Maternal properties

- Drug concentration in the maternal blood
- Uterine blood flow
- Concentration gradient on either side of placental membranes

C. Placental properties

- Lipid membrane of placenta enhances transfer
- Total surface area of placental membrane
- Functional integrity and thickness of placental barrier (p. 38)

Mechanisms involved in the transfer of substances across the placenta are:

(1) **Simple diffusion.** (2) **Facilitated diffusion** (transporter mediated) using transporter proteins in syncytiotrophoblast. (3) **Active transfer** (against concentration gradient, energy ATPase-mediated). (4) **Endocytosis:** Invagination of the cell membrane to form an intracellular vesicle which contains the extracellular molecules. (5) **Exocytosis:** Release of the molecule within vesicle to the extracellular space. Immunoglobulin IgG is taken up by endocytosis from maternal circulation and is transferred to the fetus via exocytosis. (6) **Leakage** (break in the placental membranes).

Respiratory function: Although the fetal respiratory movements are observed as early as 11 weeks, there is no gaseous exchange. Intake of oxygen and output of carbon dioxide take place by **simple diffusion** across the fetal membrane. Partial pressure gradient is the driving force for exchange between the maternal and fetal circulations. **The oxygen supply to the fetus is at the rate of 8 mL/Kg/min and this is achieved with cord blood flow of 165–330 mL/min**.

Excretory function: Waste products from the fetus such as urea, uric acid, and creatinine are excreted in the maternal blood **by simple diffusion.**

Nutritive function: The fetus obtains its nutrients from the maternal blood and when the diet is inadequate, then only depletion of maternal tissue storage occurs.

- **Glucose** which is the principal source of energy is transferred to the fetus **by facilitated diffusion.** There are transporter proteins (GLUT-1) for facilitated diffusion. GLUT-1 is located in syncytiotrophoblast. Glucose transfer from mother to fetus is not linear. Transfer rates decrease as maternal glucose concentration increases. Fetal glucose level is lower than that of the mother indicating rapid rate of fetal utilization of glucose.
- **Lipids** for fetal growth and development are transferred across the fetal membrane or synthesized in the fetus. **Triglycerides and fatty acids** are directly transported from the mother to the fetus in early pregnancy but probably are synthesized in the fetus later in pregnancy. Essential fatty acids are transferred more than the non-essential fatty acids. **Cholesterol** is capable of direct transfer. **Thus, fetal fat has got a dual origin**.
- **Amino acids** are transferred by active transport (energy requiring transport) through enzymatic mechanism (ATPase). Amino acid concentration is **higher in the fetal blood** than in the maternal blood. Some proteins (IgG), cross by the process of endocytosis. Fetal proteins are synthesized from the transferred amino acids and the level is lower than in mother.
- **Water and electrolytes** such as sodium, potassium and chloride cross through the fetal membrane by simple diffusion, whereas calcium, phosphorus and iron cross **by active transport** (active transporter proteins) against a concentration gradient, since their levels are higher in fetal than in maternal blood. **Water soluble vitamins** are transferred by active transport, but the **fat soluble vitamins** are transferred slowly so that the latter remains at a low level in fetal blood.

- **Hormones** — Insulin, steroids from the adrenals, thyroid, chorionic gonadotrophin or placental lactogen cross the placenta at a very slow rate, so that their concentration in fetal plasma are appreciably lower than in maternal plasma. **Neither parathormone nor calcitonin crosses the placenta**.

ENZYMATIC FUNCTION: Numerous enzymes are elaborated in the placenta, mentioning only few of them are: (1) diamine oxidase which inactivates the circulatory pressure (or pressor) amines, (2) oxytocinase which neutralizes the oxytocin, (3) phospholipase A_2 which synthesizes arachidonic acid, etc. **Placental endocrinology** is discussed in the Chapter 6 (p. 66).

BARRIER FUNCTION: Fetal membrane has long been considered as a protective barrier to the fetus against noxious agents circulating in the maternal blood. In general, substances of high molecular weight of more than 500 daltons are held up, but there are exceptions. Antibody and antigens in immunological quantities can traverse across the placental barrier in both directions. The transfer of the larger molecule is probably facilitated by pinocytosis. The race of drug transfer is increased in late pregnancy.

Maternal infections during pregnancy by virus (rubella, chickenpox, measles, mumps, poliomyelitis), bacteria (*Treponema pallidum, Tubercle bacillus*) or protozoa (*Toxoplasma gondii*, malaria parasites) may be transmitted to the fetus across the so-called placental barrier and affect the fetus in utero. Similarly, **almost any drug used in pregnancy can cross the placental barrier and may have deleterious effect on the fetus.**

IMMUNOLOGICAL FUNCTION (for details see p. 196, 719): The fetus and the placenta contain paternally determined antigens, which are foreign to the mother. In spite of this, there is no evidence of graft rejection. Placenta probably offers immunological protection against rejection. **The exact mechanism is yet speculative, but the interest is centered on the following:**

- Placental hormones, proteins (SP1), early pregnancy factor (EPF), PAPP-A, steroids and chorionic gonadotropin have got some **immunosuppressive effect**.
- Villous trophoblasts (Fig. 3.5) do not express HLA Class I or Class II molecules. **Extravillous trophoblast (Fig. 3.7) only express HLA Class I molecules and no HLA Class II molecules (see p. 26).**
- Though anti-HLA antibodies and sensitized T cells against paternal antigens have been detected in the maternal serum, they have **no significant effects on pregnancy**.
- There is a **shift of maternal response from cell-mediated (T helper 1) to humoral (T helper 2) immunity,** which may be beneficial to pregnancy (see p. 720).
- Decidual **natural killer (NK) cells** and trophoblast (extravillous) HLA Class I molecules interact. The cytokines thus derived, will regulate the invasion of extravillous trophoblast cells into the spiral arteries. The spiral arteries are thus converted to low resistance, high conductance uteroplacental arteries (Fig. 3.7).
- The decidual NK cells and the extravillous interstitial trophoblast cells interact at the trophoblast myometrial junction. Excessive myometrial invasion of trophoblast cells is thus prevented.
- The immunological response of implantation and that of organ transplantation are different and not comparable.
- Syncytiotrophoblast has got trophoblast-lymphocyte cross-reactive (TLX) antigen. Consequently there is production of **antibodies (blocking antibodies)** by the mother in response to this TLX (due to maternal-paternal immunoincompatibility). These blocking antibodies protect the fetus from rejection (see p. 196).

THE FETAL MEMBRANES

It consists of two layers: **outer chorion and the inner amnion**.

CHORION: It represents the remnant of chorion laeve and ends at the margin of the placenta. It is thicker than amnion, friable and shaggy on both the sides. Internally, it is attached to the amnion by loose areolar tissue and remnant of primitive mesenchyme. Externally, it is covered by vestiges of trophoblastic layer and the decidual cells of the fused decidua capsularis and parietalis which can be distinguished microscopically (Fig. 3.4B). Therefore **human placenta is a discoid, deciduate, labyrinthine and hemochorial type** (p. 35).

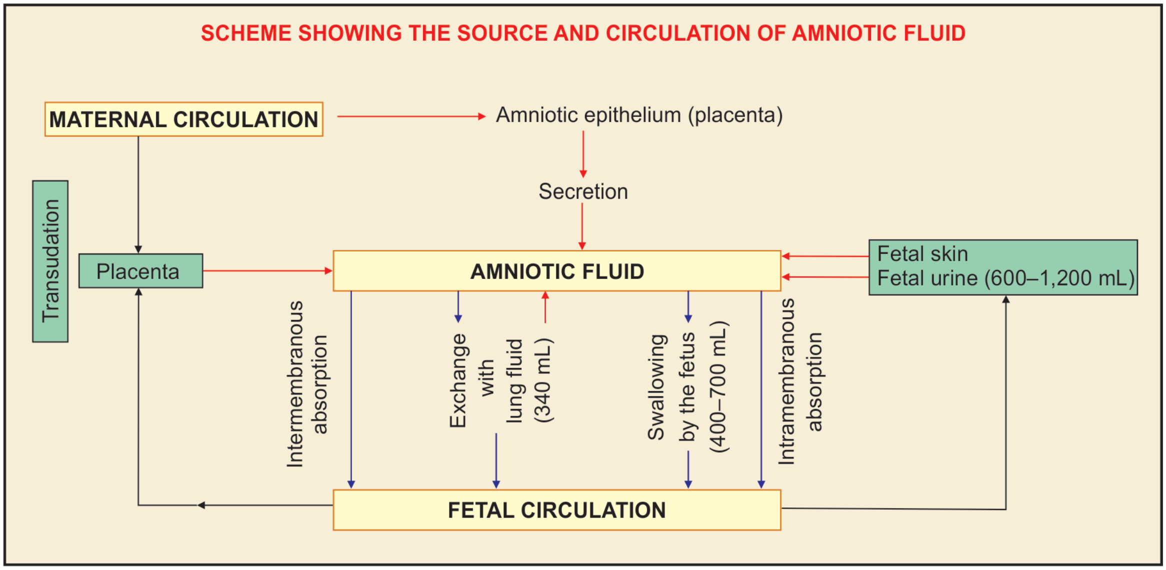
AMNION: It is the inner layer of the fetal membranes. Its internal surface is smooth and shiny and is in contact with liquor amnii. The lining epithelium is described later in the chapter. The outer surface consists of a layer of connective tissue and is apposed to the similar tissue on the inner aspect of the chorion from which it can be peeled off. **The amnion can also be peeled off from the fetal surface of the placenta except at the insertion of the umbilical cord.**

Functions: (1) Contribute to the formation of liquor amnii; (2) Intact membranes prevent ascending uterine infection; (3) Facilitate dilatation of the cervix during labor; (4) Has got enzymatic activities for steroid hormonal metabolism; (5) Rich source of glycerophospholipids containing arachidonic acid — precursor of prostaglandin E_2 and $F_{2\alpha}$.

AMNIOTIC CAVITY, AMNION AND AMNIOTIC FLUID

DEVELOPMENT: The formation of the amniotic cavity and its lining membrane, amnion has already been described with the development of the inner cell mass. Fluid accumulates slowly at first, but ultimately the fluid-filled cavity becomes large enough to obliterate the chorionic cavity; the amnion and the chorion come in loose contact by their mesenchymal layers.

Production	Removal
■ **Transudation** of maternal serum across the placental membranes ■ **Transudation** from fetal circulation across the umbilical cord or placental membranes ■ **Secretion** from amniotic epithelium ■ **Transudation** of fetal plasma through the highly permeable **fetal skin** before it is keratinized at 20th week ■ **Fetal urine** — Daily output at term is about 400–1,200 mL ■ **Fetal lung** that enters the amniotic cavity add to its volume.	♦ **Fetus swallows** about 500–1,000 mL of liquor every day. ♦ **Intramembranous absorption** of water and solutes (200–500 mL/day) from the amniotic compartment to fetal circulation through the fetal surface of the placenta.


SCHEME SHOWING THE SOURCE AND CIRCULATION OF AMNIOTIC FLUID
MATERNAL CIRCULATION
Amniotic epithelium (placenta)
Secretion
Transudation
Placenta
AMNIOTIC FLUID
Fetal skin
Fetal urine (600–1,200 mL)
Intermembranous absorption
Exchange with lung fluid (340 mL)
Swallowing by the fetus (400–700 mL)
Intramembranous absorption
FETAL CIRCULATION

Initially, the cavity is located on the dorsal surface of the embryonic disk. With the formation of the head, tail and lateral folds, it comes to surround the fetus in a manner as described in the Figs 2.9 and 2.10. Its two growing margins finally merge into the body stalk. **Thus, the liquor amnii surrounds the fetus everywhere except at its attachment with the body stalk.** The amnion is firmly attached to the umbilical cord up to its point of insertion to the placenta, but everywhere it can be separated from the underlying chorion.

STRUCTURE OF AMNION: **Fully formed amnion is 0.02–0.5 mm in thickness** and from within outwards the layers are — (1) single layer of cuboidal epithelium, (2) basement membranes, (3) compact layer of reticular structure, (4) fibroblastic layer and (5) spongy layer. **The amnion has got neither blood nor nerve supply nor any lymphatic system.**

AMNIOTIC FLUID

ORIGIN OF AMNIOTIC FLUID: The precise origin of the liquor amnii is still not well understood. It is probably of mixed maternal and fetal origin.

CIRCULATION: The water in the amniotic fluid is completely changed and **replaced in every 3 hours** as shown by the clearance of radioactive sodium injected directly into the amniotic cavity. The presence of lanugo and epithelial scales in the meconium shows that the fluid is swallowed by the fetus and some of it passes from the gut into the fetal plasma (vide scheme).

VOLUME: Amniotic fluid volume is related to gestational age. It measures about 50 mL at 12 weeks, 400 mL at 20 weeks and reaches its peak of 1 liter at 36–38 weeks. Thereafter the amount diminishes, till **at term it measures about 600–800 mL**. As the pregnancy continues post term, further reduction occurs to the extent of about 200 mL at 43 weeks.

PHYSICAL FEATURES: The fluid is faintly alkaline with low specific gravity of 1.010. It becomes highly hypotonic to maternal serum at term pregnancy. **An osmolarity of 250 mOsmol/L is suggestive of fetal maturity**. The amniotic fluid's osmolality falls with advancing gestation.

Color: In early pregnancy it is colorless, but near term it becomes pale straw colored due to the presence of exfoliated lanugo and epidermal cells from the fetal skin. It may look turbid due to the presence of vernix caseosa.

Abnormal color: Deviation of the normal color of the liquor has got clinical significance.

- **Meconium stained (green)** is suggestive of fetal distress in presentations other than the breech or transverse. Depending upon the degree and duration of the distress, it may be thin or thick or pea soup (thick with flakes). Thick with presence of flakes suggests chronic fetal distress.
- **Golden color** in Rh incompatibility is due to excessive hemolysis of the fetal RBC and production of excess bilirubin.
- **Greenish yellow (saffron)** in post maturity.
- **Dark colored** in concealed accidental hemorrhage is due to contamination of blood.
- **Dark brown (tobacco juice)** amniotic fluid is found in IUD. The dark color is due to frequent presence of old HbA.

COMPOSITION: In the first half of pregnancy, the composition of the fluid is almost identical to a transudate of plasma. But in late pregnancy, the composition is very much altered mainly due to contamination of fetal urinary metabolites. The composition includes—(1) water 98–99% and (2) solid (1–2%). **The following are the solid constituents**:

(a) ***Organic:***

Protein–0.3 mg%
Glucose–20 mg%
Urea–30 mg%
NPN–30 mg%
Uric acid–4 mg%
Creatinine–2 mg%
Total lipids–50 mg%
Hormones (prolactin, insulin and renin)

(b) ***Inorganic***—The concentration of the sodium, chloride and potassium is almost the same as that found in maternal blood. As pregnancy advances, there may be slight fall in the sodium and chloride concentration probably due to dilution by hypotonic fetal urine, whereas the potassium concentration remains unaltered.

(c) ***Suspended particles include***—Lanugo, exfoliated squamous epithelial cells from the fetal skin, vernix caseosa, cast off amniotic cells and cells from the respiratory tract, urinary bladder and vagina of the fetus.

FUNCTION: Its main function is to protect the fetus.

During pregnancy: (1) It acts as a shock absorber, protecting the fetus from possible extraneous injury; (2) Maintains an even temperature; (3) The fluid distends the amniotic sac and thereby allows for growth and free movement of the fetus and prevents adhesion between the fetal parts and amniotic sac; (4) Its nutritive value is negligible because of small amount of protein and salt content; however, water supply to the fetus is quite adequate.

During labor: (1) The amnion and chorion are combined to form a hydrostatic wedge which helps in dilatation of the cervix; (2) During uterine contraction, it prevents marked interference with the placental circulation so long as the membranes remain intact; (3) It guards against umbilical cord compression; (4) It flushes the birth canal at the end of first stage of labor and by its aseptic and bactericidal action protects the fetus and prevents ascending infection to the uterine cavity.

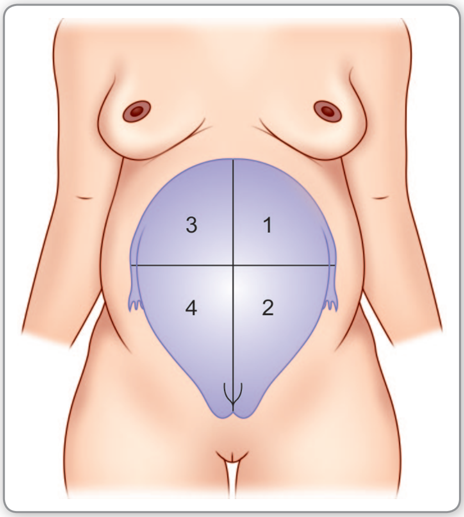


Fig. 3.12 : Amniotic fluid volume is measured to determine AFI

CLINICAL IMPORTANCE: (1) Study of the amniotic fluid provides useful information about the well being and also maturity of the fetus; (2) Intra-amniotic instillation of chemicals is used as method of induction of abortion; (3) Excess or less volume of liquor amnii is assessed by **amniotic fluid index (AFI)** (Fig. 3.12) p. 247, 535. Maternal abdomen is divided into quadrants taking the umbilicus, symphysis pubis and the fundus as the reference points. With ultrasound, the largest vertical pocket in each quadrant is measured. The sum of the four measurements (cm) is the AFI. **It is measured to diagnose the clinical condition of polyhydramnios or oligohydramnios respectively**; (4) Rupture of the membranes with drainage of liquor is a helpful method in induction of labor (p. 601).

THE UMBILICAL CORD

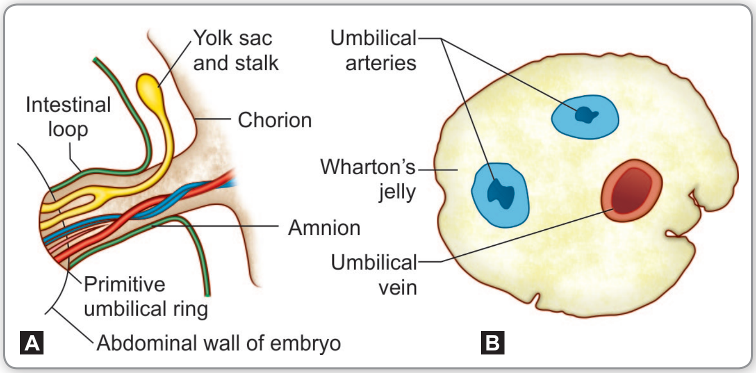
The umbilical cord or funis forms the connecting link between the fetus and the placenta through which the fetal blood flows to and from the placenta. It extends from the fetal umbilicus to the fetal surface of the placenta.

DEVELOPMENT: The umbilical cord is developed from the connective stalk or body stalk, which is a band of mesoblastic tissue stretching between the embryonic disk and the chorion. Initially, it is attached to the caudal end of the embryonic disk, but as a result of cephalocaudal folding of the embryo and simultaneous enlargement of the amniotic cavity the amnioectodermal junction converges on the ventral aspect of the fetus. As the amniotic cavity enlarges out of proportion to the embryo and becomes distended with fluid, the embryo is carried more and more into the amniotic cavity with simultaneous elongation of the connective stalk, the future umbilical cord.

STRUCTURES: The constituents of the umbilical cord when fully formed are as follows (Fig. 3.13).

1. ***Covering epithelium:*** It is lined by a single layer of amniotic epithelium but shows stratification like that of fetal epidermis at term.
2. ***Wharton's jelly:*** It consists of elongated cells in a gelatinous fluid formed by mucoid degeneration of the extraembryonic mesodermal cells. It is rich in mucopolysaccharides and has got protective function to the umbilical vessels.

3. ***Blood vessels:*** Initially, there are four vessels — two arteries and two veins. The arteries are derived from the internal iliac arteries of the fetus and carry the venous blood from the fetus to the placenta. Of the two umbilical veins, **the right one disappears by the 4th month, leaving behind one vein** which carries oxygenated blood from the placenta to the fetus. **Presence of a single umbilical artery is often associated with fetal congenital abnormalities (see p. 254).**



Figs 3.13A and B: Schematic drawing of the primitive umbilical cord of (A) Ten week embryo and (B) Cross section of a term umbilical cord

4. ***Remnant of the umbilical vesicle (yolk sac) and its vitelline duct:*** Remnant of the yolk sac may be found as a small yellow body near the attachment of the cord to the placenta or on rare occasion, the proximal part of the duct persists as Meckel's diverticulum.

5. ***Allantois:*** A blind tubular structure may be occasionally present near the fetal end which is continuous inside the fetus with its **urachus and bladder**.

6. ***Obliterated extraembryonic coelom:*** In the early period, intraembryonic coelom is continuous with extraembryonic coelom along with herniation of coils of intestine (midgut). The condition may persist as **congenital umbilical hernia or exomphalos**.

CHARACTERISTICS: It is about 40 cm in length with a usual variation of 30–100 cm. Its diameter is of average 1.5 cm with variation of 1–2.5 cm. Its thickness is not uniform but presents nodes or swelling at places. These swellings **(false knots)** may be due to kinking of the umbilical vessels or local collection of Wharton's jelly. True knots (1%) are rare. Long cord may form loop around the neck (20–30%). It shows a spiral twist from the left to right from as early as 12th week due to spiral turn taken by the vessels—vein around the arteries. **The umbilical arteries do not possess an internal elastic lamina but have got well-developed muscular coat.** These help in effective closure of the arteries due to reflex spasm soon after the birth of the baby. **Both the arteries and the vein do not possess vasa vasorum.**

ATTACHMENT: In the early period, the cord is attached to the ventral surface of the embryo close to the caudal extremity, but as the coelom closes and the yolk sac atrophies the point of attachment is moved permanently to the center of the abdomen at 4th month. Unlike the fetal attachment, the placental attachment is inconsistent. It usually attaches to the fetal surface of the placenta somewhere between the center and the edge of the placenta, called **eccentric insertion**. The attachment may be **central**, **marginal** or even on the chorion laeve at a varying distance away from the margin of the placenta, called **velamentous insertion**. The anomalies and various abnormalities of the umbilical cord are discussed in chapter 17 (p. 253).

QUESTIONS

1. Discuss in brief the source and circulation of amniotic fluid? (p. 43)
2. Mention the functions of amniotic fluid and its clinical significance? (p. 44)
3. Describe in brief the placenta at term? (p. 33)
4. Describe the circulation in the intervillous space? (p. 35)
5. Discuss in brief the functions of the placenta? (p. 39)

Write Short Notes on:

A. Fetal membranes (p. 41)
B. Placental barrier and its functions (p. 38)
C. Constituents of the umbilical cord at term pregnancy (p. 45)

Chapter 4

The Fetus

Three periods are distinguished in the prenatal development of the fetus. (1) **Ovular period or germinal period**—which lasts for first 2 weeks following ovulation. In spite of the fact that the ovum is fertilized, it is still designated as ovum. (2) **Embryonic period**—begins at 3rd week following ovulation and extends up to 10 weeks of gestation (8 weeks post conception). The crown-rump length (CRL) of the embryo is 4 mm. (3) **Fetal period** begins after 8th week following conception and ends in delivery. **The chronology in the fetal period is henceforth expressed in terms of menstrual age and not in embryonic age**.

LENGTH OF THE FETUS: To determine the length of the fetus, the measurement is commonly taken from the vertex to the coccyx (crown-rump length) in earlier weeks. While, from the end of 20th week onwards, the measurement is taken from the vertex to the heel (crown-heel length).

Calculation of the length: The crown heel (CH) measurement of the first 5 months is calculated by squaring the number of the lunar months to which the pregnancy belongs. In the second half, the same is calculated by multiplying the lunar months by 5. The length is expressed in centimeters.

Table 4.1: Principal Events of Embryonic and Fetal Development

Days 14–21 postconception	Notochord develops. Ectoderm thickens to form neural plate and neural folds.
Days 21–28 postconception	Neural folds fuse to form neural tube, four primitive cardiac chambers, first heart beats on D-21
Weeks 4–6 postconception *(4–15 mm embryo)*	Optic vesicles appear, complete neural tube closure (D-30) Limb buds appear. Formation of face
Weeks 6–8 postconception *(15–30 mm embryo)*	All major structures form, complete ventricular septum (D-46), recognizably human
Weeks 8–12 postconception *(30–60 mm embryo)*	External genitalia develop
Weeks—20	Skin is covered with lanugo. Vernix caseosa is present
Weeks—28	Testes descend to the internal inguinal ring. Baby is viable
Weeks—36	One testicle usually descends into the scrotum. Lanugo tends to disappear
Weeks—40	Both the testicles descend into the scrotum. Nails project beyond the finger tips. Posterior fontanel is closed

AGE OF THE FETUS: *Gestational age* is the duration of pregnancy calculated from the ***first day of last menstrual period (LMP)***. It is greater than the postconception (fertilization) age by 2 weeks. The length is more reliable criterion than the weight to calculate the age of the fetus. In the **first trimester**, CRL (mm) + 6.5 = gestational age in weeks. Assessment of gestational age by sonography has been discussed on page 734.

GROWTH OF THE FETUS: Normal fetal growth is characterized by cellular hyperplasia followed by hyperplasia and hypertrophy and lastly by hypertrophy alone. The fetal growth increases linearly until 37th week. It is controlled by genetic factor in the first half and by environmental factors in the second half of pregnancy. **The important physiological factors are:** Race (European babies are heavier than Indians); Sex (male baby weighs > female); Parental height and weight (tall and heavier mother have heavier babies); Birth order (weight rises from first to second pregnancy) and Socioeconomic factors (heavier babies in social class I and II). Fetal growth is predominantly controlled by IGF-1, insulin and other growth factors. Growth hormone is essential for postnatal growth. **At term, the average fetal weight in India varies from 2.5 kg to 3.5 kg**. Pathological factors affect it adversely (p. 534).

FETAL PHYSIOLOGY

NUTRITION: There are three stages of fetal nutrition following fertilization:

(1) ***Absorption:*** In the early postfertilization period, nutrition is stored in deutoplasm within cytoplasm and the very little extra nutrition needed is supplied from the tubal and uterine secretion.

(2) ***Histotrophic transfer:*** Following nidation and before the establishment of uteroplacental circulation, nutrition is derived from eroded decidua by diffusion and later on from the stagnant maternal blood in the trophoblastic lacunae.

(3) ***Hematotrophic:*** With the establishment of the fetal circulation, nutrition is obtained by active and passive transfer from the 3rd week onwards.

The fetus is a separated physiological entity and it takes what it needs from the mother even at the cost of reducing her resources. While all the nutrients are reaching the fetus throughout the intrauterine period, the demand is not squarely distributed. **Two-thirds of the total calcium, three-fifths of the total proteins and four-fifths of the total iron are drained from the mother during the last 3 months**. Thus, in preterm births, the store of the essential nutrients to the fetus is much low. The excess iron reserve is to compensate for the low supply of iron in breast milk which is the source of nutrients following birth.

FETAL BLOOD: Hematopoiesis is demonstrated in the embryonic phase **first in the yolk sac** by 14th day. By 10th week, the liver becomes the major site. The great enlargement of the early fetal liver is due to its erythropoietic function. Gradually, the red cell production sites extend to the spleen and bone marrow and near term, the bone marrow becomes the major site of red cell production.

In the early period, the erythropoiesis is megaloblastic but near term it becomes normoblastic. The fetal blood picture at term shows RBC 5–6 million/cu mm; Hb = 16.5–18.5 gm%, reticulocytes –5% and erythroblast –10%. During the first half, the hemoglobin is of fetal type (α-2, γ-2) but from 24 weeks onwards, adult type of hemoglobin (α-2, β-2) **appears and at term about 75–80% of the total hemoglobin is of fetal type** (HbF). Between 5 and 8 weeks, the embryo manufactures some additional hemoglobin: Hb Gower 1 (ξ- and ε-chains), Hb Gower 2 (α- and ε-chains) and Hb Portland (ξ- and γ-chains). Between 6–12 months after birth, the fetal hemoglobin is completely replaced by adult hemoglobin. **The fetal hemoglobin has got a greater affinity to oxygen due to lower binding of 2, 3-diphosphoglycerate compared to adult hemoglobin.** It is also resistant to alkali in the formation of alkaline hematin. **Total fetoplacental blood volume at term** is estimated to be 125 mL/kg body weight of the fetus. The red cells develop their group antigen quite early and the **presence of Rh factor has been demonstrated** in the fetal blood from as early as 38 days after conception. **The life span of the fetal RBC** is about two-thirds of the adult RBC, i.e. about 80 days. The activities of all glycolytic enzymes in fetal erythrocytes except phosphofructokinase and 6-phosphogluconate dehydrogenase are higher than those of adults or term or premature infants.

Cord blood level of iron, ferritin, vitamin B_{12} and folic acid is consistently higher than maternal blood.

LEUKOCYTES AND FETAL DEFENCE: Leukocytes appear after 2 months of gestation. The white cell count rises to about 15–20 thousand/cu mm at term. Thymus and spleen soon develop and produce lymphocytes, a major source of antibody formation. The fetus, however, rarely forms antibody because of relatively sterile environment. **Maternal immunoglobulin G (IgG) crosses the placenta from 12th week onwards** to give the fetus a passive immunity which increases with the increase in gestation period. At term fetal IgG level is 10% higher than the mother.

IgM is predominantly of fetal origin and its detection by cordocentesis may be helpful in diagnosis of intrauterine infection. IgA is produced only after birth in response to antigens of enteric infection.

URINARY SYSTEM: By the end of the first trimester, the nephrons become active and secrete urine. **Near term, the urine production rises to 650 mL/day**. However, kidneys are not essential for survival of the fetus in utero but are important in regulation of the composition and volume of the liquor amnii. Oligohydramnios may be associated with renal hypoplasia or obstructive uropathy.

SKIN: At 16th week, lanugo (downy thin colorless hairs) appears but near term almost completely disappears. **Sebaceous glands** appear at 20th week and the sweat glands somewhat later. **Vernix caseosa**—the secretion of the sebaceous glands mixed with the exfoliated epidermal cells is abundantly present smearing the skin. **The horny layer of the epidermis is absent before 20th week** which favors transudation from the fetal capillaries into the liquor amnii.

GASTROINTESTINAL TRACT: As early as 10–12 week, the fetus swallows amniotic fluid. **The meconium appears from 20th week** and at term, it is distributed uniformly throughout the gut up to the rectum indicating the presence of intestinal peristalsis. In intrauterine hypoxia (vagal stimulation), the anal sphincter is relaxed and the meconium may be voided into the liquor amnii.

Composition of the meconium: It is chiefly composed of the waste products of the hepatic secretion. It contains lanugo, hairs and epithelial cells from the fetal skin which are swallowed with the liquor amnii. Mucus, exfoliated intestinal epithelium and intestinal juices are added to the content. **The greenish black color is due to the bile pigments, specially biliverdin**.

RESPIRATORY SYSTEM: In the early months, the lungs are solid. At 28th week, alveoli expand and are lined by cuboidal epithelium. There is intimate contact with the endothelium of the capillaries. At 24th week, **lung surfactant related to phospholipids—phosphatidylcholine (lecithin) and phosphatidylglycerol appear. Surfactant is secreted by type-II alveolar cells. These substances lower the surface tension of the lung fluid so** that the alveoli can be opened up easily when breathing starts following delivery. **A lecithin:sphingomyelin (L:S) ratio of 2:1 in the liquor amnii signifies full maturity of the fetal lung. Fetal cortisol is the natural trigger for augmented surfactant synthesis**. Fetal growth restriction and prolonged rupture of membranes also accentuates surfactant synthesis.

Breathing movements are identified by 11 weeks but are irregular until 20th week. Their frequency varies from 30–70 per minute and is dependent on the maternal blood sugar concentration. Hypoxia and maternal cigarette smoking reduces FBM while hyperglycemia increases it. The tracheobronchial tree is filled up with liquor amnii.

FETAL ENDOCRINOLOGY: Growth hormone, ACTH, prolactin, TSH and gonadotrophic hormones are produced by the fetal pituitary **as early as the 10th week**. Vasopressor and oxytocic activity of the posterior pituitary have also been demonstrated as early as **12 weeks**. **Fetal adrenals show hypertrophy of the reticular zone (fetal zone)** which is the site of synthesis of estriol precursor, cortisol and dehydroepiandrosterone. This fetal zone is absent in anencephaly. The adrenal medulla produces small amount of catecholamines. Fetal thyroid starts synthesizing small amount of thyroxine by 11th week. **While the fetal ovaries remain inactive, the fetal testicles mediate the development of the male reproductive structures.** Fetal pancreas secretes **insulin as early as 12th** week and **glucagon by 8 weeks**.

THE FETAL CIRCULATION

The umbilical vein carrying the oxygenated blood (80% saturated) from the placenta, enters the fetus at the umbilicus and runs along the free margin of the falciform ligament of the liver. In the liver, it gives off branches to the left lobe of the liver and receives the deoxygenated blood from the portal vein. The greater portion of the oxygenated blood, mixed with some portal venous blood, short circuits the liver through the **ductus venosus** to enter the inferior vena cava (IVC) and thence to right atrium of the heart. The O_2 content of this mixed blood is thus reduced. Although both the ductus venosus and hepatic portal/fetal trunk bloods enter the right atrium through the IVC, there is little mixing. The terminal part of the IVC receives blood from the right hepatic vein.

In the right atrium, most of the well oxygenated (75%) ductus venosus blood is preferentially directed into the foramen ovale by the valve of the inferior vena cava and crista dividens and passes into the left atrium. Here it is mixed with small amount of venous blood returning from the lungs through the pulmonary veins. This left atrial blood is passed on through the mitral opening into the left ventricle.

Remaining lesser amount of blood (25%), after reaching the right atrium via the superior and inferior vena cava (carrying the venous blood from the cephalic and caudal parts of the fetus respectively) passes through the tricuspid opening into the right ventricle (Fig. 4.1).

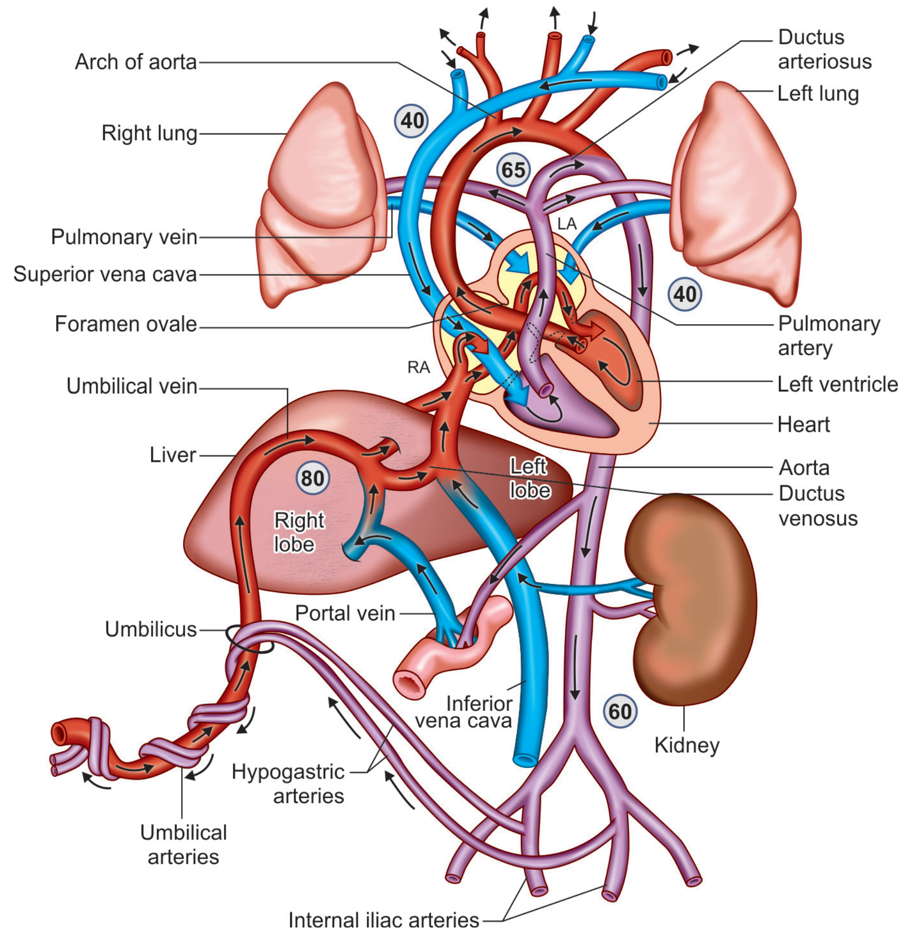
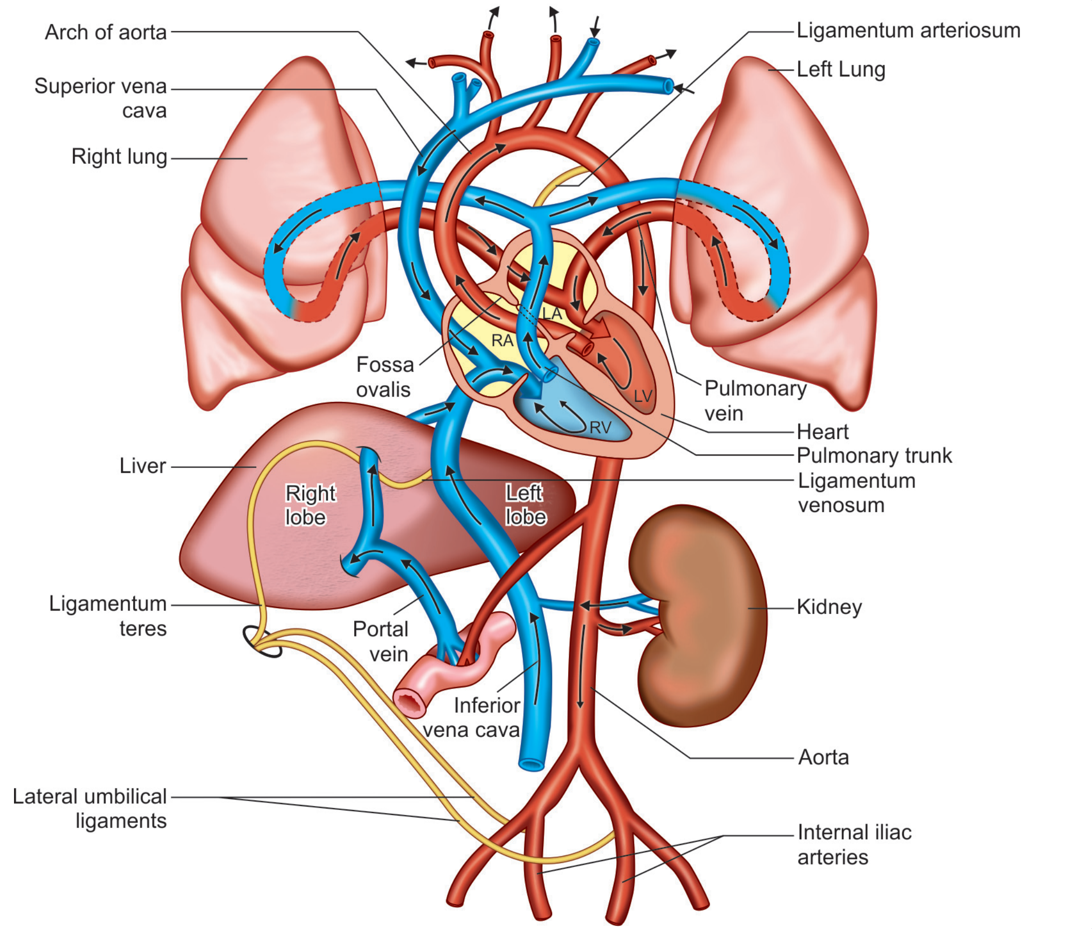

Arch of aorta
Ductus arteriosus
Left lung
Right lung
40
65
LA
Pulmonary vein
Superior vena cava
40
Foramen ovale
Pulmonary artery
RA
Umbilical vein
Left ventricle
Heart
Liver
80
Left lobe
Aorta
Ductus venosus
Right lobe
Portal vein
Umbilicus
Inferior vena cava
60
Kidney
Hypogastric arteries
Umbilical arteries
Internal iliac arteries

Fig. 4.1: Fetal circulation. (Number inside the circle indicates the percentage saturation of O_2)

During ventricular systole, the left ventricular blood is pumped into the ascending and arch of aorta and distributed by their branches to the heart, head, neck, brain and arms. The right ventricular blood with low oxygen content is discharged into the pulmonary trunk. Since the resistance in the pulmonary arteries during fetal life is very high, the main portion of the blood passes directly through the **ductus arteriosus** into the descending aorta bypassing the lungs where it mixes with the blood from the proximal aorta. 70% of the cardiac output (60% from right and 10% from left ventricle) is carried by the ductus arteriosus to the descending aorta. About 40% of the combined output goes to the placenta through the umbilical arteries. **The deoxygenated blood leaves the body by way of two umbilical arteries to reach the placenta** where it is oxygenated and gets ready for recirculation. **The mean cardiac output is comparatively high in fetus and is estimated to be 350 mL/kg/min**.

CHANGES OF THE FETAL CIRCULATION AT BIRTH

The hemodynamics of the fetal circulation undergoes profound changes soon after birth (Fig. 4.2) due to—(1) cessation of the placental blood flow and (2) initiation of respiration. **The following changes occur in the vascular system (Fig. 4.2)**:



Arch of aorta
Superior vena cava
Right lung
Fossa ovalis
Liver
Right lobe
Left lobe
Ligamentum teres
Portal vein
Inferior vena cava
Lateral umbilical ligaments
Ligamentum arteriosum
Left Lung
LA
RA
LV
RV
Pulmonary vein
Heart
Pulmonary trunk
Ligamentum venosum
Kidney
Aorta
Internal iliac arteries

Fig. 4.2: Change in the fetal circulation after birth

1. ***Closure of the umbilical arteries*:** Functional closure is almost instantaneous preventing even slight amount of the fetal blood to drain out. Actual obliteration takes about 2–3 months. **The distal parts form the lateral umbilical ligaments and the proximal parts remain open as superior vesical arteries.**

2. ***Closure of the umbilical vein*:** **The obliteration occurs a little later than the arteries,** allowing few extra volume of blood (80–100 mL) to be received by the fetus from the placenta. The ductus venosus collapses and the venous pressure of the inferior vena cava falls and so also the right atrial pressure. **After obliteration, the umbilical vein forms the ligamentum teres and the ductus venosus becomes ligamentum venosum (Fig. 4.2).**

3. ***Closure of the ductus arteriosus*:** Within few hours of respiration, the muscle wall of the ductus arteriosus contracts probably in response to rising oxygen tension of the blood flowing through the duct. The effects of variation of the O_2 tension on ductus arteriosus are thought to be mediated through the action of prostaglandins. Prostaglandin antagonists given to the mother may lead to the premature closure of the ductus arteriosus. **Whereas functional closure of the ductus may occur soon after the establishment of pulmonary circulation, the anatomical obliteration takes about 1–3 months and becomes ligamentum arteriosum.**

4. ***Closure of the foramen ovale*:** This is caused by an increased pressure of the left atrium combined with a decreased pressure on the right atrium. **Functional closure occurs soon after birth but anatomical closure occurs in about 1 year time.** During the first few days, the closure may be reversible. This is evidenced clinically by the cyanotic look of the baby during crying when there is shunting of the blood from right to left.

Within 1 or 2 hours following birth, the cardiac output is estimated to be about 500 mL/min and the heart rate varies from 120–140 per minute.

QUESTIONS

1. Mention the important physiological factors for fetal growth? (p. 47)
2. Outline in brief the fetal hematopoiesis? (p. 47)

Write Short Notes on:

A. Changes in fetal respiratory systems and its clinical importance (p. 48)
B. Fetal circulation and the changes at birth (p. 49-50)

Chapter 5

Physiological Changes During Pregnancy

During pregnancy there is progressive anatomical, physiological and biochemical change not only confined to the genital organs but also to all systems of the body. This is principally a phenomenon of maternal adaptation to the increasing demands of the growing fetus. Unless well understood, these physiological adaptations of normal pregnancy can be misinterpreted as pathological.

GENITAL ORGANS

VULVA: Vulva becomes edematous and more vascular; superficial varicosities may appear especially in multiparae. Labia minora are pigmented and hypertrophied.

VAGINA: Vaginal walls become hypertrophied, edematous and more vascular. Increased blood supply of the venous plexus surrounding the walls gives the bluish coloration of the mucosa **(Jacquemier's sign)**. The length of the anterior vaginal wall is increased.

Secretion: The secretion becomes copious, thin and curdy white due to marked exfoliated cells and bacteria. The pH becomes acidic (3.5–6) due to more conversion of glycogen into lactic acid by the *Lactobacillus acidophilus* consequent on high estrogen level. The acidic pH prevents multiplication of pathogenic organisms.

Cytology: There is preponderance of **navicular cells in cluster** (small intermediate cells with elongated nuclei) and plenty of lactobacillus.

UTERUS

There is enormous growth of the uterus during pregnancy. The uterus which in nonpregnant state weighs about 60 g, with a cavity of 5–10 mL and measures about 7.5 cm in length, **at term**, **weighs 900–1,000 g and measures 35 cm in length**. The capacity is increased by 500–1,000 times. Changes occur in all the parts of the uterus—body, isthmus and cervix.

BODY OF THE UTERUS: There is increase in growth and enlargement of the body of the uterus.

Enlargement: The enlargement of the uterus is affected by the following factors:

- **Changes in the muscles**—(1) **Hypertrophy and hyperplasia**: Not only the individual muscle fiber increases in length and breadth but also there is limited addition of new muscle fibers. These occur under the influence of the hormones—estrogen and progesterone—limited to the first half of pregnancy but pronounced up to 12 weeks. (2) **Stretching**: The muscle fibers further elongate beyond 20 weeks due to distension by the growing fetus. The wall becomes thinner and, at term, measures about 1.5 cm or less. The uterus feels soft and elastic in contrast to firm feel of the nongravid uterus.

Arrangement of the muscle fibers: Three distinct layers of muscle fibers are evident:

(1) **Outer longitudinal**—It follows a hood-like arrangement over the fundus; some fibers are continuous with the round ligaments. (2) **Inner circular**—It is scanty and sphincter like arrangement around the tubal orifices and internal os. (3) **Intermediate**—It is the thickest and strongest layer arranged in crisscross fashion through which the blood vessels run. Apposition of two double curve muscle fibers give the figure of '8' form. Thus, when the muscles contract, they occlude the blood vessels running through the fibers and hence called the **living ligature (Figs 5.1 and 5.2)**.

- **There is simultaneous increase in number and size** of the supporting fibrous and elastic tissues.

Vascular system—Whereas in the nonpregnant state, the blood supply to the uterus is mainly through the uterine and least through the ovarian but, in the pregnant state, the latter carries as much the blood as the former. There is marked spiraling of the arteries, reaching the maximum at 20 weeks; thereafter, they straighten out. Doppler velocimetry has shown uterine artery diameter becomes double, and blood flow increases by eightfold at 20 weeks of pregnancy. This vasodilatation is mainly due to estradiol and progesterone. Veins become dilated and are valveless. Numerous lymphatic channels open up. The vascular changes are most pronounced at the placental site.

The uterine enlargement is not a symmetrical one. **The fundus enlarges more than the body**. It is evident by the low down attachment of the round ligaments or insertion of the uterine end of the Fallopian tubes at term.

Weight: The increase in weight is due to the increased growth of the uterine muscles, connective tissues and vascular channels.

Relation: **Shape**—Nonpregnant pyriform shape is maintained in early months. It becomes globular at 12 weeks. As the uterus enlarges, the shape once more becomes pyriform or ovoid by 28 weeks and changes to spherical beyond 36th week (see Fig. 7.5).

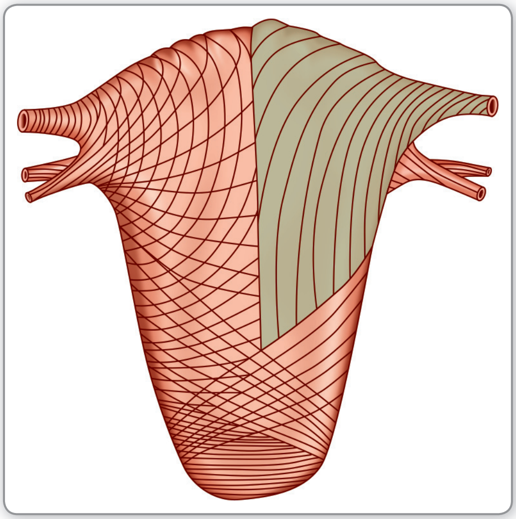
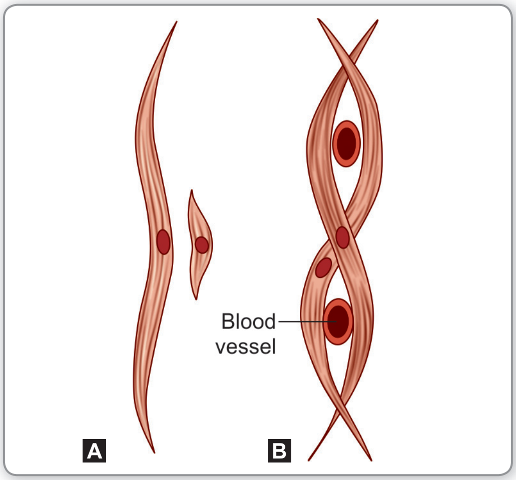


Fig. 5.1: Schematic presentation of the longitudinal, oblique and circular muscle fibers of the pregnant uterus



Figs 5.2A and B: (A) Marked elongation of the muscle fibers during pregnancy, (B) Blood vessels in between interlacing muscle fibers

Position: Normal anteverted position is exaggerated up to 8 weeks. Thus, the enlarged uterus may lie on the bladder rendering it incapable of filling, clinically evident by frequency of micturition. Afterwards, it becomes erect, the long axis of the uterus conforms more or less to the axis of the inlet. As the term approaches, especially in multiparae with lax abdominal wall, there is a tendency of anteversion. But in primigravidae with good tone of the abdominal muscles, it is held firmly against the maternal spine.

Lateral obliquity: As the uterus enlarges to occupy the abdominal cavity, it usually rotates on its long axis to the right **(dextrorotation)**. This is due to the occupation of the rectosigmoid in the left posterior quadrant of the pelvis. This makes the anterior surface of the uterus to turn to the right and brings the left cornu closer to the abdominal wall. **The cervix, as a result, is deviated to the left side (levorotation) bringing it closer to the ureter**.

Uterine peritoneum: The peritoneum maintains the relation proportionately with the growing uterus. The uterosacral ligaments and the bases of the broad ligament rise up to the level of the pelvic brim. This results in deepening of the pouch of Douglas. Large areas of the lower lateral walls of the uterus remain uncovered by peritoneum. These places are filled up by loose and vascular connective tissues.

***Contractions (Braxton-Hicks)*:** Uterine contraction in pregnancy has been named after *Braxton-Hicks* who first described its entity during pregnancy. From the very early weeks of pregnancy, the uterus undergoes spontaneous contraction. This can be felt during bimanual palpation in early weeks or during abdominal palpation when the uterus feels firmer at one moment and softer at another. Although spontaneous, the contractions may be excited by rubbing the uterus. **The contractions are irregular, infrequent, spasmodic and painless without any effect on dilatation of the cervix**. The patient is not conscious about the contractions. Intrauterine pressure remains below 8 mm Hg. Near term, the contractions become frequent with increase in intensity so as to produce some discomfort to the patient. Ultimately, it merges with the painful uterine contractions of labor. **In abdominal pregnancy, Braxton-Hicks contraction is not felt**.

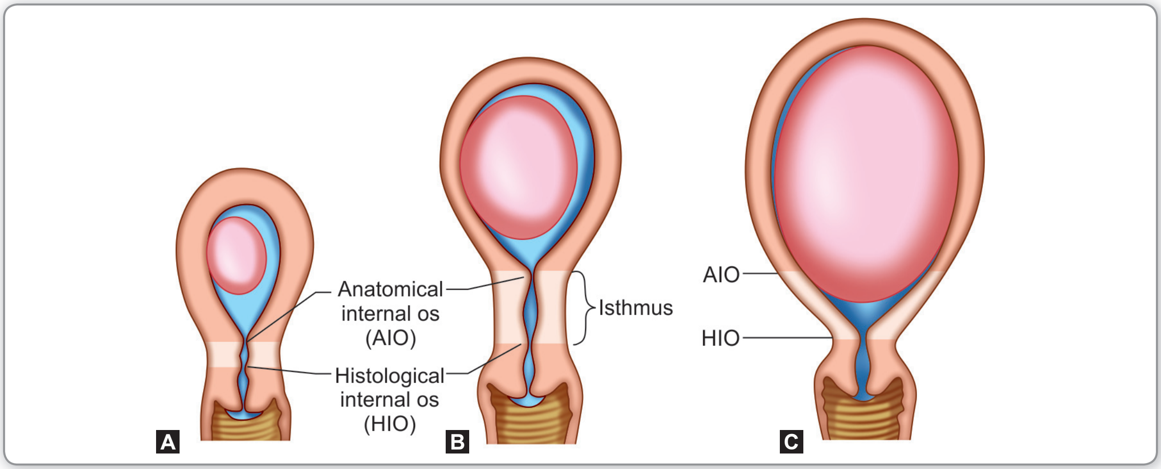
During contraction there is complete closure of the uterine veins with partial occlusion of the arteries in relation to intervillous space resulting in stagnation of blood in the space. This diminishes the placental perfusion, causing transient fetal hypoxia, which leads to fetal bradycardia coinciding with the contraction.

***Endometrium*:** The changes of the endometrium of the nonpregnant uterus into decidua of pregnancy have already been described (p. 27).

ISTHMUS

There are important structural and functional changes in the isthmus during pregnancy.

During the first trimester, isthmus hypertrophies and elongates to about 3 times its original length. It becomes softer. With advancing pregnancy beyond 12 weeks, it progressively unfolds from above, downward until it is incorporated into the uterine cavity. The circularly arranged muscle fibers in the region



Figs 5.3A to C: Elongation and formation of the lower uterine segment: (A) at 8 weeks; (B) at 12 weeks; (C) at 16 weeks

function as a sphincter in early pregnancy and thus help to retain the fetus within the uterus. Incompetency of the sphincteric action leads to mid-trimester abortion and the encirclage operation done to rectify the defect is based on the principle of restoration of the retentive function of the isthmus (Fig. 5.3).

CERVIX

Stroma**: There are hypertrophy and hyperplasia** of the elastic and connective tissues. **Fluids accumulate** inside and in between the fibers. **Vascularity** is increased especially beneath the squamous epithelium of the portio vaginalis which is responsible for its bluish coloration. There are marked hypertrophy and hyperplasia of the glands which occupy about half the bulk of the cervix. **All these lead to marked softening of the cervix (Goodell's sign)** which is evident as early as 6 weeks. It begins at the margin of the external os and then spreads upward. It not only provides diagnostic aid in pregnancy but also the changes in the cervix facilitate its dilatation during labor.

Epithelium**:** There is marked proliferation of the endocervical mucosa with downward extension beyond the squamocolumnar junction (Fig. 5.4).

This gives rise to clinical appearance of ectopy (erosion) cervix. **Sometimes, the squamous cells also become hyperactive and the mucosal changes simulate basal cell hyperplasia or cervical intraepithelial neoplasia (CIN)**. These changes are hormone induced (estrogen) and regress spontaneously after delivery.

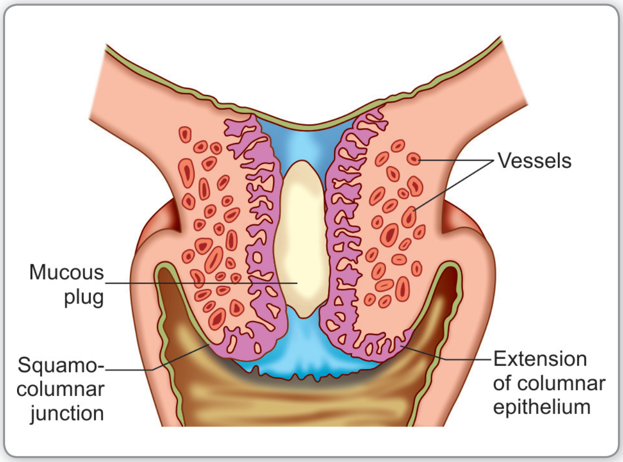


Fig. 5.4: Changes of the cervix during advanced pregnancy

Secretion**:** The secretion is copious and tenacious-physiological leukorrhea of pregnancy. This is due to the effect of progesterone. This mucous is rich in immunoglobulins and cytokines. The mucus not only fills up the glands but also forms a thick plug effectively sealing the cervical canal. Microscopic examination shows fragmentation or crystallization (beading) due to progesterone effect.

Anatomical**:** The length of the cervix remains unaltered but becomes bulky. The cervix is directed posteriorly but after the engagement of the head, directed in line of vagina. There is no alteration in the relation of the cervix. There is unfolding of the isthmus; beginning 12 weeks onwards and takes part in the formation of the lower uterine segment. Variable amount of effacement is noticed near term in primigravidae. In multiparae, the canal is slightly dilated.

FALLOPIAN TUBE

As the uterine end rises up and the fimbrial end is held up by the infundibulopelvic ligament, it is placed almost vertical by the side of the uterus. At term, its attachment to the uterus is placed at the lower end of the upper one-third, because of marked growth of the fundus. The total length is somewhat increased. The tube becomes congested. Muscles undergo hypertrophy. Epithelium becomes flattened, and patches of decidual reaction are observed.

OVARY

The growth and function of the corpus luteum reaches its maximum at 8th week when it measures about 2.5 cm and becomes cystic. It looks bright orange, later on becomes yellow and, finally pale. Regression occurs following decline in the secretion of human chorionic gonadotropin (hCG) from the placenta. Colloid degeneration occurs at 12th week which later becomes calcified at term. Hormones—

estrogen and progesterone—secreted by the corpus luteum maintain the environment for the growing ovum before the action is taken over by the placenta. These hormones not only control the formation and maintenance of decidua of pregnancy but also inhibit ripening of the follicles. **Thus both the ovarian and uterine cycles of the normal menstruation remain suspended**. **Luteoma of pregnancy** results from exaggerated luteinization reaction of the ovary.

***Decidual reaction*:** There may be patchy sheet of decidual cells on the outer surface of the ovary. **These are metaplastic changes due to high hormonal stimulation**. The same stimulus may also produce luteinization of atretic or partially developed follicles.

BREASTS

The changes in the breasts are best evident in a primigravida. In multipara, who has once lactated, the changes are not clearly defined.

SIZE: Increased size of the breasts becomes evident even in early weeks. This is due to marked hypertrophy and proliferation of the ducts (estrogen) and the alveoli (estrogen and progesterone) which are marked in the peripheral lobules. There is also hypertrophy of the connective tissue stroma. Myoepithelial cells become prominent. Vascularity is increased which results in appearance of bluish veins running under the skin. Quite often, the "**axillary tail**" (prolongation of the breast tissue under cover of the pectoralis major) becomes enlarged and painful. There may be evidence of striation due to stretching of the cutis.

NIPPLES AND AREOLA: The nipples become larger, erectile and deeply pigmented. Variable number of sebaceous glands (5–15) which remain invisible in the nonpregnant state in the areola, become hypertrophied and are called **Montgomery's tubercles**. Those are placed surrounding the nipples. Their secretion keeps the nipple and the areola moist and healthy. An outer zone of less marked and irregular pigmented area appears in second trimester and is called **secondary areola**.

SECRETION: Secretion (colostrum) can be squeezed out of the breast at about 12th week which at first becomes sticky. Later on, by 16th week, it becomes thick and yellowish. **The demonstration of secretion from the breast of a woman who has never lactated is an important sign of pregnancy**. In latter months, colostrum may be expressed from the nipples. For normal changes and lactation see page 72.

CUTANEOUS CHANGES

PIGMENTATION: The distribution of pigmentary changes is selective.

1. ***Face*** (chloasma gravidarum or pregnancy mask): It is an extreme form of pigmentation around the cheek, forehead and around the eyes. It may be patchy or diffuse; disappears spontaneously after delivery.
2. ***Breast*:** The changes are already described (vide supra).
3. ***Abdomen*:**
   - **Linea nigra:** It is a brownish black pigmented area in the midline stretching from the xiphisternum to the symphysis pubis (Fig. 7.7B). The pigmentary changes are probably due to **melanocyte stimulating hormone** from the anterior pituitary. However, estrogen and progesterone may be related to it as similar changes are observed in women taking oral contraceptives. The pigmentation disappears after delivery.
   - **Striae gravidarum:** These are slightly depressed linear marks with varying length and breadth found in pregnancy. They are predominantly found in the abdominal wall below the umbilicus, sometimes over the thighs and breasts. **These stretch marks represent the scar tissues in**

the deeper layer of the cutis. Initially, these are pinkish but after the delivery, the scar tissues contract and obliterate the capillaries and they become glistening white in appearance and are called **striae albicans**. Apart from the mechanical stretching of the skin, increase in aldosterone production during pregnancy are the responsible factors. Controlled weight gain during pregnancy and massaging the abdominal wall by lubricants like olive oil may be helpful in reducing their formation. **Apart from pregnancy, it may form in cases of generalized edema, marked obesity or in Cushing syndrome (see Fig. 8.5)**.

OTHER CUTANEOUS CHANGES: These include **vascular spider** and **palmar erythema** which are due to high estrogen level. Mild degrees of hirsutism may be observed and in puerperium the excess hair is lost.

WEIGHT GAIN

WEIGHT GAIN: In normal pregnancy, variable amount of weight gain is a constant phenomenon. In early weeks, the patient may lose weight because of nausea or vomiting. During subsequent months, the weight gain is progressive until the last 1 or 2 weeks, when the weight remains static. **The total weight gain during the course of a singleton pregnancy for a healthy woman averages 11 kg (24 lb)**. This has been distributed to 1 kg in first trimester and 5 kg each in second and third trimester. The total weight gain at term is distributed approximately as follows:

I. Reproductive Weight Gain: 6 kg	II. Net Maternal Weight Gain: 6 kg
■ Fetus—3.3 kg, placenta—0.6 kg and liquor—0.8 kg	♦ Increase in blood volume—1.3 kg
■ Uterus—0.9 kg and breasts—0.4 kg	♦ Increase in extracellular fluid—1.2 kg
■ Accumulation of fat (mainly) and protein—3.5 kg	

During pregnancy, there is variable amount of retention of electrolytes—sodium (1,000 mEq), potassium (10 g) and chlorides. The sodium is osmotically active and partially controls the distribution of water in various compartments of the body. **Causes of increased sodium retention during pregnancy are:** (1) increased estrogen and progesterone, (2) increased aldosterone consequent on the activation of the renin-angiotensin system and possibly (3) due to increased antidiuretic hormone. **The amount of water retained during pregnancy at term is estimated to be 6.5 liters.**

The increased accumulation of fluid in the tissue spaces mainly below the uterus is due to—(1) diminished colloid osmotic tension due to hemodilution driving the fluid out of the vessels and (2) increased venous pressure of the inferior extremities. Thus, slight edema of the legs is not uncommon, in otherwise normal pregnancy.

Importance of weight checking: Single weight checking is of little value except to identify the overweight or underweight patient. Periodic and regular weight checking is of importance to detect abnormality.

- ♦ **Rapid gain in weight** of more than 0.5 kg (1 lb) a week or more than 2 kg (4 lb) a month in later months of pregnancy may be the early manifestation of preeclampsia and need for careful supervision.
- ♦ **Stationary or falling weight** may suggest intrauterine growth retardation or intrauterine death of fetus.

Obese women are in increased risk of complications in pregnancy, labor and puerperium (see page 400).

Ideally weight gain should depend on pre-pregnancy body mass index (BMI) level (see p. 400). Weight gain for a woman with normal BMI (20–26) is 11–16 kg. An obese woman (BMI > 30) should not gain more than 7 kg, whereas an underweight woman (BMI < 19) may be allowed to gain up to 18 kg.

Maternal nutrition and weight gain during pregnancy are directly related to the newborn weight. However, it may not be a specific indicator as there are other factors for low birth weight infant.

BODY WATER METABOLISM

During pregnancy, the amount of **water retained at term is about 6.5 liters**. The water content of the fetus, placenta and amniotic fluid is about 3.5 liters. **Pregnancy is a state of hypervolemia**. There is active retention of sodium (900 mEq), potassium (300 mEq) and water. **The important causes of sodium retention and volume overload are:** (i) changes in maternal osmoregulation, (ii) increased estrogen and progesterone, (iii) increase in renin-angiotensin-aldosterone system (RAAS) activity, (iv) increased aldosterone, deoxycorticosterone, (v) control by arginine vasopressin (AVP) from posterior pituitary and (vi) atrial natriuretic peptide. Serum sodium level and plasma osmolality decreases. **There is resetting of the osmotic thresholds for thirst and AVP (ADH) secretion.** Plasma levels of AVP (ADH) remain unchanged, in spite of increased production. This is due to increased metabolic clearance of AVP by the enzyme vasopressinase from the placenta. **Increase in water intake due to lowered osmotic threshold for thirst causes polyuria in early pregnancy**. The threshold for AVP secretion has been reset after 8 weeks for a new steady state of osmolality. Thereafter polyuria decreases.

Atrial and brain natriuretic peptides, secreted by atrial myocytes and brain ventricles. These act as diuretics, natriuretics and vasorelaxants. Both the peptides are antagonist to RAAS.

HEMATOLOGICAL CHANGES

BLOOD VOLUME: During pregnancy, there is increased vascularity of the enlarging uterus with the interposition of uteroplacental circulation. The activities of all the systems are increased. **Blood volume is markedly raised during pregnancy**. The rise is progressive and inconsistent. All the constituents of blood are affected with increased blood volume. The blood volume starts to increase from about 6th week, expands rapidly thereafter to **maximum 40–50% above the nonpregnant level at 30–34 weeks**. The level remains almost static till delivery (Table 5.1).

PLASMA VOLUME: It starts to increase by 6 weeks and it plateaus at 30 weeks of gestation. The rate of increase almost parallels to that of blood volume but **the maximum is reached to the extent of 50%**. **Total plasma volume increases to the extent of 1.25 liters**. The increase is greater in multigravida, in multiple pregnancy and with large baby.

RBC AND HEMOGLOBIN: The RBC mass is increased to the extent of 20–30%. The total increase in volume is about 350 mL. This increase is regulated by the increased demand of oxygen transport during pregnancy. RBC mass begins to increase at about 10 weeks and continues till term without plateauing. Iron supplementation increases the RBC mass by 30%. Reticulocyte count increases by 2%. Erythropoietin level is raised.

Table 5.1: Principal Blood Changes during Pregnancy

Parameters	Nonpregnant	Pregnancy Near Term	Total Increment	Change
Blood volume (mL)	4000	5500	1500	+ 30–40%
Plasma volume (mL)	2500	3750	1250	+ 40–50%
Red Cell volume (mL)	1400	1750	350	+ 20–30%
Total Hb (g)	475	560	85	+ 18–20%
Hematocrit (whole body)	38%	32%		Diminished

The disproportionate increase in plasma and RBC volume produces a state of hemodilution (fall in hematocrit) during pregnancy. Thus, even though the total hemoglobin mass increases during pregnancy to the extent of 18–20%, there is apparent fall in hemoglobin concentration. At term, the fall is about 2 g% from the nonpregnant value. There is simultaneous fall in number of red cells by 15–20% and the hematocrit level. The excess of circulating hemoglobin left behind after delivery yields iron for storage.

The advantages of relative hemodilution are: (1) Diminished blood viscosity ensures optimum gaseous exchange between the maternal and fetal circulation. This is facilitated by lowered oxygen affinity of maternal red cells observed in later half of pregnancy. (2) It protects the woman against the adverse effects of supine and erect posture. (3) Protection of the mother against the adverse effects of blood loss during delivery.

LEUKOCYTES and IMMUNE SYSTEM: Neutrophilic leukocytosis occurs to the extent of 8,000/mm^3 and even to 20,000/mm^3 in labor. The increase may be due to rise in the levels of estrogen and cortisol. **The major change in the immune system** is the modulation away from cell-mediated cytotoxic immune response toward increased humoral and innate immune responses.

TOTAL PROTEIN: Total plasma protein increases from the normal 180 g (nonpregnant) to 230 g at term. However, due to hemodilution, the plasma protein concentration falls from 7 g% to 6 g%. This results in diminished viscosity of the blood and reduced colloid osmotic tension. Because of marked fall in albumin level from 4.3 g% to 3 g%—a fall of about 30% and only slight rise of globulin (mainly α globulin), **the normal albumin:globulin ratio of 1.7:1 is diminished to 1:1 (Table 5.2)**.

Table 5.2: Plasma Protein Changes during Pregnancy

Parameters	Non-pregnant	Pregnancy near term	Change
Total protein (g)	180	230	Increased
Plasma protein concentration (g/100 mL)	7	6	Decreased
Albumin (g/100 mL)	4.3	3	Decreased (30%)
Globulin (g/100 mL)	2.7	3	Slightly increased
Albumin:Globulin	1.7:1	1:1	Decreased

BLOOD COAGULATION FACTORS: Pregnancy is a hypercoagulable state. Fibrinogen level is raised by 50% from 200–400 mg/dL in nonpregnant to 300–600 mg/dL in pregnancy. As a result of rise in fibrinogen and globulin level and diminished blood viscosity, **erythrocyte sedimentation rate (ESR) gives a much higher value** (fourfold increase) during pregnancy (Table 5.3). **As such, ESR has got little diagnostic value in pregnancy**. Platelets count, however, gives a conflicting picture. Recent studies show a static or a slight fall to the extent of 15% of pre-pregnant level. **Gestational thrombocytopenia may be due to hemodilution and increased platelet consumption (p. 318)**. Fibrinolytic activity is depressed till 15 minutes after delivery. There is increase in activities of clotting factors like X, IX, VIII, VII and I. The levels of II, V and XII are either unchanged or mildly increased. The level of factors XI and XIII are slightly decreased. The clotting time does not show any significant change. These are all effective to control blood loss and hemostasis after the separation of placenta (Table 5.3). **Levels of coagulation factors normalize 2 weeks postpartum**. Leukocyte levels return to normal within 1–12 weeks of delivery.

Table 5.3: Changes in Blood Coagulation Factors

Parameters	Nonpregnant	Pregnancy near term	Change
Platelets (mm^3)	1,60,000–2,00,000	Conflicting observation	Static or 15% reduction of the count
Fibrinogen (mg%)	200–400	300–600	+ 50%
Fibrinolytic activity	—	Depressed	—
Clotting time	—	Unaffected	—
ESR	10 mm/h	40 mm/h	Marked increase (4 times)

CARDIOVASCULAR SYSTEM

ANATOMICAL CHANGES: Due to elevation of the diaphragm consequent to the enlarged uterus, the **heart is pushed upward and outward** with slight rotation to left (Table 5.4).

Abnormal clinical findings: The displacement may, at times, be responsible for palpitation. **The apex beat** is shifted to the 4th intercostal space about 2.5 cm outside the midclavicular line. Pulse rate is slightly raised, often with extrasystoles. **A systolic murmur** may be audible in the apical or pulmonary area. This is due to decreased blood viscosity and torsion of the great vessels. **A continuous hissing murmur** may be audible over the tricuspid area in the left second and third intercostal spaces called the "**mammary murmur**". It is due to increased blood flow through the internal mammary vessels. **Doppler echocardiography** shows an increase in the left ventricular end diastolic diameters. The left and right atrial diameters also increase. A third heart sound (S3) due to rapid diastolic filling and rarely a fourth heart sound may be auscultated. **ECG reveals** normal pattern except evidences of left axis deviation. **The physician should be familiar with these physiological findings and should execute a cautious approach in diagnosis of heart disease during pregnancy** (see p. 319).

CARDIAC OUTPUT: The cardiac output (CO) starts to increase from 5th week of pregnancy and reaches its peak 40–50% at about 30–34 weeks. Thereafter the CO remains static till term when the observation is made at lateral recumbent position. **CO is lowest in the sitting or supine position and highest in the right or left lateral or knee chest position.** Cardiac output **increases further** during labor (+50%) and immediately following delivery (+70%) over the pre-labor values. MAP also rises. There is squeezing out of blood from the uterus into the maternal circulation **(auto transfusion)** during labor and in the immediate postpartum. **CO returns to pre-labor values by 1 hour following delivery and to the pre-pregnant level by another 4 weeks time**.

Table 5.4: Hemodynamic Changes during Pregnancy

Parameters	Non-pregnant	Pregnancy near term	Change
Cardiac output (L/min)	4.5	6.26	+ 40%
Stroke volume (mL)	65	75	+ 27%
Heart rate (per minute)	70	85	+ 17%
Blood pressure	Unaffected or mid-pregnancy drop of diastolic pressure by 5–10 mm Hg		
Venous pressure	10 cm (femoral)	20–25 cm water	+ 100%
Colloid oncotic pressure (mm Hg)	20	18	– 14%
Systemic vascular resistance (SVR)	—	—	– 21%
Pulmonary vascular reistance (PVR)	—	—	– 34%

The increase in CO is caused by: (1) Increased blood volume. (2) To meet the additional O_2 required due to increased metabolic activity during pregnancy. CO is the product of SV and HR (CO = SV × HR). The increase in CO is chiefly affected by **increase in stroke volume and increase in pulse** rate to about 15 per minute. A normal heart got enough reserve power to cope with the increased load but a damaged heart fails to do so.

BLOOD PRESSURE: Systemic vascular resistance (SVR) decreases (–21%) due to smooth muscle relaxing effect of progesterone, NO, prostaglandins or ANP. In spite of the large increase in cardiac output, the **maternal BP (BP = CO × SVR) is decreased** due to decrease in SVR. **There is overall decrease in diastolic blood pressure (BP) and mean arterial pressure (MAP) by 5–10 mm Hg**. The decrease in maternal blood pressure parallels that of SVR.

VENOUS PRESSURE: Antecubital venous pressure remains unaffected. Femoral venous pressure is markedly raised especially in the later months. **It is due to pressure exerted by the gravid uterus on the common iliac veins, more on the right side due to dextrorotation of the uterus**. The femoral venous pressure is raised from 8–10 cm of water in nonpregnant state to about 25 cm of water during pregnancy in lying down position and to about 80–100 cm of water in standing position. **This explains the fact that the physiological edema of pregnancy subsides by rest alone**. Distensibility of the veins and stagnation of blood in the venous system explain the development of edema, varicose veins, piles and deep vein thrombosis.

CENTRAL HEMODYNAMICS: In pregnancy, there is **no significant change in CVP, MAP and PCWP** although there is increase in blood volume, cardiac output and heart rate. The reasons are: there is significant fall in SVR, pulmonary vascular resistance (PVR) and colloidal osmotic pressure.

SUPINE HYPOTENSION SYNDROME (POSTURAL HYPOTENSION): During late pregnancy, the gravid uterus produces a compression effect on the inferior vena cava when the patient is in supine position. This, however, results in opening up of the collateral circulation by means of paravertebral and azygos veins. In some cases (10%), when the collateral circulation fails to open up, the venous return of the heart may be seriously curtailed. This results in production of hypotension, tachycardia and syncope. **The normal blood pressure is quickly restored by turning the patient to lateral position.** The augmentation of the venous return during uterine contraction prevents the manifestation from developing during labor.

REGIONAL DISTRIBUTION OF BLOOD FLOW: Uterine blood flow is increased from 50 mL/min in nonpregnant state to about 750 mL near term. Nonpregnant uterus receives 2% of CO and breasts 1%. Increase in blood flow going to the organs is about 50% due to overall increase in CO. The increase is due to the combined effect of uteroplacental and fetoplacental vasodilatation (see p. 36). The **vasodilatation** is due to the smooth muscle relaxing effects of progesterone, estrogen, nitric oxide (endothelium derived factor), prostaglandins and atrial natriuretic peptide (ANP). In a normal pregnancy, vascular system becomes refractory to angiotensin II, endothelin I and other pressure agents (see p. 257). **Pulmonary blood flow** (normal 6,000 mL/min) is increased by 2,500 mL/min. **Renal blood flow** (normal 800 mL) increases by 400 mL/min at 16th week and remains at this level till term. **The blood flow through the skin and mucous membranes** reaches a maximum of 500 mL/min by 36th week. **Heat sensation, sweating or stuffy nose complained by the pregnant women can be explained by the increased blood flow**.

METABOLIC CHANGES

GENERAL METABOLIC CHANGES: Total metabolism is increased due to the needs of the growing fetus and the uterus. Basal metabolic rate is increased to the extent of 30% higher than that of the average for the nonpregnant women.

PROTEIN METABOLISM: There is a positive nitrogenous balance throughout pregnancy. At term, the fetus and the placenta contain about 500 g of protein and the maternal gain is also about 500 g chiefly distributed in the uterus, breasts and the maternal blood. **As the breakdown of amino acid to urea is suppressed, the blood urea level falls to 15–20 mg%**. Blood uric acid and creatinine level, however, either remain unchanged or fall slightly. Amino acids are **actively transported** across the placenta to the fetus. Pregnancy is an anabolic state.

CARBOHYDRATE METABOLISM: Transfer of increased amount of glucose from mother to the fetus is needed throughout pregnancy. **Insulin secretion is increased** in response to glucose and amino acids. There is hyperplasia and hypertrophy of beta cells of pancreas. **Sensitivity of insulin receptors is decreased** (44%) especially during later months of pregnancy. **Plasma insulin level is increased** due to a number of contra insulin factors. **These are:** estrogen, progesterone, human placental lactogen (hPL), cortisol, prolactin, free fatty acids, leptin, and TNFα. There is increased tissue resistance to insulin. **This mechanism ensures continuous supply of glucose to the fetus**.

Increased insulin level favors lipogenesis (fat storage). **During maternal fasting**, there is hypoglycemia, hypoinsulinemia, hyperlipidemia and hyperketonemia. Lipolysis generates free fatty acids (FFA) for gluconeogenesis and fuel supply. **Plasma glucagon** level remains unchanged.

Overall effect is maternal **fasting hypoglycemia** (due to fetal consumption) and **postprandial hyperglycemia and hyperinsulinemia** (due to anti-insulin factors). **Oral glucose tolerance test may show an abnormal pattern**. This helps to maintain a continuous supply of glucose and FFA to the fetus. As maternal utilization of glucose is reduced, there are gluconeogenesis and glycogenolysis. Glomerular filtration of glucose is increased to exceed the tubular absorption threshold (normal 180 mg%). So glycosuria is detected in 50% of normal pregnant women.

FAT METABOLISM: An average of 3–4 kg of fat is stored during pregnancy mostly in the abdominal wall, breasts, hips and thighs. Plasma lipids and lipoproteins increase appreciably during the latter half of pregnancy due to increased estrogen, progesterone, hPL and leptin levels.

LIPID METABOLISM: HDL level increases by 15%. LDL is utilized for placental steroid synthesis. This hyperlipidemia of normal pregnancy is **not atherogenic**. The activity of lipoprotein lipase is increased. Changes in the lipid components are tabulated in Table 5.5. Leptin, a peptide hormone, is secreted by adipose tissue and placenta. It regulates the body fat metabolism.

Table 5.5: Changes in Lipid Metabolism

Parameters	Non-pregnant	Pregnancy near term	Change
Total lipid (mg/100 mL)	650	1000	+ 50%
LDL and Cholesterol (mg/100 mL)	180	260	+ 40%
HDL (mg/100 mL)	60	70	+ 15%
Triglycerides (mg/100 mL)	80	160	+ 50%

IRON METABOLISM: Iron is absorbed in **ferrous form from duodenum and jejunum** and is released into the circulation as transferrin. About 10% of ingested iron is absorbed. Iron freed from transferrin is incorporated into hemoglobin (75%) and myoglobin or stored as ferritin or hemosiderin. Iron is transported actively across the placenta to the fetus. Iron requirement during pregnancy is considerable and is mostly limited to the second half of the pregnancy especially to the last 12 weeks. **Total iron requirement during pregnancy is estimated approximately 1,000 mg**. This is distributed in fetus and placenta 300 mg and expanded red cell mass 400 mg. (Total increase in red cell volume—350 mL and 1 mL contains 1.1 mg of iron.) There is obligatory loss of about 200 mg through normal routes. The iron in the fetus and placenta is permanently lost and a variable amount of iron in the expanded RBC volume is also lost due to blood loss during delivery (45 mg/100 mL) and the rest is returned to the store. However, there is saving of about 300 mg of iron due to amenorrhea for 10 months. (Iron loss in menstrual bleeding per cycle is 30 mg.) Iron need during lactation is 1 mg/day. **This iron need is not squarely distributed throughout the pregnancy but mostly limited to the third trimester**. (Daily iron requirement in non-menstruating women to compensate the average daily loss is 1 mg.) **Thus, in the second half, the daily requirement actually becomes very much increased to the extent of about 6–7 mg.**

The amount of the iron absorbed from the diet and that mobilized from the store are inadequate to meet the demand. In spite of the fact that absorption through the gut is enhanced during pregnancy, **serum ferritin** level actually reflects the body iron stores. **In the absence of iron supplementation, there is drop in hemoglobin, serum iron and serum ferritin concentration at term pregnancy (p. 304, Table 20.1). Thus, pregnancy is an inevitable iron deficiency state**. However, placenta transfers adequate iron to the fetus, despite severe maternal iron deficiency. Thus, there is no correlation of hemoglobin concentrations between mother and fetus.

SYSTEMIC CHANGES

RESPIRATORY SYSTEM: With the enlargement of the uterus, especially in the later months, there is elevation of the diaphragm by 4 cm. Total lung capacity is reduced by 5% due to this elevation. However, diaphragmatic excursion is increased by 1–2 cm and breathing becomes diaphragmatic. Total pulmonary resistance is reduced due to progesterone effect. The **subcostal angle increases** from 68° to 103°, the **transverse diameter** of the chest **expands** by 2 cm and the **chest circumference increases** by 5–7 cm. The mucosa of the nasopharynx becomes hyperemic and edematous. This may cause nasal stuffiness and rarely epistaxis. **A state of hyperventilation occurs** during pregnancy leading to **increase in tidal volume** and therefore respiratory minute volume by 40% (Table 5.6). **It is probably due to** progesterone acting on the respiratory center and also to increase in sensitivity of the center to CO_2. The woman feels shortness of breath.

Acid base balance—The hyperventilation causes changes in the acid base balance. The arterial $PaCO_2$ falls 38–32 mm Hg and PaO_2 rises 95–105 mm Hg. These facilitate transfer of CO_2 from fetus to mother and O_2 from mother to fetus (Table 5.7).

The pH rises in order of 0.02 unit and there is a base excess of 2 mEq/L. Thus, **pregnancy is in a state of respiratory alkalosis**. Partial renal compensation occurs through increased excretion of bicarbonate. Maternal O_2 consumption is increased by 20–40% due to increased demand of the fetus, placenta and maternal tissues.

Maternal oxygen reserve during pregnancy is reduced due to: (a) increased oxygen consumption and (b) reduced functional residual capacity. These physiological changes make a pregnant woman more susceptible to effects of apnea (during intubation).

URINARY SYSTEM: ***Kidney***—There is dilatation of the ureters, renal pelvis and the calyces. The kidneys enlarge in length by 1 cm. **Renal plasma flow** is increased by 50–75%, maximum by the 16 weeks and is maintained until 34 weeks. Thereafter it falls by 25%. **Glomerular filtration rate (GFR)** is increased by 50% all throughout pregnancy. Increased GFR causes reduction in maternal plasma levels of creatinine, blood urea nitrogen (BUN) and uric acid. Renal tubules fail to reabsorb glucose, uric acid, amino acids and water soluble vitamins completely.

Ureter: **Ureters become atonic** due to high progesterone level. **Dilatation** of the ureter above the pelvic brim with stasis is marked on the right side especially in primigravidae. **It is due to** dextrorotation of the uterus pressing the right ureter against the pelvic brim and also due to pressure by the right ovarian vein which crosses the right ureter at right angle. **The stasis is marked between 20–24 weeks**. There is marked hypertrophy of the muscle and the sheath of the ureter especially the pelvic part probably due to estrogen. **There is elongation**, **kinking and outward displacement of the ureters**. The dilatation effect resolves by 6 weeks postpartum.

Bladder: There is marked congestion with hypertrophy of the muscles and elastic tissues of the wall. **In late pregnancy**, the bladder mucosa becomes edematous due to venous and lymphatic obstruction especially in primigravidae following early engagement. Increased frequency of micturition is noticed at 6–8 weeks of pregnancy which subsides after 12 weeks. **It may be due to resetting of osmoregulation causing increased water intake and polyuria. In late pregnancy, frequency of micturition once more reappears**

Table 5.6: Changes in Respiratory System

Parameters	Non-pregnant	Pregnancy near term	Change
Respiration rate/min	15	15	Unaffected
Vital capacity (mL)	3200	3300	Almost unaltered
Tidal volume (mL)	475	675	+ 40%
Residual volume (mL)	965	765	– 20%
Inspiratory capacity (IC)	2500	2650	+10%
Minute ventilation	7.5 L/min	10.5 L/min	+40%
Total lung capacity (mL)	4200	4000	–5%

Table 5.7: Acid-Base Changes

Parameters	Non-pregnant	Pregnancy near term	Change
Arterial PO_2	95 mm Hg	106 mm Hg	Increased
Arterial PCO_2	38 mm Hg	32 mm Hg	Diminished
pH	7.40	7.42	Slightly increased
Plasma HCO_3^-	26 m mol/L	22 mmol/L	Decreased

due to pressure on the bladder as the presenting part descends down the pelvis. **Stress incontinence** may be observed in late pregnancy due to urethral sphincter weakness.

ALIMENTARY SYSTEM: **The gums** become congested and spongy and may bleed to touch. **Muscle tone and motility of the entire gastrointestinal tract are diminished** due to high progesterone level. **Cardiac sphincter** is relaxed and regurgitation of acid gastric content into the esophagus may produce chemical esophagitis and heart burn. Dyspepsia is common. There is diminished gastric secretion and delayed emptying time of the stomach. **Risk of peptic ulcer disease is reduced**. Atonicity of the gut leads to **constipation**, while diminished peristalsis facilitates more absorption of food materials.

LIVER AND GALLBLADDER: Although there is no histological change in the liver cells, but the functions are depressed. **With the exception of raised alkaline phosphatase levels, other liver function tests** (serum levels of bilirubin, AST, ALT, CPK, LDH) **are unchanged**. There is mild cholestasis (estrogen effect). There is marked atonicity of the gallbladder (progesterone effect). This, together with high blood cholesterol level during pregnancy, favors stone formation.

NERVOUS SYSTEM: Some sorts of temperamental changes are found during pregnancy and in the puerperium. Nausea, vomiting, mental irritability and sleep disorders are probably due to some psychological background. **Postpartum blues, depression or psychosis may develop in a susceptible individual (p. 511)**.

Compression of the **median nerve** underneath the flexor retinaculum over the wrist joint leading to pain and paresthesia in the hands and arm **(Carpal tunnel syndrome)** may appear in the later months of pregnancy. Similarly, paresthesia and sensory loss over the anterolateral aspect of the thigh may occur. It is due to **compression of the lateral cutaneous nerve of the thigh**.

CALCIUM METABOLISM AND SKELETAL SYSTEM: During pregnancy there is increase in the demand of calcium by the growing fetus to the extent of 28 g, 80% of which is required in the last trimester for fetal bone mineralization. **Daily requirement of calcium during pregnancy and lactation averages 1–1.5 g**. **Maternal total calcium levels fall but serum ionized calcium level is unchanged**. Fifty percent of serum calcium is ionized which is important for physiological function. Calcium absorption from intestine and kidneys are doubled due to **rise in the level of 1, 25 dihydroxy vitamin D_3**. Pregnancy does not cause hyperparathyroidism. Calcitonin levels increase by 20%. Calcitonin protects the maternal skeleton from osteoporosis. **Maternal serum phosphate level is unchanged**.

There is increased mobility of the pelvic joints due to softening of the ligaments caused mainly by hormone. This along with increased lumbar lordosis during later months of pregnancy due to enlarged uterus produces backache and waddling gait.

ENDOCRINE SYSTEM: The endocrinology in relation to pregnancy is discussed in the chapter 6.

QUESTIONS

1. Discuss in brief the important changes in the cardiovascular system in pregnancy? (p. 60)
2. Discuss in brief the important changes in the respiratory system in pregnancy? (p. 62)

Write Short Notes on:

A. Weight gain in pregnancy (p. 57)
B. Hematological changes in pregnancy (p. 58)
C. Supine hypotension syndrome (p. 61)
D. Carbohydrate metabolism in pregnancy (p. 61)

Related theory questions (Long & Short), Obstetric Case Discussions, Viva table discussions, Postoperative word round discussions, and MCQs are discussed in author's books:

1. **Bedside Clinic and Viva Voce:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

For further reading:

2. **Master pass in Obstetrics and Gynaecology**: 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

Chapter 6

Endocrinology in Relation to Reproduction

The endocrine glands play very important role in the physiology of reproduction. **Endocrinology in relation to reproduction includes the knowledge of**:

- Hormones essential for the maturation of the Graafian follicles, ovulation and maintenance of corpus luteum after fertilization.
- Following conception, transfer of function of pituitary-ovarian axis to placenta, which acts temporarily as a new powerhouse or **endocrine organ**.
- Physiological alteration of various endocrine glands namely, the pituitary, thyroid, parathyroid, adrenals and pancreas during pregnancy.
- Endocrine control of **labor**. This is discussed in Chapter 13.
- Hormonal influence during puerperium necessary for maintenance of **lactation**.

MATURATION OF GRAAFIAN FOLLICLES AND OVULATION

Follicular maturation takes place during the first half of the cycle. **The hormones essential for follicular maturation are** mainly FSH and a small proportion of LH. For continued FSH activity, estrogen is necessary.

OVULATION: Under the influence of FSH, about 20 Graafian follicles develop synchronously and only one of them will be able to ovulate. The rest become atretic and mix-up with interstitial tissue. For details see author's Textbook of Gynecology — Chapter 8.

CAUSES OF OVULATION: The possible factors involved in ovulation have been described in page 22.

CHANGES WITHIN THE FOLLICLE AFTER OVULATION: The avascular Graafian follicle becomes vascularized and the granulosa cells become luteinized. The morphologically altered Graafian follicle is now changed into **corpus luteum**. The corpus luteum secretes **progesterone**, **17α-hydroxy progesterone (luteinized granulosa cells) and estradiol**, **androstenedione (theca cells)**.

MAINTENANCE OF CORPUS LUTEUM AFTER FERTILIZATION

Function of corpus luteum is essential to maintain the early pregnancy. Corpus luteum secretes about 40 mg of progesterone a day. After implantation, human chorionic gonadotropin (hCG) and possibly human placental lactogen (hPL), secreted by the syncytiotrophoblast cells maintain the growth and function of the corpus luteum.

PLACENTAL ENDOCRINOLOGY

At 6–8 weeks, there is transfer of functions of corpus luteum to the placenta (luteal-placental shift)—which acts temporarily as a new **endocrine organ** or powerhouse of hormone production.

HORMONES OF PLACENTA

Placenta produces a variety of hormones of which **protein and steroid hormones** are significantly important. Syncytiotrophoblasts contain abundant rough endoplasmic reticulum, Golgi bodies and mitochondria. **Syncytiotrophoblasts** are the principal site of **protein and steroid hormones** in pregnancy.

HORMONES OF PLACENTA AND THEIR CYTOCHEMICAL ORIGIN

Hypothalamic-like (releasing) Hormones	Cytochemical Origin
■ Corticotropin-releasing hormone (CRH)	Cytotrophoblast and syncytiotrophoblast
■ Gonadotropin-releasing hormone (GnRH)	+
■ Thyrotropin-releasing hormone (TRH)	+
■ Growth hormone releasing hormone (GHRH)	+

Pituitary-like Hormones	Cytochemical Origin
■ Adrenocorticotropic hormone (ACTH)	Syncytiotrophoblast
■ Human chorionic gonadotropin (hCG)	+
■ Human chorionic thyrotropin (hCT)	+
■ Human placental lactogen (hPL)	+

Other Pregnancy Proteins	Cytochemical Origin
♦ PSβG, PAPP – A	Syncytiotrophoblast
♦ Relaxin	Corpus luteum, Decidua, Placenta
♦ Prolactin	Decidua
♦ Atrial natriuretic peptide (ANP)	Atrial myocyte (see p. 58)
♦ Early pregnancy factor (EPF)	Activated platelets ovaries

Growth Factors	Cytochemical Origin
♦ Inhibin, activin, transforming growth factor β (TGF-β)	Syncytiotrophoblast
IGF-1 and 2, Epidermal growth factor (EGF)	+

Steroid Hormones	Cytochemical Origin
♦ Estrogens (estriol)	Fetoplacental unit
♦ Progesterone	Before 6 weeks — corpus luteum thereafter placenta
♦ Cortisol	Decidua, adrenal

PROTEIN HORMONES

Protein hormones are similar but not necessarily identical with those produced by the pituitary. For example, placental lactogen is chemically similar to both pituitary growth hormone and prolactin, but biological activity of placental lactogen is much inferior than prolactin or growth hormone produced by pituitary.

HUMAN CHORIONIC GONADOTROPIN (hCG): hCG is a glycoprotein. Its molecular weight is 36,000–40,000 daltons. It consists of a hormone nonspecific α (92 amino acids) and a hormone specific β (145 amino acids) subunit. hCG is chemically and functionally similar to pituitary luteinizing hormone. The α subunit is biochemically similar to LH, FSH and TSH whereas the β subunit is relatively unique to hCG. Placental GnRH may have a control on hCG formation.

Functions:

(1) It acts as a stimulus for the secretion of progesterone by the corpus luteum of pregnancy. **Rescue and maintenance of corpus luteum** till 6 weeks of pregnancy is the major biological function of hCG.

(2) **hCG stimulates Leydig cells of the male fetus** to produce testosterone in conjunction with fetal pituitary gonadotropins. It is thus indirectly involved in the development of male external genitalia.

(3) It has got **immunosuppressive activity,** which may inhibit the maternal processes of immunorejection of the fetus as a homograft.

(4) **Stimulates** both adrenal and placental steroidogenesis.

(5) **Stimulates** maternal thyroid because of its thyrotropic activity.

(6) **Promotes** secretion of relaxin from the corpus luteum.

Level of hCG at different periods of pregnancy: hCG is produced by the syncytiotrophoblast of the placenta and secreted into the blood of both mother and fetus. **The plasma half-life of** hCG is about 36 hours. **By radioimmunoassay, it can be detected in the maternal serum or urine as early as 8–9 days postfertilization**. In the early pregnancy, **the doubling time of hCG** concentrations in plasma is 1.4–2 days. The blood and urine values reach **maximum levels ranging from 100 IU/mL to 200 IU/mL between 60 and 70 days of pregnancy**. The concentration falls slowly reaching a low level of 10–20 IU/mL between 100 and 130 days. Thereafter, the levels remain const-ant throughout pregnancy with a slight secondary peak at 32 weeks. High levels of hCG could be detected in—(a) multiple pregnancy, (b) hydatidiform mole or choriocarcinoma and (c) relatively high in pregnancy with a trisomy 21 fetus (Down's syndrome). **Plasma lower levels** are found in ectopic pregnancies and in spontaneous abortion. **hCG disappears from the circulation within 2 weeks following delivery (Fig. 6.1)**.

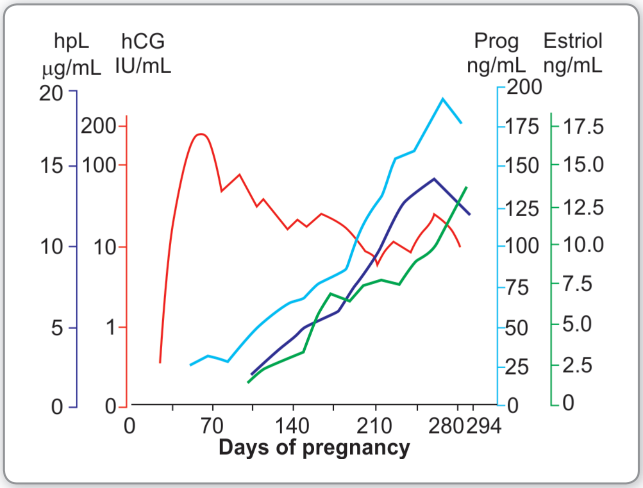
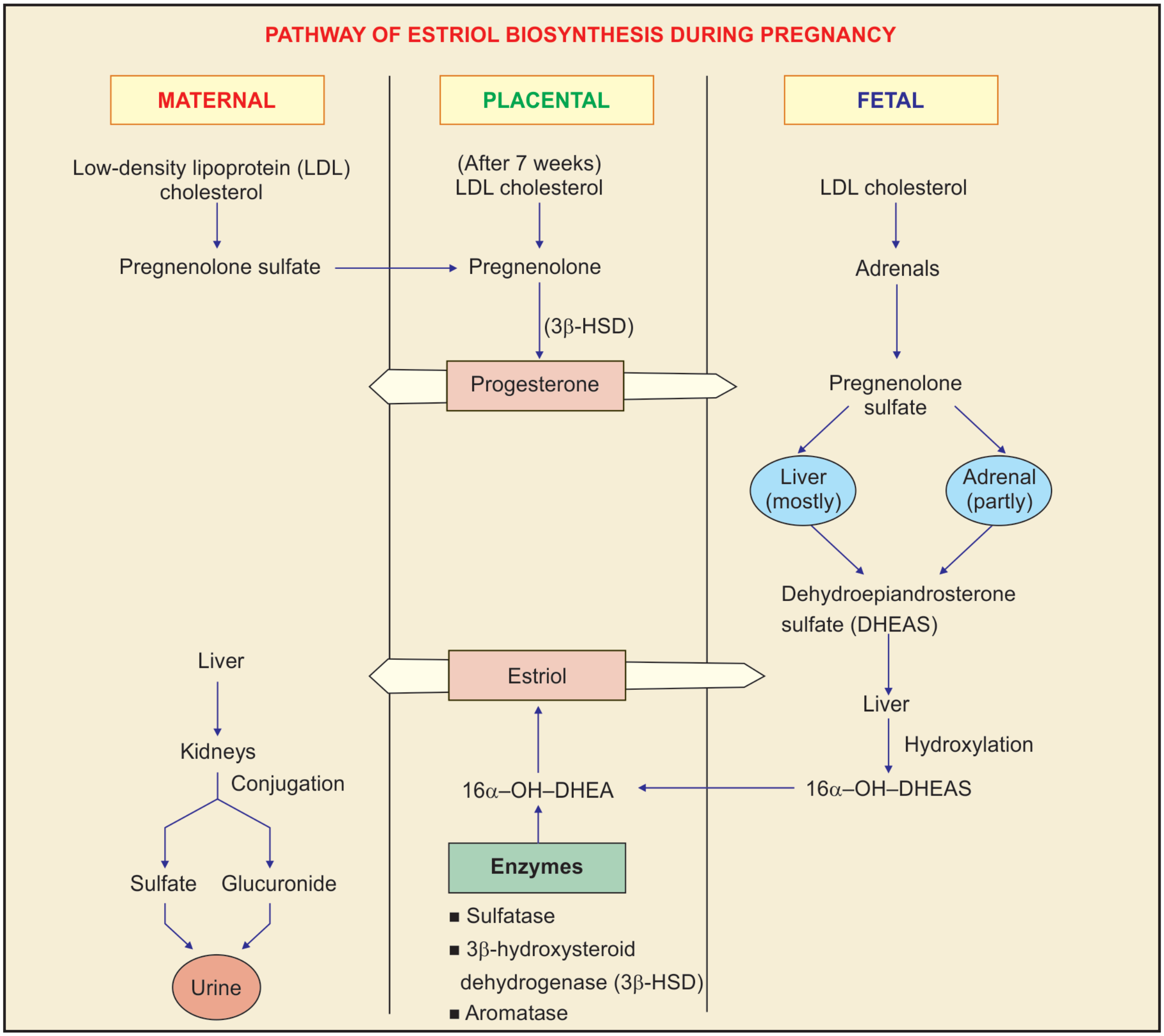


Fig. 6.1: Mean serum levels of placental hormones during pregnancy as measured by radioimmunoassay

HUMAN PLACENTAL LACTOGEN (hPL): This is also known as **human chorionic somatomammotropin (hCS)**. The hormone is synthesized by the syncytiotrophoblast of the placenta. The hormone is chemically and immunologically similar to pituitary growth hormone and prolactin. hPL in maternal serum is first detected during the 3rd week of gestation. The level rises progressively from 5 µg/mL to 25 µg/mL until about 36 weeks of gestation. **The plasma concentration of hPL is proportional to placental mass**.

Functions: hPL antagonizes insulin action. High level of maternal insulin helps protein synthesis. hPL causes maternal lipolysis and promotes transfer of glucose and amino acids to the fetus. As a potent angiogenic hormone, it helps to develop fetal vasculature. It promotes growth and differentiation of breasts for lactation.

PREGNANCY-SPECIFIC β-1 GLYCOPROTEIN (PS β-1G): It is produced by the trophoblast cells. **It can be detected in the maternal serum 18–20 days after ovulation**. PS β-1G is a potent immunosuppressor of lymphocyte proliferation and prevents rejection of the conceptus.



PATHWAY OF ESTRIOL BIOSYNTHESIS DURING PREGNANCY

MATERNAL
PLACENTAL
FETAL
Low-density lipoprotein (LDL) cholesterol
Pregnenolone sulfate
(After 7 weeks) LDL cholesterol
Pregnenolone
(3β-HSD)
Progesterone
LDL cholesterol
Adrenals
Pregnenolone sulfate
Liver (mostly)
Adrenal (partly)
Dehydroepiandrosterone sulfate (DHEAS)
Liver
Hydroxylation
16α–OH–DHEAS
16α–OH–DHEA
Estriol
Liver
Kidneys
Conjugation
Sulfate
Glucuronide
Urine
Enzymes
■ Sulfatase
■ 3β-hydroxysteroid dehydrogenase (3β-HSD)
■ Aromatase

Early pregnancy factor (EPF) is a protein, produced by the activated platelets and other maternal tissues. It is detectable in the circulation 6 – 24 hours after conception. EPF is immunosuppressant and prevents rejection of the conceptus.

Growth factors: Inhibin, activin, insulin-like growth factor (IGF-1 and 2), transforming growth factor β (TGF-β) and epidermal growth factor (EGF) are produced by the syncytiotrophoblast cells. **They have varied functions** including immunosuppressive, paracrine and steroidogenic.

Pregnancy-associated plasma protein—A (PAPP-A) is secreted by the syncytiotrophoblast. It acts as an immunosuppressant in pregnancy.

STEROIDAL HORMONES

ESTROGEN: In late pregnancy, qualitatively, estriol is the most important amongst the three major estrogens. **The site of its production is in the syncytiotrophoblast**.

Fetoplacental unit and biosynthesis of estriol:

The placenta is an incomplete endocrine organ as it has no capability of independent steroidogenesis like that of ovary. For steroidogenesis, it depends much on the precursors derived mainly from the fetal and partly from the maternal sources. Fetal adrenal gland and the placenta contain the complementary enzyme system. This is the key of Diczfalusy's concept of fetoplacental or better still **maternal-fetoplacental unit**. The biosynthesis pathway in the final formation of estriol is shown diagrammatically in the scheme above.

Estriol is first detectable at 9 weeks (0.05 ng/mL) and increases gradually to about 30 ng/mL at term. Fetal death, fetal anomalies (adrenal atrophy, anencephaly, Down's syndrome), hydatidiform moles, placental sulfatase or aromatase deficiency are associated with **low estriol**.

PROGESTERONE: Before 6 weeks of pregnancy, the corpus luteum secretes 17-hydroxyprogesterone. Following the development of trophoblast, progesterone is synthesized and secreted in increasing amount from the placenta. **Precursors from fetal origin are not necessary as in estrogen production.** The placenta can utilize cholesterol as a precursor derived from the mother for the production of pregnenolone. Pregnenolone is converted to progesterone in the endoplasmic reticulum by 3 β-hydroxy steroid dehydrogenase. The daily production rate of progesterone in late normal pregnancy is about 250 mg. **Low progesterone levels** are observed in ectopic pregnancy and in abortion. **High values** are observed in hydatidiform mole, **Rh-isoimmunization**. **After delivery, the plasma progesterone decreases rapidly and is not detectable after 24 hours**.

Functions of the steroid hormones (estrogen and progesterone):

It is indeed difficult to single out the function of one from the other.

- **Together** they play an important role in the maintenance of pregnancy. **Estrogen** causes hypertrophy and hyperplasia of the uterine myometrium, thereby increasing the accommodation capacity and blood flow of the uterus. **Progesterone** in conjunction with estrogen stimulates growth of the uterus, causes decidual changes of the endometrium required for implantation and it inhibits myometrial contraction.
- Development and hypertrophy of the **breasts during pregnancy** are achieved by a number of hormones. Hypertrophy and proliferation of the ducts are due to **estrogen**, while those of lobulo-alveolar system are due to combined action of **estrogen and progesterone** (details are given below).
- **Both the steroids** are required for the adaptation of the maternal organs to the constantly increasing demands of the growing fetus.
- **Progesterone** maintains uterine quiescence, by stabilizing lysosomal membranes and inhibiting prostaglandin synthesis. Progesterone and estrogens are antagonistic in the process of labor (see Chapter 12).
- **Estrogens** sensitizes the myometrium to oxytocin and prostaglandins. Estrogens ripen the cervix.
- **Progesterone along** with hCG and decidual cortisol inhibits T-lymphocyte mediated tissue rejection and protects the conceptus (immunomodulatory role).
- **Together** they cause inhibition of cyclic fluctuating activity of gonadotropin–gonadal axis thereby preserving gonadal function.

DIAGNOSTIC VALUE OF PLACENTAL HORMONES

(a) ***Diagnosis of pregnancy***—Presence of hCG in the plasma can be detected by radioimmunoassay and in urine either by biological or immunological tests.

Radioimmunoassay can detect minute quantity of plasma hCG β subunit soon after the implantation of blastocyst but biological and **immunological tests do not become positive at least 44 days after the last menstrual period**.

(b) ***Follow-up cases who had trophoblastic tumors:*** Radioimmunoassay for the detection of hCG β subunit is more sensitive in the follow-up study of hydatidiform mole or choriocarcinoma.

(c) ***Detection of function of fetoplacental unit:*** The placental hormones commonly studied for evaluating placental insufficiency and status of the fetus-in-utero are hPL and estriol. These biochemical changes have been largely replaced by biophysical profiles (see Chapter 11).

RELAXIN: It is a peptide hormone structurally related to insulin. The main source of production is the corpus luteum of the ovary but part of it may be also produced by the placenta and decidua. It has been claimed that relaxin relaxes myometrium, the symphysis and sacroiliac joints during pregnancy and also helps in cervical ripening by its biochemical effect.

CHANGES OF ENDOCRINE GLANDS DURING PREGNANCY

Pituitary, thyroid, adrenal cortex, parathyroid and pancreas show distinct physiological changes during pregnancy leading to increase in output of respective hormones. The basic purpose of these changes is to adjust the internal environment of the mother to meet the additional requirements imposed by metabolic changes during pregnancy as well as to meet the extra demands by the growing fetus.

The specific anatomical and physiological changes in the individual endocrine glands are described in the next page.

PITUITARY GLAND

MORPHOLOGICAL CHANGES IN NORMAL PREGNANCY: During normal pregnancy, the pituitary increases in weight by 30–50% and is enlarged to about twice its normal size. This is principally due to hyperplasia of acidophilic prolactin secreting cells. Sometimes, the pituitary enlargement may impinge on the optic chiasma causing bitemporal hemianopia. Maternal pituitary gland is not necessary for the maintenance of pregnancy. The pituitary gland during pregnancy becomes more susceptible to alterations in blood supply. Sudden hypotension following postpartum hemorrhage may cause infarction of the gland (Sheehan Syndrome).

PHYSIOLOGICAL CHANGES: Pituitary gonadotropins (FSH, LH) levels are low due to increased level of estrogens and progesterone. **Growth hormone** level is elevated due to growth hormone variant made by syncytiotrophoblast of the placenta and this explains partly the weight gain observed during normal pregnancy. **Serum prolactin** level increases by 10 times. **TSH** secretion is same as in nonpregnant state. **ACTH and corticotropin-releasing hormone (CRH)** levels increase. **ACTH** does not cross the placenta while TRH does. Plasma vasopressin **(ADH)** level remains unchanged during pregnancy. All the pregnancy-induced changes in the pituitary revert to normal within few months after delivery.

THYROID

MORPHOLOGICAL CHANGES DURING PREGNANCY: Hyperplasia of the thyroid gland occurs during normal pregnancy and causes slight generalized enlargement of the gland. However, pregnant women remain euthyroid.

PHYSIOLOGICAL CHANGES: Renal clearance of iodine is increased due to increased glomerular filtration. **Maternal serum iodine levels fall** due to increased renal loss and also due to transplacental shift to the fetus. These cause hyperplasia of the gland. Iodine intake during pregnancy should be increased from 100 μg/day to 200 μg/day (as recommended by WHO). **There is rise in the basal metabolic rate**, which begins at about the third month, reaches a value of +25% during the last trimester. The increase in BMR is probably due to increase in net oxygen consumption of mother and fetus.

There is stimulatory effect of hCG (chorionic thyrotropin) to thyroid gland mainly in the first trimester. Due to this thyrotropic effect of hCG, there may be a transient phase of hyperthyroidism in some women (gestational transient thyrotoxicosis)

The serum protein-bound iodine is increased in pregnancy, the range being 6.2–11.2 µg% instead of the usual 4–8 µg%. **Thyroxin-binding globulin (TBG) increases** and reaches a plateau by 20 weeks. It remains unchanged at that level until delivery. The increase in TBG is due to estrogen stimulation. Iodine and drugs used to treat hyperthyroidism cross the placenta freely.

Fetal thyroid starts functioning after 12 weeks. Till then the fetus is entirely dependent upon the maternal supply of T_4 through the placenta, for all neurologic development. TRH crosses the placenta but TSH crosses it very minimally. Maternal total T_4 and T_3 are increased by 18 weeks but free T_4 and T_3 levels are unchanged. Maternal TSH remains normal. Secretion of T_4 and T_3 is 20:1, but biological activity of T_3 is five times more than that of T_4. Level of calcitonin — a thyroid hormone secreted by the parafollicular cells, increases by 20%. Calcitonin protects the maternal skeleton from excess bone loss during pregnancy and lactation. Routine screening of all pregnant women for thyroid function is not recommended by ACOG. However women with high risk factors should have their serum TSH level checked in their first antenatal visit.

ADRENAL CORTEX

MORPHOLOGICAL CHANGES DURING PREGNANCY: There is slight enlargement of the adrenal cortex, particularly the thickness of the zona fasciculata is increased.

PHYSIOLOGICAL CHANGES: There is **significant increase in the serum levels of aldosterone, deoxycorticosterone (DOC), corticosteroid-binding globulin (CBG), ACTH, cortisol and free cortisol**. The increase of CBG (double) is due to high estrogen level. **The levels of total cortisol (metabolically active) nearly doubles**. The level of corticotropin-releasing hormone (CRH) increases markedly. Dehydroepiandrosterone sulfate (DHEAS) levels are decreased. Testosterone and androstenedione levels are slightly raised. The physiological correlation of CRH, ACTH and cortisol concentrations are maintained. Absence of features of Cushing's syndrome with such a high level of free cortisol might indicate that tissue target sites are less sensitive to cortisol (tissue resistance). Cortisol does cross the placenta but not ACTH.

The explanations of physiologic hypercortisolism in pregnancy are: Increased plasma cortisol half-life, delayed plasma clearance by the kidneys and resetting of hypothalamic-pituitary-adrenal feedback mechanism.

PARATHYROID GLAND

MORPHOLOGICAL CHANGES IN NORMAL PREGNANCY: Maternal parathyroid hyperplasia occurs during pregnancy.

PHYSIOLOGICAL CHANGES: The concentration of parathyroid hormone (PTH) is normal during pregnancy. The main functions of PTH are to regulate the renal synthesis of 1, 25 dihydroxy vitamin D_3 and mobilization of calcium from bone. 1, 25 dihydroxy vitamin D_3 enhances calcium reabsorption from the kidneys and small intestines. Calcitonin opposes the action of PTH and vitamin D. **PTH does not cross the placenta but the calcium ions do cross against a concentration gradient**. The marked demand of calcium (25–30 g) by the fetus during the second half of pregnancy is achieved by an increase in maternal 1, 25 dihydroxy vitamin D levels. The absorption and turnover of calcium occur well in advance of fetal skeletal mineralization. There is active transfer of maternal calcium to the fetus. Total serum calcium level during pregnancy falls slightly but ionized calcium levels remain unchanged. Blood levels of $[1,25(OH)_2D_3]$, calcitriol increase.

PANCREAS

PHYSIOLOGICAL CHANGES IN PREGNANCY: During pregnancy there is hypertrophy and hyperplasia of the β cells of islets of Langerhans in maternal pancreas. In pregnancy, there is hyperinsulinemia particularly during third trimester which coincides with the peak concentration of placental hormones. Several antinsulin factors (hPL) and other factors (CRP, IL-6, TNF-α and leptin) decrease insulin sensitivity and increase insulin resistance. Maternal blood glucose level is increased in the second half of pregnancy. This helps increased transfer of glucose from the mother to the fetus through the placenta (see p. 58).

HORMONAL INFLUENCES NECESSARY FOR MAINTENANCE OF LACTATION

The breast is a modified sweat gland. It consists of ducts, alveoli and fibrofatty connective tissue. During puberty there is proliferation of fibrofatty tissue without any change in the alveoli-ductal system.

The endocrine control of lactation can be divided into following stages:

(a) Preparation of breast **(mammogenesis),** (b) synthesis and secretion of milk by breast alveoli **(lactogenesis),** (c) ejection of milk **(galactokinesis)** and (d) maintenance of lactation **(galactopoiesis)**.

The preparation of breast development has been described in page 56. Secretion and ejection of milk and maintenance of lactation are discussed in page 172. The hormones responsible are schematically represented in Figure 6.2.

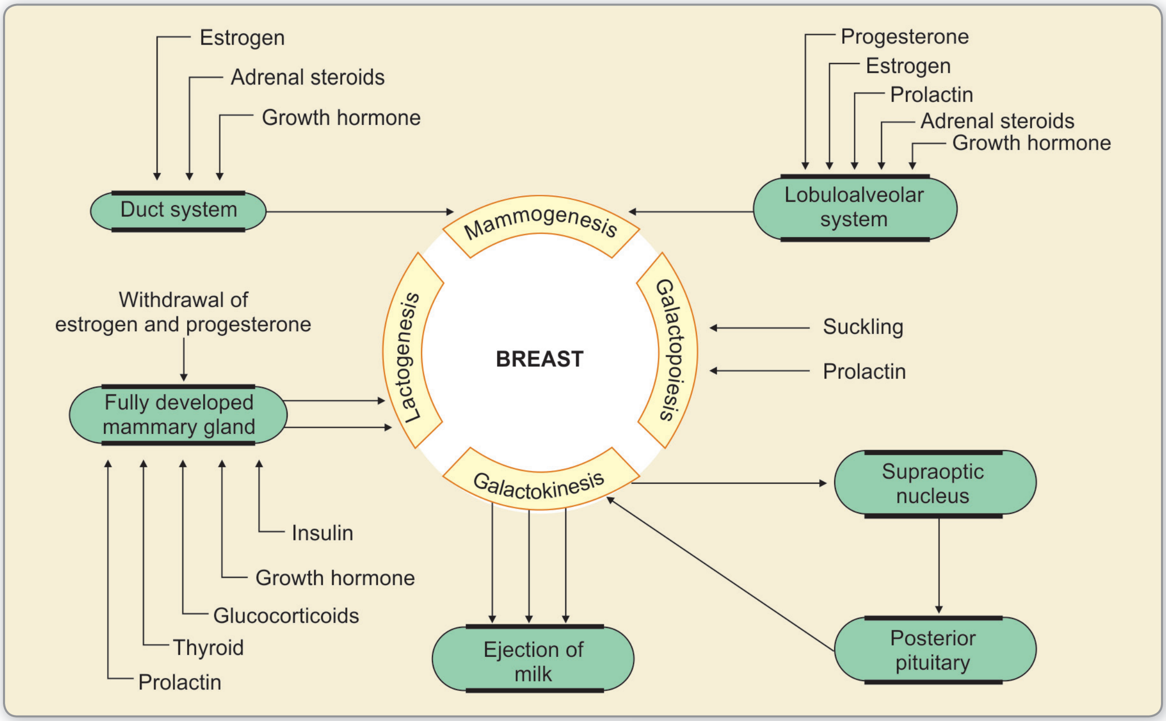

Estrogen
Adrenal steroids
Growth hormone
Duct system
Progesterone
Estrogen
Prolactin
Adrenal steroids
Growth hormone
Lobuloalveolar system
Mammogenesis
Withdrawal of estrogen and progesterone
Fully developed mammary gland
Lactogenesis
BREAST
Galactopoiesis
Suckling
Prolactin
Galactokinesis
Supraoptic nucleus
Insulin
Growth hormone
Glucocorticoids
Thyroid
Prolactin
Ejection of milk
Posterior pituitary

Fig. 6.2: Endocrine glands in relation to lactation

QUESTIONS

1. Discuss in brief the human chorionic gonadotropin (hCG) and mention its functions? (p. 66)
2. Mention the important functions of estrogen and progesterone in pregnancy? (p. 69)
3. Make an outline of the endocrine control of lactation? (p. 72)

Chapter 7

Diagnosis of Pregnancy

The reproductive period of a woman begins at menarche and ends in menopause. It usually extends from 13–45 years. While biological variations may occur in different geographical areas, pregnancy is rare below 12 years and beyond 50 years. **Lina Medina** in Lima, Peru was the youngest one, delivery by cesarean section when she was only 5 years and 7 months old and **the oldest one** at 57 years and 4 months old.

DURATION OF PREGNANCY: The duration of pregnancy has traditionally been calculated by the clinicians in terms of 10 lunar months or 9 calendar months and 7 days or 280 days or 40 weeks, **calculated from the first day of the last menstrual period. This is called menstrual or gestational age.**

But, fertilization usually occurs 14 days prior to the expected missed period and in a previously normal cycle of 28 days duration, it is about 14 days after the first day of the period. Thus, the true gestation period is to be **calculated by subtracting 14 days from 280 days, i.e. 266 days. This is called fertilization or ovulatory age** and is widely used by the embryologist.

FIRST TRIMESTER (FIRST 12 WEEKS)

SUBJECTIVE SYMPTOMS

The following are the presumptive symptoms of early months of pregnancy:

Amenorrhea during the reproductive period in an otherwise healthy individual having previous normal periods, is likely due to pregnancy unless proved otherwise. However, cyclic bleeding may occur up to 12 weeks of pregnancy, until the decidual space is obliterated by the fusion of decidua vera with decidua capsularis. Such bleeding is usually scanty, lasting for a shorter duration than her usual and roughly corresponds with the date of the expected period. **This is termed as placental sign**. This type of bleeding should not be confused with the commonly met pathological bleeding, i.e. threatened abortion. Pregnancy, however, may occur in women who are previously amenorrheic — during lactation and puberty.

Morning sickness (Nausea and vomiting) is inconsistently present in about 70% cases, more often in the first pregnancy than in the subsequent one. **It usually appears soon following the missed period and rarely lasts beyond 16 weeks**. Its intensity varies from nausea on rising from the bed to loss of appetite or even vomiting. But it usually does not affect the health status of the mother (See p. 180).

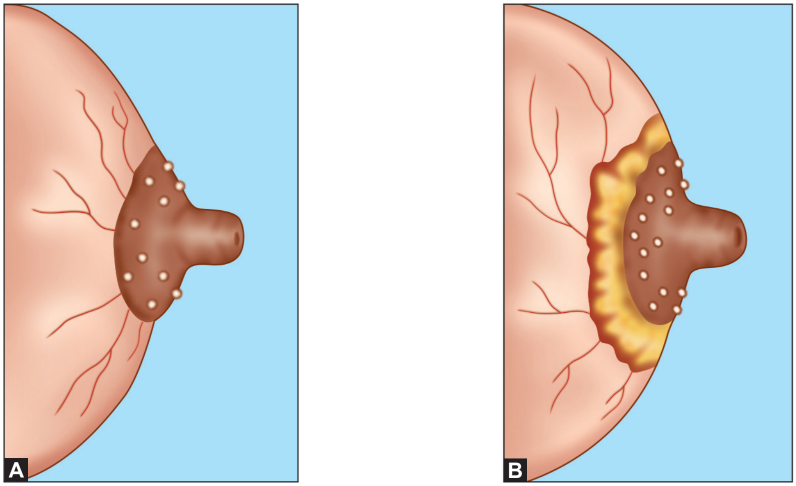
Frequency of micturition is quite troublesome symptom during 8–12th week of pregnancy. It is due to (1) resting of the bulky uterus on the fundus of the bladder because of exaggerated anteverted position of the uterus, (2) congestion of the bladder mucosa and (3) change in **maternal osmoregulation** causing increased thirst and polyuria (see p. 58). As the uterus straightens up after 12th week, the symptom disappears.

Breast discomfort in the form of feeling of fullness and 'pricking sensation' is evident as early as 6–8th week specially in primigravidae.

Fatigue is a frequent symptom which may occur early in pregnancy.

OBJECTIVE SIGNS: ♦ ***Breast changes*** are valuable only in primigravidae, as in multiparae, the breasts are enlarged and often contain milk for years. The breast changes are evident between 6 and 8 weeks. There is enlargement with vascular engorgement evidenced by the delicate veins visible under the skin (Fig. 7.1).

The nipple and the areola (primary) become more pigmented specially in dark women. Montgomery's tubercles are prominent. Thick yellowish secretion (colostrum) can be expressed as early as 12th week.



Figs 7.1A and B: Breast changes during pregnancy; (A) Pronounced pigmentation of the primary areola and nipple; (B) Appearance of secondary areola, development of Montgomery tubercles and increased vascularity

♦ ***Per abdomen*** — Uterus remains a pelvic organ until 12th week, it may be just felt per abdomen as a suprapubic bulge.

♦ ***Pelvic changes*** — The pelvic changes are diverse and appear at different periods. Collectively, these may be informative in arriving at a diagnosis of pregnancy.

- **Jacquemier's or Chadwick's sign:** It is the dusky hue of the vestibule and anterior vaginal wall visible at about 8th week of pregnancy. The discoloration is due to local vascular congestion.
- **Vaginal sign:** (a) Apart from the bluish discoloration of the anterior vaginal wall (b) The walls become softened and (c) Copious non-irritating mucoid discharge appears at 6th week (d) There is increased pulsation, felt through the lateral fornices at 8th week called **Osiander's sign.**
- **Cervical signs:** (a) Cervix becomes soft as early as 6th week **(Goodell's sign)**, a little earlier in multiparae. The pregnant cervix feels like the lips of the mouth, while in the non-pregnant state, like that of tip of the nose. (b) On speculum examination, the bluish discoloration of the cervix is visible. It is due to increased vascularity.
- **Uterine signs:** (a) *Size, shape and consistency* — The uterus is enlarged to the size of hen's egg at 6th week, size of a cricket ball at 8th week and size of a fetal head by 12th week. The pyriform

shape of the non-pregnant uterus becomes globular by 12 weeks. There may be asymmetrical enlargement of the uterus if there is lateral implantation. This is called **Piskacek's sign** where one half is more firm than the other half. As pregnancy advances, symmetry is restored. **The pregnant uterus feels soft and elastic**.

(b) *Hegar's sign:* It is present in two-thirds of cases. **It can be demonstrated between 6 and 10 weeks**, a little earlier in multiparae. **This sign is based on the fact that**: (1) upper part of the body of the uterus is enlarged by the growing fetus (2) lower part of the body is empty and extremely soft and (3) the cervix is comparatively firm. Because of variation in consistency, on bimanual examination (two fingers in the anterior fornix and the abdominal fingers behind the uterus), the abdominal and vaginal fingers seem to appose below the body of the uterus (Fig. 7.2). Examination must be gentle to avoid the risk of abortion.

(c) *Palmer's sign:* Regular and rhythmic uterine contraction can be elicited during bimanual examination as early as 4–8 weeks. Palmer in 1949, first described it and it is a valuable sign when elicited.

To elicit the test, the uterus is cupped between the internal fingers and the external fingers for about 2–3 minutes. During contraction, the uterus becomes firm and well defined but during relaxation, becomes soft and ill defined. While the contraction phase lasts for about 30 seconds, with increasing duration of pregnancy, the relaxation phase increases (Fig. 7.3). **After 10th week, the relaxation phase is so much increased that the test is difficult to perform.**

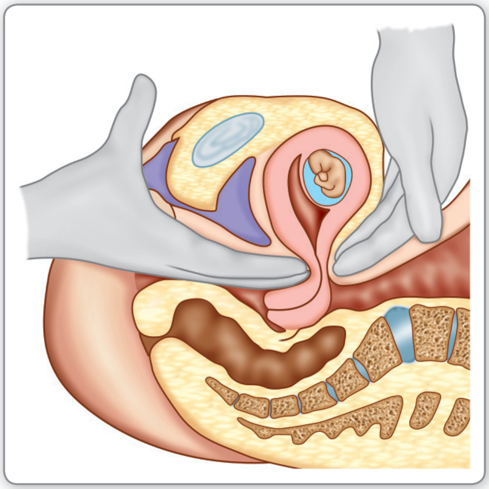


Fig. 7.2: Demonstration of Hegar's sign

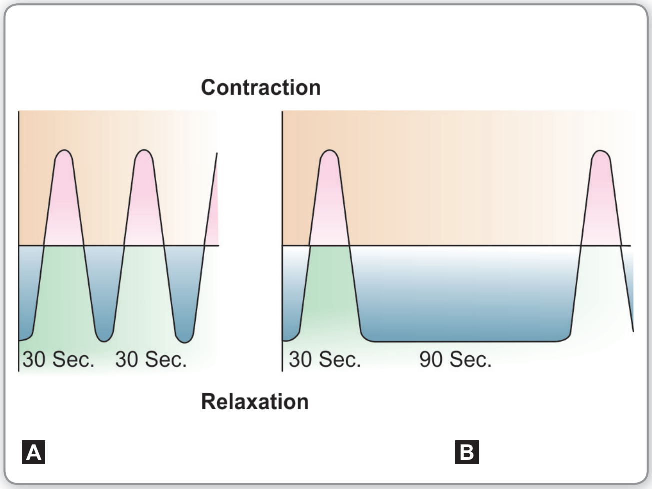


Fig. 7.3: Graphic representation of Palmer's sign — (A) at 4–8th week, (B) at 10th week

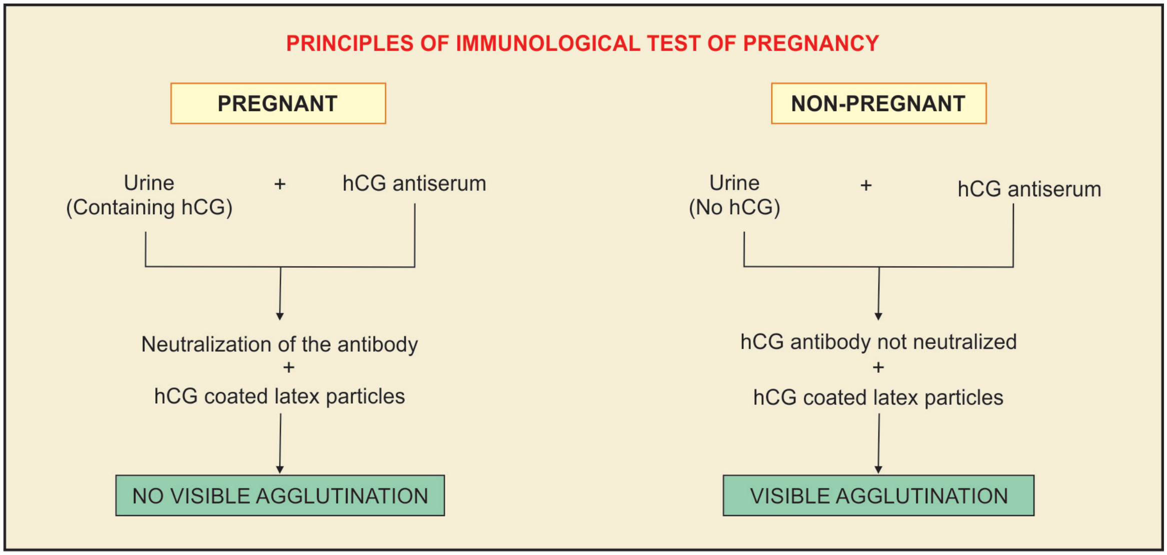
IMMUNOLOGICAL TESTS FOR DIAGNOSIS OF PREGNANCY

Principle: Pregnancy tests depend on detection of the antigen (hCG) present in the maternal urine or serum with antibody either polyclonal or monoclonal available commercially (Table 7.1).

Tests used: A. ***Immunoassays without radioisotopes***

- **Agglutination inhibition tests** — Using latex (LAI). The materials for these tests are supplied in kits containing all the reagents needed to do a test.

Principle of agglutination inhibition tests: One drop of urine is mixed with one drop of a solution that contains hCG antibody. If hCG is not present in the urine sample (e.g. the woman is not pregnant), the antibody remains


PRINCIPLES OF IMMUNOLOGICAL TEST OF PREGNANCY

free. Now one drop of another solution that contains latex particles coated with hCG is added. Agglutination of the latex particles can be observed easily this time. **Therefore, the pregnancy test is negative if there is agglutination.** On the other hand, if hCG were present in the urine sample (e.g. woman was pregnant), it would bind the available antibody. There would be no further agglutination when the solution containing hCG coated latex particles was added. Therefore, **pregnancy test is positive if there is no agglutination** (schematic presentation above).

- **Direct agglutination test (hCG direct test)** — Latex particles coated with anti-hCG monoclonal antibodies are mixed with urine. An agglutination reaction indicates a positive result when the urine sample contains hCG. Absence of agglutination (urine without hCG) indicates a negative one. The sensitivity is 0.2 IU hCG/mL.

- **Enzyme-linked immunosorbent assay (ELISA)** — It is based on one monoclonal antibody that binds the hCG in urine and serum. A second antibody that is linked with enzyme alkaline phosphatase is used to 'sandwich' the bound hCG. It is detected by color change after binding. This is more sensitive and specific. ELISA can detect hCG in serum up to 1–2 mIU/mL and as early as 5 days before the first missed period.

- **Fluoroimmunoassay (FIA)** — It is a highly precise sandwich assay. It uses a second antibody tagged with a fluorescent label. The fluorescence emitted is proportional to the amount of hCG. It can detect hCG as low as 1 mIU/mL. FIA takes 2–3 hours. It is used to detect hCG and for follow up hCG concentrations.

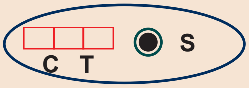
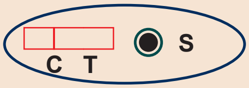
B. *Immunoassays with radioisotopes*

- **Radioimmunoassay (RIA)** — It using I^{125} ido hCG antibodies. It is more sensitive and can detect β subunit of hCG up to 0.002 IU/mL in the serum. It can detect pregnancy as early as 8–9 days after ovulation (day of blastocyst implantation). Radio receptor assay gives highest sensitivity of 0.001 IU/mL in the serum. RIAs are quantitative, so can be used for determining the doubling time of hCG (ectopic pregnancy monitoring). RIAs require 3–4 hours to perform.

- **Immunoradiometric assay (IRMA) —** It uses sandwich principle to detect whole hCG molecule. IRMAs use I_{125} labeled hCG and require only 30 minutes. It can detect hCG as low as 0.05 mIU/mL.

Selection of time: Diagnosis of pregnancy by detecting hCG in maternal serum or urine can be made by 8 to 11 days after conception. The test is not reliable after 12 weeks.

Table 7.1: Summary of Pregnancy Tests (BETA hCG)

Test	Test Sensitivity	Time Taken	Inference	Positive On
Immunological tests (Urine)				
- Agglutination inhibition test (Latex test)	0.5–1 (IU/mL) (Urine)	2 minutes	Absence of agglutination	2 days after missed period
- Direct latex agglutination test	0.2 (IU/mL) (Urine)	2 minutes	Presence of agglutination	2–3 days after missed period
- Two-site sandwich immunoassay (membrane ELISA/ card tests)	30–50 mIU/mL (Urine)	4–5 minutes	Color bands in the control as well as in test window	On the first day of the missed period (28th day of cycle)
- Various kits in card forms are available			+ Pregnant (Card test) 	– Not pregnant (Card test) 
- Enzyme-linked Immunosorbent Assay (ELISA)	1–2 mIU/mL (Serum)	2–4 hours		5 days before the first missed period
Radioimmunoassay				
- (β subunit)	0.002 IU/mL	3–4 hours		25th day of cycle
- Immunoradiometric assay (IRMA)	0.05 mIU/mL (Serum)	30 minutes		8 days after conception

Collection of urine: The patient is advised to collect the first voided urine in the morning in a clean container **(not to wash with soap)**. Kits to perform the test at home are also available.

Other uses of pregnancy tests: Apart from diagnosis of uterine pregnancy, the tests are employed in the diagnosis of ectopic pregnancy (see p. 213), **to monitor pregnancy following in vitro fertilization and embryo transfer and to follow up cases of hydatidiform mole and choriocarcinoma**. Test accuracy ranges from 98.6 – 99%. Non-pregnant level is below 1 mIU/mL.

Limitations: Test accuracy is affected due to presence of (i) hemoglobin (ii) albumin (iii) LH and (iv) immunological diseases.

Advantages: They are advantageous over the biological methods because of their speed, simplicity, accuracy and less cost. **Biological tests** were based on the classic discovery of **Aschheim and Zondek in 1927**. All these tests are of historical interest.

ULTRASONOGRAPHY: Intradecidual gestational sac (GS) is identified as early as 29 to 35 days of gestation.

Fetal viability and gestational age is determined by detecting the following structures by

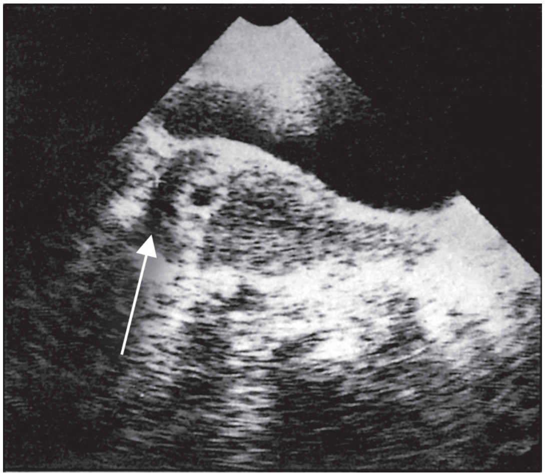


Fig. 7.4 : Gestational ring at 5th week

transvaginal ultrasonography. *Gestational sac and yolk sac* by 5 menstrual weeks (Fig. 7.4); *Fetal pole and cardiac activity* — 6 weeks; *Embryonic movements* by 7 weeks. Fetal gestational age is best determined by measuring the CRL between 7 and 12 weeks (variation ± 5 days). **Doppler effect of ultrasound can pick up the fetal heart rate reliably by 10th week.** The instrument is small, handy and cheap (see Fig. 42.49). The gestational sac (true) must be differentiated from pseudogestational sac (see p. 734).

SECOND TRIMESTER (13–28 WEEKS)

SYMPTOMS: The subjective symptoms — such as nausea, vomiting and frequency of micturition usually subside, while amenorrhea continues. **The new features that appear are**:

- **"*Quickening*" (feeling of life) denotes the perception of active fetal movements by the women**. It is usually felt about the 18th week, about 2 weeks earlier in multiparae. Its appearance is an useful guide to calculate the expected date of delivery with reasonable accuracy (see later in the chapter).
- ***Progressive enlargement*** of the lower abdomen by the growing uterus.

GENERAL EXAMINATION

- ***Chloasma:*** Pigmentation over the forehead and cheek may appear at about 24th week.
- ***Breast changes:*** (a) Breasts are more enlarged with prominent veins under the skin (b) Secondary areola specially demarcated in primigravidae, usually appears at about 20th week (c) Montgomery's tubercles are prominent and extend to the secondary areola (d) Colostrum becomes thick and yellowish by 16th week (e) Variable degree of striae may be visible with advancing weeks.

ABDOMINAL EXAMINATION

Inspection: (1) Linear pigmented zone **(linea nigra)** extending from the symphysis pubis to ensiform cartilage may be visible as early as 20th week (2) **Striae** (both pink and white) of varying degree are visible in the lower abdomen, more towards the flanks (see Fig. 8.5).

Palpation: **Fundal height** is increased with progressive enlargement of the uterus. Approximate duration of pregnancy can be ascertained by noting the height of the uterus in relation to different levels in the abdomen. **The following formula is an useful guide for the purpose (Fig. 7.5)**.

The height of the uterus is midway between the symphysis pubis and umbilicus at 16th week; at the level of umbilicus at 24th week and at the junction of the lower third and upper two-thirds of the distance between the umbilicus and ensiform cartilage at 28th week.

- **The uterus feels soft and elastic** and becomes ovoid in shape.
- **Braxton-Hicks contractions** are evident, the features of which have been mentioned in p. 54.
- **Palpation of fetal parts** can be felt distinctly by 20th week. The findings are of value not only to diagnose pregnancy but also to identify the presentation and position of the fetus in later weeks.
- **Active fetal movements** can be felt at intervals by placing the hand over the uterus as early as 20th

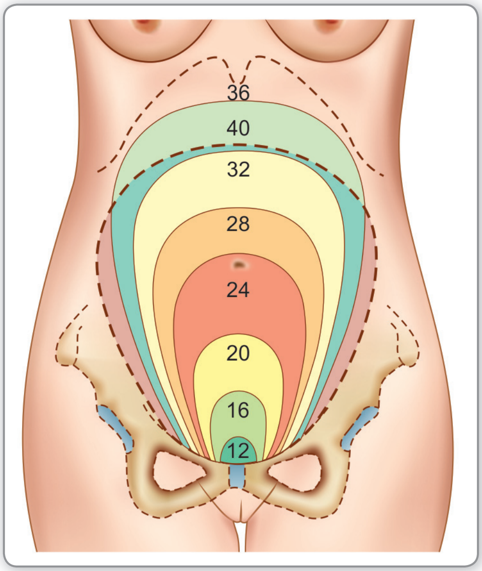


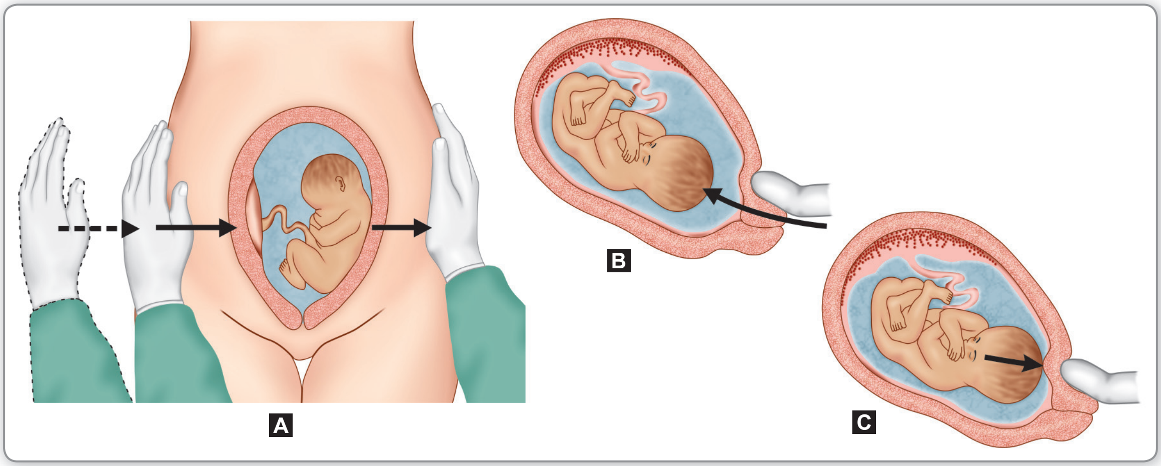
Fig. 7.5: The level of fundus uteri at different weeks. Note the change of uterine shape

week. **It not only gives positive evidence of pregnancy but of a live fetus**. The intensity varies from a faint flutter in early months to stronger movements in later months.

- **External ballottement** is usually elicited as early as 20th week when the fetus is relatively smaller than the volume of the amniotic fluid (Fig. 7.6A). It is difficult to elicit in obese patients and in cases with scanty liquor amnii. It is best elicited in breech presentation with the head at the fundus.

Auscultation

- **Fetal heart sound (FHS) is the most conclusive clinical sign of pregnancy**. With an ordinary stethoscope, it can be detected between 18–20 weeks. The sounds resemble the tick of a watch under a pillow. Its location varies with the position of the fetus. The rate varies from 110–160 beats per minute. **Two other sounds are confused with fetal heart sounds. Those are**:
- ***Uterine souffle*** is a soft blowing and systolic murmur heard low down at the sides of the uterus, best on the left side. **The sound is synchronous with the maternal pulse and is due to increase in blood flow through the dilated uterine vessels**. It can be heard in big uterine fibroid.



Figs 7.6A to C: (A) External ballottement; (B and C) Steps showing how to elicit internal ballottement

- ***Funic or fetal souffle*** is due to rush of blood through the umbilical arteries. **It is a soft, blowing murmur synchronous with the fetal heart sounds.**

VAGINAL EXAMINATION

- ***The bluish discoloration*** of the vulva, vagina and cervix is much more evident, so also softening of the cervix.
- ***Internal ballottement*** can be elicited between 16–28th week (Figs 7.6B and C). The fetus is too small before 16th week and too large to displace after 28th week. However, the test may not be elicited in cases with scanty liquor amnii, or when the fetus is transversely placed.

INVESTIGATIONS (Imaging Studies)

Sonography: Routine sonography at 18–20 weeks permits a detailed survey of fetal anatomy, placental localization and the integrity of the cervical canal. **Gestational age** is determined by measuring the biparietal diameter (BPD), head circumference (HC), abdominal circumference (AC) and femur length (FL). It is most accurate when done between 12 and 20 weeks (variation ± 8 days). BPD is measured at the level of the thalami and cavum septum pellucidum. BPD is measured from outer edge of the skull to the inner edge of the opposite side (see p. 759).

Fetal organ anatomy is surveyed to detect any malformation. ***Fetal viability*** is determined by real-time ultrasound. Absence of fetal cardiac motion confirms fetal death.

Magnetic Resonance Imaging (MRI): MRI can be used for fetal anatomy survey, biometry and evaluation of complex malformations (see p. 739).

Radiologic evidence of fetal skeletal shadow may be visible **as early as 16th week** (see p. 740).

LAST TRIMESTER (29-40 WEEKS)

SYMPTOMS: (1) **Amenorrhea** persists (2) **Enlargement of the abdomen** is progressive which produces some mechanical discomfort to the patient such as palpitation or dyspnea following exertion (3) **Lightening** — At about 38th week, specially in primigravidae, a sense of relief of the pressure symptoms is obtained due to engagement of the presenting part (4) **Frequency of micturition** reappears (5) **Fetal movements** are more pronounced.

SIGNS:

- **Cutaneous changes** are more prominent with increased pigmentation and striae.
- **Uterine shape** is changed from cylindrical to spherical beyond 36th week.
- **Fundal height:** The distance between the umbilicus and the ensiform cartilage is divided into three equal parts. The fundal height corresponds to the junction of the upper and middle third at 32 weeks, up to the level of ensiform cartilage at 36th week and it comes down to 32 week level at 40th week because of engagement of the presenting part. To determine whether the height of the uterus corresponds to 32 weeks or 40 weeks, engagement of the head should be tested. **If the head is floating, it is of 32 weeks pregnancy and if the head *is engaged, it is of 40 weeks pregnancy.***

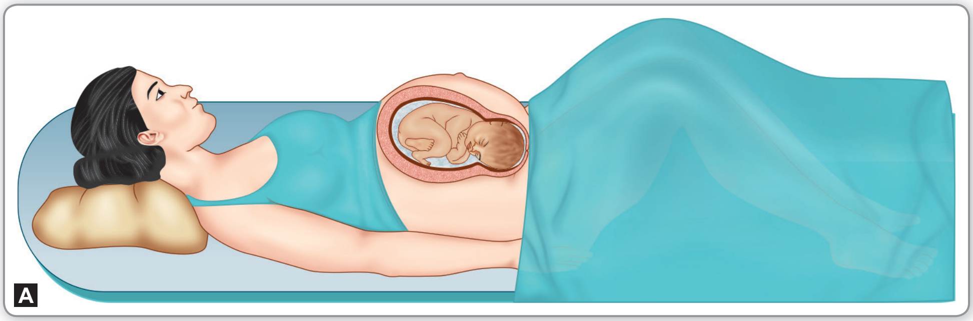


Fig. 7.7A: Position of the woman during obstetric examination

Symphysis fundal height (SFH). The upper border of the fundus is located by the ulnar border of the left hand and this point is marked. The distance between the upper border of the symphysis pubis up to the marked point is measured by a tape in centimeter (Fig. 7.7B). After 24 weeks, the SFH measured in cm corresponds to the number of weeks up to 36 weeks. A variation of ± 2 cm is accepted as normal. Variation beyond the normal range needs further evaluation (see p. 88 and p. 535).

- **Braxton-Hicks** contractions are more evident.
- **Fetal movements** are easily felt.
- **Palpation of the fetal parts** and their identification become much easier. Lie, presentation and position of the fetus are determined.

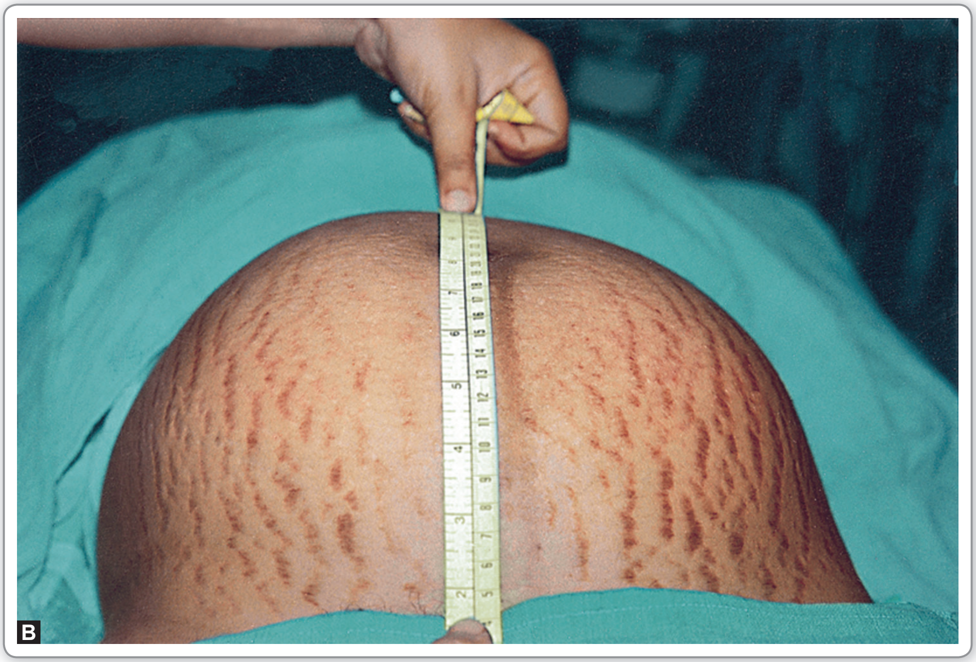


Fig. 7.7B: Symphysis fundal height (SFH)

- **FHS** is heard distinctly in areas corresponding to the presentation and position of the fetus. **FHS may not be audible in cases** of maternal obesity, polyhydramnios, occipitoposterior position and certainly in IUD.
- **Sonography** — gestational age estimation by BPD, HC, AC and FL is less accurate (variation ± 3 weeks). Fetal growth assessment can be made provided accurate dating scan has been done in first or second trimester.

  Fetal AC at the level of the umbilical vein is used to assess gestational age and fetal growth profile (IUGR or macrosomia) (see p. 535). Fetal weight estimation can be done using tables (Hadlock – 1984, p. 74). **Amniotic fluid volume** assessment (see p. 44, 247, 535) is done to detect oligohydramnios (AFI < 5) or polyhydramnios (AFI > 25).

  Placental anatomy: Location (fundus or previa), thickness (placentomegaly in diabetes) or other abnormalities (see p. 737) are noted.

  Other information: Fetal life, number, presentation and organ anatomy as done in the first and second trimester are surveyed again.

DIFFERENTIAL DIAGNOSIS OF PREGNANCY

While the clinical diagnosis of pregnancy at times becomes easy but there are occasions where the diagnosis poses a problem. The enlargement of the uterus caused by pregnancy may have to be differentiated from abdominopelvic swellings, such as **uterine fibroid**, **cystic ovarian tumor**, **encysted tubercular peritonitis**, **hematometra or even distended urinary bladder.** The confusion is accentuated by the presence of amenorrhea for some other reasons. Pregnancy may also coexist with the swellings.

Pseudocyesis (Syn: Phantom, spurious, false pregnancy): **It is a psychological disorder where the woman has the false but firm belief that she is pregnant although no pregnancy exists.** The woman often is infertile who has an intense desire to have a baby. The conspicuous feature is cessation of menstruation. Other confusing manifestations are gradual enlargement of the abdomen because of deposition of fat, secretion from the breasts and intestinal movement, imagining it to be fetal movement. In some cases, the condition continues until eventually spurious labor sets in. Obstetric examination reveals absence of positive signs of pregnancy. Examination with ultrasound and/or immunological tests for pregnancy may be required to negate the diagnosis.

Cystic ovarian tumor: **The diagnostic points are**: (1) The swelling is slow growing, usually takes months to grow (2) Amenorrhea is usually absent (3) It feels cystic or tense cystic (4) Absence of Braxton-Hicks contraction (5) Absence of positive signs of pregnancy (6) Ultrasonography shows absence of fetus.

Fibroid: (1) The tumor is slow growing, often takes years (2) Amenorrhea is absent (3) The feel is firm, more towards hard but may be cystic in cystic degeneration (4) Positive signs of pregnancy are absent (5) Ultrasonography or immunological test for pregnancy gives negative result.

Encysted peritonitis: (1) History of Koch's infection (2) Amenorrhea of longer duration may be present (3) Swelling is ill defined (4) Absence of positive signs of pregnancy (5) Internal examination reveals normal uterus separated from the swelling (6) Ultrasonography — absence of fetus.

Distended urinary bladder: In chronic retention of urine due to retroverted gravid uterus, the distended bladder may be mistaken as ovarian cyst or acute hydramnios. Catheterization of the bladder solves the problem.

SUMMARY OF DIAGNOSIS OF PREGNANCY

- ***Positive or absolute signs:*** (1) Palpation of fetal parts and perception of active fetal movements by the examiner at about 20th week (2) Auscultation of fetal heart sounds (3) Ultrasound evidence of embryo as early as 6th week and later on the fetus (4) Radiological demonstration of the fetal skeleton at 16th week and onwards.
- ***Presumptive symptoms and signs:*** It includes the features mainly appreciated by the women. (1) Amenorrhea (2) Frequency of micturition (3) Morning sickness (4) Fatigue (5) Breast changes (6) Skin changes (7) Quickening.
- ***Probable signs:*** (1) Abdominal enlargement (2) Braxton-Hicks contractions (3) External ballottement (4) Outlining the fetus (5) Changes in the size, shape and consistency of the uterus (6) Jacquemier's sign (7) Softening of the cervix (8) Osiander's sign (9) Internal ballottement (10) Immunological test.

CHRONOLOGICAL APPEARANCE OF SPECIFIC SYMPTOMS AND SIGNS OF PREGNANCY

AT 6–8 WEEKS: *Symptoms* — Amenorrhea, morning sickness, frequency of micturition, fatigue, breast discomfort.

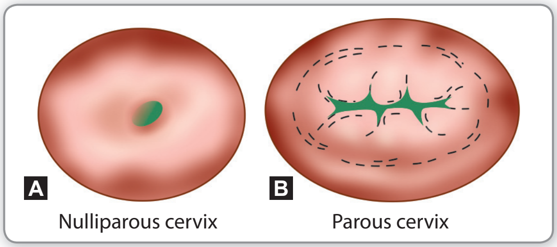
Signs: Breast enlargement, engorged veins visible under the skin; nipples and areola more pigmented. Internal examination reveals — positive Jacquemier's sign, softening of the cervix, bluish discoloration of the cervix and Osiander's sign; positive Hegar's and Palmer's sign. Uterine enlargement varies from hen's egg to medium size orange. Immunological tests will be positive. Sonographic evidence of gestational ring.

AT 16TH WEEK: ***Symptoms*** — Except amenorrhea, all the previous symptoms disappear.

Signs: Breast changes — pigmentation of primary areola and prominence of Montgomery's tubercles, colostrum. Uterus-midway between pubis and umbilicus, Braxton-Hicks contractions, uterine souffle, internal ballottement. X-ray shows fetal shadow. Sonographic diagnosis.

AT 20TH WEEK: ***Symptoms*** — Amenorrhea, quickening (18th week).

Signs: Appearance of secondary areola (20th week), linea nigra (20 weeks), uterus at the level of umbilicus at 24 weeks, Braxton-Hicks contractions, external ballottement (20th week), fetal parts (20 weeks), fetal movements (20 weeks), FHS (20 weeks), internal ballottement (16–28 weeks). X-ray shows fetal shadow. Sonographic diagnosis.



Figs 7.8A and B: Appearance of external os — (A) Nulliparous; (B) Parous

SIGNS OF PREVIOUS CHILD BIRTH

The following are the features which are to be considered in arriving at a diagnosis of having a previous birth.

Breasts become more flabby; nipples are prominent whoever breast-fed their infant; primary areolar pigmentation still remains and so also the white striae.

Abdominal wall is more lax and loose. There may be presence of silvery white striae and linea alba.

Uterine wall is less rigid and the contour of the uterus is broad and round, rather than ovoid.

Perineum is lax and evidence of old scarring from previous perineal laceration or episiotomy may be found.

Introitus is gaping and there is presence of carunculae myrtiformes.

Vagina is more roomy.

Cervix: Nulliparous cervix is conical with a round external os. In parous women, it becomes cylindrical and the external os is a transverse patulous slit and may admit the tip of the finger (Fig. 7.8). However, as a result of operative manipulation even a nulliparous cervix may be torn and resembles a multiparous cervix.

ESTIMATION OF GESTATIONAL AGE AND PREDICTION OF EXPECTED DATE OF DELIVERY

Estimation of gestational age and thereby forecasting the EDD is not only the concern of the individual but it is invaluable in the diagnosis of intrauterine growth retardation of the fetus or management of high risk pregnancy.

Gestational age is about 280 days calculated from the first day of the last normal menstrual period (LMP). Accurate LMP is the most reliable parameter for estimation of gestational age.

But in significant number of cases (20–30%), the patients either fail to remember the LMP or report inaccurately. The matter becomes complicated when the conception occurs during lactation amenorrhea or soon following withdrawal of contraceptive pills (ovulation may be delayed for 4–6 weeks) or in cases with bleeding in early part of pregnancy. **The following parameters either singly or in combination are useful in predicting the gestational age with fair degree of accuracy.**

PATIENT'S STATEMENT

- ***Date of fruitful coitus:*** If the patient can remember the date of the single fruitful coitus with certainty, it is quite reliable to predict the expected date of delivery with accuracy of 50% within 7 days on either side. As previously mentioned, 266 days are to be added to the date of the single fruitful coitus to calculate the expected date. This is not much practicable except when the pregnancy occurs in instances of sudden death or absence of the husband or rape.
- ***Naegele's formula:*** Provided the periods are regular, it is very useful and commonly practiced means to calculate the expected date. Its prediction range is about 50% with 7 days on either side of EDD. **If the interval of cycles is longer, the extra days are to be added and if the interval is shorter, the lesser days are to be subtracted to get the EDD.**
- ***Date of quickening:*** A rough idea about the probable date of delivery can be deduced by adding 22 weeks in primigravidae and 24 weeks in multiparae to the date of quickening.

PREVIOUS RECORDS: **The required weeks are to be added to make it 40 weeks.**

A. ***Clinical:***

- **Size of the uterus** prior to 12 weeks more precisely corresponds with the period of amenorrhea.
- **Palpation of fetal parts** at the earliest by 20th week.
- **Auscultation of FHR** at the earliest by 18–20 weeks using ordinary stethoscope and that using Doppler principle at 10th week.

B. ***Investigation records:*** Investigation records during first half of pregnancy are invaluable.

- **Recording of positive pregnancy test** using immunological principle at first missed period by earliest.

- **Ultrasonographic findings** at the earliest are: (a) Gestation sac — at 5 weeks. (b) Measurement of crown rump length (CRL) detected at 7 weeks, approximates 10 mm; at 10 weeks – 34 mm (CRL in cm + 6.5 = weeks of pregnancy).

OBJECTIVE SIGNS

- **Height of the uterus** above the symphysis pubis in relation to the landmarks on the abdominal wall or SFH (see p. 80).
- **Lightening:** Following the appearance of the features suggestive of lightening (see p. 137), the labor is likely to commence within 3 weeks.
- **Size of the fetus**, change in the uterine shape, volume of liquor amnii, hardening of the skull and girth of the abdomen are of value in assessing the maturity of the fetus specially if the examinations are done by the same person at intervals.
- **Vaginal examination:** If the cervix becomes shorter and dilated, the labor is fairly not far off. But labor may start even with long and closed cervix.

INVESTIGATIONS: Sonography — The following parameters are of use.

- **First trimester** — See p. 81; Crown — Rump Length (CRL) is most accurate. (Variation ± 5 days).
- **Second trimester** by BPD, HC, AC and FL measurement. Most accurate when done between 12 and 20 weeks (variation ± 8 days).
- **Third trimester** — Less reliable, variation ± 16 days.

Gestational age determined by sonographic measurement (in first or second trimester) should be compared with the menstrual age. **In clinical practice, when the difference between the two is less than 10 days, the EDD derived from the LMP is confirmed. When the difference is more, the EDD should be based on ultrasonographic fetal biometry.**

X-ray — Appearance and density of ossification centers in the upper end of the tibia (38–40 weeks) and lower end of femur (36–37 weeks).

ESTIMATION OF FETAL WEIGHT

Approximate prediction of the fetal weight is more important than the mere estimation of the uterine size. This is more important prior to induction of labor or elective cesarean section. The following methods are useful when considered together to have a rough idea about the size of the fetus:

A. **Fetal growth velocity** is maximum (26.9 g/day) over the 32–36 weeks of pregnancy. It declines gradually to 24 g/day over the 36–40 weeks of gestation. Individual fetal growth varies considerably. **Conditional centiles depending on individual fetal growth velocity is thought to be more important.**

B. **Johnson's formula:** Height of the uterus above the symphysis pubis in centimeters minus 12, if the vertex is at or above the level of ischial spines or minus 11, if the vertex is below the level of ischial spines — multiplied by 155 in either case gives the weight of the fetus in grams. **Example** — Height of the uterus above the symphysis pubis = 32 cm and the station of the head is at — 2. The weight of the fetus will be (32–12) × 155 = 3100 g. This is, however, applicable only in vertex presentation. However, the approximate size of the fetus is modified by the amount of liquor amnii and thickness of the abdominal wall.

C. **Sonography:** Fetal weight has been estimated by combining a number of biometric data, e.g. BPD, HC, AC and FL. **Tables (Hadlock, Shepard) are currently in use (computer software)**. Estimated fetal weight likely to be within 10 percent of actual weight.
   - **Shepard's formula:** $\log_{10} \text{EFW (g)} = 1.2508 + (0.166 \times \text{BPD}) + 0.046 \times \text{AC}) - (0.002646 \times \text{AC} \times \text{BPD})$.
   - **Hadlock's formula:** $\log_{10} \text{EFW (g)} = 1.3596 - 0.00386\,(\text{AC} \times \text{FL}) + 0.0064\,(\text{HC}) + 0.00061\,(\text{BPD} \times \text{AC}) + 0.0425\,(\text{AC}) + 0.174\,(\text{FL})$.

QUESTIONS

1. How you can make the diagnosis of pregnancy in the first trimester? (p. 73)
2. Mention the benefits of ultrasonography in the first trimester of pregnancy? (p. 77, p. 734)

Write Short Notes on:

A. Symphysis Fundal Height (p. 80)
B. Braxton Hicks Contractions (p. 54, p. 78, p. 80)

Chapter 8

The Fetus-in-Utero

The fetus lies inside the uterus in a closed sac filled with liquor amnii. It has enough freedom of movement until the later months of pregnancy, when it becomes relatively fixed. Till then, periodic examination is essential to note its lie, presentation, position and attitude. Incidental idea can be gained about the size of the fetus or amount of liquor amnii.

LIE: The lie refers to the relationship of the long axis of the fetus to the long axis of the centralized uterus or maternal spine, the most common lie being longitudinal (99.5%). The lie may be transverse or oblique; sometimes the lie is unstable until labor sets in, when it becomes either longitudinal or transverse (Fig. 8.1 and Table 8.1).

PRESENTATION: The part of the fetus which occupies the lower pole of the uterus (pelvic brim) is called the presentation of the fetus. Accordingly, the presentation may be cephalic (96.5%), podalic (3%) or shoulder and other (0.5%). When more than one part of the fetus present, it is called **compound presentation**.

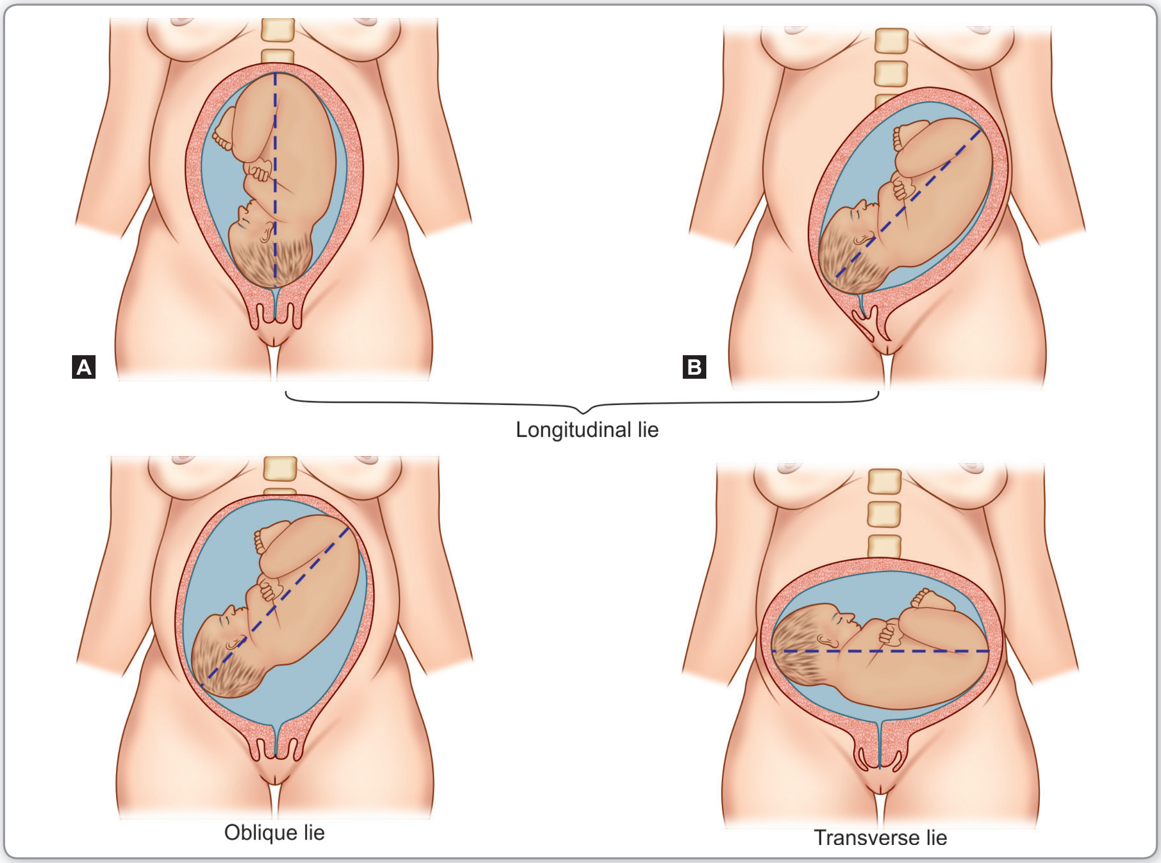
PRESENTING PART: The presenting part is defined as the part of the presentation which overlies the internal os and is felt by the examining finger through the cervical opening. Thus, in cephalic presentation, the presenting part may be vertex (most common), brow or face, depending upon the degree of flexion of the head (Fig. 8.2).

Table 8.1: Preponderance of Lie, presentation and presenting part

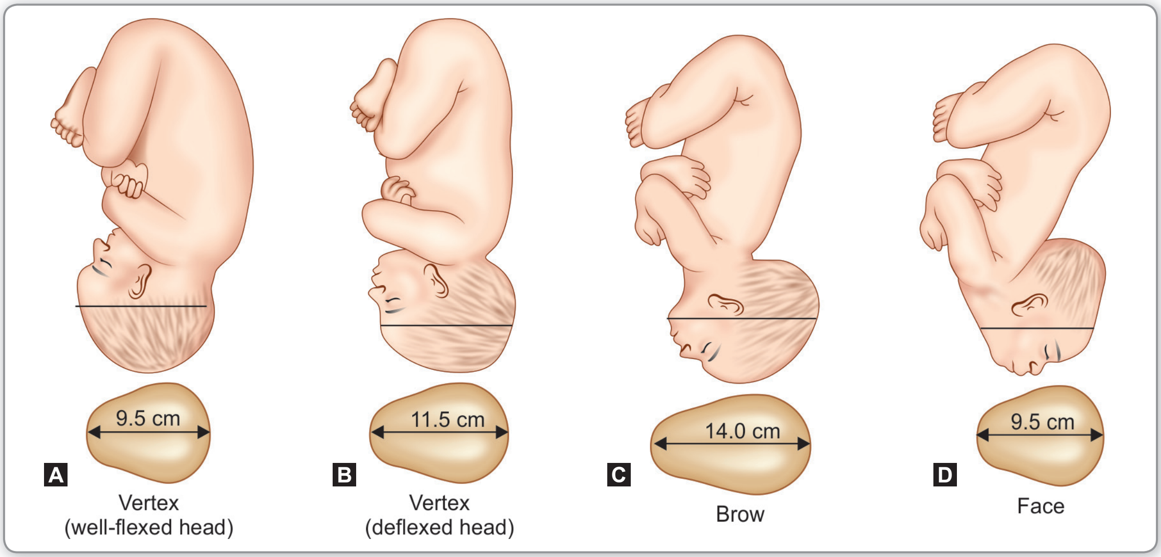
Lie	Presentation	Presenting Part of Cephalic
Longitudinal (99.5%)	Cephalic (96.5%)	Vertex – (96%)
Transverse **Oblique** **Unstable** } (0.5%)	Breech (3%) Shoulder (0.5%) }	Brow Face } (0.5%)

Similarly, the fetal legs in a breech presentation may be flexed (complete breech), extended (frank breech) or a foot may be present (footling). However, the term presentation and presenting part are often used synonymously and expressed more commonly in clinical practice according to the latter definition.

ATTITUDE: The relation of the different parts of the fetus to one another is called attitude of the fetus. The universal attitude is that of flexion. During the later months, the head, trunk and limbs of the fetus maintain the attitude of flexion on all joints and form an ovoid mass that corresponds approximately to the shape of uterine ovoid. The characteristic flexed attitude may be modified by the amount of liquor amnii. There may be exceptions to this universal attitude and extension of the head may occur (deflexed vertex, brow or face presentation, according to the degree of extension), or the legs may become extended in breech. The course of labor in such circumstances may be modified accordingly.


A
B
Longitudinal lie
Oblique lie
Transverse lie

Figs 8.1A and B: Fetal lie. (B), the fetus seems to lie in oblique position in relation to the maternal spine but remains in longitudinal lie in relation to uterine axis. Correction of the uterine obliquity rectifies apparent oblique lie of the fetus (A)


9.5 cm
A
Vertex
(well-flexed head)
11.5 cm
B
Vertex
(deflexed head)
14.0 cm
C
Brow
9.5 cm
D
Face

Figs 8.2A to D: Varieties of cephalic presentations in different attitude

DENOMINATOR: It is an arbitrary bony fixed point on the presenting part which comes in relation with the various quadrants of the maternal pelvis. **The following are the denominators of the different presentations**—occiput in vertex, mentum (chin) in face, frontal eminence in brow, sacrum in breech and acromion in shoulder.

POSITION: It is the relation of the denominator to the different quadrants of the pelvis. For descriptive purpose, the pelvis is divided into equal segments of 45° to place the denominator in each segment. Thus, theoretically, there are 8 positions with each presenting part (Fig. 8.3).

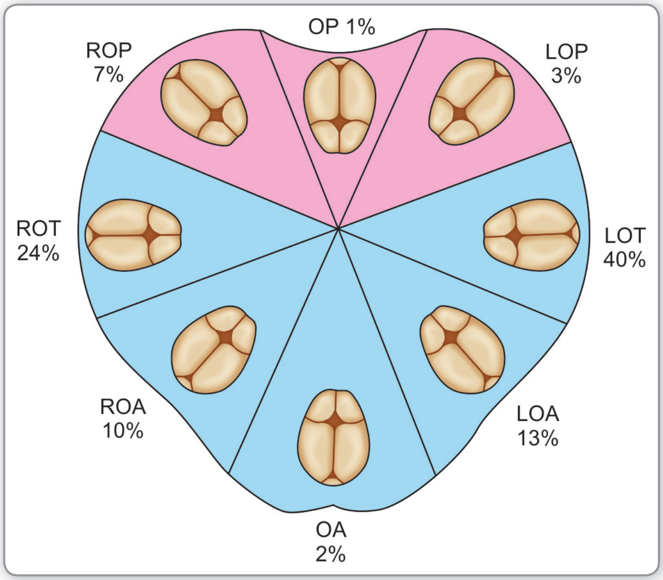


Fig. 8.3: The position and relative frequency of the vertex at the onset of labor

Anterior, posterior, right or left position is referred in relation to the maternal pelvis, with the mother in erect position. However, some have retained the conventional description of four vertex positions. **Vertex occupying the left anterior quadrant of the pelvis is the most common one** and is called left occipitoanterior (LOA). This is the first vertex position. Similarly, right occipitoanterior (ROA) is the second vertex; right occipitoposterior (ROP) third vertex and left occipitoposterior (LOP) is the fourth vertex position.

CAUSES OF PREPONDERANCE OF LONGITUDINAL LIE AND CEPHALIC PRESENTATION

The fetus in the attitude of flexion assumes a shape of an ovoid with its long vertico-podalic axis measuring about 25 cm (10") at term (Fig. 8.4).

The fetus accommodates comfortably along the long axis of the ovoid shape of the uterine cavity at term. Hence, there is preponderance of longitudinal lie.

The cephalic presentation, being the absolute majority amongst the longitudinal lie, can be explained by: (1) Gravitation—the head being heavier comes down to the bottom. (2) Adaptation — the smallest circumference of the flexed head is about 27.5 cm (11") and the circumference of the breech with both thighs flexed is about 32.5 cm (13"). Thus the cephalic and the podalic poles can be comfortably accommodated in the narrow lower pole and the wider fundal area of the uterus respectively.

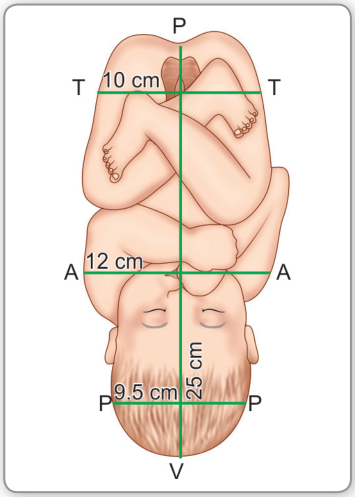


Fig. 8.4: Diagrammatic representation of a fetus in flexed attitude
V–P—Vertico-podalic diameter;
A–A—Bisacromial diameter;
T–T—Bitrochanteric diameter
P–P—Biparietal diameter

METHODS OF OBSTETRICAL EXAMINATION

ABDOMINAL EXAMINATION: A thorough and systemic abdominal examination beyond 28 weeks of pregnancy can reasonably diagnose the lie, presentation, position and the attitude of the fetus. It is not unlikely that the lie and presentation of the fetus might change, especially in association with excess liquor amnii and hence periodic check up is essential.

***Preliminaries:* Verbal consent for examination is taken.** The patient is asked to evacuate the bladder. She is then made to lie in dorsal position with the thighs slightly flexed (see Fig. 7.7A, p. 80). Abdomen is fully exposed. The examiner stands on the right side of the patient.

Inspection: To note (1) whether the uterine ovoid is longitudinal or transverse or oblique (2) contour of the uterus—fundal notching, convex or flattened anterior wall, cylindrical or spherical shape (3) undue enlargement of the uterus (4) skin condition of abdomen for evidence of ringworm or scabies and (5) any incisional scar mark on the abdomen.

Palpation: **Height of the uterus**: The uterus is to be centralized, if it is deviated. The ulnar border of the left hand is placed on the upper most level of the fundus and an approximate duration of pregnancy is ascertained in terms of weeks of gestation (Fig. 8.5). Alternatively, the SFH can be measured with a tape (see Fig. 7.7B).

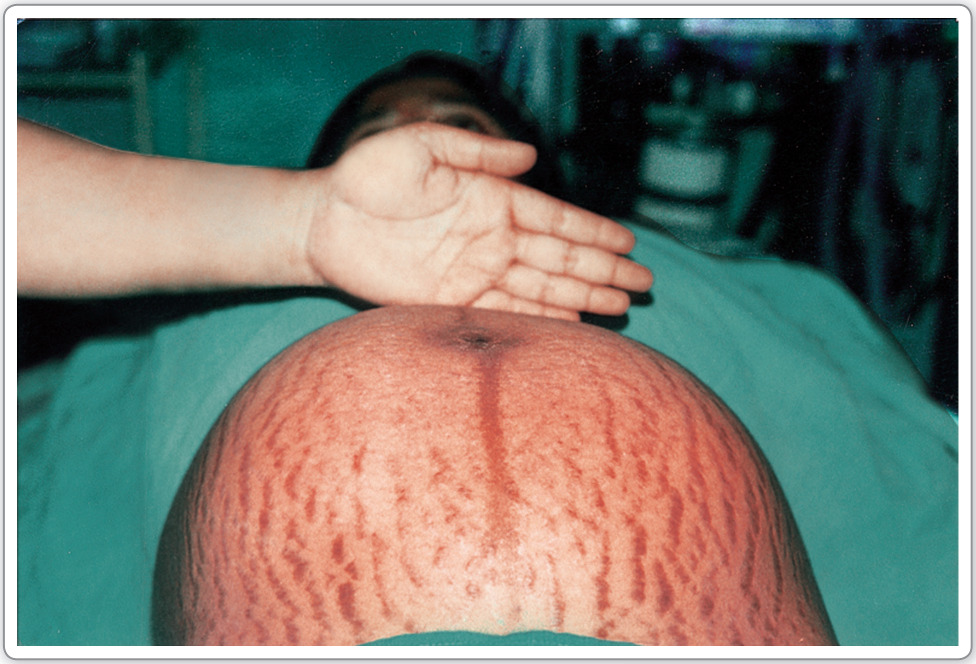


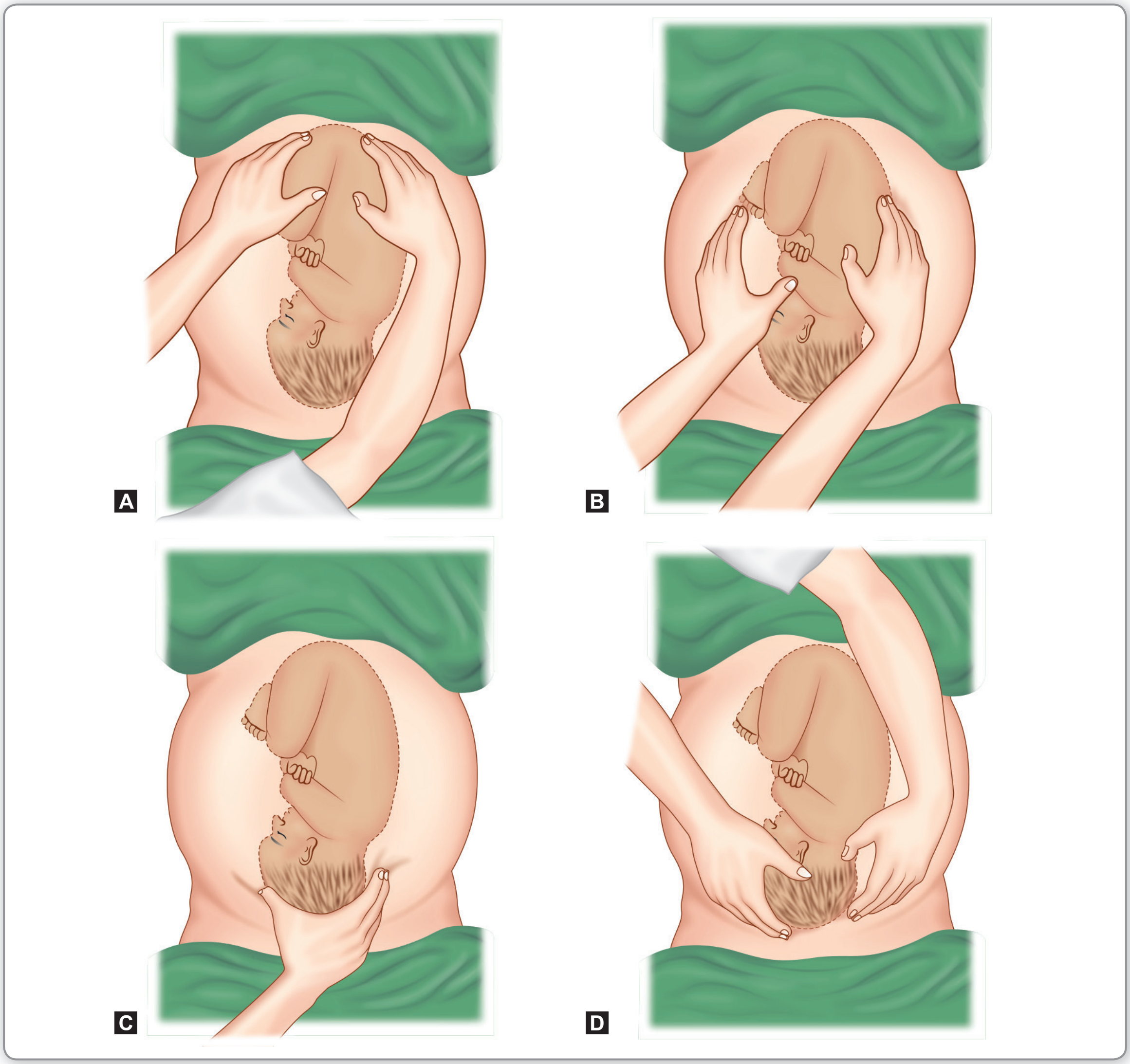
Fig. 8.5: To note the height of the uterus. Linea nigra and striae gravidarum are also visible

There are conditions where the height of the uterus may not correspond with the period of amenorrhea. **The conditions where the height of the uterus is more than the period of amenorrhea are**: (1) mistaken date of the last menstrual period (2) twins (3) polyhydramnios (4) big baby (5) pelvic tumors—ovarian or fibroid (6) hydatidiform mole and (7) concealed accidental hemorrhage. **The condition where the height of the uterus is less than the period of amenorrhea are:** (1) mistaken date of the last menstrual period (2) scanty liquor amnii (3) fetal growth retardation and (4) intrauterine fetal death.

Obstetric grips (Leopold maneuvers) (Fig. 8.6): Palpation should be conducted with utmost gentleness. Clumsy and purposeless palpation is not only uniformative but may cause undue uterine irritability. **During Braxton-Hicks contraction or uterine contraction in labor, palpation should be suspended**.

(i) **Fundal grip:** The palpation is done facing the patient's face. The whole of the fundal area is palpated using both hands laid flat on it to find out which pole of the fetus is lying in the fundus: (a) broad, soft and irregular mass suggestive of breech, or (b) smooth, hard and globular mass suggestive of head. In transverse lie, neither of the fetal poles are palpated in the fundal area.

(ii) **Lateral or umbilical grip:** The palpation is done facing the patient's face. The hands are to be placed flat on either side of the umbilicus to palpate one after the other, the sides and front of the uterus **to find out the position of the back, limbs and the anterior shoulder**. The back is suggested by smooth curved and resistant feel. The 'limb side' is comparatively empty and there are small knob like irregular parts. After the identification of the back, it is essential to note its position whether placed anteriorly or towards the flank or placed transversely. Similarly, the disposition of the small parts, whether placed to one side or placed anteriorly occupying both the sides, is to be noted. **The position of the anterior shoulder is to be sought for**. It forms a well-marked prominence in the lower part of the uterus above the head. It may be placed near the midline or well away from the midline.



Figs 8.6A to D: Obstetric grips (**Leopold maneuvers**): (A) Fundal grip (**first Leopold**); (B) Lateral grip (**second Leopold**); (C) Pawlik's grip (**third Leopold**); (D) Pelvic grip (**fourth Leopold**)

(iii) **Pawlik's grip (Third Leopold):** The examination is done facing toward the patient's face. The overstretched thumb and four fingers of the right hand are placed over the lower pole of the uterus keeping the ulnar border of the palm on the upper border of the symphysis pubis. When the fingers and the thumb are approximated, the presenting part is grasped distinctly (if not engaged) and also the mobility from side to side is tested. **In transverse lie, Pawlik's grip is empty**.

(iv) **Pelvic grip (Fourth Leopold): The examination is done facing the patient's feet**. Four fingers of both the hands are placed on either side of the midline in the lower pole of the uterus and parallel to the inguinal ligament. The fingers are pressed downward and backward in a manner of approximation of finger tips to palpate the part occupying the lower pole of the uterus (presentation). **If it is head, the characteristics to note are**: (1) precise presenting area (2) attitude and (3) engagement.

To ascertain the presenting part, the greater mass of the head (cephalic prominence) is carefully palpated and its relation to the limbs and back is noted. **The attitude** of the head is inferred by noting the relative position of the sincipital and occipital poles (Fig. 8.8). **The engagement** is ascertained noting the presence or absence of the sincipital and occipital poles or whether there is convergence or divergence of the finger tips during palpation (Fig. 8.9). **This pelvic grip using both the hands is favored** as it is most comfortable for the woman and gives most information.

Auscultation: Auscultation of distinct fetal heart sounds (FHS) not only helps in the diagnosis of a live baby, but its location of maximum intensity can resolve doubt about the presentation of the fetus (Fig. 8.7A).

The fetal heart sounds are best audible through the back (left scapular region) in vertex and breech presentation, where the convex portion of the back is in contact with the uterine wall. However, in face presentation, the heart sounds are heard through the fetal chest.

As a rule, the maximum intensity of the FHS is below the umbilicus in cephalic presentation and around the umbilicus in breech. In different positions of the vertex, the location of the FHS depends on the position of the back and the degree of descent of the head. In occipitoanterior position, the FHS is located in the middle of the spinoumbilical line of the same side. In occipitolateral position, it is heard

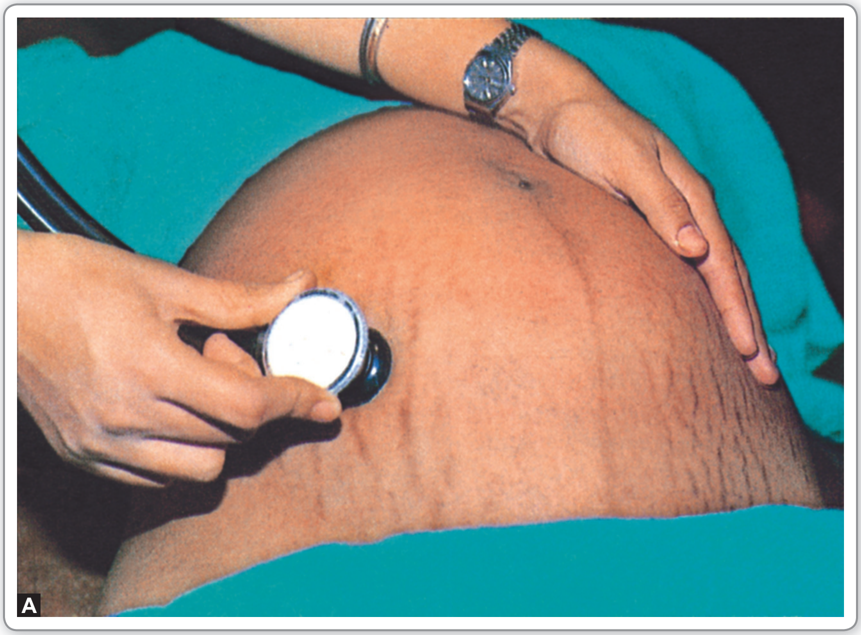


Fig. 8.7A: Auscultation of distinct fetal heart sounds (FHS)

more laterally and in occipitoposterior position, well back toward the mother's flank on the same side. In left occipitoposterior position, it is most difficult to locate the FHS (Fig. 8.7B).

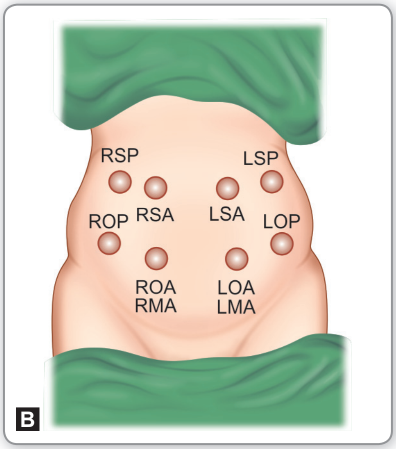


Fig. 8.7B: Location of the fetal heart sound in different presentation of position of the fetus

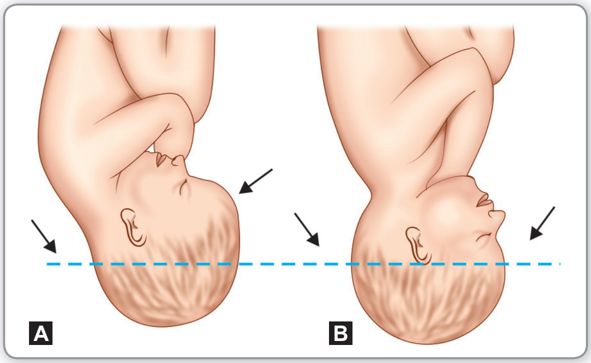
INTERNAL EXAMINATION: The diagnosis of the presentation and position of the fetus may not be accurate by internal examination during pregnancy when the cervix remains closed. However, during labor, accurate information may be obtained by palpation of the sagittal suture and fontanelles through the open cervix. Stress for strict aseptic precautions during vaginal examination needs no emphasis.

ULTRASONOGRAPHY: The diagnosis of the lie, presentation and position may be difficult in the presence of marked obesity, irritable uterus, excessive liquor amnii and deeply engaged head, especially in primigravidae. Ultrasonography can locate the head and the body (see p. 732). Alternatively, straight X-ray may be needed to arrive at a diagnosis in such cases.

INFERENCES: As the vertex is the most common presentation, the relevant information in relation to the vertex is only mentioned.

Lie: The longitudinal lie is evident from: (1) Longitudinal uterine ovoid on inspection (2) the poles of the fetal ovoid—cephalic and podalic are placed, one at the lower and the other at the upper part of the uterine cavity, as evident from the fundal and first pelvic grips.

Presentation: The cephalic presentation is evident from the first pelvic grip—smooth, hard and globular mass.



Figs 8.8A and B: Relative position of the sincipital and the occipital pole as felt in first pelvic grip: (A) Well flexed (B) Deflexed

Attitude: From the first pelvic grip, the relative positions of the sincipital and occipital poles are determined. **In well-flexed head**, the sincipital pole is placed at a higher level but **in deflexed state**, both the poles remain at a same level (Fig. 8.8).

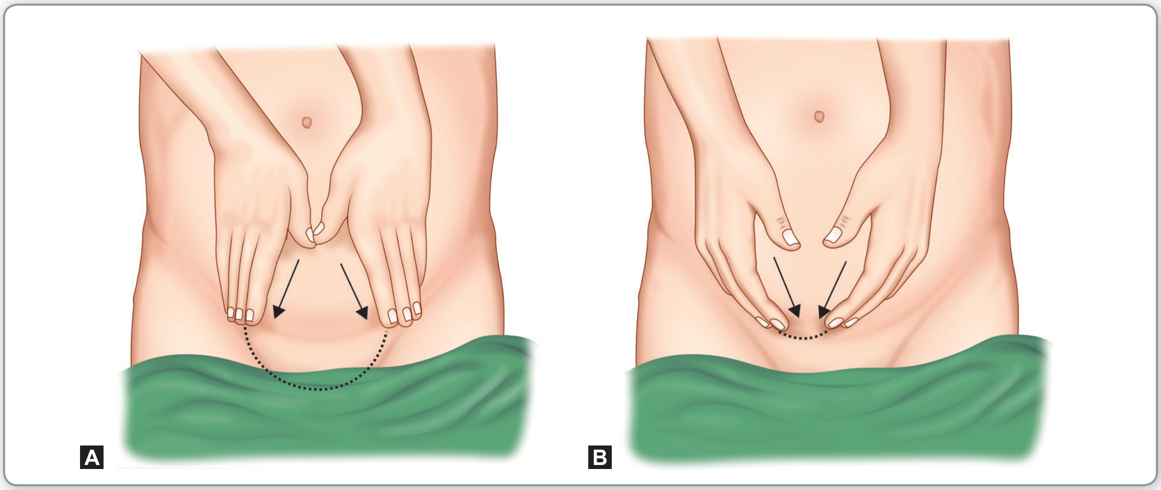
Presenting part: Vertex is diagnosed from the first pelvic grip. The cephalic prominence, **being the sinciput, is placed on the same side toward which limbs lie**.

Position: The occipitoanterior position is diagnosed by: (1) Inspection—convexity of the uterine contour. (2) Lateral grip—(a) The back is placed not far from the midline to the same side of the occiput (b) The anterior shoulder is near the midline (3) Auscultation—maximum intensity of the FHS is close to the spinoumbilical line on the same side of the back.

Right or left position is to be determined by: (1) Position of the back (2) Position of the occiput and (3) Location of the FHS.

ENGAGEMENT: When the greatest horizontal plane, the biparietal, has passed the plane of the pelvic brim, the head is said to be engaged.

Diagnosis: First pelvic grip: (1) Both the poles (sinciput and occiput) are not felt per abdomen. However, the sincipital pole can be felt with difficulty even though the head is engaged (2) Divergence of the examining fingers of both the hands while trying to push downward on the lower abdomen (Fig. 8.9).



Figs 8.9A and B: Abdominal palpation to determine engagement of the head: (A) Divergence of fingers—engaged head, (B) Convergence fingers—not engaged

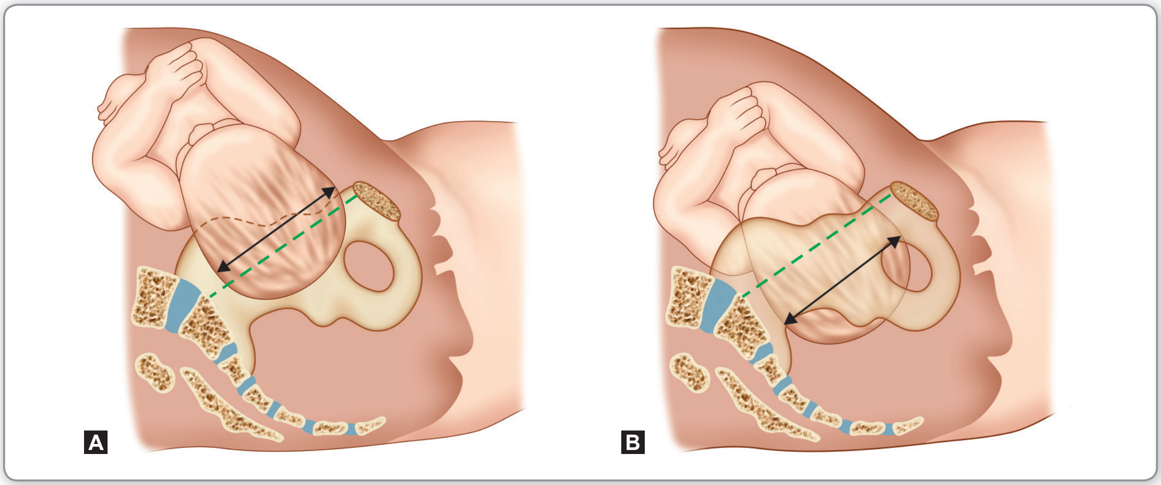
Convergence of the fingers while palpating the lateral aspects of the fetal head indicates that the head is not yet engaged.

Vaginal Examination: **Lower pole of the unmolded head** is usually at or below the level of the ischial spines. The distance between the pelvic inlet and ischial spines is about 5 cm. But the distance between the biparietal plane of the unmolded head to the vertex is about 3 cm (Fig. 8.10).

Imaging: Lateral view sonography is confirmatory.

Significance: Engagement of the head always excludes disproportion at the brim, as the head is the best pelvimeter.

The traditional concept that in primigravidae, the engagement occurs by 38 weeks is not corroborative in clinical practice. In majority, the engagement occurs between 38–42 weeks or even during first stage of labor. In multigravidae, however, the engagement occurs late in first stage of labor



Figs 8.10A and B: The relationship of the biparietal diameter to the pelvic brim and that of lower pole of the head to the ischial spines in: (A) Non-engaged head; (B) Engaged head

after the rupture of the membranes. **However, if the head fails to engage in primigravidae even at 38th week, the causes are to be sought for**. **Common causes are**: (1) Deflexed head bringing the larger diameter to engage (2) Cephalopelvic disproportion or big head or a combination of both (3) Polyhydramnios (4) Poor formation or yielding of lower uterine segment—preventing the head to sink into the pelvis, (5) Hydrocephalus (6) Placenta previa (7) Pelvic tumors—ovarian or fibroid (8) High pelvic inclination (9) Functional — when no cause can be detected (20%).

Fixed head: The word 'fixed' should not be used to designate an engaged head. Whereas, an engaged head is fixed but conversely, the fixed head is not necessarily engaged. When an egg is placed on the egg cup, it remains fixed yet the maximum diameter does not pass through the rim (Fig. 8.11).

Similarly, the head may be fixed to the brim but that does not mean that the maximum diameter of the head (biparietal) will pass through the brim. As such, the use of the term 'fixed' should be abandoned. Similarly, the term 'engaging' should be withheld. **A clear statement is to be made as to whether the head is engaged or not**.

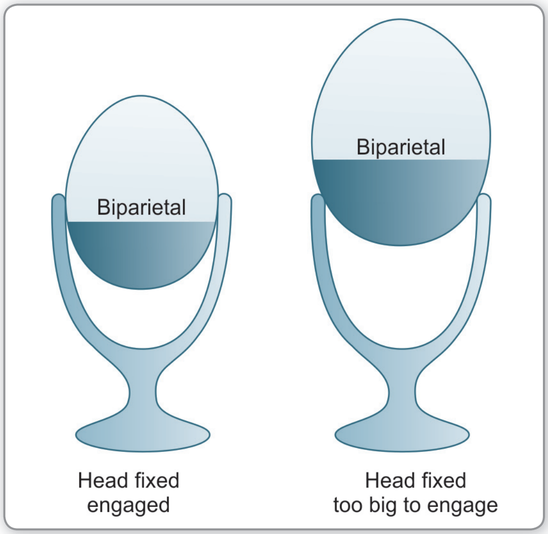


Fig. 8.11: Diagrammatic representation showing the difference between an engaged and a fixed head by use of egg cups and eggs

QUESTIONS

1. What is lie? What are the different types of lie? (p. 85)

Write Short Notes on:

A. Engagement (p. 91)
B. Presentation (p. 85)

Chapter 9

Fetal Skull and Maternal Pelvis

FETAL SKULL

Fetal skull is to some extent compressible and made mainly of thin pliable tabular (flat) bones forming the vault. This is anchored to the rigid and incompressible bones at the base of the skull.

AREAS OF SKULL: The skull is arbitrarily divided into several zones of obstetrical importance (Fig. 9.1). These are:

- **Vertex :** It is a quadrangular area bounded anteriorly by the bregma and coronal sutures behind by the lambda and lambdoid sutures and laterally by lines passing through the parietal eminences.
- **Brow :** It is an area bounded on one side by the anterior fontanel and coronal sutures and on the other side by the root of the nose and supraorbital ridges of either side.

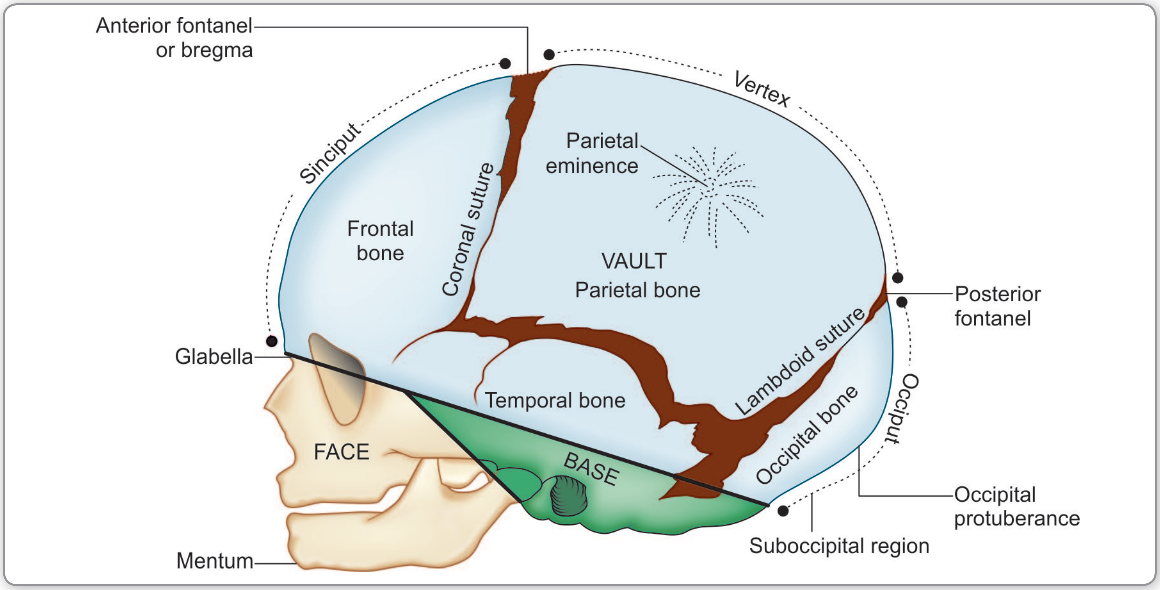

Anterior fontanel or bregma
Vertex
Sinciput
Parietal eminence
Frontal bone
Coronal suture
VAULT
Parietal bone
Posterior fontanel
Glabella
Lambdoid suture
Temporal bone
Occiput
Occipital bone
FACE
BASE
Occipital protuberance
Suboccipital region
Mentum

Fig. 9.1: Fetal skull showing different regions and landmarks of obstetrical significance

- **Face** : It is an area bounded on one side by root of the nose and supraorbital ridges and on the other, by the junction of the floor of the mouth with neck.

Sinciput is the area lying in front of the anterior fontanel and corresponds to the area of brow and the occiput is limited to the occipital bone.

Flat bones of the vault are united together by non-ossified membranes attached to the margins of the bones. These are called sutures and fontanels. Of the many sutures and fontanels, the following are of obstetric significance.

SUTURES: (Figs 9.1 and 9.2)

- **The sagittal or longitudinal suture** lies between two parietal bones.
- **The coronal sutures** run between parietal and frontal bones on either side.
- **The frontal suture** lies between two frontal bones.
- **The lambdoid sutures** separate the occipital bone and the two parietal bones.

Importance: (1) It permits gliding movement of one bone over the other during molding of the head, a phenomenon of significance while the head passes through the pelvis during labor. (2) Digital palpation of sagittal suture during internal examination in labor gives an idea of the manner of engagement of the head (asynclitism or synclitism), degree of internal rotation of the head and degree of molding of the head.

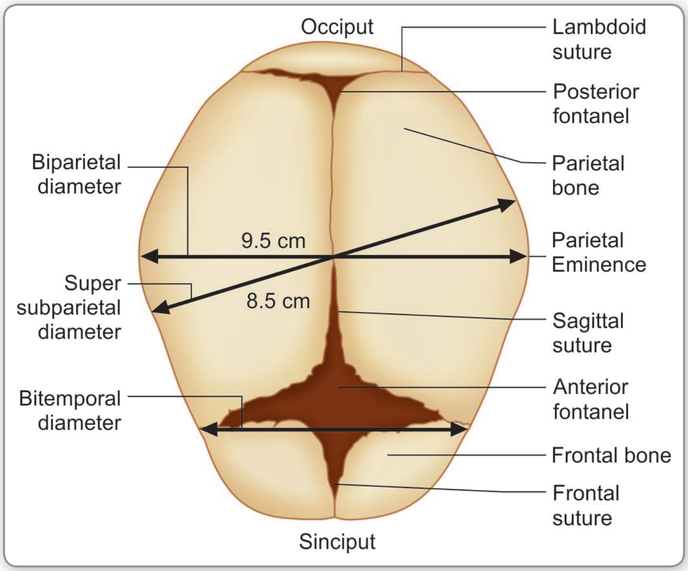


Fig. 9.2: Fetal skull showing important sutures, fontanels and diameters of obstetric significance

FONTANELS: Wide gap in the suture line is called fontanel. Of the many fontanels (6 in number), two are of obstetric significance: (1) Anterior fontanel or bregma and (2) Posterior fontanel or lambda.

Anterior fontanel (Fig. 9.2): **It is formed by** joining of the four sutures in the midplane. The sutures are anteriorly frontal, posteriorly sagittal and on either side, coronal. **The shape** is like a diamond. Its anteroposterior and transverse diameters measure approximately 3 cm each. **The floor** is formed by a membrane and it becomes ossified 18 months after birth. It becomes pathological, if it fails to ossify even after 24 months.

Importance:

- Its palpation through internal examination denotes the degree of flexion of the head.
- It facilitates molding of the head.
- As it remains membranous long after birth, it helps in accommodating the marked brain growth; the brain becoming almost double its size during the first year of life.
- Palpation of the floor reflects intracranial status—depressed in dehydration, elevated in raised intracranial tension.
- Collection of blood and exchange transfusion, on rare occasion, can be performed through it via the superior longitudinal sinus.
- Cerebrospinal fluid can be drawn, although rarely, through the angle of the anterior fontanel from the lateral ventricle.

Posterior fontanel: **It is formed by** junction of three suture lines — sagittal suture anteriorly and lambdoid suture on either side. **It is triangular in shape and measures** about 1.2 × 1.2 cm (1/2" × 1/2"). **Its floor** is membranous but becomes bony at term. Thus, truly its nomenclature as fontanel is misnomer. It denotes the position of the head in relation to maternal pelvis.

Sagittal fontanel: It is inconsistent in its presence. When present, it is situated on the sagittal suture at the junction of anterior two-third and posterior one-third. It has got no clinical importance.

DIAMETERS OF SKULL (Fig. 9.3): The engaging diameter of the fetal skull depends on the degree of flexion present. **The anteroposterior diameters of the head which may engage are:**

Table 9.1: Important Diameters of the Skull

Diameters	Measurement in Cm (inches)	Attitude of the Head	Presentation
Suboccipitobregmatic — extends from the nape of the neck to the center of the bregma	9.5 cm (3 ¾")	Complete flexion	Vertex
Suboccipito-frontal — extends from the nape of the neck to the anterior end of the anterior fontanel or center of the sinciput	10 cm (4")	Incomplete flexion	Vertex
Occipitofrontal — extends from the occipital eminence to the root of the nose (Glabella)	11.5 cm (4 ½")	Marked deflexion	Vertex
Mento-vertical — extends from the midpoint of the chin to the highest point on the sagittal suture	14 cm (5 ½")	Partial extension	Brow
Submentovertical — extends from junction of floor of the mouth and neck to the highest point on the sagittal suture	11.5 cm (4 ½")	Incomplete extension	Face
Submentobregmatic — extends from junction of floor of the mouth and neck to the center of the bregma	9.5 cm (3 ¾")	Complete extension	Face

The transverse diameters which are concerned in the mechanism of labor are (Fig. 9.2 and Table 9.1):

- ***Biparietal diameter***—9.5 cm (3 ¾"): It extends between two parietal eminences. Whatever may be the position of the head, this diameter nearly always engages.
- ***Super-subparietal***—8.5 cm (3 ½"): It extends from a point placed below one parietal eminence to a point placed above the other parietal eminence of the opposite side.
- ***Bitemporal diameter***—8 cm (3 ¼"): It is the distance between the antero-inferior ends of the coronal suture.
- ***Bimastoid diameter*** — 7.5 cm (3"): It is the distance between the tips of the mastoid processes. The diameter is incompressible and it is impossible to reduce the length of the bimastoid diameter by obstetrical operation.

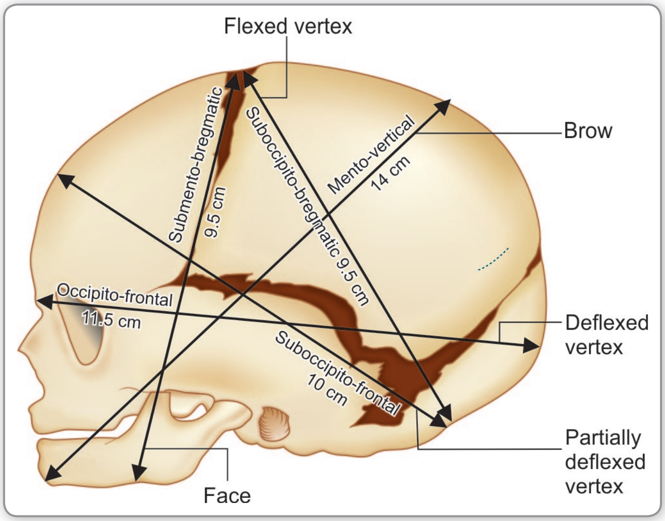


Fig. 9.3: The important landmarks of fetal skull

CIRCUMFERENCES: Circumference of the plane of the diameter of engagement differs according to the attitude of the head (Table 9.2).

Table 9.2: Circumferences of the Head in Different Attitudes

Attitude of the Head	Plane of Engagement	Circumference
Complete flexion	Biparietal-suboccipitobregmatic (almost round shape)	27.5 cm (11")
Deflexed	Biparietal-occipito-frontal (oval shape)	34 cm (13 ½")
Incomplete extension	Biparietal-mento-vertical (bigger oval shape)	37.5 cm (15")
Complete extension	Biparietal-submentobregmatic (almost round shape)	27.5 cm (11")

** Conversion of centimeters into inches is approximate*

MOLDING: It is the alteration of the shape of the fore-coming head while passing through the resistant birth passage during labor. There is, however, very little alteration in size of the head, as volume of the content inside the skull is incompressible although small amount of cerebrospinal fluid and blood escape out in the process. During normal delivery, an alteration of 4 mm in skull diameter commonly occurs.

Mechanism: There is compression of the engaging diameter of the head with corresponding elongation of the diameter at right angle to it (Fig. 9.4).

Thus, in well flexed head of the anterior vertex presentation, the engaging suboccipitobregmatic diameter is compressed with elongation of the head in mento-vertical diameter which is at right angle to suboccipitobregmatic (Fig. 9.5).

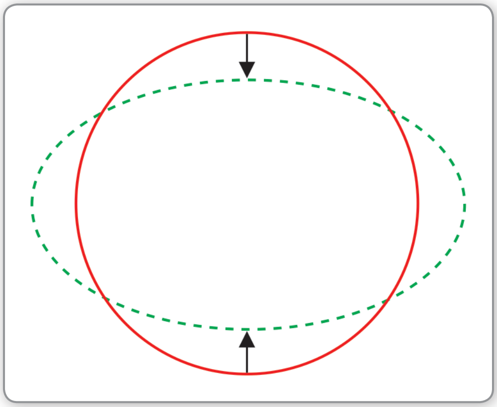


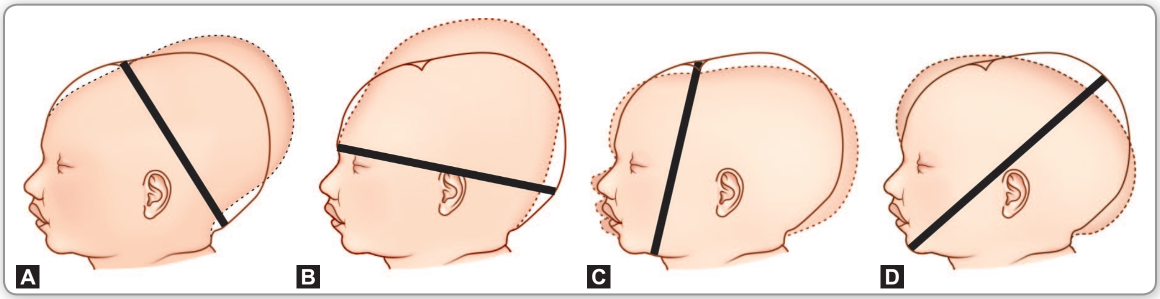
Fig. 9.4: Diagrammatic representation showing the principle of moulding of the head

During the process, the parietal bones tend to overlap the adjacent bones, viz. the occipital bone behind, the frontal bones in front and the temporal bones at the sides. In first vertex position, the right parietal bone tends to override the left one and this becomes reverse in second vertex position. **Molding disappears within few hours after birth**.

Grading: There are three gradings. Grade-1—the bones touching but not overlapping, Grade-2—overlapping but easily separated and Grade-3—fixed overlapping.

Importance:

- Slight molding is inevitable and beneficial. It enables the head to pass more easily, through the birth canal.
- Extreme molding as met in disproportion may produce severe intracranial disturbance in the form of tearing of tentorium cerebelli or subdural hemorrhage.
- Shape of the molding can be a useful information about the position of the head occupied in the pelvis.



Figs 9.5A to D: Types of molding in cephalic presentations (shown by dotted line): (A) Vertex presentation with well flexed head; (B) Vertex presentation with deflexed head (sugar loaf head); (C) Face presentation; (D) Brow presentation

CAPUT SUCCEDANEUM: It is the formation of swelling due to stagnation of fluid in the layers of the scalp beneath the girdle of contact. The girdle of contact is either bony or the dilating cervix or vulval ring. The swelling is diffuse, boggy and is not limited by the suture line (Fig. 9.6). It may be confused with cephalhematoma (see ch. 33). It disappears spontaneously within 24 hours after birth.

Mechanism of formation: While the head descends to press over the dilating cervix or vulval ring, the overlying scalp is free from pressure, but the tissues in contact with the full circumference of the girdle of contact is compressed. This interferes with venous return and lymphatic drainage from the unsupported area of scalp → stagnation of fluid and appearance of a swelling in the scalp (Fig. 9.6). **Caput usually occurs after rupture of the membranes**.

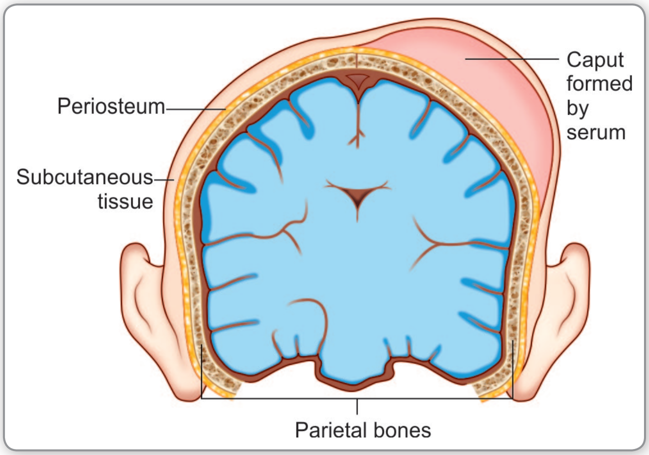


Fig. 9.6: Formation of caput succedaneum

Importance:

- It signifies static position of the head for a long period of time.
- Location of the caput gives an idea about the position of the head occupied in the pelvis and the degree of flexion achieved. In left position, the caput is placed on right parietal bone and in right position, on left parietal bone. **With increasing flexion**, **the caput is placed more posteriorly**.

PELVIS

From the obstetrical standpoint, it is useful to consider the bony pelvis as a whole rather than separately. For descriptive purpose, an articulated pelvis is composed of four bones—two innominate bones, sacrum and coccyx. These are united together by four joints—two sacroiliac joints, sacrococcygeal joint and the symphysis pubis.

The pelvis is anatomically divided into a false pelvis and a true pelvis, the boundary line being the brim of the pelvis. **The bony landmarks on the brim of the pelvis from anterior to posterior on each side are**—upper border of symphysis pubis, pubic crest, pubic tubercle, pectineal line, iliopubic eminence, iliopectineal line, sacroiliac articulation, anterior border of the ala of sacrum and sacral promontory (Fig. 9.7).

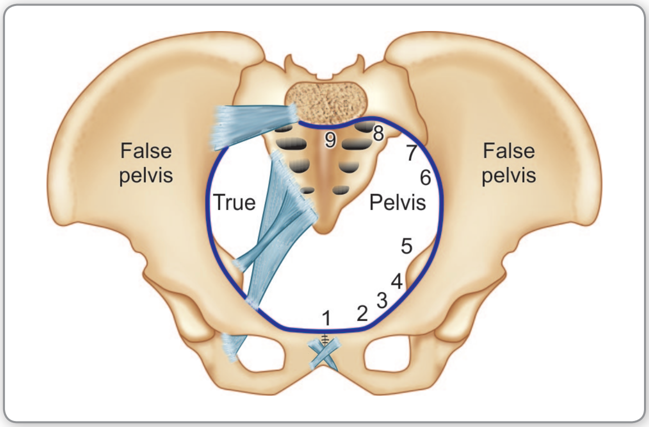


Fig. 9.7: Bony landmarks on the brim of the pelvis separating the true from the false pelvis; 1-Symphysis pubis. 2-Pubic crest. 3-Pubic tubercle. 4-Pectineal line. 5-Iliopubic eminence. 6-Iliopectineal line. 7-Sacroiliac articulation. 8-Anterior border of the ala of sacrum and 9-Sacral promontory

FALSE PELVIS

The false pelvis is formed by the iliac portions of the innominate bones and is limited above by the iliac crests. It has got little obstetric significance except that its measurements can to a certain extent, predict the size and configuration of the true pelvis. Its only obstetric function is to support the enlarged uterus during pregnancy. Its boundaries are: posteriorly—lumbar vertebrae, laterally—iliac fossa and anteriorly—anterior abdominal wall.

TRUE PELVIS

This part of the pelvis is chiefly of concern to the obstetricians, as it forms the canal through which the fetus has to pass. It is shallow in front, formed by symphysis pubis and measures 4 cm (1 ½") and deep posteriorly, formed by the sacrum and coccyx and measures 11.5 cm (4 ½"). **For descriptive purpose, it is divided into inlet, cavity and outlet**.

The pelvic measurements given in the text are average when measured radiologically and vary within a limited degree in different countries. The conversion of centimeters into inches is approximate.

INLET

As the inlet is the brim of the pelvis, the circumference of the inlet is formed by the bony landmarks mentioned previously.

Shape: It is almost round (gynecoid) with the anteroposterior diameter being the shortest. **Other different shapes of the inlet are anthropoid, android and platypelloid (p. 402)**.

Plane: It is an imaginary flat surface bounded by the bony points mentioned as those of the brim. It is not strictly a mathematical plane and is, therefore, **often referred to as superior strait**.

Inclination: In the erect posture, the pelvis is tilted forward. As such, the plane of the inlet makes an angle of about 55° with the horizontal and is called angle of inclination. Another way of measuring the inclination radiographically is to take the angle between the plane of the inlet and the front of the body of the fifth lumbar vertebra. The angle is normally about 135° (Fig. 9.8).

When the angle of inclination is increased due to sacralization of fifth lumbar vertebra, it is called **high inclination**. **High inclination has got obstetric significances:**

1. There is delay in engagement because the uterine axis fails to coincide with that of inlet
2. It favors occipitoposterior position
3. There is difficulty in descent of the head due to long birth canal and flat sacrum interfering with internal rotation.

The angle of inclination may be lessened in case of lumbarization of first piece of sacral vertebra and is called **low inclination**. **It has got no obstetric significance**. It actually facilitates early engagement.

Sacral angle: It is the angle formed by the true conjugate with the first two pieces of the sacrum (Fig. 9.8). Normally, it is greater than 90°. A sacral angle of lesser degree suggests funnelling of the pelvis.

Axis: It is a mid-perpendicular line drawn to the plane of the inlet (Fig. 9.17). **Its direction is downward and backward**. When extended, the line passes through the umbilicus to coccyx. It is important that the uterine axis should coincide with

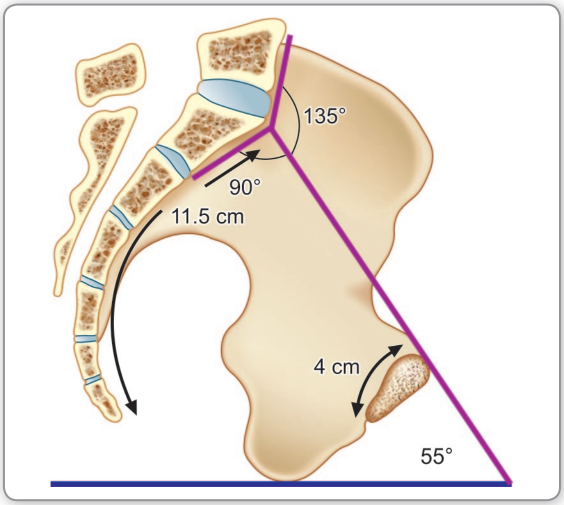


Fig. 9.8: Diagrammatic representation showing plane of the inlet; inclination of the pelvis, sacral angle and depth of the pelvis

the axis of the inlet so that the force of the uterine contractions will be spread in the right direction, to force the fetus to pass through the brim.

Diameters: The measurements of the diameters are all approximate and minor variation is the rule rather than the exception.

Anteroposterior *(Syn: true conjugate, anatomical conjugate, conjugate vera)*: It is the distance between the midpoint of the sacral promontory to the inner margin of the upper border of symphysis pubis (Fig. 9.9). **It measures 11 cm (4 ¼")**. It is not the shortest diameter of the inlet in the anteroposterior plane. In practice, the true conjugate cannot be estimated directly. However, **its measurement is inferred** by subtracting 1.2 cm (½") from the diagonal conjugate thus allowing for the inclination, thickness and height of the symphysis pubis.

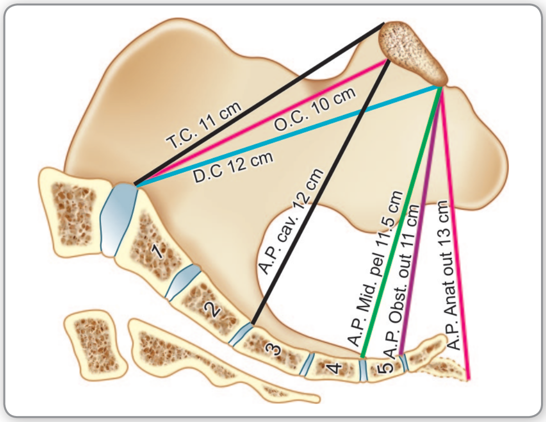


Fig. 9.9: Sagittal section of the pelvis showing anteroposterior diameters in different planes

Obstetric conjugate: It is the distance between the midpoint of the sacral promontory to prominent bony projection in the midline on the inner surface of the symphysis pubis (Fig. 9.9).

The point is somewhat below its upper border. It is the shortest anteroposterior diameter in the anteroposterior plane of the inlet. **It measures 10 cm (4")**. It cannot be clinically estimated but is to be inferred from the diagonal conjugate—1.5–2 cm (¾") to be deducted or by lateral radiopelvimetry.

Diagonal conjugate: It is the distance between the lower border of symphysis pubis to the midpoint on the sacral promontory. **It measures 12 cm (4 ¾") (Fig. 9.9)**.

It is measured clinically during pelvic assessment in late pregnancy or in labor. **Obstetric conjugate is computed** by subtracting 1.5–2 cm from the diagonal conjugate depending upon the height, thickness and inclination of the symphysis pubis.

How to measure? The patient is placed in dorsal position. Two fingers are introduced into the vagina taking aseptic precautions. The fingers are to follow the anterior sacral curvature. In normal pelvis, it is difficult to feel the sacral promontory or at best can be felt with difficulty. However, in order to reach the promontory, the elbow and the wrist are to be depressed sufficiently while the fingers are mobilized in upward direction. The point at which the bone recedes from the fingers is the sacral promontory. The fingers are then mobilized under the symphysis pubis and a marking is placed over the gloved index finger by the index finger of the left hand (Fig. 9.10).

The internal fingers are removed and the distance between the marking and the tip of the middle finger gives the measurement of diagonal conjugate. **For practical purpose, if the middle finger fails to reach the promontory or touches it with difficulty, it is likely that the conjugate is adequate for an average size head to pass through.**

Transverse diameter: It is the distance between the two farthest points on the pelvic brim over the iliopectineal lines. **It measures 13 cm (5 ¼") (Figs 9.11 and 9.12)**.

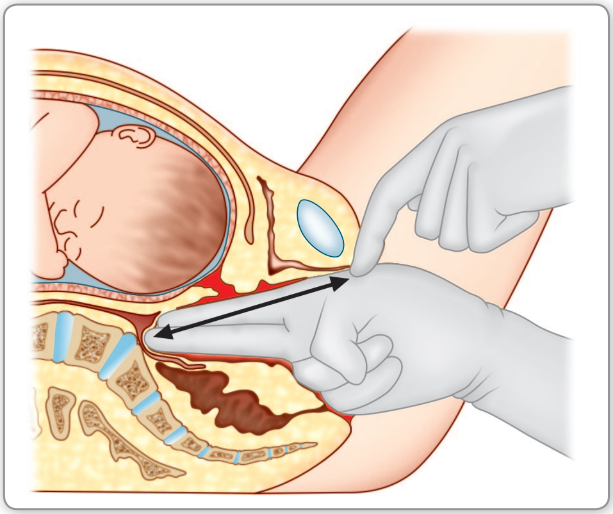


Fig. 9.10: Measurement of diagonal conjugate

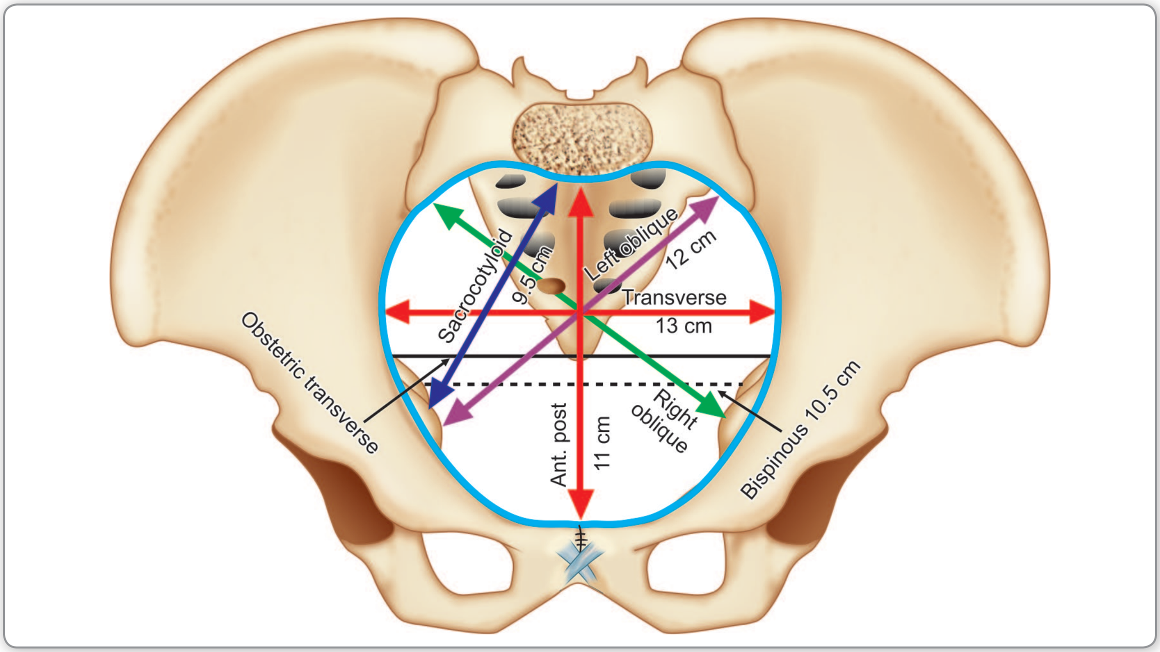


Fig. 9.11: Different diameters of the inlet of obstetrical significance. Bispinous diameter is also demonstrated

The diameter usually lies slightly closer to sacral promontory and divides the brim into anterior and posterior segment. The head negotiates the brim through a diameter, called **available or obstetrical transverse**. This is described as a diameter which bisects the anteroposterior diameter in the midpoint. Thus the obstetrical transverse is either equal or less than the anatomical transverse.

Oblique diameters: There are two oblique diameters—right and left. Each one extends from one sacroiliac joint to the opposite iliopubic eminence and **measures 12 cm (4 ¾")**. **Right or left denotes the sacroiliac joint from which it starts (Figs 9.11 and 9.12)**.

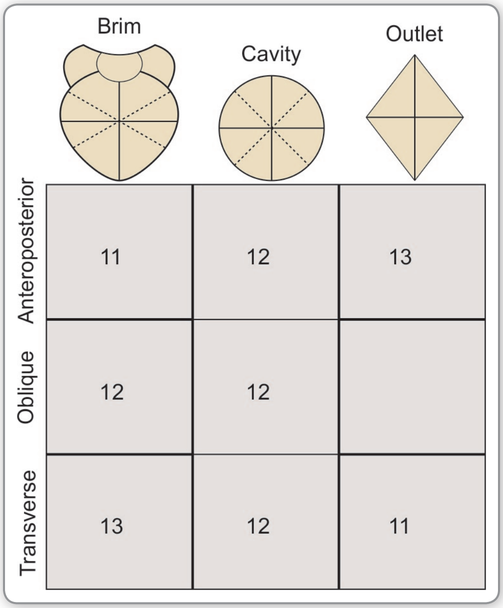
Sacrocotyloid—9.5 cm (3 ¾"): It is the distance between the midpoint of the sacral promontory to iliopubic eminence (Fig. 9.11). It represents the space occupied by the biparietal diameter of the head while negotiating the brim in flat pelvis.

CAVITY

Cavity is the segment of the pelvis bounded above by the inlet and below by plane of least pelvic dimensions.

Shape: It is almost round.

Plane: The plane extends from the midpoint of posterior surface of symphysis pubis to the junction of second and third sacral vertebrae (Fig. 9.17). It is called **plane of greatest pelvic dimensions**. It is the most roomy plane of the pelvis and is almost round in shape.



	Brim	Cavity	Outlet
Anteroposterior	11	12	13
Oblique	12	12	
Transverse	13	12	11

Fig. 9.12: Composite measurements of the diameters of the inlet, cavity and outlet in centimeters

Axis: It is the mid-perpendicular line drawn to the plane of the cavity. Its direction is almost downward (Fig. 9.17).

Diameters: **Anteroposterior (12 cm or 4 ¾"):** It measures from the midpoint on the posterior surface of the symphysis pubis to the junction of second and third sacral vertebrae (Fig. 9.9).

Transverse (12 cm or 4 ¾"): It cannot be precisely measured as the points lie over the soft tissues covering the sacrosciatic notches and obturator foramina.

OUTLET

Obstetrical outlet: **It is the segment of the pelvis bounded above by the plane of least pelvic dimensions and below by the anatomical outlet (Fig. 9.13)**.

Obstetric Significance of Plane of Least Pelvic Dimension

- It is the **narrowest** plane in the pelvis
- This plane corresponds roughly to the **origin of levator ani** muscles (Fig. 9.16)
- It is at this plane that the **internal rotation** of the fetal head occurs
- It marks the **beginning of the forward curve** of the pelvic axis (Fig. 9.17)
- It is a landmark used for **pudendal nerve block** analgesia
- This level of ischial spines indicate **Station 'O'** (see p. 156) Ch.13
- It is **irregularly oval** and notched on each side by ischial spine.

Its anterior wall is deficient at the pubic arch; its lateral walls are formed by ischial bones and the posterior wall includes whole of the coccyx.

Shape: It is anteroposteriorly oval.

Plane: The plane is otherwise known as **plane of least pelvic dimensions or narrow pelvic plane**. The plane extends from the lower border of the symphysis pubis to the tip of ischial spines and posteriorly to meet the tip of the fifth sacral vertebra.

Diameters: **Transverse—Syn: Bispinous (10.5 cm or 4 $^{1}/_{5}$")**: It is the distance between the tip of two ischial spines.

Anteroposterior (11 cm or 4 ¼"): It extends from the inferior border of the symphysis pubis to the tip of the sacrum (Fig. 9.9).

Posterior sagittal (5 cm or 2"): It is the distance between the tip of the sacrum and the midpoint of bispinous diameter.

Axis: It is represented by a line joining the center of the plane with the sacral promontory. Its direction is almost vertical.

Anatomical Outlet: It is otherwise known as bony outlet. It is bounded in front by the lower border of the symphysis pubis; laterally by the ischiopubic rami, ischial tuberosity and sacrotuberous ligament and posteriorly by the tip of coccyx (Fig. 9.14). Thus, it consists of two triangular planes with a common base formed by a line joining the ischial tuberosities. The apex of the anterior triangle is formed by the inferior border of the pubic arch and that of the posterior triangle by the tip of the coccyx.

Shape: It is diamond-shaped.

Plane: It is formed by a line joining the lower border of the symphysis pubis to the tip of the coccyx (Fig. 9.17). It forms an angulation of 10° with the horizontal.

Axis: It is a mid-perpendicular line drawn to the plane of the outlet. Its direction is downward and forward (Fig. 9.17).

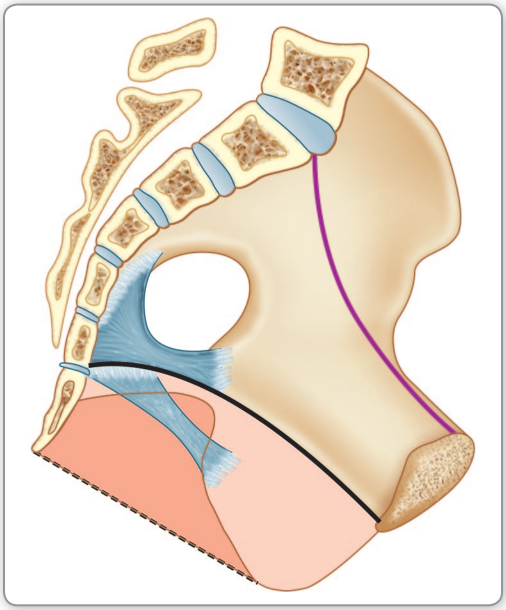
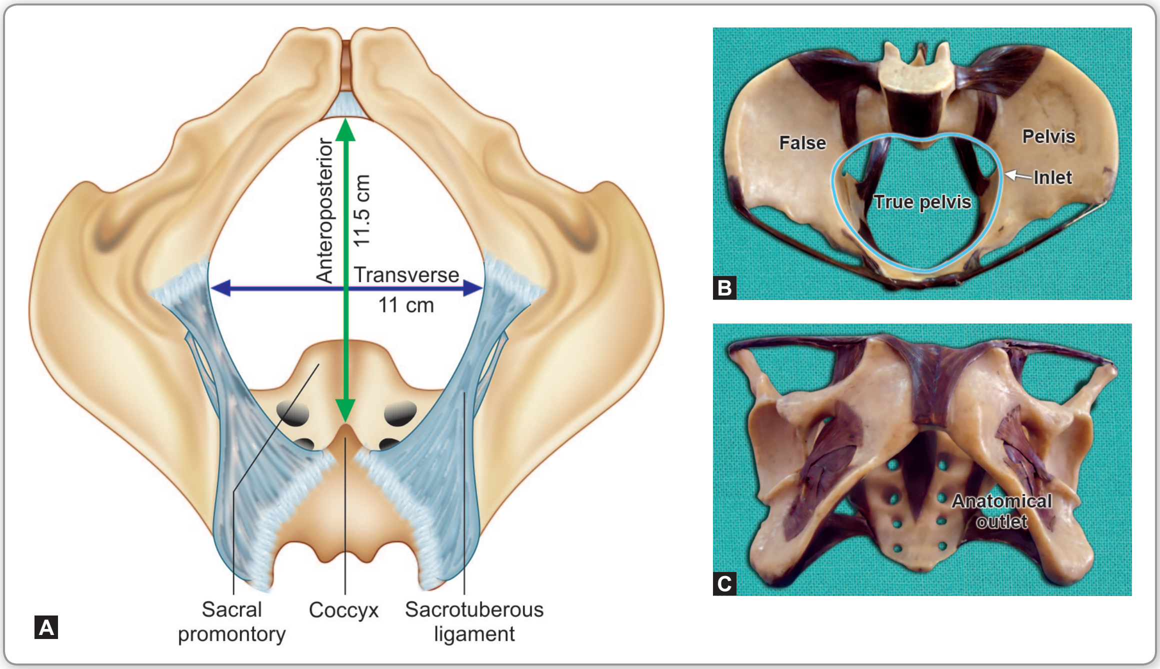


Fig. 9.13: Obstetrical outlet (*shaded area*)



Figs 9.14A to C: (A) Boundary of the anatomical outlet with its measurements; (B) Dummy showing false pelvis, true pelvis and the brim of the pelvis; (C) Dummy showing anatomical outlet of the pelvis

Diameters: **Anteroposterior:** It extends from the lower border of the symphysis pubis to the tip of the coccyx. It measures 13 cm or 5 ¼" with the coccyx pushed back by the head when passing through the introitus in the second stage of labor; with the coccyx in normal position, the measurement will be 2.5 cm less (Fig. 9.9).

Transverse — Syn : Intertuberous (11 cm or 4 ¼"): It measures between inner borders of ischial tuberosities.

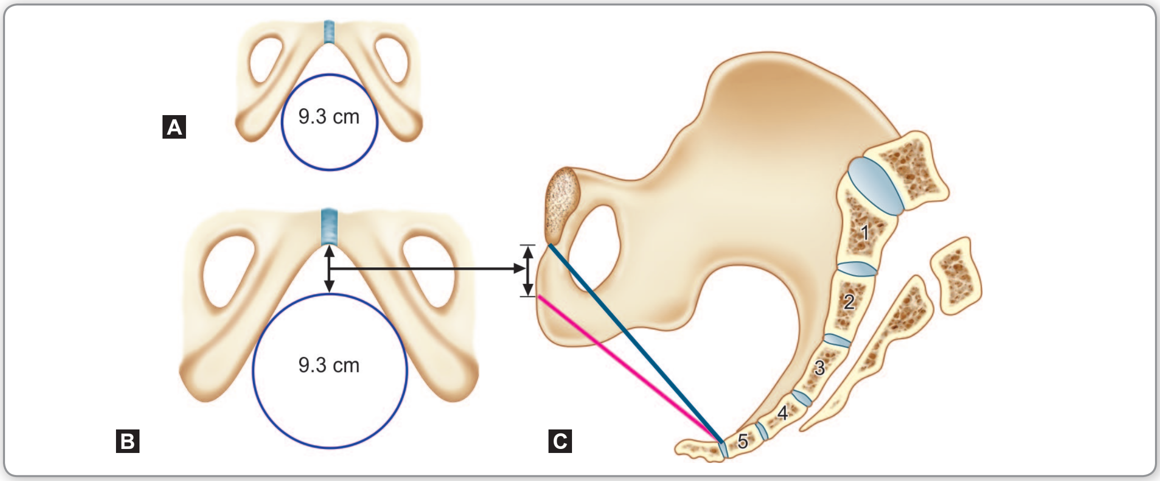
Posterior sagittal diameter (8.5 cm or 3 ½"): It is the anteroposterior distance between the sacrococcygeal joint and the midpoint of transverse diameter of outlet (TDO). It is clinically measured by the distance between the sacrococcygeal joint and anterior margin of the anus.

Subpubic angle: It is formed by the approximation of the two descending pubic rami. In normal female pelvis, it measures 85°.

Pubic arch: Arch formed by the descending rami of both the sides is of obstetric importance. Normally, it measures 6 cm in between the pubic rami at a level of 2 cm below the apex of the subpubic arch. Clinically, it is assessed by placing 3 fingers side by side.

The narrower the pubic arch, the more is the fetal head displaced backward and the less the room available for it. Normally, the subpubic arch is rounded and less space is wasted under the symphysis pubis. When a round disk of 9.3 cm diameter (diameter of well flexed fetal head) is placed under the arch, the distance between the symphysis pubis and the circumference of the disk is measured. This measurement is the **waste space of Morris** and should not exceed 1 cm in a normal pelvis.

Available anteroposterior diameter: When the **waste space of Morris** is more than 1 cm, the anterior point of the anteroposterior diameter of the outlet extends below the symphysis pubis on the pubic rami for a distance equivalent to the waste space of Morris (Fig. 9.15). The distance between the said point and the tip of the sacrum is called available anteroposterior diameter of the outlet. It is through this diameter that the head escapes out of the bony outlet.



Figs 9.15A to C: Diagrammatic representation of — (A) Normal pubic arch; (B) Waste space of Morris; (C) Available anteroposterior diameter of the outlet

MIDPELVIS

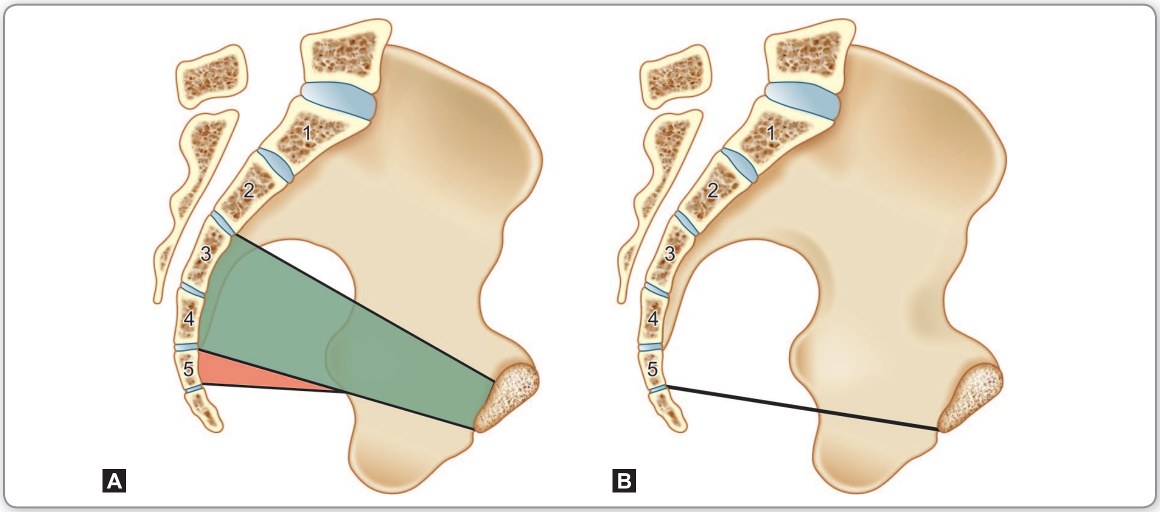
Midpelvis is the segment of the pelvis bounded above by the plane of greatest pelvic dimensions and below by a plane known as midpelvic plane.

Midpelvic plane: The midpelvic plane extends from the lower margin of the symphysis pubis through the level of ischial spines to meet either the junction of S_4 and S_5 or tip of the sacrum depending upon the configuration of the sacrum. If the plane meets the tip of the fifth sacrum, it coincides with the plane of least pelvic dimensions. If the plane meets the junction of S_4 and S_5, the plane becomes a wedge posteriorly (Fig. 9.16).

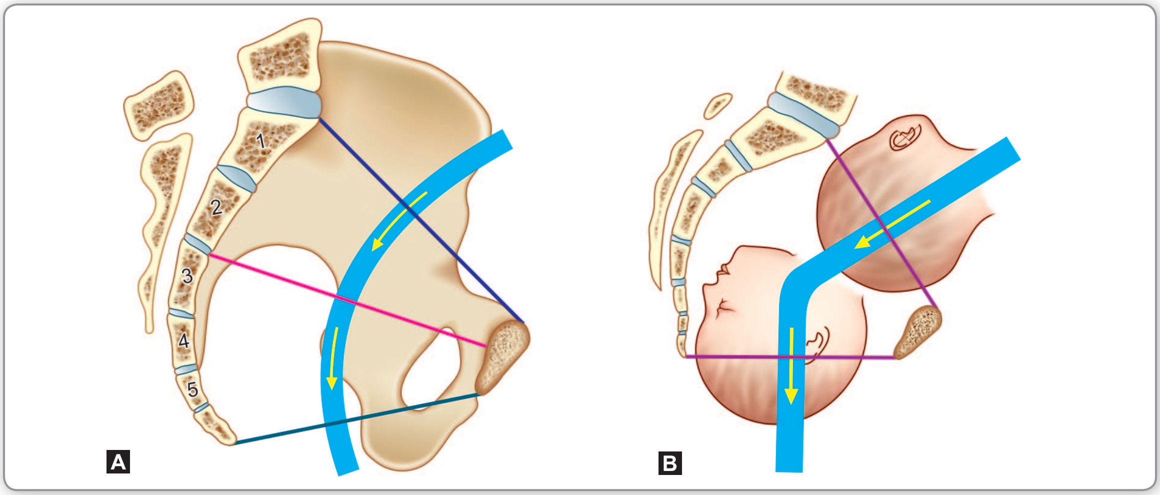
Diameters: **Transverse diameter — Syn : bispinous (10.5 cm)** : It measures between the two ischial spines.

Anteroposterior diameter (11.5 cm): It extends from the lower border of the symphysis pubis to the point on the sacrum at which the midpelvic plane meets.

Posterior sagittal diameter (4.5 cm): It extends from the midpoint of the bispinous diameter to the point on the sacrum at which the midpelvic plane meets.



Figs 9.16A and B: Diagrammatic representation of midpelvis and midpelvic plane: (A) Zone of midpelvis (shaded area) with the midpelvic plane becomes a wedge posteriorly; (B) Midpelvic plane coincides with the plane of least pelvic dimensions



Figs 9.17A and B: Pelvic axis — (A) Axes of inlet, cavity and outlet are shown by arrows drawn perpendicular to the planes. The shaded area over the axes is the anatomical pelvic axis (curve of Carus); (B) Obstetric pelvic axis — as shown by the shaded area is directed downward and backward up to ischial spines and then directed forward

PELVIC AXIS: ***Anatomical (curve of Carus):*** Anatomical pelvic axis is formed by joining the axes of inlet, cavity and outlet. It is uniformly curved with the convexity fitting with the concavity of the sacrum. The fetus does not, however, transverse the uniform curved path (Fig. 9.17).

Obstetrical: It is through this axis that the fetus negotiates the pelvis. It is not uniformly curved. Its direction is first downward and backward up to the level of ischial spines and then directed abruptly forward (Fig. 9.17).

PELVIC JOINTS: ***Symphysis pubis:*** It is a secondary fibrocartilaginous joint. It has got no capsule and no synovial cavity. The articular surfaces are covered with hyaline cartilage. Due to softening of the ligaments during pregnancy, there is considerable amount of gliding movement.

Sacroiliac articulation: It is a synovial joint and is an articulation between the articular surface of the ilium and sacrum. The articulating surfaces are not alike. It has got a capsule and a synovial cavity.

Engagement to diagnose, it is better to palpate gently with two hands facing down over the abdomen (Fig. 8.6) than to prod around with Pawlik's grip, which in non-experienced hands is painful.

Sacrococcygeal joint is a synovial hinge joint. It allows both flexion and extension. Extension increases the anteroposterior diameter of the outlet.

PHYSIOLOGICAL ENLARGEMENT OF PELVIS DURING PREGNANCY AND LABOR

Imaging studies show an increase in width and mobility of the symphysis pubis during pregnancy which returns to normal following delivery. The pubic bones may separate by 5–10 mm. Similar changes also occur in sacroiliac joints. There is gliding movement of the symphysis pubis near term. Relaxation of the pelvic joints is due to progesterone and relaxin. There is increase of the anteroposterior diameter of the inlet during labor by the rotatory movement of the sacroiliac joints. In dorsal lithotomy position, the anteroposterior diameter of the outlet may be increased to 1.5–2 cm. Furthermore, the coccyx is pushed back while the head descends down to the perineum.

QUESTIONS

1. Describe the plane of least pelvic dimension. What is the obstetric significance of this plane? (p. 102)

Related theory questions (Long & Short), Obstetric Case Discussions, Viva table discussions, Postoperative word round discussions, and MCQs are discussed in author's books:

Bedside Clinic and Viva Voce: 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

For further reading:

Master pass in Obstetrics and Gynaecology: 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

Chapter 10

Antenatal Care, Preconceptional Counseling and Care

Systematic supervision (examination and advice) of a woman during pregnancy is called antenatal (prenatal) care. The supervision should be regular and periodic in nature according to the need of the individual. Actually prenatal care is the care in continuum that starts before pregnancy and ends at delivery and the postpartum period. **Antenatal care comprises of**:

- Careful history taking and examinations (general and obstetrical)
- Advice given to the pregnant woman.

AIMS AND OBJECTIVE

The aims are: (1) to screen the "high risk" cases (see p. 716), (2) to prevent or to detect and treat at the earliest any complication, (3) to ensure continued risk assessment and to provide ongoing primary preventive health care, (4) to educate the mother about the physiology of pregnancy and labor by demonstrations, charts and diagrams (mothercraft classes), so that fear is removed and psychology is improved, (5) to discuss with the couple about the place, time and mode of delivery, provisionally and care of the newborn, (6) to motivate the couple about the need of family planning and also appropriate advice to couple seeking medical termination of pregnancy.

The objective is to ensure a normal pregnancy with delivery of a healthy baby from a healthy mother.

The criteria of a normal pregnancy are delivery of a single baby in good condition at term (between 38 and 42), with fetal weight of 2.5 kg or more and with no maternal complication. As such, a normal pregnancy is a retrospective term.

PROCEDURE AT THE FIRST VISIT

The first visit should not be deferred beyond the second missed period. It may be earlier if the patient desires to terminate the pregnancy.

OBJECTIVES: (1) To assess the health status of the mother and fetus. (2) To assess the fetal gestational age and to obtain baseline investigations. (3) To organize continued obstetric care and risk assessment.

Components of routine prenatal care are recorded in a standardized pro forma (antenatal record book).

HISTORY TAKING

Vital statistics

Name: ....

Date of first examination: .... *Address*: ....

Age: A woman having her first pregnancy at the age of 30 or above (FIGO – 35 years) is called **elderly primigravida**. Extremes of age (teenage and elderly) are obstetric risk factors (see p. 398).

Gravida and parity: **Gravida** denotes a pregnant state both present and past, irrespective of the period of gestation. **Parity** denotes a state of previous pregnancy beyond the period of viability.

Gravida and para refer to pregnancies and not to babies. As such, a woman who delivers twins in first pregnancy is still a gravida one and para one. A pregnant woman with a previous history of two abortions and one term delivery can be expressed as fourth gravida but primipara. It is customary in clinical practice to summarize the past obstetric history by two digits (the first one relates with viable births and the second one relates with abortion) connected with a plus sign affixing the letter "P". **Thus, P_{2+1} denotes** the patient had two viable births and one abortion. In some centers, it is expressed by four digits connected by dashes. **P**A-B-C-D, where A **denotes** number of term (37–42 weeks) pregnancies, B: number of preterm (28 to <37 weeks) pregnancies, C: number of miscarriages (<28 weeks) and D: the number of children alive at present. A pregnant woman with a previous history of four births or more is called **grand multipara**.

Terminology

- **A nullipara** is one who has never completed a pregnancy to the stage of viability. She may or may not have aborted previously.
- **A primipara** is one who has delivered one viable child. Parity is not increased even if the fetuses are many (twins, triplets).
- **A multigravida** is one who has previously been pregnant. She may have aborted or have delivered a viable baby.
- **A parturient** is a women in labor.
- **A nulligravida** is one who is not now and never has been pregnant.
- **A primigravida** is one who is pregnant for the first time.
- **Multipara** is one who has completed two or more pregnancies to the stage of viability or more.
- **A puerpera** is a woman who has just given birth.

Duration of marriage: This is relevant to note the fertility or fecundity. A pregnancy long after marriage without taking recourse to any method of contraception is called low fecundity and soon after marriage is called high fecundity. A woman with low fecundity is unlikely to conceive frequently.

Religion: ....

Occupation: It is helpful in interpreting symptoms of fatigue due to excess physical work or stress or occupational hazards. Such women should be informed to reduce such activities.

Occupation of the husband: A fair idea about the socioeconomic condition of the patient can be assessed. This knowledge is of value: (a) to anticipate the complications likely to be associated with low social status such as anemia, preeclampsia, prematurity, etc. (b) to give reasonable and realistic antenatal advice during family planning guidance.

Period of gestation: The duration of pregnancy is to be expressed in terms of completed weeks. A fraction of a week of more than 3 days is to be considered as completed week. In calculating the weeks of gestation in early part of pregnancy, counting is to be done from the **first day of last normal menstrual period (LNMP)** and in later months of pregnancy, counting is to be done from expected date of delivery (EDD).

Most reliable clinical parameter of gestational age assessment is an accurate LMP. In some cases, LMP may be inaccurate, unknown or following the use of oral contraceptives (OC). In the case of OC use, ovulation may not have occurred 2 weeks after the LMP. In such a situation, ultrasonography in first trimester of pregnancy is more reliable to estimate the gestational age.

Complaints**:** Categorically, the genesis of the complaints is to be noted. Even if there is no complaint, enquiry is to be made about the sleep, appetite, bowel habit and urination.

History of present illness**:** Elaboration of the chief complaints as regard their onset, duration, severity, use of medications and progress is to be made.

History of present pregnancy**:** The important complications in different trimesters of the present pregnancy are to be noted carefully. These are hyperemesis and threatened abortion in first trimester, features of pyelitis in second trimester and anemia, preeclampsia and antepartum hemorrhage in the last trimester. Number of previous antenatal visits (booking status), immunization status, has to be noted. Any medication or radiation exposure in early pregnancy or medical-surgical events during pregnancy should be enquired.

Obstetric history**:** This is only related with multigravidae. The previous obstetric events are to be recorded chronologically as per the pro forma given on the next page. To be relevant, enquiry is to be made whether she had antenatal and intranatal care before.

No.	**Year and date**	**Pregnancy events**	**Labor events**	**Methods of delivery**	**Puerperium**	**Baby** - *Weight and sex* - *Condition at birth (Apgar score)* - *Breast-feeding* - *Immunization*
1.	2011 January	Miscarriage at 8 weeks		Evacuation done	Uneventful	
2.	2011 July	Well covered antenatally, uneventful	Uneventful	Spontaneous vaginal	Uneventful	Baby-boy weight 2.6 kg Cried at birth Breast-fed (6 months), alive and well.
3.	2013 July	—do—	—do—	—do—	—do—	Girl weight 2.7 kg Cried at birth Breast-fed (6 months) Both the babies fully immunized, alive and well.

The obstetric history is to be summed up as: Status of gravida, parity, number of deliveries (term, preterm), miscarriage, pregnancy termination (MTP) and living issue [e.g. Mrs R.L, ($P_{2+0+1+2}$) G4, P2, miscarriage 1, living 2 at 36 weeks of present pregnancy].

An undue long gap between the last and the present pregnancy requires careful supervision during pregnancy and labor. **The minimum spacing** between first birth and subsequent pregnancy should be 2 years.

Menstrual history**:** Cycle, duration, amount of blood flow and **first day of the last normal menstrual period (LNMP) are to be noted** (spontaneous). From the LNMP, the expected date of delivery (EDD) has to be calculated. The first day of the menstruation being the important event can be remembered precisely while the last day of the period is often tailed off and hence may be forgotten.

Calculation of the expected date of delivery (EDD): This is done according to **Naegele's formula** (1812) by adding 9 calendar months and 7 days *to the first day of the last normal (28 days cycle) period.* Alternatively, one can count back 3 calendar months from the first day of the last period and then add 7 days to get the expected date of delivery; the former method is commonly employed.

Example: The patient had her first day of last menstrual period on 1st January. By adding 9 calendar months it comes to 1st October and then add 7 days, i.e. 8th October, which becomes the expected date of delivery. For **IVF pregnancy** date of LMP is 14 days prior to date of embryo transfers (266 days).

***Past medical history*:** Relevant history of past medical illnesses (urinary tract infections, tuberculosis) is to be elicited.

***Past surgical history*:** Previous surgery—general or gynecological, if any, is to be enquired.

***Family history*:** Family history of hypertension, diabetes, tuberculosis, blood dyscrasia, known hereditary disease, if any, or twinning is to be enquired.

***Personal history*:** Contraceptive practice prior to pregnancy, smoking or alcohol habits are to be enquired. LMP may be a withdrawal bleed following pill usage. The first ovulation may be delayed for 4–6 weeks (see p. 108). Smoking or alcohol abuse has got some relation with low birth weight of the baby. Previous history of blood transfusion, corticosteroid therapy, any drug allergy and immunization against tetanus or prophylactic administration of anti-D immunoglobulin are to be enquired.

EXAMINATION

General Physical Examination

***Build*:** Obese/average/thin. ***Nutrition*:** Good/average/poor

***Height*:** Short stature is likely to be associated with a small pelvis.

Thus, in primigravidae, the height is to be measured to screen out the short stature. While an arbitrary measurement of 5 feet. is considered as short stature in western countries, it is 4' 7" in India considering the low average height.

***Weight*:** Weight should be taken in all cases in an accurate weighing machine. Repeated weight checking in subsequent visit should preferably be done in the same weighing machine. The importance of weight checking has already been discussed (see p. 57).

***Pallor*:** The sites to be noted are lower palpebral conjunctiva, dorsum of the tongue and nail beds.

***Jaundice*:** The sites to be noted are bulbar conjunctiva, under surface of the tongue, hard palate and skin.

***Tongue, teeth, gums and tonsils*:** Evidences of malnutrition are evident from glossitis and stomatitis. Evidence of any source of infection in the mouth is to be eradicated least there be a chance of autogenous infection in puerperium.

***Neck*:** Neck veins, thyroid gland or lymph glands are looked for any abnormality. Slight physiological enlargement of the thyroid gland occurs during pregnancy in 50% of cases.

***Edema of legs*:** Both the legs are to be examined. The sites for evidence of edema are over the medial malleolus and anterior surface of the lower one-third of the tibia. The area is to be pressed with the thumb for at least 5 seconds. Varicosity in the legs, if any, is to be noted.

Causes of edema in pregnancy: (1) Physiological (2) Preeclampsia (3) Anemia and hypoproteinemia (4) Cardiac failure (5) Nephrotic syndrome.

Dependent edema is physiological in pregnancy but generalized edema (anasarca) or facial edema can be a first sign of disease.

Physiological edema: The cause of physiological edema is due to increased venous pressure of the inferior extremities by the gravid uterus pressing on the common iliac veins. **The features of the physiological edema are:** (1) slight degree (ankle edema), usually confined to one leg, more on the right, (2) unassociated with any other features of preeclampsia or proteinuria, (3) disappears on rest alone, (4) other pathologies of cardiac, renal and hematological are absent.

***Pulse*:**

Blood pressure: Disappearance of sounds (Korotkoff 5) rather than muffling of sounds (Korotkoff 4) is the best representation of diastolic pressure during pregnancy.

***Systemic examination*:**

Heart, Lungs, Liver and Spleen:

Breasts: **Examination of the breasts** helps to note the presence of pregnancy changes but also to note the nipples (cracked or depressed) and skin condition of the areola. The purpose is to correct the abnormality; if any, so that there will be no difficulty in breastfeeding immediately following delivery.

Obstetrical examination (see details p. 87): *Abdominal*: Tone of the abdominal muscles, presence of any incisional scar or presence of herniation and skin condition of the abdomen are to be looked for. Fundus of the uterus is just palpable above the symphysis pubis at 12 weeks.

Vaginal: Examination is done in the antenatal clinic when the patient attends the clinic for the first time before 12 weeks. **It is done:** (1) to diagnose the pregnancy, (2) to corroborate the size of the uterus with the period of amenorrhea and (3) to exclude any pelvic pathology. Internal examination is, however, omitted in cases with previous history of miscarriage, occasional vaginal bleeding in present pregnancy. **Ultrasound examination** has replaced routine internal examination. It is more informative and without any known adverse effect.

Steps of vaginal examination: Vaginal examination is done in the antenatal clinic. **The patient must empty her bladder prior to examination** and is placed in the dorsal position with the thighs flexed along with the buttocks placed on the foot-end of the table. Hands are washed with soap and a sterile glove is put on the examining hand (usually right).

Inspection: By separating the labia—using the left two fingers (thumb and index), the character of the vaginal discharge, if any, is noted. Presence of cystocele or uterine prolapse or rectocele is to be elicited.

Speculum examination: This should be done prior to bimanual examination, especially when the smear for exfoliative cytology or vaginal swab is to be taken. A bivalve speculum is used. The cervix and the vault of the vagina are inspected with the help of good light source placed behind. **Cervical smear for exfoliative cytology** or a vaginal swab from the upper vagina, in presence of discharge, may be taken.

Bimanual: Two fingers (index and middle) of the right hand are introduced deep into the vagina while separating the labia by left hand. **The left hand is now placed suprapubically.** Gentle and systematic examinations are to be done to note: (1) **Cervix**: consistency, direction and any pathology. (2) **Uterus**: size, shape, position and consistency. Early pregnancy is the best time to correlate accurately uterine size and duration of gestation. (3) **Adnexa**: any mass felt through the fornix. If the introitus is narrow, one finger may be introduced for examination. **No attempt should be made to assess the pelvis at this stage.**

Routine investigations:

- **Blood**: Hemoglobin, hematocrit, ABO, Rh grouping, blood glucose and VDRL are done. Serology (antibody) screening is done in selected cases (see p. 336).
- **Urine:** Protein, sugar and pus cells. If significant proteinuria is found, "clean catch" specimen of midstream urine is collected for culture and sensitivity test. **To collect the midstream urine**, the patient is advised to clean the vulva and to collect the urine in a clean container during the middle of the act of urination. Presence of nitrites and/or leukocyte esterase by **dipstick** indicates urinary tract infection (p. 743).
- **Cervical cytology study** by Papanicolaou stain has become a routine in many clinics.

***Special investigations*:**

(a) **Serological tests for rubella, hepatitis B virus and HIV:** antibodies to detect rubella immunity and screening for hepatitis B virus and HIV (with consent) (see Chapter 20).

(b) **Genetic screen: Maternal serum alpha-fetoprotein** (MSAFP), triple test at 15–18 weeks for mother at risk of carrying a fetus with neural tube defects, Down's syndrome or other chromosomal anomaly (see p. 129).

(c) **Ultrasound examination: First trimester scan** either transabdominal (TAS) or transvaginal (TVS) helps to detect: (i) early pregnancy, (ii) accurate dating, (iii) number of fetuses, (iv) gross fetal anomalies, (v) any uterine or adnexal pathology (see p. 734). Use of ultrasound should be selective rather than a routine.

Booking (18–20 weeks) scan has got advantages in addition to first trimester scan: (i) detailed fetal anatomy survey and to detect any structural abnormality including cardiac, (ii) placental localization. Ultrasound examination is also very reassuring to the couple (see p. 734).

Ultrasound examination is performed as a routine at 18–20 weeks though doubt remains about its absolute benefit.

***Repetition of the investigations*:** (1) Hemoglobin estimation is repeated at 28th and 36th week. (2) Urine is tested (dipstick) for protein and sugar at every antenatal visit.

PROCEDURE AT THE SUBSEQUENT VISITS

Generally, checkup is done at interval of 4 weeks up to 28 weeks; at interval of 2 weeks up to 36 weeks and thereafter weekly till delivery. Ideally, this should be more flexible depending on the need and the convenience of patient. In the developing countries, as per **WHO** recommendation, the visit may be curtailed to **at least 4**; first in second trimester **around 16 weeks**, second **between 24 and 28 weeks**, the third visit **at 32 weeks** and the fourth visit **at 36 weeks**.

***Objectives*:** (A) **To assess:** (1) fetal well-being, (2) lie, presentation, position and number of fetuses, (3) anemia, preeclampsia, amniotic fluid volume and fetal growth, (4) to organize specialist antenatal clinics for patients with problems like cardiac disease and diabetes. (B) **To select**, **time for** ultrasonography, amniocentesis or chorion villus biopsy when indicated (see p. 129-30).

***History*:** To note: (1) appearance of any new symptom (headache, dysuria), (2) date of quickening.

***Examination*:**

General: In each visit, the following are checked and recorded: (1) weight, (2) pallor, (3) edema legs, (4) blood pressure.

***Abdominal examination*:** *Inspection:* Abdominal enlargement, pregnancy marks—linea nigra, striae, surgical scars (midline or suprapubic). *Palpation:* (a) To note the height of the fundus above the symphysis pubis (see p. 88). (b) **In the second trimester**, to identify the fetus by external ballottement, fetal movements, palpation of fetal parts and auscultation of fetal heart sounds. (c) **In the third trimester,** abdominal palpation will help to identify fetal lie, presentation, position, growth pattern, volume of liquor and also any abnormality. Examination also helps to detect whether the presenting part is engaged or not. Girth of abdomen is measured at the level of umbilicus. The girth increases by about 2.5 cm per week beyond 30 weeks and at term, measures about 95–100 cm. (d) **Others**—any uterine mass (fibroid) or tenderness. Fetal activity (movements) is also recorded.

***Vaginal examination*:** **Vaginal examination in the later months of pregnancy (beyond 37 weeks)** with an idea to assess the pelvis is not informative. Pelvic assessment is best done with the onset of labor or just before induction of labor. Methods of vaginal examination for assessment of the pelvis and test for cephalopelvic disproportion are described in Chapter 24. **Any history of vaginal bleeding contraindicates vaginal examination.**

Ongoing assessment and counseling is important as prenatal care has an **educational opportunity**. The woman should be informed about the list of **warning signs so** that she can contact the hospital or avail the nearby health-care facilities in time (see below).

Warning Signs are:

- Leakage of fluid from vagina
- Vaginal bleeding
- Abdominal pain: distressing in nature
- Headache, visual changes
- Decrease or loss in fetal movements
- Fever, rigor, excess vomiting, diarrhea

ANTENATAL ADVICE

PRINCIPLES: (1) **To counsel the women** about the importance of regular checkup. (2) **To maintain or improve** the health status of the woman to the optimum till delivery by judicious advice regarding diet, drugs and hygiene. (3) **To improve** the psychology and to remove the fear of the unknown by counseling the woman.

DIET: The diet during pregnancy should be adequate to provide: (a) good maternal health, (b) optimum fetal growth, (c) the strength and vitality required during labor and (d) successful lactation. During pregnancy, there is increased calorie requirement due to increased growth of the maternal tissues, fetus, placenta and increased basal metabolic rate. **The increased calorie requirement is to the extent of 300 over the nonpregnancy state during second half of pregnancy**. Generally, the **diet in pregnancy** should be of woman's choice as regard the quantity and the type. Woman with normal BMI should eat adequately so as to gain the optimum weight (11 kg). Overweight women with BMI between 26 and 29 should limit weight gain to 7 kg and obese women (BMI > 29) should gain less weight. Excessive weight gain increases antepartum and intrapartum complications including fetal macrosomia.

Table 10.1: Daily Dietary Allowances for a Woman of Reproductive Age, Pregnancy and Lactation

Dietary components	Nonpregnant	Pregnancy second half	Lactation	Sources
Energy (kcal)	2,200 kcal	2,500 kcal	2,600 kcal	Protein, fat, carbohydrate
Protein (g)	50 g	60 g	65 g	Meat, fish, poultry, dairy product
Iron (mg)	18 mg	40 mg*	30 mg*	Meat, egg, grains [* to be supplemented]
Calcium (mg)	500 mg	1,000 mg	1,500 mg	Dairy products
Zinc (mg)	12 mg	15 mg	19 mg	Meat, egg, seafood
Iodine (µg)	150 µg	175 µg	200 µg	Iodized salt, seafood
Vitamin A (IU)	5,000 IU	6,000 IU	8,000 IU	Vegetables, liver, fruits
Vitamin D (IU)	200 IU	400 IU	400 IU	Dairy products
Thiamine (mg)	1.1 mg	1.5 mg	Almost same as in pregnancy	Grains, cereals
Riboflavin (mg)	1.1 mg	1.6 mg		Meat, liver, grains
Nicotinic acid (mg)	15 mg	17 mg		Meat, nuts, cereals
Ascorbic acid (mg)	60 mg	70 mg		Citrus fruits, tomato
Folic acid (µg)	200 µg	400 µg		Leafy vegetables, liver
Vitamin B_{12} (µg)	2 µg	2.2 µg		Animal proteins

The pregnancy diet ideally should be light, nutritious, easily digestible and rich in protein, minerals and vitamins. In terms of figures, the daily requirement during pregnancy and lactation is given in Table 10.1. It is not an absolute recommendation but simply a guide. The diet should consist in addition to the principal food at least half liter, if not, 1 liter of milk (1 liter of milk contains about 1 g of calcium), plenty of green vegetables and fruits. The amount of salt should be of sufficient amount to make the food tasty. At least, half of the total protein should be first class containing all the amino acids and majority of the fat should be animal type which contains vitamins A and D.

Dietetic advice should be given with due consideration to the socioeconomic condition, food habits and taste of the individual. **Woman with normal BMI** (see p. 400) should eat as to maintain the schedule weight gain in pregnancy (see p. 57). **The instruction about diet should be reasonable and realistic to individual women**.

Supplementary nutritional therapy**:** As previously mentioned, there is negative iron balance during pregnancy and the dietetic iron is not enough to meet the daily requirement especially in the second half of the pregnancy. Thus, **supplementary iron therapy is needed for all pregnant mothers from 16 weeks onwards**. Above 10 g% of hemoglobin, 1 tablet of ferrous sulfate (Fersolate) containing 60 mg of elemental iron is enough. The dose should be proportionately increased with lower hemoglobin level to 2–3 tablets a day. **Three tablets provide 45 mg of absorbable iron**. As the essential vitamins are either lacking in the foods or are destroyed during cooking, supplementary vitamins are to be given daily from 20th week onwards (Table 10.1).

ANTENATAL HYGIENE: In otherwise uncomplicated cases, the following advices are to be given:

Rest and sleep**:** The patient may continue her usual activities throughout pregnancy. However, excessive and strenuous work should be avoided especially in the first trimester and the last 4 weeks. Recreational exercise (prenatal exercise class) is permitted as long as she feels comfortable.

There is individual variation of the amount of sleep required. However, on an average, the patient should be in bed for about 10 hours (8 hours at night and 2 hours at noon), especially in the last 6 weeks. In late pregnancy, lateral posture is more comfortable.

Bowel**:** Constipation is common. It may cause backache and abdominal discomfort. Regular bowel movement may be facilitated by regulation of diet taking plenty of fluids, vegetables and milk or prescribing stool softeners at bedtime. There may be rectal bleeding, painful fissures or hemorrhoids due to hard stool.

Bathing**:** The patient should take daily bath but be careful against slipping in the bathroom due to imbalance.

Clothing, shoes and belt**:** The patient should wear loose but comfortable garments. High heel shoes should better be avoided in advanced pregnancy when the center of balance alters. Constricting belt should be avoided.

Dental care**:** Good dental and oral hygiene should be maintained. The dentist should be consulted, if necessary. This will facilitate extraction or filling of the caries tooth, if required, comfortably in the second trimester.

Care of the breasts**:** Breast engorgement may cause discomfort during late pregnancy. A well-fitting brassiere can give relief.

Coitus**:** Generally, coitus is not restricted during pregnancy. Release of prostaglandins and oxytocin with coitus may cause uterine contractions. Women with increased risk of miscarriage or preterm labor should avoid coitus if they feel such increased uterine activity.

Travel**:** Travel by vehicles having jerks is better to be avoided, especially in first trimester and the last 6 weeks. The long journey is preferably to be limited to the second trimester. Rail route is preferable to bus route. Travel in pressurized aircraft is safe up to 36 weeks. **Air travel is contraindicated** in cases with placenta previa, preeclampsia, severe anemia and sickle cell disease. Prolonged sitting in a car or aeroplane should be avoided due to the risk of venous stasis and thromboembolism. Seat belt should be under the abdomen.

Smoking and alcohol**:** In view of the fact that smoking is injurious to health, it is better to stop smoking not only during pregnancy but even thereafter. Heavy smokers have smaller babies and there is also more chance of abortion. Similarly, alcohol consumption is to be drastically curtailed or avoided, so as to prevent fetal maldevelopment or growth restriction (see p. 537, 589).

IMMUNIZATION: Fortunately, most of life-threatening epidemics are rare. In the developing countries, immunization in pregnancy is a routine for tetanus; others are given when epidemic occurs or traveling to an endemic zone or for traveling overseas.

Live virus vaccines (rubella, measles, mumps, varicella, yellow fever) are contraindicated. **Rabies, hepatitis A and B vaccines, toxoids can be given as in nonpregnant state**.

However in certain circumstances, risk or benefit assessment should be made before making decision.

***Tetanus*: Immunization against tetanus not only protects the mother but also the neonates**. In unprotected women, 0.5 mL tetanus toxoid is given intramuscularly at 6 weeks interval for 2 such, the first one to be given between 16 and 24 weeks. Women who are immunized in the past, a booster dose of 0.5 mL IM is given in the last trimester.

Prenatal classes are found to be helpful and valuable (see p. 155).

***Drugs*:** Almost all the drugs given to mother will cross the placenta to reach the fetus. Possibility of pregnancy should be kept in mind while prescribing drugs to any woman of reproductive age (see p. 587).

GENERAL ADVICE: The patient should be persuaded to attend for antenatal checkup positively on the schedule date of visit. **She is instructed** to report to the physician even at an early date if some untoward symptoms arise such as intense headache, disturbed sleep with restlessness, urinary troubles, epigastric pain, vomiting and scanty urination.

She is advised to come to hospital for consideration of admission in the following circumstances:

- Painful uterine contractions at interval of about 10 minutes or earlier and continued for at least 1 hour—suggestive of onset of labor.
- Sudden gush of watery fluid per vaginam—suggestive of premature rupture of the membranes.
- Active vaginal bleeding, however slight it may be.

MINOR AILMENTS IN PREGNANCY

***Nausea and vomiting*:** Nausea and vomiting especially in the morning, soon after getting out of bed, are usually common in primigravidae. They usually appear following the first or second missed period and subside by the end of first trimester. 50% women have both nausea and vomiting, 25% have nausea only and 25% are unaffected.

Three main measures can reduce the problem. **Dietary changes:** To take dry toast, biscuits and protein rich meals. Frequent small foods are helpful. Fatty foods are avoided. **Behavior modification:** To avoid personal triggering factors. The woman can identify herself, this factor. Initial supplementation with vitamin B_1 and B_6 is started. **Medications** are discussed on page 587.

***Backache*:** It is a common problem (50%) in pregnancy. Physiological changes that contribute to backache are: joint ligament laxity (relaxin, estrogen), weight gain, hyperlordosis and anterior tilt of the pelvis. Other factors may be faulty posture and high heel shoes, muscular spasm, urinary infection or constipation. Excessive weight gain should be avoided. Rest with elevation of the legs to flex the hips may be helpful. Improvement of posture, well-fitted pelvic girdle belt which corrects the lumbar lordosis during walking and rest in hard bed often relieve the symptom. Massaging the back muscles, analgesics and rest relieve the pain due to muscle spasm.

***Constipation*:** Constipation is a quite common ailment during pregnancy. Atonicity of the gut due to the effect of progesterone, diminished physical activity and pressure of the gravid uterus on the pelvic colon are the possible explanations. Regular bowel habit may be restored with advice mentioned earlier.

***Leg cramps*:** It may be due to deficiency of diffusible serum calcium or elevation of serum phosphorus. Supplementary calcium therapy in tablet or syrup after the principal meals may be effective. Massaging the leg, application of local heat and intake of vitamin B_1 (30 mg) daily may be effective.

***Acidity and heartburn*:** Heartburn is common in pregnancy due to relaxation of the esophageal sphincter. Patient is advised to avoid over eating and not to go to bed immediately after the meal. Liquid antacids may be helpful. Hiatus hernia which is common during the pregnancy can also produce heartburn, especially when the patient is in lying down position. Sleeping in semi-reclining position with high pillows relieves the symptoms of hiatus hernia.

***Varicose veins*:** Varicose veins in the legs and vulva (varicosities) or rectum (hemorrhoids) may appear for the first time or aggravate during pregnancy, usually in the later months. It is due to obstruction in the venous return by the pregnant uterus. For leg varicosities, elastic crepe bandage during movements and elevation of the limbs during rest can give symptomatic relief. **Specific therapy is better to be avoided**. Varicosities usually disappear following delivery.

Hemorrhoids: It may cause annoying complications like bleeding or may get prolapsed. Regular use of laxative to keep the bowel soft, local application of hydrocortisone ointment and replacement of the piles if prolapsed are essential. **Surgical treatment is better to be withheld** as the condition sharply improves following delivery.

***Carpal tunnel syndrome (10%)*:** Woman presents with pain and numbness in the thumb, index and the middle finger. There is weakness in the muscles for thumb movements. This is due to compression effect on the median nerve. Physiological changes in pregnancy with retention of excess fluid are the common cause. Treatment is mostly symptomatic. A splint is applied during sleep time to the slightly flexed wrist to give relief. Corticosteroid injection or surgical decompression is rarely needed. It resolves spontaneously following delivery.

***Round ligament pain*:** Stretching of the round ligaments during movements in pregnancy may cause sharp pain in the groins. This pain may be unilateral or bilateral. It is usually felt in second trimester onwards. This is more common in right side as a result of dextrorotation of uterus. Pain may be awakening at night time because of sudden roll over movements during sleep. Pain may be reduced by making movements gradual instead of sudden. Local heat application is helpful. Analgesics are rarely needed.

***Ptyalism*:** Increased secretion of saliva is observed during pregnancy. It may be associated with increased intake of starch, though actual cause is not known. This problem is usually self-limiting and may be overcome by decreasing intake of carbohydrates. It is not associated with any adverse pregnancy outcome.

***Syncope*:** It is often seen in a woman following prolonged standing or standing upright abruptly. This is due to pooling of blood in the veins of the lower extremities. There is the effect of compression of the pelvic veins by the gravid uterus also. Other causes may be dehydration, hypoglycemia or overexertion. The woman presents with dizziness or light headedness on standing upright abruptly or following standing for a prolonged period.

Syncope usually resolves rapidly on lying in left lateral position. Syncope in supine position is also managed by resting in lateral recumbent position. Recurrent syncope needs cardiological evaluation.

Ankle edema: Excessive fluid retention as evidenced by marked gain in weight or evidences of preeclampsia has to be excluded. No treatment is required for physiological edema or orthostatic edema.

Edema subsides on rest with slight elevation of the limbs. **Diuretics should not be prescribed.**

Vaginal discharge: Assurance to the patient and advice for local cleanliness are all that are required. Presence of any infection (*Trichomonas, Candida, Bacterial vaginosis*) should be treated with vaginal application of metronidazole or miconazole (see p. 356).

EXERCISE IN PREGNANCY

A low impact exercise may be continued throughout the period of a normal pregnancy. However, physiologic changes of pregnancy may restrict certain types of exercises.

Limits of moderate intensity physical activity in pregnancy:

- Exercise should be regular (30 min/day), of low impact, and as a part of daily activites.
- Exercise should avoid any symptoms of breathlessness, fatigue or dizziness.
- Exercise should be done in a cool area without becoming uncomfortable and warm.
- Prolonged supine position, any compression to the uterus or risk of injury (fall) should be avoided.

Contraindications of Exercise and to Limit Physical Activity

■ Fetal growth restriction (FGR)	■ Vaginal bleeding (APH)
■ Cardiac or pulmonary disease	■ Hypertension in pregnancy
■ Cervical insufficiency	■ Risk for preterm labor

VALUES OF ANTENATAL CARE

The value of antenatal supervision is so much tested and recognized that it is needless to stress its importance. It should be borne in mind that a successful obstetric outcome depends on continued careful supervision which starts in pregnancy and ends in puerperal period. **Inadequacy of one cannot be compensated by the other.** The chief values are:

- **To screen the high risk cases.** Medical disorders and obstetric complications are sorted out at the earliest (Ch. 20). **Risk assessment is a continued process and not once only.**
- **Detection of high risk factors** deserves no credit unless proper steps are taken to rectify it. Cases need to be admitted, investigated and treated.
- **Pregnancy should be regularly supervised.** Casual antenatal visit or inadequate care is worse than no care at all. Efficacy of prenatal care depends on the **quality of care** given to the woman.
- **Antenatal care is said to be the strategy; the intranatal care is the tactic in obstetrics.** One is indispensible from the other to achieve a good result. Care should be **thorough** and based on **individual woman's need**.
- **Acceptance of advice:** During pregnancy, advice regarding diet, drugs, family planning guidance and immunization schedule are better followed than in the nonpregnant state.
- **It is an opportunity to make the patient realize** that childbirth is a physiological process and to boost up the psychology so that the patient finds herself confident during the ordeal of labor.
- **The net effect is marked reduction in maternal mortality** (about one-seventh) and morbidity. Similarly, there is significant **reduction in perinatal mortality (about one-fifth) and morbidity.**

DRAWBACKS

1. Trifling abnormality may be exaggerated for which unnecessary medication or risky operative interference is prescribed.
2. Unless quality of care is maintained in the antenatal clinic, the benefits of antenatal care are not obtained.
3. Good antenatal care only cannot reduce maternal and neonatal mortality and morbidity unless the woman gets good care during labor and postnatal period.

LIMITATIONS: Many complications in obstetrics often arise as emergency and without any warning during pregnancy, labor and puerperium. These are hemorrhage (APH, PPH), hypertension (eclampsia), premature rupture of membranes, unexplained intrauterine fetal death, cord prolapse or shoulder dystocia. These are the important causes of maternal morbidity and mortality in India. Simultaneous availability of **emergency obstetric care (EmOC)** should be (p. 683) there to combat these complications. **Therefore good antenatal care and efficient EmOC are complementary to each other for successful obstetric outcome.**

PRECONCEPTIONAL COUNSELING AND CARE

When a couple is seen and counseled about pregnancy, its course and outcome well before the time of actual conception is called **preconceptional counseling**. **Objective is to ensure that a woman enters pregnancy with an optimal state of health which would be safe both for herself and the fetus**. Organogenesis is completed by the first trimester. By the time the woman is seen first in the antenatal clinic, it is often too late to advice because all the adverse factors have already begun to exert their effects.

Preconceptual phase is the time to identify any risk factor that could potentially affect the perinatal outcome adversely. The woman is informed about the risk factor and at the same time care is provided to reduce or to eliminate the risk factor in an attempt to improve the pregnancy outcome. **Virtually preconceptional counseling is a part of preventive medicine**.

PRECONCEPTIONAL VISIT, RISK ASSESSMENT AND EDUCATION

- **Identification of high risk factors** by detailed evaluation of obstetric, medical, family and personal history. Risk factors are assessed by laboratory tests, if required.
- **Base level health status** including blood pressure is recorded.
- **Rubella and hepatitis immunization** in a nonimmune woman is offered (see p. 337, 349).
- **Folic acid supplementation** (4 mg a day) starting 4 weeks prior to conception up to 12 weeks of pregnancy (see p. 472), is advised. This can reduce the incidence of neural tube defects.
- **Maternal health is optimized preconceptionally**. Problems of overweight, underweight, anemia, abnormal papanicolaou smears are evaluated and treated appropriately.
- **Fear of the incoming pregnancy** is removed by preconceptional education.
- **Patient with medical complications** should be educated about the effects of the disease on pregnancy and also the effects of pregnancy on the disease. In extreme situation, the pregnancy is discouraged. Preexisting chronic diseases (hypertension, diabetes, epilepsy) are stabilized in an optimal state by intervention.
- **Drugs used before pregnancy** are verified and changed if required so as to avoid any adverse effect on the fetus during the period of organogenesis (see p. 587). For example, anticonvulsant drugs are checked (see p. 584), warfarin is replaced with heparin and oral antidiabetic drugs are replaced with insulin.
- **Woman should be urged to stop** smoking, taking alcohol and abusing drugs. Addicted woman is given specialized care.
- **Inheritable genetic diseases** (sickle cell disease, cystic fibrosis) are screened before conception and risk of passing on the condition to the offspring is discussed (see p. 316).
- **Importance of prenatal diagnosis** for chromosomal or genetic diseases is discussed (see p. 129, 132).
- **Inheritable genetic diseases** could be managed either by **primary prevention** (eliminating the causal factor) or **by secondary prevention** (terminating the affected fetus).
- **Couples with history of recurrent fetal loss** (see p. 195) or with family history of congenital abnormalities (genetic, chromosomal or structural) are investigated and counseled appropriately. There may be some **untreatable factors** (see p. 128).

Educational classes include discussion as regard delivery, timing, method and possible interventions (ventouse/forceps or cesarean delivery). Such prenatal classes are found helpful and valuable.

The counseling should be done by primary health-care providers. The help of an obstetrician, physician and geneticist may be required and should be extended.

LIMITATIONS: Unfortunately, only a small percentage of women take the advantage of preconceptual care. The important reasons are: (i) lack of public awareness, (ii) many pregnancies are unplanned.

Summary of Antenatal (Prenatal) Care

- **Careful history taking**
- **Thorough clinical examination** (general and obstetric)
- **Investigations** (routine and special)
- **Advice:** Diet (nutrition), hygiene, specific to any problem
- **Subsequent visits:** Every 4 weeks until 28 weeks then every 2 weeks until 36 weeks and weekly till delivery
  - A. **Maternal health:**
    - Weight, BP, Pallor, symptom analysis (nausea, dysuria, etc.). Any other abnormality
    - Identification of high risk factors
  - B. **Fetal health:** Fetal growth, fundal height, fetal heart rate, amniotic fluid volume, presentation and fetal activity
- **Couple education,** counseling and advice
- **Preparation for childbirth:** Couple is informed about labor course, delivery, the need of operations, anesthesia and the complications.

KEY POINTS

- **Pregnancy is a physiologic event. Most pregnancies are normal.**
- **Risk assessment**, early detection of risk factors and management, health education, advocacy, all are the key elements in antenatal care.
- **Preconceptional folic acid supplementation** (0.4 mg) should be given to all women planning pregnancy from 4 weeks before and to be continued at least 3 months thereafter.
- **Ultrasound examination** between 16 and 20 weeks is done to assess accurate gestational age, to detect fetal abnormality, viability and multiple pregnancy.
- Women should be monitored for optimum weight gain (24 lb or 11 kg) in pregnancy.
- Women should do their normal activities. Heavy weight lifting or excessive physical activity should be avoided.
- **Antenatal care is a continued primary and preventive health care**. Subsequent visits are done to assess maternal and fetal well-being (see p. 106). List of warning signs should be explained to her (p. 111).
- **Diet in pregnancy** should ideally be light, frequent, easily digestible and rich in protein, minerals and vitamins (p. 112).

QUESTIONS

1. Define antenatal care? What are the aims and objectives of antenatal care? (p. 106)
2. Mention the different investigations done during antenatal care? (p. 110)
3. Discuss in brief the preconception counseling and care? (p. 116)

Chapter 11

Antenatal Assessment of Fetal Well-Being

Majority (80%) of fetal deaths occur in the antepartum period. **The important causes of deaths are:** (i) Chronic fetal hypoxia (IUGR). (ii) maternal complications, e.g. diabetes, hypertension, infection; (iii) fetal congenital malformation and (iv) unexplained cause.

There is progressive decline in maternal deaths all over the world. Currently, more interest is focused to evaluate the fetal health. **The primary objective of antenatal fetal assessment is to avoid fetal death**. As such simultaneously with good maternal care during pregnancy and labor, the fetal health in utero should be supervised with equal vigilance.

Aims of antenatal fetal monitoring:

- To ensure satisfactory growth and well-being of the fetus throughout pregnancy.
- To screen out the high-risk factors that affect the growth of the fetus.

Rationality of Antenatal Fetal Tests

- Tests must provide information superior to that of clinical evaluation
- Test results should be helpful in management to improve perinatal outcome
- Benefits of tests must outweigh the potential risks and the costs

The Measures That can be Taken When a Fetus is Found Compromised

- Bed rest
- Fetal surveillance
- Drug therapy
- Urgent delivery of the fetus—term or preterm
- Neonatal intensive care (NIC)
- Termination of pregnancy for fetal congenital anomaly

CLINICAL EVALUATION OF FETAL WELL-BEING AT ANTENATAL CLINIC

FIRST VISIT

The initial antenatal examination should be carried out in the first trimester. At this examination a record is kept of the size of the uterus following bimanual examination or by ultrasonography. This is of immense help in estimating the correct duration of gestation in the last trimester.

Fetal well-being depends on satisfactory maternal health throughout pregnancy. After a thorough clinical examination of the mother, the investigations are initiated as early as possible (see p. 109).

SUBSEQUENT VISITS

At every antenatal visit, the following clinical parameters are taken into account for assessment of satisfactory progress of gestation.

1. **Maternal weight gain:** During the second half of pregnancy, the average weight gain is 1 kg a fortnight. Any excess weight gain may be due to excess fluid retention and could be the first sign of pre-eclampsia. If the weight gain is less than normal, stationary or even falling, one should be on the look-out for intrauterine growth restriction.

2. **Blood pressure:** Initial recording of blood pressure prior to 12 weeks helps to differentiate a pre-existing chronic hypertension from a pregnancy-induced hypertension developing later on. Hypertension, pre-existing or pregnancy-induced, may impair the fetal growth (p. 277).

3. **Assessment of the size of the uterus and height of the fundus:** In early weeks, the size of the uterus is of great value in confirming the calculated duration of gestation. The height of the fundus should be documented at each visit. *The top of the uterine fundus is measured from the superior border of the symphysis pubis (bladder should be empty)* using a tape. **After 24 weeks of pregnancy, the distance measured in cm normally corresponds to the period of gestation in weeks**. A variation of 1–2 cm is acceptable.

Provided the patient is sure about her **date of last normal menstrual period,** a measurement of **symphysis-fundal height** in later month of pregnancy is a useful **screening test for further investigation**. The measurement is compared to the expected distance plotted on a chart (Fig. 11.1). **If the measurement falls below the 10th centile**, fetal growth restriction is suspected and more specific investigation should be done.

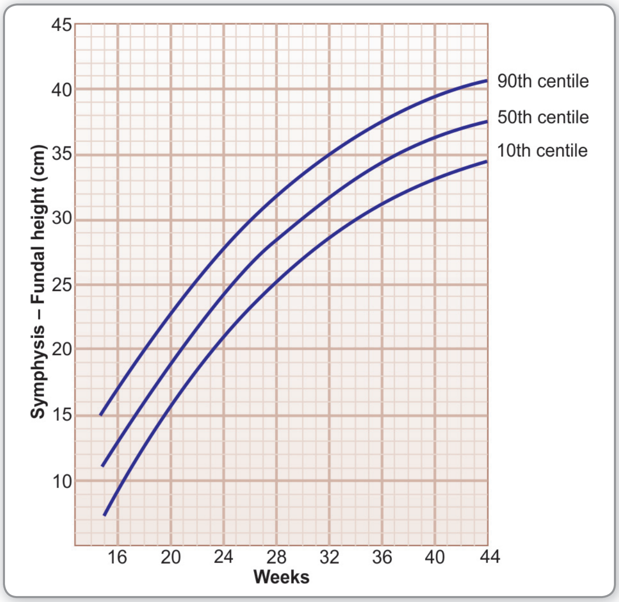


Fig. 11.1: Gestational age chart estimated from symphysis-fundal height

4. **Clinical assessment of excess liquor** should be recorded, as well as any **scanty liquor** in the last trimester. Evidence of scanty liquor may indicate placental insufficiency and the need for undertaking other placental function tests.

5. **Documentation of the girth of the abdomen in the last trimester** of pregnancy should form a routine part of abdominal examination. This is measured at the lower border of the umbilicus. Normally, the girth increases steadily up to term. If the girth gradually diminishes beyond term or earlier, it arouses suspicion of placental insufficiency. This is of particular value in suspecting placental insufficiency in the high-risk cases such as pre-eclampsia, chronic hypertension and IUGR.

SPECIAL INVESTIGATIONS

About 30% of antepartum fetal deaths are due to asphyxia (IUGR, post-dates), 30% due to maternal complications (pre-eclampsia, placental abruption, diabetes mellitus), 15% due to congenital malformations and chromosomal abnormalities and 5% due to infection. About 20% of stillbirths have no obvious cause. **About 50% of first trimester spontaneous abortions and about 5% of stillborn infants have chromosomal abnormalities**.

Congenital abnormalities may be: (1) **Chromosomal**: numerical (47 XXX) or structural (translocations), (2) **single gene** (cystic fibrosis), (3) **polygenic and multifactorial** (NTDs) and (4) **teratogenic disorders** (drugs).

Apart from **clinical evaluation**, **biochemical and biophysical methods** have also been used for the diagnosis. Some of these methods carry risks to the mother and/or the fetus and are also expensive. **Therefore, their application should provide definite benefits that clearly outweigh the potential risks and the costs**.

EARLY PREGNANCY

♦ **Biochemical** ♦ **Biophysical** ♦ **Cytogenetic**

Antenatal assessment of fetal well-being in early pregnancy is primarily designed to detect fetal congenital abnormalities. Therefore, this chapter should be read in conjunction with chapter 12 (p. 128). The candidates for **prenatal screening** are mentioned in p. 128. Women who are screen positive should be offered fetal karyotyping for confirmation.

ANTEPARTUM FETAL SURVEILLANCE (LATE PREGNANCY)

OBJECTIVES ARE (ACOG)—(1) Prevention of fetal death and (2) avoidance of unnecessary interventions.

METHODS: ▪ **Clinical** ▪ **Biochemical** ▪ **Biophysical**

CLINICAL: The clinical assessment of fetal growth can be evaluated by the parameters mentioned earlier in the chapter. **They may be useful as screening test for further investigation**.

BIOCHEMICAL: Biochemical tests are mainly done for assessment of **pulmonary maturity** (see p. 124).

BIOPHYSICAL: Principle—*Biophysical profile* is a **screening test** for utero-placental insufficiency. The fetal biophysical activities are initiated, modulated and regulated through fetal nervous system. The fetal CNS is very much sensitive to diminished oxygenation. Hypoxia → metabolic acidosis → CNS depression → changes in fetal biophysical activity.

The following biophysical tests are used:

(1) Fetal movement count
(4) Non-stress test (NST)
(7) Vibroacoustic stimulation test
(2) Ultrasonography
(5) Fetal biophysical profile (BPP)
(8) Contraction stress test (CST)
(3) Cardiotocography
(6) Doppler ultrasound
(9) Amniotic fluid volume

Fetal movement count—Any of the two methods can be applied:

- **Cardif "count 10" formula:** The patient counts fetal movements starting at 9 am. The counting comes to an end as soon as 10 movements are perceived. **She is instructed to report the physician if—**(i) less than 10 movements occur during 12 hours on 2 successive days or (ii) no movement is perceived even after 12 hours in a single day.
- **Daily fetal movement count (DFMC):** Three counts each of 1 hour duration (morning, noon and evening) are recommended. The total counts multiplied by four gives daily (12 hour) fetal movement count (DFMC). **If there is diminution of the number of "kicks" to less than 10 in 12 hours** (or less than 3 in each hour), it indicates fetal compromise.
- Mothers perceive 88% of the fetal movements detected by Doppler imaging. **The count should be performed daily starting at 28 weeks of pregnancy**.

Loss of fetal movements is commonly followed by disappearance of FHR within next 24 hours. In either of the earlier methods, if the result is ominous, the candidate is subjected to NST. Maternal hypoglycemia is associated with increased fetal movements. **Maternal perception of fetal movements may be reduced** with fetal sleep (quiet), fetal anomalies (CNS), anterior placenta, hydramnios, obesity, drugs (narcotics), chronic smoking and hypoxia.

Non-stress test (NST): In non-stress test, a continuous electronic monitoring of the fetal heart rate along with recording of fetal movements (cardiotocography) is undertaken. **There is an observed association of FHR acceleration with fetal movements, which when present, indicates a healthy fetus.** It can reliably be used as a **screening test**. The accelerations of the FHR associated with fetal movements are presumably reflex mediated. It should be emphasized that **the test is valuable to identify the fetal wellness rather than illness**.

Interpretation

- **Reactive (Reassuring)—**When two or more accelerations of more than 15 beats per minute above the baseline and longer than 15 seconds in duration are present in a 20 minute observation (see p. 693).
- **Non-reactive (Non-reassuring)**—Absence of any fetal reactivity.

A **reactive NST** is associated with perinatal death of about 5 per 1,000. But perinatal death is about 40 per 1,000 when the **NST is nonreactive**. Testing should be started after 30 weeks and frequency should be twice weekly. The test has a *false negative rate of 0.5% and false positive rate of 50%.*

Vibroacoustic stimulation (VAS) is used (see p. 696) to change the fetal sleep state from quiet (non-REM) to active (REM) sleep. **A reactive NST after VAS indicates a reactive fetus**. The procedure is harmless.

Fetal Biophysical Profile (BPP)—considers several parameters (see Tables 11.1 and 11.2). BPP using real time ultrasonography has a high predictive value.

Indications—Non-reactive NST, high-risk pregnancy. **Test frequency** weekly after a normal NST, and twice weekly after an abnormal test.

Modified Biophysical Profile consists of NST and ultrasonographically determined amniotic fluid index (AFI). Modified BPP is considered abnormal (nonreassuring) when the NST is non-reactive and/or the AFI is < 5.

Table 11.1: Biophysical Profile Scoring (Manning—1992)

Observation for 30 minutes
Normal score = 2; Abnormal = 0

Parameters	Minimal Normal Criteria	Score
Non-stress Test (NST)	Reactive pattern (p. 122)	2
Fetal breathing movements	≥ 1 episode lasting > 30 second	2
Gross body movements	≥ 3 discrete body/limb movements	2
Fetal muscle tone	≥ 1 episode of active extension (limb or trunk) with return of flexion; opening and closing of hand, considered normal	2
Amniotic fluid	≥ 1 pocket measuring 2 cm in two perpendicular planes (2 × 2 cm pocket)	2

Table 11.2: BPP Scoring, Interpretation and Management

BPP Score	Interpretation	Management
8–10	Normal; Less risk of fetal asphyxia	Repeat testing at weekly interval or more
6	Suspect chronic asphyxia	If ≥ 36 weeks → deliver; but if L/S < 2.0 repeat test in 4–6 hours
4	Suspect chronic asphyxia ↓	If ≥ 36 weeks deliver, if < 32 weeks repeat testing in 4–6 hours
0–2	Strongly suspect asphyxia	Test for 120 minutes ↓ persistent score ≤ 4 ↓ deliver regardless of gestational age

An abnormal score of 4 or less is associated with fetal acidemia. Abnormal BPP is associated with high risks of stillbirth and perinatal mortality.

Fetal Cardiotocography (CTG): A normal tracing after 32 weeks, would show baseline heart rate of 110–160 beats per minute (bpm) with an amplitude of baseline variability 5–25 bpm. There should be no deceleration or there may be early deceleration of very short duration. Importantly, **there should be two or more accelerations during a 20-minute period** (see p. 693).

Ultrasonography: IUGR can be diagnosed accurately with serial measurement of BPD, AC, HC, FL and amniotic fluid volume. **AC is the single measurement which best reflects fetal nutrition**. The average increase of biparietal diameter beyond 34 weeks is 1.7 mm per week. When the HC/AC ratio is elevated (> 1.0) after 34 weeks, IUGR is suspected (see p. 535). Ultrasound examination is the main diagnostic tool to assess fetal growth.

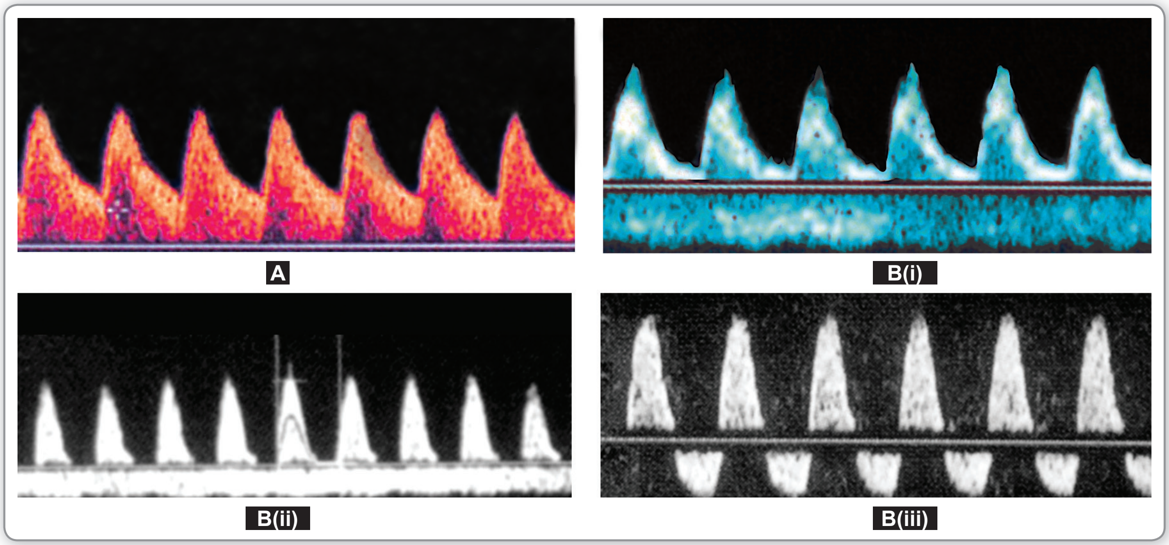
Amniotic fluid volume (AFV): Amniotic fluid volume is primarily dependent upon the fetal urine output, pulmonary fluid production and fetal swallowing. Decreasing AFV may be the result of fetal hypoxia and placental insufficiency. A vertical pocket of amniotic fluid ≥ 2 cm is considered normal. Amniotic fluid index (AFI) is the sum of vertical pockets from four quadrant of uterine cavity (see p. 44). AFI ≤ 5 is associated with increased risk of perinatal mortality and morbidity.

Doppler Ultrasound Velocimetry:

Doppler flow velocity waveforms are obtained from arterial and venous beds in the fetus (Figs 11.2A and B). **Arterial Doppler (umbilical artery)** waveforms are helpful to assess the downstream vascular resistance. The arterial Doppler waveform is used to measure the peak systolic (S), peak diastolic (D) and mean (M) volumes. From these values **S/D ratio**, **pulsatility index (PI)** [PI = (S–D)/M] or **resistance index (RI)** [RI = (S–D)/S] are calculated.

In a normal pregnancy the S/D ratio, PI and RI decreases as the gestational age advances. Higher values greater than 2 SDs above the gestational age mean indicate reduced diastolic velocities and increased placental vascular resistance. These features are at increased risk for adverse pregnancy outcome.

Venous Doppler (Ductus Venosus, Umbilical Vein) parameters provide information about cardiac forward function (cardiac compliance, contractility and after-load). Fetuses with abnormal cardiac function show pulsatile flow in the umbilical vein (UV). Normal UV flow is monophasic (see Table 11.3).



Figs 11.2A and B: Umbilical artery flow velocity waveform: (A) Normal; (B) Abnormal—(i) Reduced end-diastolic flow; (ii) Absent end-diastolic flow; (iii) Reversed end-diastolic flow

Table 11.3: Antenatal Doppler Ultrasound Changes and the Suggestive Features of a Compromised Fetus

Vessel	Change	Pathophysiological Basis	Clinical Significance
Umbilical artery (UA)	Reduced or absent or reversed end diastolic flow (Fig. 11.2)	Failure of villous trophoblast invasion (see p. 37, 39)	↑ resistance in fetoplacental circulation → IUGR, pre-eclampsia
Middle cerebral artery (MCA)	↑ Diastolic velocity; ↓ S/D or Pulsatory index	Dilatation of cerebral vessels	"Brain Sparing" effect in response to hypoxemia
Ductus venosus (DV)	↑ Doppler index*; Absent/ Reversed flow (a-wave)	↑ Central venous pressure (CVP)	Fetal acidemia
Umbilical vein (UV)	↑ Doppler index; Pulsatile flow	↑ CVP or ↓ Cardiac compliance	Fetal acidemia

* Increased Doppler indices means there is increased vascular flow resistance.

The fetuses having UA Doppler flow abnormalities (AEDV or REDV) are at higher risk of intrauterine hypoxia. Risk of stillbirth is high when the Doppler flow in the ductus venosus (venous parameter) is abnormal.

Use of ultrasonography for fetal biometry and Doppler study for umbilical artery flow velocimetry has reduced perinatal mortality and unnecessary early intervention significantly.

Contraction stress test (CST) is based to observe the response of the fetus at risk for uteroplacental insufficiency in relation to uterine contractions (see p. 576).

OTHER INVESTIGATIONS IN LATE PREGNANCY

Amniocentesis in late pregnancy:

- **Test for fetal pulmonary maturity**
- **Assessment of severity of Rh iso-immunization**

Pulmonary maturity: Confirmation of lung maturation reduces the incidence of **respiratory distress syndrome (RDS) in the newborn**. The risk of RDS (p. 547) is high for infants that are delivered preterm (< 37 weeks). **RDS is caused by** the deficiency of pulmonary surfactant (see p. 547), which is synthesized by the type II alveolar cells. Surfactant is packaged in lamellar bodies → discharged in the lung alveoli → carried in the pulmonary fluid → carried into the amniotic fluid.

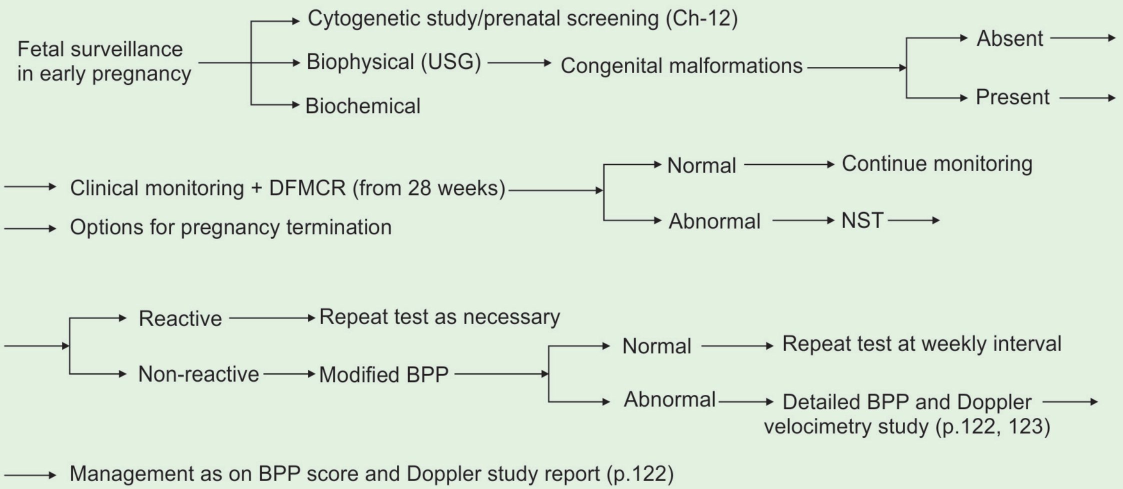
Assessment of fetal pulmonary maturity:

1. For evaluation of fetal pulmonary maturity the sample of amniotic fluid should be obtained by amniocentesis. Amniocentesis should be done under ultrasound guidance (p. 742).
2. **Estimation of pulmonary surfactant** by lecithin/sphingomyelin (L/S) ratio. Amniotic fluid L/S ratio at 31–32 weeks is 1; at 35 weeks L/S ratio is 2. L/S ratio ≥ 2 indicates pulmonary maturity.
3. **Shake test or Bubble test (Clement's):** This is a useful bedside test, rapidly performed with a fair degree of accuracy. The test is based on the ability of pulmonary surfactant to form a foam or bubble, on shaking which remains stable for at least 15 minutes. Increasing dilutions of amniotic fluid are mixed with 96% ethanol, shaken for 15 seconds and inspected after 15 minutes for the presence of **a complete ring of bubbles at the meniscus. If it is present, the test is positive and indicates maturity of the fetal lungs**.
4. **Foam Stability Index (FSI)** is based on surfactant detection by *shake test* (see above). FSI is calculated by utilizing serial dilutions of amniotic fluid to quantitate the amount of surfactant present. FSI > 47 virtually excludes the risk of RDS.
5. **Presence of phosphatidylglycerol (PG)** in amniotic fluid reliably indicates lung maturation. PG is tested by thin layer chromatography similar to L:S measurement.

6. **Saturated phosphatidylcholine** ≥ 500 ng/mL indicates pulmonary maturity.
7. **Fluorescence polarization:** This test utilizes polarized light to quantitate surfactant in the amniotic fluid. The ratio of surfactant to albumin is measured by an automatic analyzer. Presence of 55 mg of surfactant per gram of albumin indicates fetal lung maturity.
8. **Amniotic fluid optical density** at 650 nm greater than 0.15 indicates lung maturity.
9. **Lamellar body** is the storage form of surfactant in the amniotic fluid. They can be counted as the size is same as that of platelets. A lamellar body count > 30,000/μL indicates pulmonary maturity.
10. **Orange colored cells**—Desquamated fetal cells obtained from the centrifuged amniotic fluid are stained with 0.1% Nile blue sulfate. Presence of orange colored cells > 50% suggests pulmonary maturity.
11. **Amniotic fluid turbidity:** During first and second trimesters, amniotic fluid is yellow and clear. At term it is turbid due to vernix.

Assessment of severity of Rh–isoimmunization is done by amniocentesis for estimation of bilirubin in the amniotic fluid by spectrophotometric analysis. The optical density difference at 450 nm gives the prediction of the severity of fetal hemolysis (see p. 392).

SUMMARY: ANTENATAL ASSESSMENT OF FETAL WELL-BEING


Fetal surveillance in early pregnancy
Cytogenetic study/prenatal screening (Ch-12)
Biophysical (USG)
Biochemical
Congenital malformations
Absent
Present
Clinical monitoring + DFMCR (from 28 weeks)
Options for pregnancy termination
Normal
Continue monitoring
Abnormal
NST
Reactive
Repeat test as necessary
Non-reactive
Modified BPP
Normal
Repeat test at weekly interval
Abnormal
Detailed BPP and Doppler velocimetry study (p.122, 123)
Management as on BPP score and Doppler study report (p.122)

KEY POINTS

- About 20% of cases with fetal death remain unexplained. Risk increases with increasing gestational age.
- **DFMC** is a simple, widely used method for monitoring of fetal well-being. The principle is—there is decrease in fetal movements when there is fetal hypoxemia.
- **Fetal movement count** by the mother is an ideal first-line screening test both for high-risk and low-risk patients. A healthy fetus should have minimum 10 movements in 12-hour period. Count should be done daily beginning at 28 weeks (p. 121). Mothers perceive 88% of fetal movements detected by sonography.
- **Fetal cardiac accelerations** are associated with fetal movements in more than 85% of the time. A reactive NST requires at least two accelerations of FHR in 20 minutes of monitoring (p. 122).
- **At term** a fetus spends approximately 25% of its time in quiet sleep state (NREM) and 70% in active sleep state (REM).

contd...

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- **Vibroacoustic stimulation** (VAS) can change fetal state from quiet (non-REM) sleep to active (REM) sleep and it is harmless. A reactive NST after VAS indicates a healthy fetus (see p. 122).
- **The observation of FHR** accelerations in response to fetal activity or stimulation to the fetus, indicates fetal well being.
- **NST** should be done twice weekly in complicated pregnancies (diabetes mellitus, IUGR). **NST** has a low false negative rate (< 1%) but a high false positive rate (> 50%) (p. 122).
- **Ultrasound examination** is an essential tool for pregnancy dating, detecting structural and/or chromosomal anomalies, growth profile and well-being of the fetus (p. 733). Important parameters for fetal growth assessment are: BPD, HC, AC, FL and amniotic fluid volume.
- **FBPP** is assessed by using real time sonography. The fetal biophysical activities that appear first are the last to disappear with fetal hypoxia. BPP correlates well with fetal acid base status. False negative rate of normal BPP is less than 0.1%.
- **Fetal BPP** includes NST, fetal breathing and gross body movement. Management is based on total score (see p. 122). A score of 6 is suspicious and should be repeated.
- **Modified BPP** includes NST and AFI. It takes less time. BPP has lower false positive rate than NST and high positive predictive value (p. 122).
- **Cardiotocography** is the cornerstone of antenatal fetal assessment (see p. 693). A normal trace should have a baseline fetal heart rate of 110-160 bpm, variability of 5–25 bpm and at least two accelerations in a 20-minute period (p. 122).
- **In a normal pregnancy the S/D ratio and the resistance index (RI)** decrease as pregnancy advances (p. 123).
- **Absent or reversed end-diastolic flow velocity** in the umbilical artery is associated with an increase in perinatal mortality and morbidity (p. 123).
- **Abnormal Doppler flow in the ductus venosus** is associated with increased perinatal morbidity and mortality (p. 687).
- **Ultrasonography and Doppler flow velocimetry** when used in antenatal fetal well being assessment, the risk of perinatal mortality and unnecessary early intervention for delivery, could be reduced significantly.
- **Fetal pulmonary maturity** can be accurately predicted by amniotic fluid tests (L/S ratio, Lamellar body count).

QUESTIONS

1. How antenatal assessment of fetal well-being could be done clinically? (p. 121)
2. Mention the different biophysical tests for antenatal fetal surveillance? (p. 121)

Write Short Notes on:

A. Nonstress test (p. 122)

B. Biophysical profile (p. 122)

Chapter 12

Prenatal Genetic Counseling, Screening and Diagnosis

Genetic Counseling: Nearly 3% of newborns have major congenital anomaly. Usually genetic factors are responsible. Chromosomal abnormalities are observed in majority of all first trimester miscarriages and about 5% of all stillborns. The different etiologic factors for fetal malformations are:

1. Chromosomal abnormalities (numeric or structural)
2. Single gene disorders (cystic fibrosis)—1%
3. Polygenic or multifactorial disorders
4. Teratogenic disorders due to exposure of exogenous factors (drugs).

Prenatal genetic counseling, screening and diagnosis are done to evaluate a fetus with risk of chromosomal, genetic abnormality or a structural anomaly. Couple is communicated with the basic knowledge of genetic abnormalities. Different possible causes are discussed. Written information (leaflets) may be handed over as that allows the couple for discussion among themselves. Couples are encouraged to ask questions. Women's or couples' risk assessment for having a baby with increased risk of genetic disease should be done based on the ethnicity, race, personal (age, drug history) or family history. In cases where the risk is high, couple needs additional counseling by a genetic counselor. **Noninvasive prenatal screening for aneuploidy or neural tube defects is offered to all women regardless of age.**

The means to diagnose such abnormality is to obtain fetal tissue or cells by: (a) chorionic villus sampling (CVS), (b) amniocentesis, (c) cordocentesis, (d) fetal cells from maternal blood, (e) free fetal DNA (ffDNA) from maternal plasma/blood, (f) ultrasonography (USG) and (g) fetal echocardiography.

PRENATAL GENETIC SCREENING

Aims: Detection and identification of couples (individuals) who are at high risk for having a child with an inherited (chromosomal or genetic) disorder.

Noninvasive screening for chromosomal anomaly (trisomy 21, 18, 13) should be a routine to all pregnant women, irrespective of their age. Women who are screen positive should be offered fetal karyotyping for confirmation.

Table 12.1: Risk Factors for Prenatal Genetic Screening

Maternal Risk Factors	Prenatal Risk Factors
■ Maternal age > 35 years	■ Oligohydramnios (see p. 250)
■ Family history of neural tube defects	■ Polyhydramnios (see p. 250)
■ Previous baby born with neural tube defect	■ Severe symmetrical fetal growth restriction (see p. 534)
■ Previous child with chromosomal anomaly	■ Abnormal ultrasound findings (structural anomalies)
■ One or both parents—carriers of sex-linked or autosomal traits	■ Uncontrolled diabetes mellitus in the periconceptional period (see p. 325)
■ One parent is known to carry a balanced translocation	■ Contact with infection (teratogenic), e.g. rubella, cytomegalovirus (see p. 348) or intake of teratogenic drugs (see p. 587)
■ History of recurrent miscarriage	■ Presence of soft tissue markers of chromosomal anomaly on ultrasonography (see p. 735)
	■ Abnormal maternal serum screening (see p. 128)

PROCEDURES FOR EARLY DETECTION OF FETAL (I) Genetic; (II) Chromosomal and (III) Structural Abnormalities

- Triple test (combined test): MSAFP, unconjugated estriol (uE3) and hCG (see p. 106)
- Quadruple test: (MSAFP, uE3, hCG, inhibin A)
- Integrated test: NT, PAPP-A + hCG (see p. 106)
- Cell-free fetal DNA from maternal plasma/blood
- Maternal serum alpha fetoprotein (MSAFP) see p. 106
- Chorionic villus biopsy (see p. 129)
- Cordocentesis (see p. 130)
- Amniocentesis
- Fetal cell isolation from maternal blood—(from isolated fetal nucleated red blood cells or trophoblast cells)
- High resolution ultrasonography (see p. 732) for (nuchal thickening, nasal bone)
- Peri-implantation genetic diagnosis (see p. 132)
- 3D or 4D ultrasound with increased resolution
- Fetoscopy (see p. 132)

BIOCHEMICAL ANALYTES

a. Maternal serum alpha fetoprotein (MSAFP): AFP is an oncofetal protein (molecular weight 70,000). It is produced by yolk sac and fetal liver. Highest level of AFP in fetal serum and amniotic fluid is reached around 13 weeks and thereafter it decreases. Maternal serum level reaches a peak around 32 weeks. **MSAFP level is elevated** in a number of conditions: (a) wrong gestational age, (b) open neural tube defects (NTDs), (c) multiple pregnancy, Rh isoimmunization, (d) IUFD, (e) anterior abdominal wall defects and (f) renal anomalies. **Low levels** are found in trisomies (Down's syndrome), gestational trophoblastic disease.

b. Inhibin A is a dimeric glycoprotein. It is produced by the corpus luteum and the placenta. Serum level of inhibin A is raised in women carrying a fetus with Down's syndrome.

c. Others: hCG (see p. 213); uE3 (see p. 129); PAPP (see p. 68).

SCREENING METHOD

First Trimester Screening

Screening parameters are: (A) **Biophysical:** (i) ultrasound measurement of nuchal translucency (NT), (ii) Nasal bone, (B) **Biochemical:** (i) free β-hCG, (ii) PAPP-A (Pregnancy Associated Plasma Protein-A).

Time of Test: Between 11 weeks and 14 weeks.

Values: PAPP-A—reduced; β-hCG—increased; NT—measurement increased in trisomy 21.

NT is the fluid-filled space (detected by USG) between the fetal skin and the underlying soft tissue at the region of the fetal neck. NT ≥ 3 mm is abnormal. Combined tests can detect tristomy 21 in 92% cases with a false-positive rate of 5%.

First trimester screening is either equal or even superior to second trimester screening.

Advantages: Once a woman is screen positive, diagnostic tests should be done early.

A targeted ultrasound examination during the second trimester and fetal echocardiography are to be done when NT is ≥ 3 mm.

Second Trimester Screening

It is done between 15 weeks and 22 weeks.

MSAFP: This test is done between 15 weeks and 20 weeks. MSAFP value of 2.5 multiples of the median (MOM) when adjusted with maternal weight and ethnicity is taken as cut-off point. Elevated MSAFP detects 85% of all neural tube defects. Cases with such high values are considered for high resolution ultrasound imaging and/or amniocentesis. Very low MSAFP levels are associated with increased rates of miscarriage, stillbirth and neonatal death.

Triple Test: It is a combined biochemical test which includes MSAFP, hCG and uE3 (unconjugated estriol). Maternal age in relation to confirmed gestation age is also taken into account. It is used for detection of Down's syndrome. In an affected pregnancy, levels of MSAFP and uE3 tend to be low while that of hCG is high. It is performed at 15–22 weeks. It gives a risk ratio and for confirmation CVS/amniocentesis has to be done. The result is considered to be screen positive if the risk ratio is 1:250 or greater.

Quadruple (Quad) Screening includes four biochemical analytes: (1) Maternal Serum Alpha Fetoprotein (MSAFP), (2) Unconjugated estriol (uE3), (3) dimeric inhibin-A and (4) hCG.

Quadscreen can detect trisomy 21 in 85% of cases with a false-positive rate of 0.9%. Levels of serum analytes in cases with trisomy 21: hCG—increased; uE3—reduced; inhibin A—elevated; MSAFP—reduced.

Adjustments are to be made for maternal age, weight and ethnic group.

Best screening procedure is combined first and second trimester procedures (ACOG).

PRENATAL DIAGNOSIS

Screen positive women are offered fetal karyotyping. Fetal tissues are obtained for confirmation of diagnosis. The procedures are: (a) invasive and (b) noninvasive.

Invasive Procedures for Prenatal Diagnosis:

- **Chorionic Villus Sampling (CVS)**
- **Amniocentesis**
- **Cordocentesis or Percutaneous Umbilical Blood Sampling (PUBS)**

■ **Chorionic Villus Sampling (CVS)** is performed for prenatal diagnosis of **genetic disorders**. It is carried out **transcervically** between 10 weeks and 13 weeks and **transabdominally** from 10 weeks to term. Diagnosis can be obtained by 24 hours, and as such, if termination is considered, it can be done in the first trimester safely. A few villi are collected from the chorion frondosum under ultrasonic guidance with the help of a long malleable polythylene catheter with a metal obturator introduced transcervically (TC-CVS) along the extraovular space. The obturator is then withdrawn. About 15–25 mg of villi are aspirated in a 20 mL syringe creating a negative pressure. The tissues are obtained in a tissue culture media within the syringe. Transabdominal (TA-CVS) is done using a spinal needle (18–20 gauge) under ultrasound guidance. It provides earlier diagnosis than amniotic fluid studies. **Complications are:** fetal loss (1–2%), oromandibular limb deformities or vaginal bleeding. False-positive results (2–3%) are there due to placental mosaics and maternal cell contamination. In such a situation, amniocentesis should be performed to confirm the diagnosis. **Limb reduction deformity (LRD)** is low when CVS is performed after 9 completed weeks of gestation. CVS performed between 10 weeks and 13 weeks of gestation is safe and accurate as that of amniocentesis. Placenta biopsy has mostly replaced cordocentesis. This procedure is of low risks, technically easier and cytogenetic results are obtained within 24–48 hours. Pregnancy termination when needed can be done a safely in the early weeks of gestation.

TC-CVS is avoided in cases with, cervical myoma, acutely angulated uterus, uterine malformations or in presence of infections, such as the genital herpes or cervicitis or in presence of vaginal bleeding. Anti-D immunoglobulin 50 μg IM should be administered following the procedure to a Rh-negative woman. The information obtained by CVS, amniocentesis or cordocentesis is discussed below.

- **Cytogenetic diagnosis:** Fetal trophoblast cells from CVS or the desquamated fetal cells in the amniotic fluid obtained by amniocentesis or fetal blood cells obtained by cordocentesis are cultured, G-banded and examined to make a diagnosis of chromosomal anomalies, e.g. trisomy 21 (Down's syndrome), monosomy X (Turner's syndrome) and others.
- **DNA analysis:** Single gene disorders (cystic fibrosis, Tay-Sachs disease) can be diagnosed using specific DNA probes. DNA amplification is done by polymerase chain reaction (PCR) or chromosomal microarrays. The specific chromosomal region containing the mutated gene can be indentified.
- **Biochemical:** Amniotic fluid AFP level is high when the fetus suffers from open neural tube defects. This is also confirmed by ultrasound scanning. The normal AFP concentration in liquor amnii at the 16th week is about 20 mg/L. Amniotic fluid level of 17-hydroxy-progesterone is raised in congenital adrenal hyperplasia.

Structural chromosomal abnormalities (translocations, inversions, mutations) can be detected by **fluorescence in situ hybridization (FISH)**. Chromosome-specific probes can be used to detect the unknown DNA.

■ **Genetic Amniocentesis** is an invaisve procedure. It is performed after 15 weeks under ultrasonographic guidance (see p. 741). The fetal cells obtained in this procedure are subjected for cytogenetic analysis.

Early amniocentesis has been carried out at 12–14 weeks of gestation. Amnifiltration has been used to increase the cell yield. Genetic amniocentesis before 13 weeks is not recommended (ACOG).

Table 12.2: Prenatal Diagnosis: CVS, Amniocentesis and Cordocentesis

	Chorionic Villus Sampling	Amniocentesis	Cordocentesis
Time	Transcervical 10–13 weeks, Transabdominal 10 weeks to term	After 15 weeks (early 12–14 weeks)	18–20 weeks
Materials for study	Trophoblast cells	• Fetal fibroblasts • Fluid for biochemical study (see p. 741)	• Fetal white blood cells (others—infection and biochemical study)
Karyotype result	• Direct preparation: 24–48 hours. • Culture: 10–14 days	• Culture: 3–4 weeks	• Culture: 24–48 hours
Fetal loss	0.5–1%	0.5%	1–2%
Accuracy	Accurate; may need amniocentesis for confirmation	Highly accurate	Highly accurate
Termination of pregnancy when indicated	1st trimester—safe	2nd trimester—risky	2nd trimester–risky
Maternal effects following termination of pregnancy	Very little	More traumatic; physically and psychologically	Same as amniocentesis

FETAL BLOOD SAMPLING

■ Cordocentesis (Percutaneous Umbilical Blood Sampling)

A 22-gauge spinal needle, 13 cm in length, is inserted through the maternal abdominal and uterine wall under real-time ultrasound guidance using a curvilinear probe. The needle tip is progressed carefully and it punctures the umbilical vein approximately 1–2 cm from the placental insertion. Umbilical vein is preferred. The advantages are: (a) vein is larger in size (b) causes less bradycardia and (c) less hemorrhage.

Generally, 0.5–2 mL of fetal blood is collected. It is performed under local anesthetic usually from 18 weeks of gestation.

RISKS: This invasive procedure may lead to abortion, preterm labor and intrauterine fetal death. These may be due to bleeding, cord hematoma formation, infection (amnionitis), fetomaternal hemorrhage or preterm rupture of membranes. Overall fetal loss is 1–2%. Anti-D immunoglobulin 100 µg IM should be given to Rh-negative, yet unimmunized woman.

All the information as obtained in amniocentesis or chorion villus sampling, could be gathered. Additional values are mentioned below.

Hematological	—	For fetal anemia (see p. 388), bleeding disorders (autoimmune thrombocytopenia), Rh disease (see p. 386) and hemoglobinopathies
Fetal infections	—	Toxoplasmosis, viral infections (see p. 345)
Fetal blood gas and acid-base status	—	In fetal growth restriction (see p. 533)
Fetal therapy	—	Blood transfusion (see p. 132), drug therapy (see p. 572)

NONINVASIVE METHOD OF PRENATAL TESTING FROM MATERNAL PLASMA/BLOOD

Fetal DNA comes in the maternal circulation from the placenta. Fetal DNA can be detected in maternal plasma and whole blood from the first trimester onward. This is rapidly cleared from the maternal circulation after delivery. Cell-free fetal DNA (cff-DNA) is a reliable source for prenatal diagnosis. Approximately 5% of cell free DNA in the maternal blood is fetal. The cff DNA is a reliable source for prenatal diagnosis. The amount of cff-DNA in maternal blood increases with gestational age. The test is generally done from 10 weeks of pregnancy. The smaller fetal DNA fragments (50–200 base pairs) are separated from the maternal cell-free DNA for confirmation of success. The newer technology of Massively Parallel Sequencing (MPS) allows virtually all DNA molecules in the plasma to be analyzed.

Testing for cff-DNA is highly sensitive and specific. Detection rates for fetal trisomy 13, trisomy 18 and trisomy 21 are greater than 98%, with a very low false-positive rate (< 0.5 %). However, a woman with a positive test result should be referred for genetic counseling and should be offered invasive prenatal diagnosis for confirmation of test results. (ACOG-2012).

Conditions for Diagnosis with cff-DNA

1. Fetal Rh-D typing using cff-DNA to determine fetal blood group status. This is done without amniocentesis (see p. 392).
2. Single gene disorders can be diagnosed when the father has a mutation and that is not present in the mother (Marfan syndrome, cystic fibrosis).
3. Fetal aneuploidy: Trisomy 21.

Intact fetal cells: Fetal trophoblasts, lymphocytes, granulocytes or nucleated red blood cells can be isolated from maternal blood. Analysis of intact fetal cells by FISH with specific chromosome probes can diagnose fetal aneuploidy for other chromosomes besides trisomy 21. However, intact fetal cells are rare in maternal blood (1 per 1–10 million maternal cells).

BIOPHYSICAL: Ultrasonographic examination of the fetus in the early (10–14 weeks) pregnancy can detect fetal anomalies. **Crown-rump length (CRL) smaller than the gestational age** is associated with the risk of chromosomal anomalies (trisomy or triploidy). Increased **nuchal translucency** (NT) at 10–14 weeks is associated with many chromosomal abnormalities (trisomy, monosomy, triploidy—see Fig. 41.8). Detection rate is about 70–80% with a false-positive rate of 5–6%. **Absence of nasal bone (NB)** on USG at 10–12 weeks is associated with fetal Down's syndrome. When NB and NT were combined, detection rate of trisomy 21 was 92% with a false-positive rate of 3.5%. For details of biophysical markers, see p. 735.

Table 12.3: Prenatal Diagnosis: Biochemical and Biophysical Screening Tests

	β-hCG + PAPP-A + NT	**MSAFP**	**MSAFP, hCG, uE$_3$ (Triple Test)**	**MSAFP, uE$_3$, hCG, Inhibin A**	**Soft Tissue Marker (Nuchal Translucency; Nasal Bone)**
Time (weeks)	11–14	15–20	15–18	15–20	11–14
Observation	β-hCG (↑), PAPP-A (↓)	MSAFP (↑)	MSAFP (↓); uE$_3$ ↓; hCG (↑)	MSAFP (↓); uE$_3$ ↓; hCG (↑); Inhibin (↑)	Nuchal thickness (NT) > 3 mm, Nasal bone absent
Anomaly to detect	**Down's syndrome**	**Open neural tube defects**	**Down's syndrome**	**Down's syndrome**	**Down's syndrome, Turner's syndrome and others**
Comment	A cut off value 1 in 300 is screen positive	Cut-off level of 2.5 MOM can detect 90% of anencephaly, 80% open spina bifida	A cut-off value of 1 in 200 is screen positive	Detection rate is high	Detection rate of Down's syndrome is high (92%), when NT and NB are combined (see p. 515)
Detection rate	85–92%	85%	73%	85–92%	85–92%
False-positive rate	5%	3–5%	5%	0.9%	3–5%

Women who are screen positive should be offered fetal karyotyping by invasive methods (see p. 129)

Magnetic resonance imaging (MRI): Information superior to ultrasonography could be obtained (see p. 739).

Peri-implantation genetic diagnosis (PGD) is done by: (a) polar body biopsy, (b) blastomere biopsy (from 6–8 cell embryo) and (c) trophectoderm biopsy (5–6 days blastocyst). Diagnostic accuracy in PGD is high (98–99%) both for cytogenetic and single gene disorders. FISH technique is used for detection of aneuploidy, translocation and other chromosomal rearrangements. PGD may be preferred to usual prenatal diagnosis (CVS or amniocentesis) where pregnancy termination is not accepted.

Polar body biopsy: It is done by removing the first or second polar body in the preconceptional phase. Paternal genotype is not assessed here.

Blastomere biopsy: One or two cells are aspirated through a hole made in the zona pellucida by mechanical, laser or chemical means. This does not affect the normal embryonic development.

Fetal therapy: Intrauterine fetal tranfusion for fetal anemia (alloimmunization, thalassemia) is done. Fetal medical therapy is done for various conditions through maternal medication. Medicines are carried transplacentally to the fetus. Maternal oral therapy with propylthiouracil for fetal hyperthyroidism, digoxin or flecainide for fetal tachyarrhythmias and oral dexamethasone for congenital adrenal hyperplasia of a female fetus have been found effective. Fetal stem cell transplantation and fetal gene therapy could be used for many hematological, metabolic, immunological and inherited diseases. Intrauterine fetal surgery has been attempted in few selected cases. **Common fetoscopic surgeries done are: laser therapy for TTTS (see p. 240), cystoscopic laser for posterior urethral valves, fetal tracheal occlusion for congenital diaphragmatic hernia and release of amniotic bands**.

KEY POINTS

- **Approximately 3% of live-born infants** have a major birth defcect. Majority (80%) of fetal deaths occur **antenatally**.
- **Birth defect may be**—(a) *Chromosomal*: numerical or structural, (b) *Single gene disorder,* (c) *Polygenic or multifactorial,* or (d) *Teratogenic disorder* (drugs). About half of chromosomal abnormalities are due to autosomal trisomy and remaining half is due to sex chromosomal abnormalities.
- **Screening for prenatal diagnosis** should be offered to all pregnancies. *MSAFP estimation is* done between 15–18 weeks. Value of 2.5 MOM adjusted with maternal age is taken as cut-off point. Elevated level can detect 85% of all open NTDs (see p. 128).
- **Triple test** (MSAFP, hCG, uE_3) is used for detection of Down's syndrome. It is done between 15 weeks and 18 weeks.
- **First trimester screening** with biochemical analytes (↓) PAPP-A and (↑) hCG and USG measurement of NT (↑) can improve detection rate (87%) of Down's syndrome (see p. 106). For confirmation, prenatal genetic study (CVS, amniocentesis or cordocentesis) has to be performed.
- **Second trimester screening (quad screening)** at 15–18 weeks: (↓) MSAFP, (↓) uE3, (↑) inhibin A and (↑) hCG can detect trisomy 21 in 85% of cases with a false-positive rate of 0.9%.
- **Screen positive women** are offered fetal karyotyping for confirmation. Fetal tissues are obtained from CVS, amniocentesis or cordocentesis. All these are invasive procedures.
- **Invasive procedures** carry risks. CVS is comparable to amniocentesis in terms of fetal loss rate and diagnostic accuracy. To avoid the problems of LRD, CVS should be done after 9 completed weeks. The complications of cordocentesis appear to be 1–2% (see p. 131).
- **Single gene disorders** can be detected by enzymatic analysis and or by molecular genetics. Direct analysis is done when gene sequence is known otherwise linkage analysis is done.
- **PGD** can be done by (see p. 132) removing a single cell from the embryo. Molecular genetics including FISH can detect genetic or chromosomal disorder accurately and safely. Currently **implantation rate** is only 20–30% in most IVF centers. After genetic screening, implantation rate increases by 50%.
- **Cell-free fetal DNA** (cff DNA) can be obtained from maternal plasma and whole blood. cff-DNA is a reliable source for prenatal diagnosis. It is a noninvasive procedure. Fetal aneuploidy (trisomy 21) and single gene disorders can be diagnosed.
- **Intact fetal cells** have also been recovered from maternal circulation. Genetic and chromosomal disorders are detected from a fetal cell using DNA probes and FISH or comparative genomic hybridization (CGH) and chromosomal microarrays.

QUESTIONS

1. Discuss in brief the important methods of prenatal genetic screening? (p. 128)
2. Mention the different methods of prenatal genetic diagnosis? (p. 129)

Write Short Notes on:

A. Cell Free Fetal DNA (cff DNA) (p. 131)

B. Chorionic Villous Sampling (p. 129)

Chapter 13

Normal Labor

DEFINITION: Series of events that take place in the genital organs in an effort to expel the viable products of conception (fetus, placenta and the membranes) out of the womb through the vagina into the outer world is called Labor. It may occur prior to 37 completed weeks, when it is called the preterm labor. Expulsion of a previable live fetus occurs through the same process but in a miniature form and is called mini-labor. Labor is characterized by the presence of regular uterine contractions with effacement and dilatation of the cervix and fetal descent. A parturient is a patient in labor and parturition is the process of giving birth. **Delivery** is the expulsion or extraction of a viable fetus out of the womb. It is not synonymous with labor; delivery can take place without labor as in elective cesarean section. Delivery may be vaginal, either spontaneous or aided, or it may be abdominal.

NORMAL LABOR (EUTOCIA): Labor is called normal if it fulfills the following criteria. (1) Spontaneous in onset and at term. (2) With vertex presentation. (3) Without undue prolongation. (4) Natural termination with minimal aids. (5) Without having any complications affecting the health of the mother and/or the baby.

ABNORMAL LABOR (DYSTOCIA): Any deviation from the definition of normal labor is called Abnormal labor. Thus, labor in a case with presentation other than vertex or having some complications even with vertex presentation affecting the course of labor or modifying the nature of termination or adversely affecting the maternal and/or fetal prognosis is called abnormal labor.

DATE OF ONSET OF LABOR: It is very much unpredictable to foretell precisely the exact date of onset of labor. It not only varies from case to case but even in different pregnancies of the same individual. Calculation based on Naegele's formula can only give a rough guide. **Based on the formula, labor starts approximately** on the expected date in 4%, 1 week on either side in 50%, 2 weeks earlier and 1 week later in 80%, at 42 weeks in 10%, and at 43 weeks plus in 4%.

CAUSES OF ONSET OF LABOR

The precise mechanism of initiation of human labor is still obscure. Endocrine, biochemical and mechanical stretch pathways as obtained from animal experiments, however, put forth the following hypotheses.

- **Uterine distension:** Stretching effect on the myometrium by the growing fetus and liquor amnii can explain the onset of labor at least in twins or polyhydramnios. **Uterine stretch** increases gap junction proteins, receptors for oxytocin and specific contraction associated proteins (CAPs).
- **Fetoplacental contribution:** Cascade of events activate **fetal hypothalamic-pituitary-adrenal axis** prior to onset of labor → increased CRH → increased release of ACTH → fetal adrenals → increased

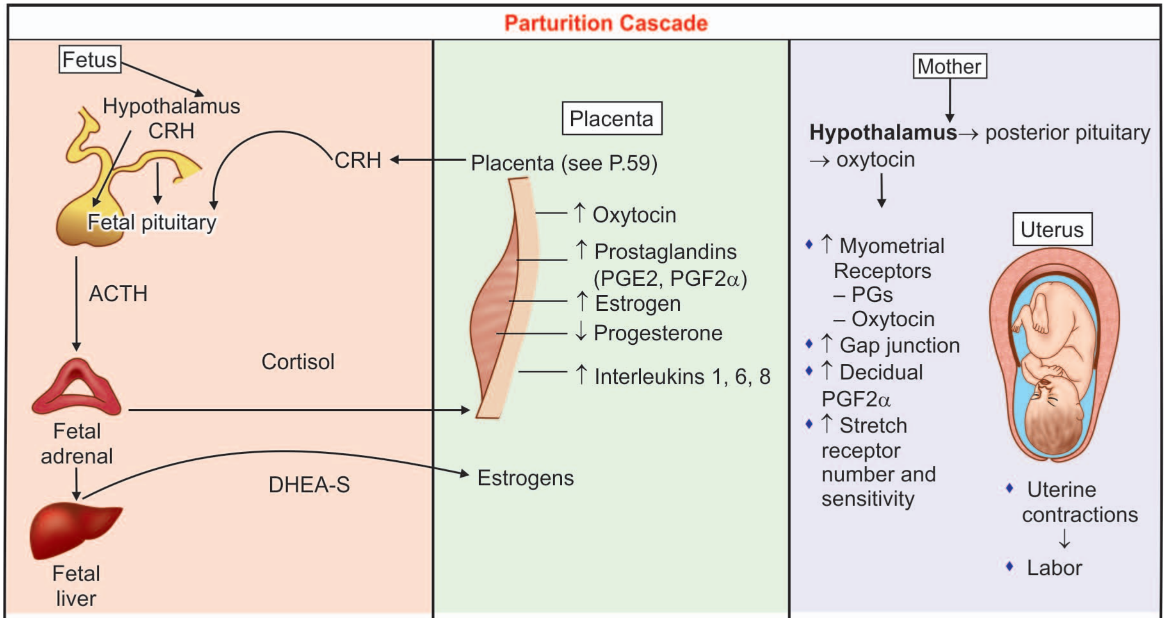

Parturition Cascade
Fetus
Hypothalamus
CRH
Fetal pituitary
CRH
ACTH
Cortisol
Fetal adrenal
DHEA-S
Fetal liver
Placenta
Placenta (see P.59)
↑ Oxytocin
↑ Prostaglandins (PGE2, PGF2α)
↑ Estrogen
↓ Progesterone
↑ Interleukins 1, 6, 8
Estrogens
Mother
Hypothalamus→ posterior pituitary → oxytocin
♦ ↑ Myometrial Receptors
– PGs
– Oxytocin
♦ ↑ Gap junction
♦ ↑ Decidual PGF2α
♦ ↑ Stretch receptor number and sensitivity
Uterus
♦ Uterine contractions
↓
♦ Labor

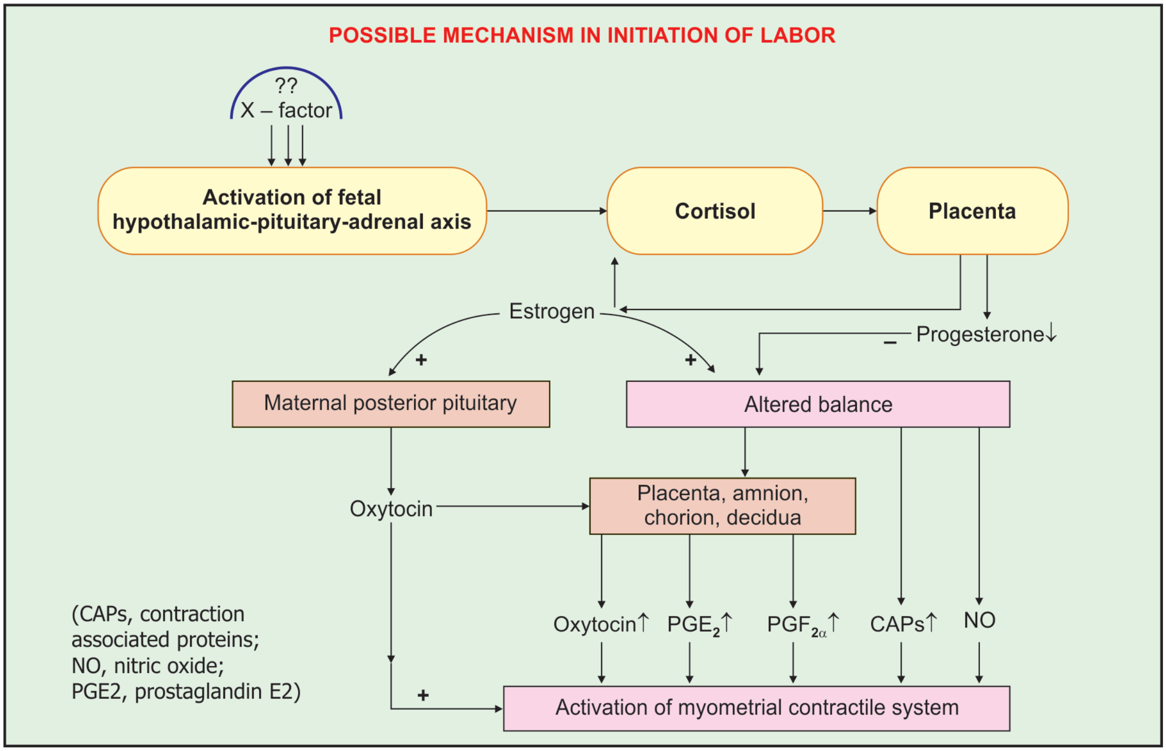
Fig. 13.1: Initiation of parturition

cortisol secretion → accelerated production of estrogen and prostaglandins from the placenta (Fig. 13.1).

- **Estrogen:** The probable mechanisms are:
  - — **Increases** the release of oxytocin from maternal pituitary.
  - — **Promotes** the synthesis of myometrial receptors for oxytocin (by 100–200 folds), prostaglandins and increase in gap junctions in myometrial cells.
  - — **Accelerates** lysosomal disintegration in the decidual and amnion cells resulting in increased prostaglandin (PGF$_{2\alpha}$) synthesis.
  - — **Stimulates** the synthesis of myometrial contractile protein—actomyosin through cAMP.
  - — **Increases** the excitability of the myometrial cell membranes.
- **Progesterone:** Increased fetal production of dehydroepiandrosterone sulfate (DHEA-S) and cortisol inhibits the conversion of fetal pregnenolone to progesterone. Progesterone levels therefore fall before labor. **It is the alteration** in the estrogen : progesterone ratio rather than the fall in the absolute concentration of progesterone, which is linked with prostaglandin synthesis.
- **Prostaglandins:** Prostaglandins are the important factors, which initiate and maintain labor. **The major sites of synthesis of prostaglandins are**—amnion, chorion, decidual cells and myometrium. **Synthesis is triggered by**—rise in estrogen level, glucocorticoids, mechanical stretching in late pregnancy, increase in cytokines (IL–1, 6, TNF), infection, vaginal examination, and separation or rupture of the membranes. Prostaglandins enhance gap junction (intermembranous gap between two cells through which stimulus flows) formation.

Biochemical Mechanisms Involved in the Synthesis of Prostaglandins

Phospholipase A_2 in the lysosomes of the fetal membranes near term → esterified arachidonic acid → formation of free arachidonic acid → synthesis of prostaglandins through prostaglandin synthetase. Prostaglandins (E_2 and $F_{2\alpha}$) diffuse in the myometrium → act directly at the sarcoplasmic reticulum → inhibit intracellular cAMP generation → increase local free calcium ions → uterine contraction. Once the arachidonic acid cascade is initiated, prostaglandins themselves will activate lysosomal enzyme systems. **The prostaglandin synthesis reaches a peak** during the birth of placenta probably contributing to its expulsion and to the control of postpartum hemorrhage.


POSSIBLE MECHANISM IN INITIATION OF LABOR
??
X – factor
Activation of fetal hypothalamic-pituitary-adrenal axis
Cortisol
Placenta
Estrogen
Progesterone↓
–
+
+
Maternal posterior pituitary
Altered balance
Oxytocin
Placenta, amnion, chorion, decidua
Oxytocin↑
PGE$_2$↑
PGF$_{2\alpha}$↑
CAPs↑
NO
+
Activation of myometrial contractile system
(CAPs, contraction associated proteins;
NO, nitric oxide;
PGE2, prostaglandin E2)

- ***Oxytocin and myometrial oxytocin receptors:*** (i) Large number of oxytocin receptors are present in the fundus compared to the lower segment and the cervix. (ii) **Receptor number increases during pregnancy reaching maximum during labor**. (iii) Receptor sensitivity increases during labor. (iv) Oxytocin stimulate synthesis and release of PGs (E_2 and $F_{2\alpha}$) from amnion and decidua. Vaginal examination and amniotomy cause rise in maternal plasma oxytocin level (Ferguson reflex). *Fetal plasma oxytocin* level is found increased during spontaneous labor compared to that of mother. Its role in human labor is not yet established.
- ***Neurological factor:*** Although labor may start in denervated uterus, labor may be also initiated through nerve pathways. Both α and β adrenergic receptors are present in the myometrium; estrogen causing the α receptors and progesterone the β receptors to function predominantly. The contractile response is initiated through the α receptors of the postganglionic nerve fibers in and around the cervix, and the lower part of the uterus. This is based on observation that onset of labor occurs following stripping or low rupture of the membranes.

CONTRACTILE SYSTEM OF THE MYOMETRIUM

The basic elements involved in the uterine contractile systems are: (a) actin, (b) myosin, (c) adenosine triphosphate (ATP), (d) the enzyme myosin light chain kinase (MLCK), and (e) Ca^{++}.

Structural unit of a myometrial cell is myofibril which contains the proteins—**actin and myosin.** The interaction of myosin and actin is essential for muscle contraction. The key process in actin-myosin interaction is myosin light chain phosphorylation. This reaction is controlled by **myosin light chain kinase (MLCK).** Oxytocin acts on myometrial receptors and activates phospholipase C, which increases intracellular calcium level. Calcium is essential for the activation of MLCK and binds to the kinase as **calmodulin–calcium** complex. Intracellular calcium levels are regulated by two general mechanisms: (1) influx across the cell membrane and (2) release from intracellular storage sites. Calcium is stored within the cells in the sarcoplasmic reticulum and in mitochondria. Progesterone and cAMP promote calcium storage at these sites. $PGF_{2\alpha}$, E_2 and oxytocin on the other hand stimulate its release.

- Intracellular Ca^{++} → calmodulin Ca^{++} → MLCK → phosphorylated myosin + actin → myometrial contraction.
- Decease of intracellular Ca^{++} (or its shift to the storage sites) → dephosphorylation of myosin light chain → inactivation of myosin light chain kinase → myometrial relaxation.

Uterine muscles have two types of adrenergic receptors—(1) α receptors, which on stimulation, produce a decrease in cyclic AMP (adenosine monophosphate) and result in contraction of the uterus and (2) β receptors, which on stimulation, produce rise in cyclic AMP and result in inhibition of uterine contraction.

FALSE PAIN: *(Synonym: false labor, spurious labor)*: It is found more in primigravidae than in parous women. It usually appears prior to the onset of true labor pain by 1 or 2 weeks in primigravidae and by a few days in multiparae. Such pains are probably due to stretching of the cervix and lower uterine segment with consequent irritation of the neighboring ganglia.

PRELABOR: *(Synonym: premonitory stage)*: The premonitory stage may begin 2–3 weeks before the onset of true labor in primigravidae and a few days before in multiparae. **The features are inconsistent and may consist of the following:**

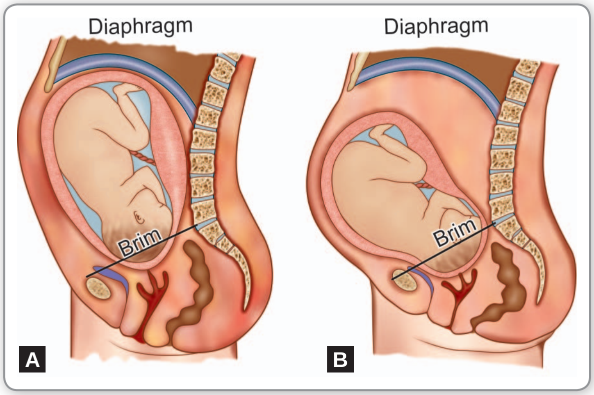
- ***"Lightening"*:** A few weeks prior to the onset of labor especially in primigravidae, the presenting part sinks into the true pelvis. It is due to active pulling up of the lower pole of the uterus around the presenting part. It signifies incorporation of the lower uterine segment into the wall of the uterus. This diminishes the fundal height and hence minimizes the pressure on the diaphragm (Fig. 13.2). The mother experiences a sense of relief from the mechanical cardiorespiratory embarrassment. There may be frequency of micturition or constipation due to mechanical factor—pressure by the engaged presenting part. **It is a welcome sign** as it rules out cephalopelvic disproportion and other conditions preventing the head from entering the pelvic inlet.
- ***Cervical changes*:** A few days prior to the onset of labor, cervix becomes ripe. **A ripe cervix is** (a) soft, (b) 80% effaced (<1.5 cm in length), (c) admits one finger easily, and (d) cervical canal is dilatable.
- ***Appearance of false pain*** (see below).

True labor pain is characterized by: (i) Painful uterine contractions at regular intervals, (ii) frequency of contractions increase gradually, (iii) intensity and duration of contractions increase progressively, (iv) associated with "show", (v) progressive effacement and dilatation of the cervix, (vi) descent of the presenting part, (vii) formation of the "bag of forewaters" and (viii) not relieved by enema or sedatives.

False labor pain is: (i) Dull in nature, (ii) confined to lower abdomen and groin, (iii) not associated with hardening of the uterus, (iv) they have no other features of true labor pain as discussed above and (v) usually relieved by enema or sedative.

***Labor pain*:** Throughout pregnancy, painless Braxton Hicks contractions with simultaneous hardening of the uterus occur. These contractions change their character, become more powerful, intermittent and are associated with pain. Pain more often felt in front of the abdomen or radiating toward the thighs.

***Show*:** With the onset of labor, there is profuse cervical secretion. Simultaneously, there is slight oozing of blood from rupture of capillary vessels of the cervix and from the raw decidual surface caused by separation of the membranes due to stretching of the lower uterine segment. **Expulsion of cervical mucus plug mixed with blood is called "show".**



Figs 13.2A and B: Showing phenomenon of "lightening". (A) Before and (B) after lightening

***Dilatation of internal os*:** With the onset of labor pain, the cervical canal begins to dilate more in the upper part than in the lower, the former being accompanied by corresponding stretching of the lower uterine segment.

***Formation of "bag of waters"*:** Due to stretching of the lower uterine segment, the membranes are detached easily because of its loose attachment to the poorly formed decidua. With the dilatation of the cervical canal, the lower pole of the fetal membranes becomes unsupported and tends to bulge into the cervical canal. As it contains liquor, which has passed below the presenting part, it is called "bag of waters". During uterine contraction with consequent rise of intra-amniotic pressure, this bag becomes tense and convex. After the contractions pass off, the bulging may disappear completely. **This is almost a certain sign of onset of labor**. However, in some cases the membranes are so well applied to the head that the finding may not be detected.

STAGES OF LABOR: Conventionally, events of labor are divided into three stages:

- ***First stage*:** It starts from the onset of true labor pain and ends with full dilatation of the cervix. It is, in other words, the "cervical stage" of labor. Its average duration is 12 hours in primigravidae and 6 hours in multiparae.
- ***Second stage*:** It starts from the full dilatation of the cervix (not from the rupture of the membranes) and ends with expulsion of the fetus from the birth canal. It has got two phases—(1) **The propulsive phase**—starts from full dilatation up to the descent of the presenting part to the pelvic floor. (2) **The expulsive phase** is distinguished by maternal bearing down efforts and ends with delivery of the baby. Its average duration is 2 hours in primigravidae and 30 minutes in multiparae.
- ***Third stage*:** It begins after expulsion of the fetus and ends with expulsion of the placenta and membranes (afterbirths). Its average duration is about 15 minutes in both primigravidae and multiparae. The duration is, however, reduced to 5 minutes in active management.
- ***Fourth stage*:** It is the stage of observation for at least 1 hour after expulsion of the afterbirths. During this period maternal vitals, uterine retraction and any vaginal bleeding are monitored. Baby is examined. These are done to ensure that both the mother and baby are well.

PHYSIOLOGY OF NORMAL LABOR

During pregnancy there is marked hypertrophy and hyperplasia of the uterine muscle and the enlargement of the uterus. At term, the length of the uterus measures about 35 cm including cervix. The fundus is wider both transversely and anteroposteriorly than the lower segment. The uterus assumes pyriform or ovoid shape. The cervical canal is occluded by a thick, tenacious and mucus plug.

UTERINE CONTRACTION IN LABOR: Throughout pregnancy there is irregular involuntary spasmodic uterine contractions which are painless (Braxton Hicks) and have no effect on dilatation of the cervix (see p. 53). The character of the contractions changes with the onset of labor. The **pacemaker of the uterine contractions** is situated in the region of the tubal ostia from where waves of contractions spread downward. While there are wide variations in frequency, intensity and duration of contractions, they remain usually within normal limits in the following patterns.

- There is good synchronization of the contraction waves from both halves of the uterus and also between upper and lower uterine segments.
- There is fundal dominance of contractions that diminish gradually in duration and intensity through midzone down to lower segment. It takes about 10–20 seconds.
- The waves of contraction follow a regular pattern.
- The upper segment of the uterus contracts more strongly and for a longer time than the lower part.

- Intra-amniotic pressure rises beyond 20 mm Hg during uterine contraction.
- Good relaxation occurs in between contractions to bring down the intra-amniotic pressure to less than 8 mm Hg. Contractions of the fundus last longer than that of the midzone.

During contraction, uterus becomes hard and somewhat pushed anteriorly to make the long axis of the uterus in line with that of pelvic axis. Simultaneously, the patient experiences pain which is situated more on the hypogastric region, often radiating to the thighs. **Probable causes of pain are**: (a) Myometrial hypoxia during contractions (as in angina), (b) stretching of the peritoneum over the fundus, (c) stretching of the cervix during dilatation, (d) stretching of the ligaments surrounding the uterus and (e) compression of the nerve ganglion. **Pain of uterine contractions is distributed along the cutaneous nerve distribution of T_{10} to L_1**. Pain of cervical dilatation and stretching is referred to the back through the sacral plexus.

***Tonus*: It is the intrauterine pressure in between contractions.** During pregnancy, as the uterus is quiescent (inactive), the tonus is of 2–3 mm Hg. During the first stage of labor, it varies from 8 mm Hg to 10 mm Hg. It is inversely proportional to relaxation. **The factors which govern the tonus are:** (i) Contractility of uterine muscles, (ii) intra-abdominal pressure, and (iii) overdistension of uterus as in twins and hydramnios.

***Intensity*: The intensity of uterine contraction describes the degree of uterine systole.** The intensity gradually increases with advancement of labor until it becomes maximum in the second stage during delivery of the baby. **Intrauterine pressure is raised to 40–50 mm Hg during first stage and about 100–120 mm Hg in second stage of labor during contractions. In spite of diminished pain in third stage, the intrauterine pressure is probably the same as that in the second stage.** The diminished pain is due to lack of stretching effect.

***Duration*:** In the first stage, the contractions last for about 30 seconds initially but gradually increase in duration with the progress of labor. Thus in the second stage, the contractions last longer than in the first stage.

***Frequency*:** In the early stage of labor, the contractions come at intervals of 10–15 minutes. The intervals gradually shorten with advancement of labor until in the second stage, when it comes every 2–3 minutes.

It is important to note that all the features of uterine contractions mentioned are very effective only when they are in combination.

RETRACTION: Retraction is a phenomenon of the uterus in labor in which the muscle fibers are permanently shortened. Unlike any other muscles of the body, **the uterine muscles have this property to become shortened once and for all**. Contraction is a temporary reduction in length of the fibers, which attain their full length during relaxation. In contrast, retraction results in permanent shortening and the fibers are shortened once and for all (Fig. 13.3). **The net effects of retraction in normal labor are:**

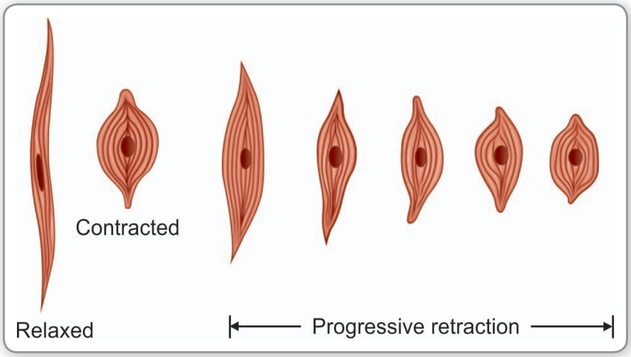


Fig. 13.3: Showing phenomenon of contraction and retraction of uterine muscle fibers during labor

— Essential property in the formation of lower uterine segment and dilatation and effacement of the cervix.

— To maintain the descent of the presenting part made by the uterine contractions and to help in ultimate expulsion of the fetus.

— To reduce the surface area of the uterus favoring separation of placenta.

— Effective hemostasis after the separation of the placenta.

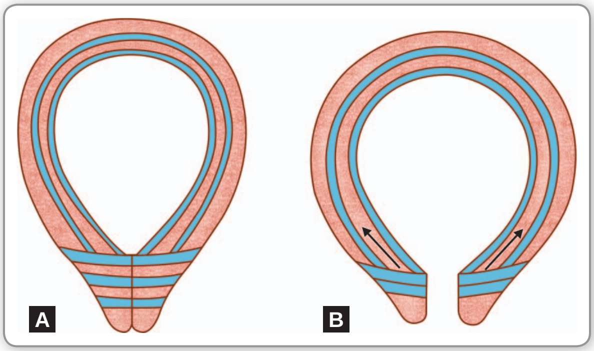
EVENTS IN FIRST STAGE OF LABOR

The first stage is chiefly concerned with the preparation of the birth canal so as to facilitate expulsion of the fetus in the second stage. **The main events that occur in the first stage are**—(a) dilatation and effacement of the cervix and (b) full formation of lower uterine segment.

DILATATION OF THE CERVIX: Prior to the onset of labor, in the prelabor phase (Phase-1) there may be a certain amount of dilatation of cervix, especially in multiparae and in some primigravidae. Important structural components of the cervix are—(a) smooth muscle (5–20%), (b) collagen and (c) the ground substance. **Predisposing factors which favor smooth dilatation are**—(a) softening of the cervix, (b) fibromusculoglandular hypertrophy, (c) increased vascularity, (d) accumulation of fluid in between collagen fibers, (e) breaking down of collagen fibrils by enzymes collagenase and elastase, and (f) change in the various glycosaminoglycans (e.g. increase in hyaluronic acid, decrease in dermatan sulfate) in the matrix of the cervix. These are under the action of hormones—estrogen, progesterone and relaxin. Too much fibrosis as in chronic cervicitis or prolapse or organic lesion in the cervix as in carcinoma, results in deficiency of these factors. As a result, cervix may fail to dilate.

Actual Factors Responsible are:

♦ ***Uterine contraction and retraction***—The longitudinal muscle fibers of the upper segment are attached with circular muscle fibers of the lower segment and upper part of the cervix in a bucket-holding fashion (Fig. 13.4). Thus, with each uterine contraction, not only the canal is opened up from above down but also it becomes shortened and retracted. There is some co-ordination between fundal contraction and cervical dilatation called "**polarity of uterus**". While the upper segment contracts, retracts and pushes the fetus, the lower segment and the cervix dilate in response to the forces of contraction of upper segment.



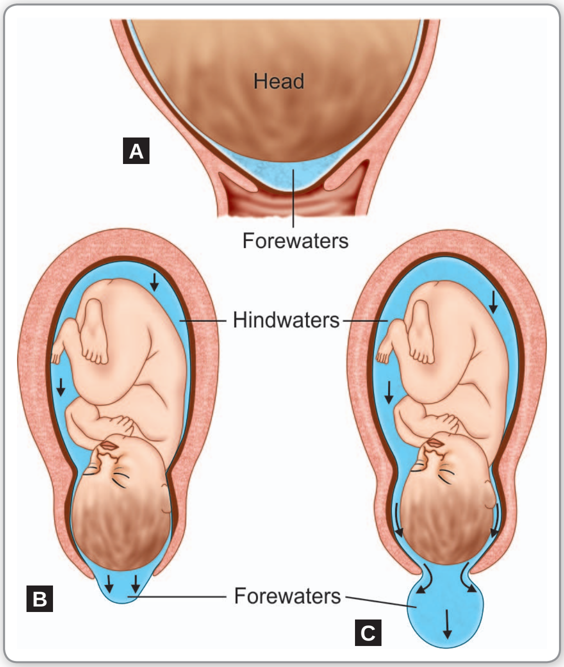
Figs 13.4A and B: Diagrammatic representation showing dilatation of the cervix by the pull of the longitudinal muscles of the uterus: (A) Before labor; (B) After labor

♦ ***Fetal axis pressure*****:** In labor with longitudinal lie and with well-fitted (flexed) fetal head on the cervix, fetal vertebral column is straightened by the contractions of the circular muscle fibers of the body of the uterus. This allows the fundal strong contraction force to be transmitted through the fetal podalic pole and vertebral column to the well-fitted fetal head. This causes mechanical stretching of the lower segment and opening up (dilatation) of the cervical canal. With each uterine contraction, there is elongation of the uterine ovoid and decrease in the transverse diameter. In transverse lie fetal axis pressure is absent. With progressive contraction and retraction, the upper segment becomes shorter and thicker while the lower segment becomes thinner and wider. The cervical canal starts dilating.

♦ ***Bag of membranes*****:** The membranes (amnion and chorion) are attached loosely to the decidua lining the uterine cavity except over the internal os. In vertex presentation, the girdle of contact of the head (that part of the circumference of the head which first comes in contact with the pelvic brim) being spherical, may well fit with the wall of the lower uterine segment. Thus, the amniotic cavity is divided into two compartments (Fig. 13.5). The part above the girdle of contact contains the fetus with bulk of the liquor called **hindwaters**, and the one below it containing small amount of liquor called **forewaters**. With the onset of labor, the membranes attached to the lower uterine segment are detached and with the rise of intrauterine pressure during contractions there is herniation of the membranes through the cervical canal. There is ball-valve like action by the well-flexed head. Uterine contractions generate hydrostatic pressure in the forewaters that in turn dilate the cervical canal like a wedge. When the bag of forewaters is absent (PROM) the pressure of the presenting part pushes the cervix centrifugally.

♦ ***Vis-a-tergo*:** The final phase of dilatation and retraction of the cervix is achieved by downward thrust of the presenting part of the fetus and upward pull of the cervix over the lower segment. This phenomenon is lacking in transverse lie where a thin cervical rim fails to disappear.

EFFACEMENT OR TAKING UP OF CERVIX: **Effacement** is the process by which the muscular fibers of the cervix are pulled upward and merges with the fibers of the lower uterine segment. The cervix becomes thin during first stage of labor or even before that in primigravidae. **In primigravidae**, effacement precedes dilatation of the cervix, **whereas in multiparae**, both occur simultaneously (Fig. 13.6). Expulsion of mucus plug is caused by effacement.

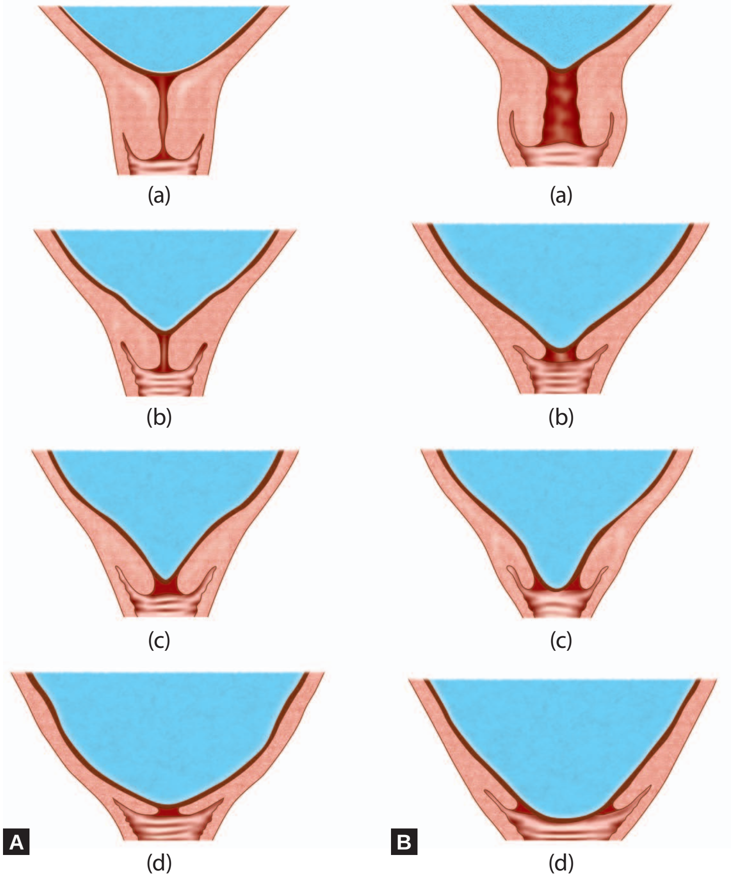


Figs 13.5A to C: (A) Formation of bag of membranes and forewaters; (B) Well fitting presenting part dividing the forewaters from hindwater; (C) Ill fitting presenting part allows the hindwaters to force into the bag of membranes during contraction which may lead to its early rupture

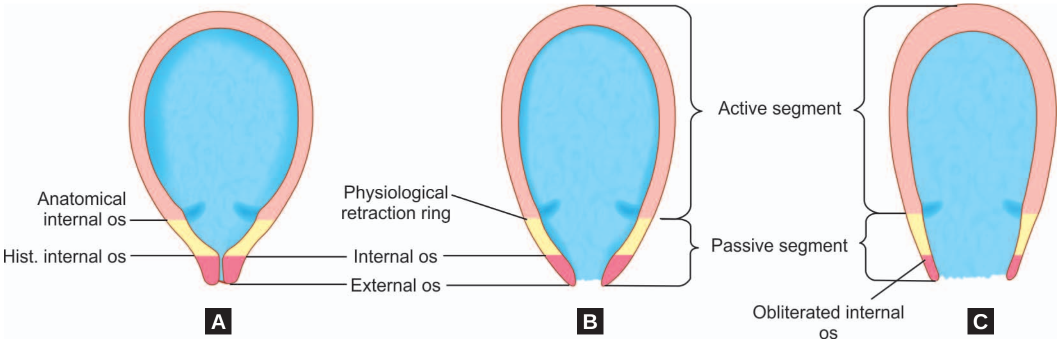
LOWER UTERINE SEGMENT: Before the onset of labor, there is no complete anatomical or functional division of the uterus. During labor the demarcation of an active upper segment and a relatively passive lower segment is more pronounced. The wall of the upper segment becomes progressively thickened with progressive thinning of the lower segment (Fig. 13.7). This is pronounced in late first stage, especially after rupture of the membranes and attains its maximum in second stage. A distinct ridge is produced at the junction of the two, called **physiological retraction ring** which should not be confused with the **pathological retraction ring**—a feature of obstructed labor (see p. 421-2). Lower segment of uterus is characterized by following features (Table 13.1):

Table 13.1: Lower Segment of Uterus and the Clinical Significance

Anatomical Features	Clinical Significance
▪ It is developed from the isthmus of the (nonpregnant) uterus, which is bounded above anatomical and below by histological internal os. ▪ In labor, it is bounded above by the physiological retraction ring (see p. 141-2) and below by the fibromuscular junction of cervix and uterus. ▪ This segment is formed maximally during labor and the peritoneum is loosely attached anteriorly. ▪ It measures 7.5–10 cm when fully formed and becomes cylindrical during the second stage of labor (Figs 13.7B, C). ▪ The wall becomes gradually thin due to: (i) Relaxation of the muscle fibers to allow elongation, (ii) the muscle fibers are drawn up by the muscle fibers of the upper uterine segment by contraction and retraction during labor (see p. 140) and (iii) descent of the presenting part causes further stretching and thinning out of wall (see p. 141-2). ▪ This segment has got poor retractile property compared to the upper segment.	▪ The phenomenon of receptive relaxation enables expulsion of the fetus by formation of complete birth canal along with the fully dilated cervix (Fig. 13.17). ▪ Implantation of placenta in lower segment is known as placenta previa (see p. 282). ▪ It is through this segment that cesarean section is performed. ▪ Poor decidual reaction in this segment facilitates morbid adherent placenta (see p. 288), once the placenta is implanted here. ▪ In obstructed labor, the lower segment is very much stretched and thinned out and ultimately gives way (ruptures) especially in multiparae (see p. 496). ▪ It is entirely the passive segment of the uterus. Because of poor retractile property, there is chance of postpartum hemorrhage if placenta is implanted over the area.


(a)
(a)
(b)
(b)
(c)
(c)
A
(d)
B
(d)

Figs 13.6A and B: Diagrammatic representation of the dilatation and "taking up" of the cervix in (A) primigravida and (B) multipara. (A) a – cervix before labor; b, c – progressive "taking up" of the cervix without much dilatation; d – cervix completely taken up with external os still remaining undilated; (B) a – cervix before labor, to note the patulous cervix; b, c – progressive and simultaneous dilatation and "taking up" of the cervix; d – taking up and dilatation of the external os occur simultaneously


Anatomical internal os
Hist. internal os
Physiological retraction ring
Internal os
External os
Active segment
Passive segment
Obliterated internal os
A
B
C

Figs 13.7A to C: Sequence of development of the active and passive segments of the uterus. (A) Uterus at term; (B) In early labor; (C) Late second stage

EVENTS IN SECOND STAGE OF LABOR

The second stage begins with the complete dilatation of the cervix and ends with the expulsion of the fetus. **This stage is concerned with the descent and delivery of the fetus through the birth canal**.

Second stage has two phases: (1) **Propulsive**—from full dilatation until head touches the pelvic floor. (2) **Expulsive**—since the time mother has irresistible desire to *"bear down"* and push until the baby is delivered.

With the full dilatation of the cervix, the membranes usually rupture and there is escape of good amount of liquor amnii. The volume of the uterine cavity is thereby reduced. Simultaneously, uterine contraction and retraction become stronger. The uterus becomes elongated during contraction, while the anteroposterior and transverse diameters are reduced. The elongation is partly due to the contractions of the circular muscle fibers of the uterus to keep the fetal axis straight.

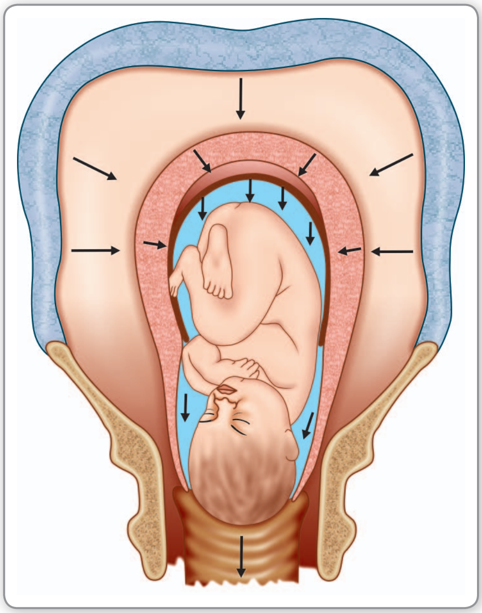


Fig. 13.8: Diagram showing the expulsive forces in the second stage. Increased intra-abdominal pressure augments the downward expulsive force of uterine contraction

Delivery of the fetus is accomplished by the downward thrust offered by uterine contractions supplemented by voluntary contraction of abdominal muscles (Fig.13.8) against the resistance offered by bony and soft tissues of the birth canal. There is always a tendency to push the fetus back into the uterine cavity by the elastic recoil of the tissue of the vagina and the pelvic floor. **This is effectively counterbalanced by the power of retraction**. Thus, with increasing contraction and retraction, the upper segment becomes more and more thicker with corresponding thinning of lower segment. **Endowed with power of retraction**, **the fetus is gradually expelled from the uterus against the resistance offered by the pelvic floor**. After the expulsion of the fetus, the uterine cavity is permanently reduced in size only to accommodate the afterbirths.

The expulsive force of uterine contractions is added by voluntary contraction of the abdominal muscles called **"bearing down" efforts**. For details see p. 153.

EVENTS IN THIRD STAGE OF LABOR

The third stage of labor comprises the phase of **placental separation**; its descent to the lower segment and finally its **expulsion** with the membranes.

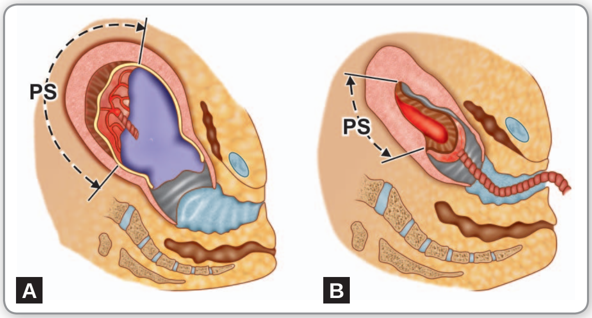
PLACENTAL SEPARATION: At the beginning of labor, the placental attachment roughly corresponds to an area of 20 cm (8") in diameter. There is no appreciable diminution of the surface area of the placental attachment during first stage. During the second stage, there is slight but progressive diminution of the area following successive retractions, which attains its peak immediately following the birth of the baby.

After the birth of the baby, the uterus measures about 20 cm (8") vertically and 10 cm (4") anteroposteriorly, the shape becomes discoid. The wall of the upper segment is much thickened while thin and flabby lower segment is thrown into folds. The cavity is much reduced to accommodate only the afterbirths.

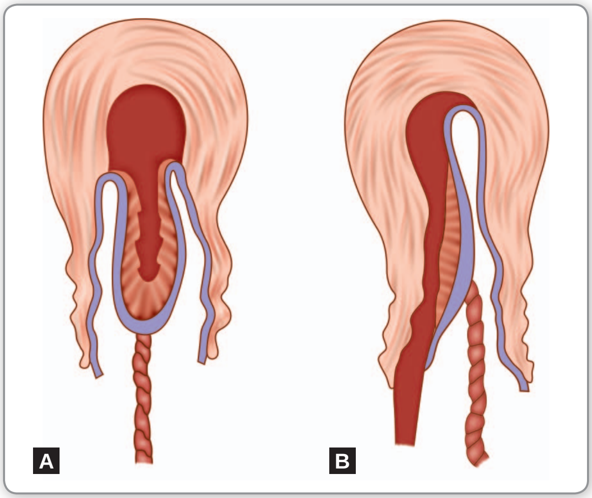
Mechanism of separation: Marked retraction reduces effectively the surface area at the placental site to about its half. But as the placenta is inelastic, it cannot keep pace with such an extent of diminution resulting in its buckling (Fig. 13.9). A shearing force is instituted between the placenta and the placental site which brings about its ultimate separation. **The plane of separation runs through deep spongy layer of decidua basalis** so that a variable thickness of decidua covers the maternal surface of the separated placenta. **There are two ways of separation of placenta (Fig. 13.10)**.

(1) **Central separation (Schultze):** Detachment of placenta from its uterine attachment starts at the center resulting in opening up of few uterine sinuses and accumulation of blood behind the placenta (retroplacental hematoma). With increasing contraction, more and more detachment occurs facilitated by weight of the placenta and retroplacental blood until whole of the placenta gets detached.

(2) **Marginal separation (Mathews-Duncan):** Separation starts at the margin as it is mostly unsupported. With progressive uterine contraction, more and more areas of the placenta get separated. Marginal separation is found more frequently.



Figs 13.9A and B: Diagram showing area of placental site—(A) Before the delivery of the baby and (B) after the delivery of the baby. Note the reduction of the surface area of the placental site resulting in buckling of the placenta. PS = Placental surface

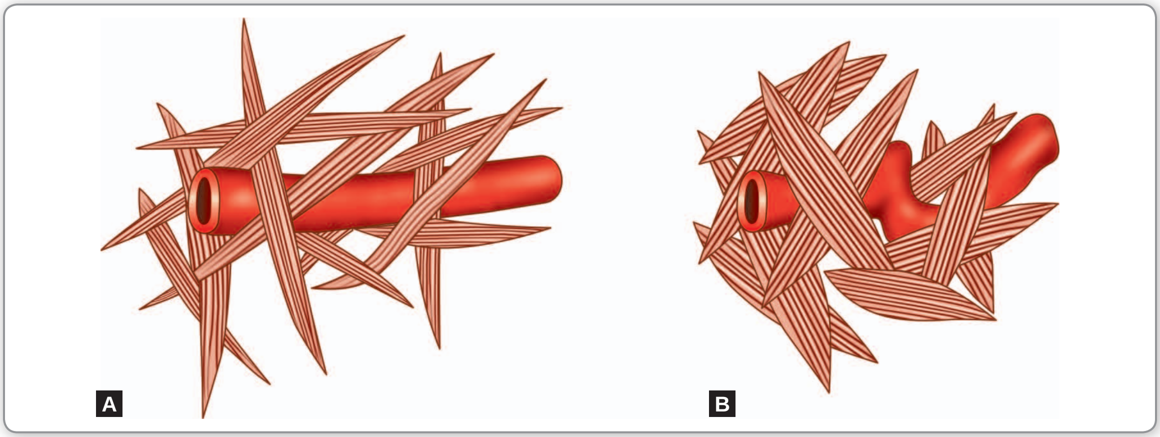


Figs 13.10A and B: Types of separation of the placenta—(A) Schultze method and (B) Mathews-Duncan method

SEPARATION OF THE MEMBRANES: The membranes, which are attached loosely in the active part, are thrown into multiple folds. Those attached to the lower segment are already separated during its stretching. **The separation is facilitated partly** by uterine contraction and mostly by weight of the placenta as it descends down from the active part. The membranes so separated carry with them remnants of decidua vera giving the outer surface of the chorion its characteristic roughness.

EXPULSION OF PLACENTA: After complete separation of the placenta, it is forced down into the flabby lower uterine segment or upper part of the vagina by effective contraction and retraction of the uterus. Thereafter, it is expelled out either by voluntary contraction of abdominal muscles (bearing down efforts) or by manual procedure (see p. 163).

***Mechanism of control of bleeding*:** After placental separation, innumerable torn sinuses which have free circulation of blood from uterine and ovarian vessels have to be obliterated. **The occlusion is affected by complete retraction** whereby the arterioles, as they pass tortuously through the interlacing intermediate layer of the myometrium, are literally clamped (Fig. 13.11). **It (living ligature) is the principal mechanism of hemostasis**. However, **thrombosis** occurs to occlude the torn sinuses, a phenomenon, which is facilitated by hypercoagulable state of pregnancy. **Apposition of the walls of the uterus** following expulsion of the placenta (myotamponade) also contributes to minimize blood loss.



Figs 13.11A and B: Blood vessels: (A) Running through the interlacing muscle fibers; (B) literally clamped due to effective retraction of the uterine muscles

MECHANISM OF NORMAL LABOR

DEFINITION: The series of movements that occur on the head in the process of adaptation during its journey through the pelvis is called mechanism of labor. It should be borne in mind that while the principal movements are taking place in the head, the rest of the fetal trunk is also involved in it, either participating in or initiating the movement.

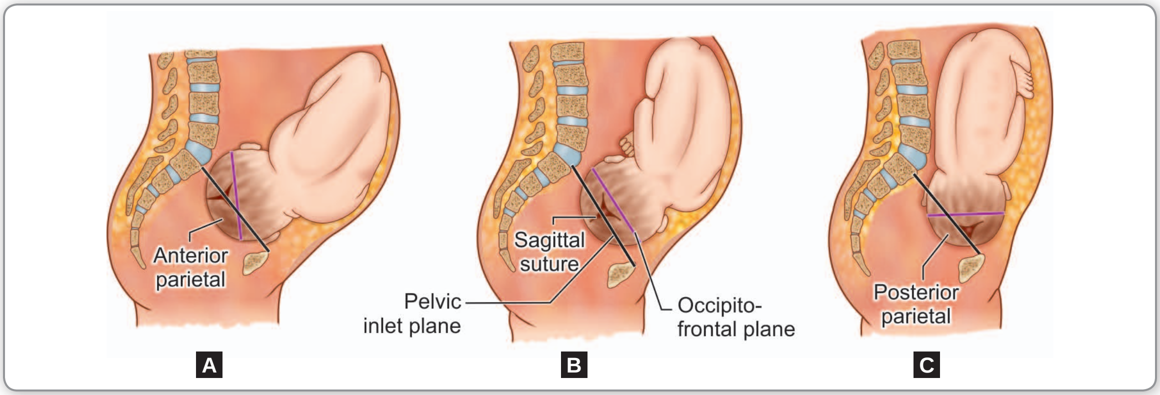
MECHANISM: In normal labor, the head enters the brim more commonly through the available transverse diameter (70%) and to a lesser extent through one of the oblique diameters. Accordingly, the position is either occipitolateral or oblique occipitoanterior. Left occipitoanterior is little more common than right occipitoanterior as the left oblique diameter is encroached by the rectum. **The engaging anteroposterior diameter** of the head is either suboccipitobregmatic 9.5 cm (3 3/4") or in slight deflexion—the suboccipitofrontal 10 cm (4"). **The engaging transverse diameter is biparietal 9.5 cm (3.74")**. **As the occipitolateral position is the most common, the mechanism of labor in such position will be described**. **The principal movements are:** (1) engagement, (2) descent, (3) flexion, (4) internal rotation, (5) crowning, (6) extension, (7) restitution, (8) external rotation, and (9) expulsion of the trunk. Although the various movements are described separately but in reality, the movements at least some, may be going on simultaneously.

***Engagement*:** Head brim relation prior to the engagement as revealed by imaging studies shows that due to lateral inclination of the head, the sagittal suture does not strictly correspond with the available transverse diameter of the inlet. Instead, it is either deflected anteriorly toward the symphysis pubis or posteriorly toward the sacral promontory (Fig. 13.12). Such deflection of the head in relation to the pelvis is called **asynclitism**.

When the sagittal suture lies anteriorly, the posterior parietal bone becomes the leading presenting part and is called **posterior asynclitism or posterior parietal presentation**. This is more frequently found in primigravidae because of good uterine tone and a tight abdominal wall.

In others, the sagittal suture lies more posteriorly with the result that the anterior parietal bone becomes the leading presenting part and is then called **anterior parietal presentation or anterior asynclitism**. It is more commonly found in multiparae.

Mild degrees of asynclitism are common but severe degrees indicate cephalopelvic disproportion.



Figs 13.12A to C: Head brim relation prior to engagement: (A) Anterior parietal presentation; (B) Head in synclitism; (C) Posterior parietal presentation

Posterior lateral flexion of the head occurs to glide the anterior parietal bone past the symphysis pubis in posterior parietal presentation. Lateral flexion in the reverse direction occurs to glide the posterior parietal bone past the sacral promontory in anterior parietal presentation. **After this movement which occurs early in labor, not only the head enters the brim but also synclitism occurs**. However, in about 25% of cases, the head enters the brim in synclitism, i.e. the sagittal suture corresponds to the diameter of engagement.

Advantages of Asynclitism

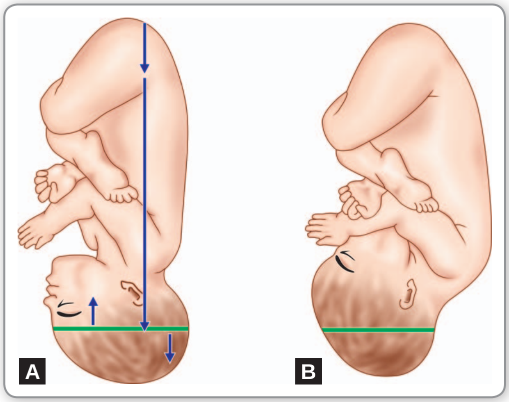
- Engagement of head with asynclitism, the two parietal eminences cross the brim one at a time. This helps lesser diameter (super subparietal: 8.5 cm), to cross the pelvic brim instead of larger biparietal diameter (9.5 cm) for engagement in synclitism.
- Asynclitism is beneficial in the mechanism of engagement of head.
- Marked and persistent asynclitism is abnormal and indicates cephalopelvic disproportion.

In primigravidae, engagement occurs in a significant number of cases before the onset of labor **while in multiparae**, the same may occur in late first stage with rupture of the membranes.

Descent: Provided there is no undue bony or soft tissue obstruction, descent is a continuous process. It is slow or insignificant in first stage but pronounced in second stage. It is completed with the expulsion of the fetus. In primigravidae, with prior engagement of the head, there is practically no descent in first stage; while in multiparae, descent starts with engagement. Head is expected to reach the pelvic floor by the time the cervix is fully dilated. **Factors facilitating descent are**—(1) uterine contraction and retraction, (2) bearing down efforts and (3) straightening of the ovoid fetal especially after rupture of the membranes.

Flexion: While some degree of flexion of the head is noticeable at the beginning of labor but complete flexion is rather uncommon. As the head meets the resistance of the birth canal during descent, full flexion is achieved. Thus, if the pelvis is adequate, **flexion is achieved either due to the resistance offered by the unfolding cervix, the walls of the pelvis or by the pelvic floor**. It has been seen that flexion precedes internal rotation or at least coincides with it. Flexion is essential for descent, since it reduces the shape and size of the plane of the advancing diameter of the head.

Flexion is explained by the two-arm lever theory—the fulcrum represented by the occipito-allantoid joint of the head, the short arm extends from the condyles to the occipital protuberance, and the long arm extends from condyles to the chin. When resistance is encountered, by ordinary law of mechanics, the short arm descends and the long arm ascends resulting in flexion of the head (Fig. 13.13).



Figs 13.13A and B: Lever action producing flexion of the head reducing the engaging diameter of the head from—(A) occipitofrontal to (B) suboccipitobregmatic

Internal rotation: It is a movement of great importance without which there will be no further descent. The mechanism of internal rotation is very complex, although easy to describe. **The theories which explain the anterior rotation of the occiput are:**

- ***Slope of pelvic floor*:** Two halves of levator ani form a gutter and viewed from above, the direction **of the fibers is backward and toward the midline**. Thus, during each contraction, the head, occiput in particular, in well-flexed position, stretches the levator ani, particularly that half which is in relation to the occiput. After the contraction passes off, elastic recoil of the levator ani occurs bringing the occiput forward toward the midline. The process is repeated until the occiput is placed anteriorly. **This is called rotation by law of pelvic floor (Hart's rule)**.
- ***Pelvic shape*:** Forward inclination of the side walls of the cavity, narrow bispinous diameter and long anteroposterior diameter of the outlet result in putting the long axis of the head to accommodate in the maximum available diameter, i.e. anteroposterior diameter of the outlet leaving behind the smallest bispinous diameter.
- ***Law of unequal flexibility (Sellheim and Moir)*:** The internal rotation is primarily due to inequalities in the flexibility of the component parts of the fetus.

In occipitolateral position, there will be anterior rotation by two-eighths of a circle of the occiput (Fig. 13.14) whereas in oblique anterior position, rotation will be one-eighth of a circle forward, placing the occiput behind the symphysis pubis. There is always an accompanying movement of descent with internal rotation. **Thus, prerequisites of anterior internal rotation of the head are** well-flexed head, efficient uterine contraction, favorable shape at the midpelvic plane, and tone of the levator ani muscles.

The level at which internal rotation occurs is variable. Rotation in the cervix although favorable is a less frequent occurrence. **In majority of cases, rotation occurs at the pelvic floor**. Rarely, it occurs as late as crowning of the head.

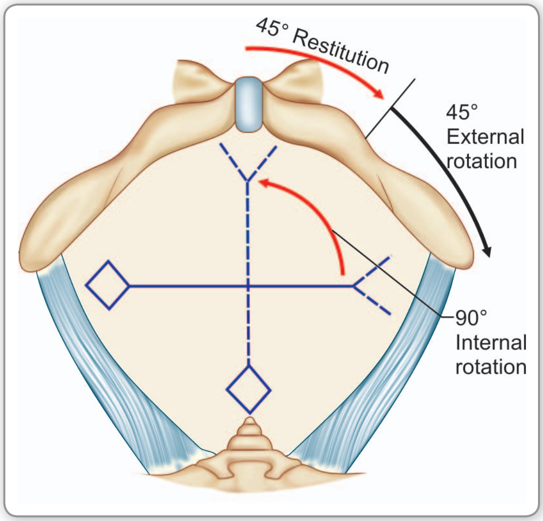


Fig. 13.14: Degree of internal rotation, restitution and external rotation of the head in left occipitolateral position

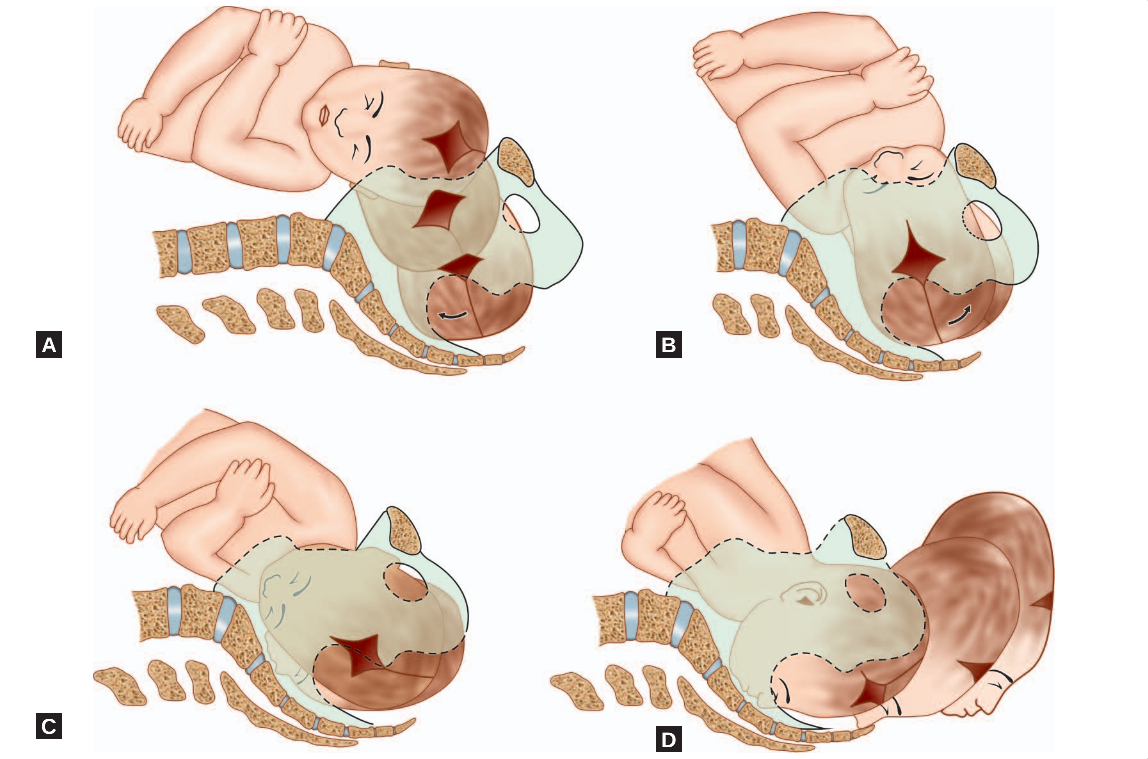
Torsion of the neck: Torsion of the neck is an inevitable phenomenon during internal rotation of the head. If the shoulders remain in the anteroposterior diameter, the neck has to sustain a torsion of two-eighths of a circle corresponding with the same degree of anterior rotation of the occiput. But the neck fails to withstand such major degree of torsion and as such there will be some amount of simultaneous rotation of the shoulders in the same direction to the extent of one-eighth of a circle placing the shoulders to lie in the oblique diameter with one-eighth of torsion still left behind. **Thus, the shoulders move to occupy the left oblique diameter in left occipitolateral position and right oblique diameter in right occipitolateral position**. **In oblique occipitoanterior position**, there is no movement of the shoulders from the oblique diameter as the neck sustains a torsion of only one-eighth of a circle.

Crowning**:** After internal rotation of the head, further descent occurs until the subocciput lies underneath the pubic arch. At this stage, **the maximum diameter of the head (biparietal diameter) stretches the vulval outlet without any recession of the head even after the contraction is over—called "crowning of the head".**

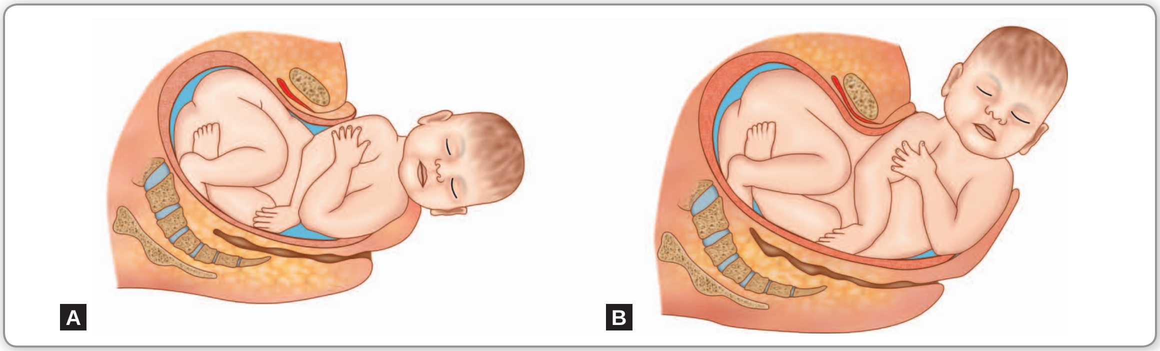
Extension**: Delivery of the head takes place by extension through "couple of force" theory**. The driving force pushes the head in a downward direction while the pelvic floor offers a resistance in the upward and forward direction. The downward and upward forces neutralize and remaining forward thrust helping in extension (Fig. 13.15). **The successive parts of the fetal head to be born** through the stretched vulval outlet are vertex, brow and face. Immediately following the release of the chin through the anterior margin of the stretched perineum, the head drops down, bringing the chin in close proximity to the maternal anal opening.

Restitution**: It is the visible passive movement of the head due to untwisting of the neck sustained during internal rotation**. Movement of restitution occurs rotating the head through one-eighth of a circle in the direction opposite to that of internal rotation (Fig. 13.14). **The occiput thus points to the maternal thigh of the corresponding side to which it originally lay (Fig. 13.25)**.

External Rotation**: It is the movement of rotation of the head visible externally due to internal rotation of the shoulders**. As the anterior shoulder rotates toward the symphysis pubis from the oblique diameter, it carries the head in a movement of external rotation through one-eighth of a circle in the same direction as restitution. The shoulders now lie in the anteroposterior diameter. **The occiput points directly toward the maternal thigh corresponding to the side to which it originally directed at the time of engagement (Fig. 13.14 and Fig. 13.25)**.



Figs 13.15A to D: Lateral view showing mechanism of labor in left occipitolateral position. (A and B) Posterior parietal presentation, posterior lateral flexion of the head and engagement; (C and D) Internal rotation of the head with movement of the shoulders; descent and delivery of the head by extension



Figs 13.16A and B: Delivery of the shoulders by lateral flexion. (A) Anterior shoulder; (B) Posterior shoulder

SUMMARY OF MECHANISM OF LABOR

(Occipito-lateral Position)

Diameter of engagement : Available transverse diameter of the inlet

Engaging diameter of the head : Suboccipitobregmatic 9.5 cm (3 ¾”)
or
Suboccipitofrontal 10 cm (4”)

DESCENT

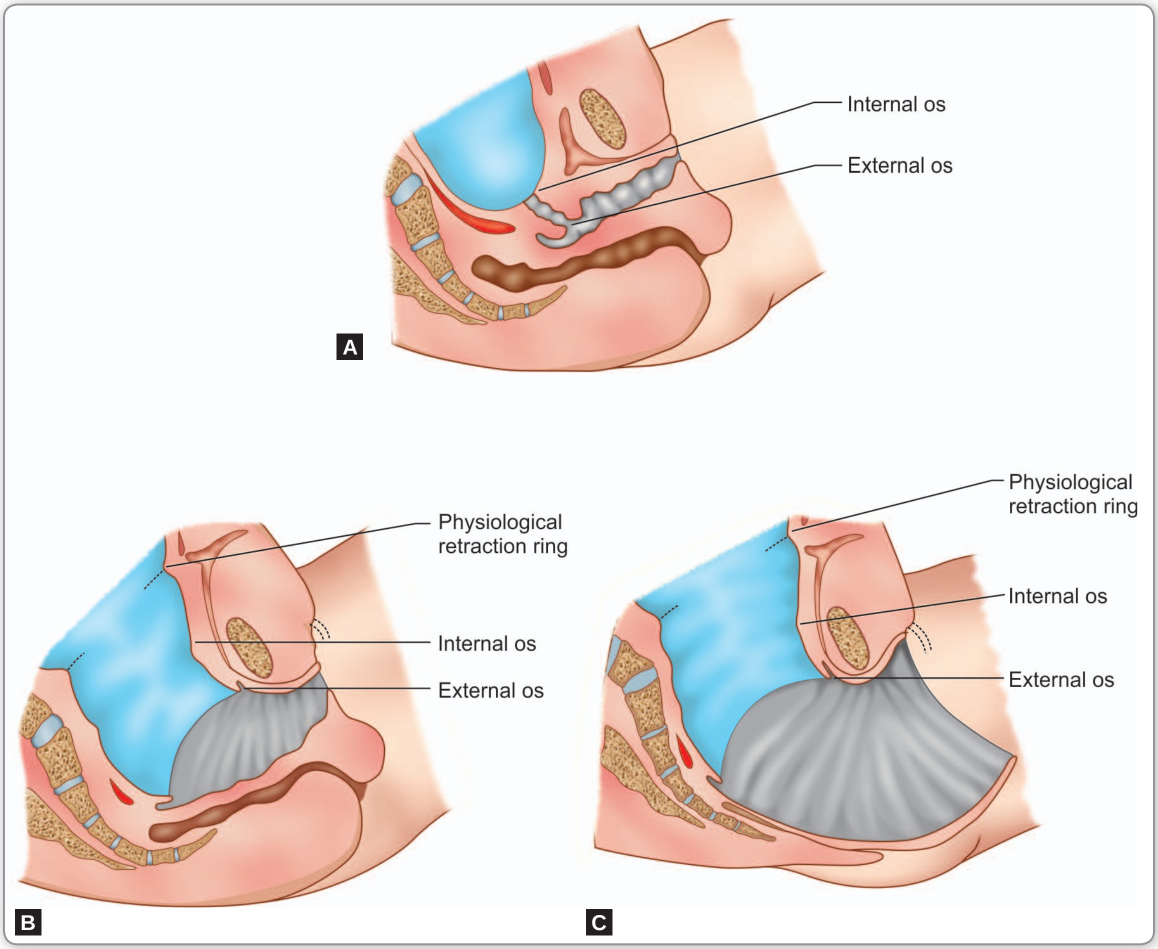
Engagement
↓
Increasing flexion
↓
Internal rotation of occiput anteriorly to 2/8th of circle
Simultaneous rotation of the shoulders to 1/8th of circle
↓
Crowning
↓
Delivery of the head by extension
↓
Restitution
↓
External rotation
↓
Delivery of the shoulders and trunk by lateral flexion

Birth of Shoulders and Trunk (Fig. 13.16): After the shoulders are positioned in anteroposterior diameter of the outlet, further descent takes place until the anterior shoulder escapes below the symphysis pubis first. By a movement of lateral flexion of the spine, the posterior shoulder sweeps over the perineum. Rest of the trunk is then expelled out by lateral flexion.

ANATOMY OF LABOR

As labor advances, the body of uterus, cervix and vagina together form a uniformly curved canal called the **birth canal**. Normally, at the onset of labor when the head is not engaged, the pelvic structures anterior to the vagina are urethra and bladder, and those posterior to the vagina are the pouch of Douglas with coils of intestine, rectum, anal canal, perineum and anococcygeal raphe.

As the head descends down with progressive dilatation of the vagina, it displaces the anterior structures upward and forward, and the posterior structures downward and backward, as if the head is passing through a swing door (Fig. 13.17). The bladder which remains a pelvic organ throughout the first stage becomes an abdominal organ in the second stage of labor. **However, there is no stretching of the urethra** as was previously thought. **Rather, the urethra is pushed anteriorly** with the neck of the bladder still lying in the vulnerable position behind the symphysis pubis. The changes in the posterior structures due to downward and backward displacement are marked when the head is sufficiently low down and in the stage of "crowning". The perineum which is a triangular area of about 4 cm thickness becomes a thinned out, membranous structure of less than 1 cm thickness. The anus, from being a closed opening, becomes dilated to the extent of 2–3 cm. The anococcygeal raphe is also thinned and stretched. Thus, the posterior wall of the birth canal becomes about 23 cm (9") in length, 11.5 cm (4 1/2") for the depth of the sacrum, and 11.5 cm (4 1/2") for the stretched soft tissue, while its anterior wall remains the same 4 cm (1 1/2") in length. The canal becomes almost a semicircle.



Figs 13.17A to C: (A) The relative position of the bladder, urethra and the genital organs at the beginning of labor; (B) Formation of birth canal with the cervix fully dilated. **Note the forward displacement of the urethra and bladder neck behind the pubis**; (C) Marked stretching with downward and backward displacement of the posterior wall of the canal as the head descends down

CLINICAL COURSE OF FIRST STAGE OF LABOR

The first symptom to appear is intermittent painful uterine contractions followed by expulsion of blood-stained mucus (show) per vaginam. Only few drops of blood mixed with mucus is expelled and any excess should be considered abnormal.

PAIN: Pains are felt more anteriorly with simultaneous hardening of the uterus. Initially, pains are not strong enough to cause discomfort and come at varying intervals of 15–30 minutes with duration of about 30 seconds. But gradually the interval becomes shortened with increasing intensity and duration so that in late first stage the contraction comes at intervals of 3–5 minutes and lasts for about 45 seconds. The relation of pain with uterine contraction is of great clinical significance. **In normal labor**, pains are usually felt shortly after the uterine contractions begin and pass off before complete relaxation of the uterus. **Clinically pains are said to be good** if they come at intervals of 3–5 minutes and at the height of contraction the uterine wall cannot be indented by the fingers.

DILATATION AND EFFACEMENT OF THE CERVIX: Progressive anatomical changes in the cervix, such as dilatation and effacement, are recorded following each vaginal examination. **Cervical dilatation relates** with dilatation of the external os and **effacement is determined by** the length of the cervical canal in the vagina. In primigravidae, the cervix may be completely effaced, feeling like a paper although not dilated enough to admit a fingertip. It may be mistaken for one that is fully dilated. While in multiparae, dilatation and taking up occur simultaneously which are more abrupt following rupture of the membranes. **The anterior lip of the cervix is the last to be effaced**. The first stage is said to be completed only when the cervix is completely retracted over the presenting part during contractions.

Cervical dilatation is expressed either in terms of fingers—1, 2, 3 or fully dilated or better in terms of centimeters (10 cm when fully dilated). It is usually measured with fingers but recorded in centimeters. One finger equals to 1.6 cm on average. Simultaneously, effacement of the cervix is expressed in terms of percentage, i.e. 25%, 50% or 100% (cervix less than 0.25 cm thick). **The term “rim” is used** when the depth of the cervical tissue surrounding the os is about 0.5–1 cm.

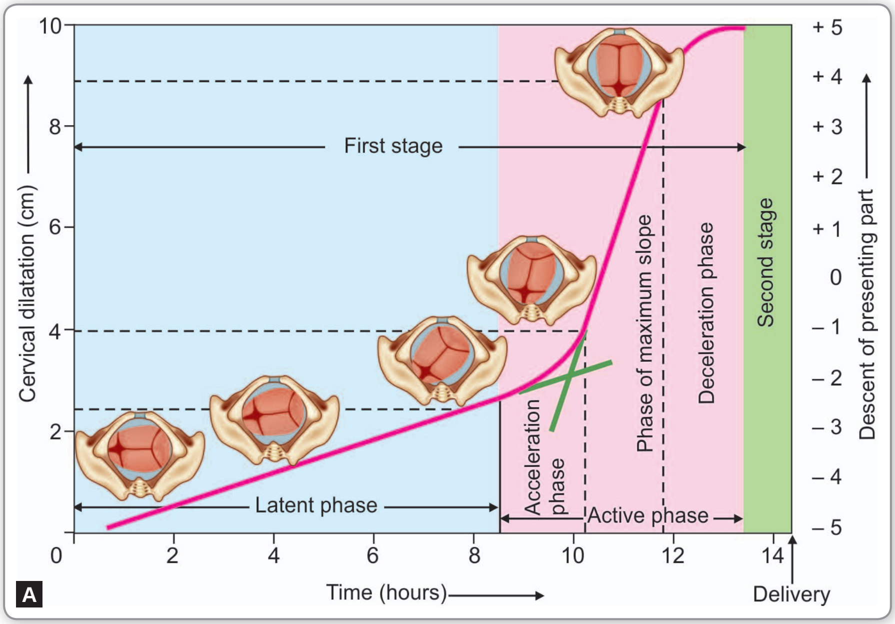
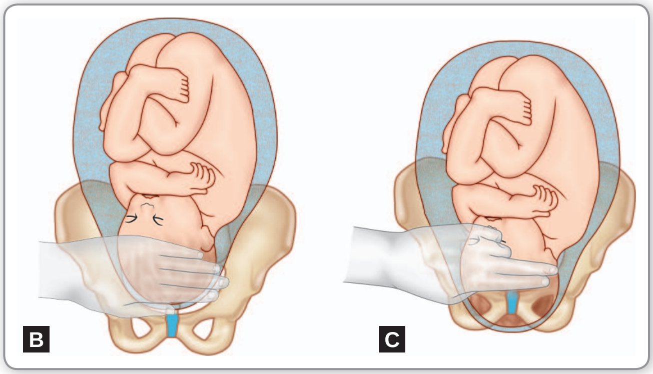


Fig. 13.18A: Composite partographic representation of different phases of labor showing progressive cervical dilatation, descent and rotation of the head

Partograph (see fig. 35.4): Friedman (1954) first devised it. Partograph is a composite graphical record of cervical dilatation and descent of head against duration of labor in hours. It also gives information about fetal and maternal condition, which are all recorded on a single sheet of paper (details see p. 607). **Cervical dilatation is a sigmoid curve and the first stage of labor has got two phases—(1) Latent phase and (2) Active phase (Fig. 13.18A)**.



Figs 13.18B and C: (B) Head is palpable five-fifths above the symphysis pubis. Head is mobile, free and not engaged; (C) Head is palpable two-fifths above and three-fifths has gone down the brim. Head is engaged. See also Figure 13.20

Latent phase of labor is defined as the period between the onset of true labor pain and the point when the cervical dilatation becomes 3–4 cm. Normal duration of latent phase of labor in a primigravida is about 20 hours (average 8.6 hours) and 14 hours (average 5.3 hours) in a multipara.

The active phase has got three components.

(i) Acceleration phase with cervical dilatation of 3–4 cm.
(ii) Phase of maximum slope of 4–9 cm dilatation. (iii) Phase of deceleration of 9–10 cm dilatation.

In primigravidae, the latent phase is often long (about 8 hours) during which effacement occurs; the cervical dilatation averaging only 0.35 cm/h. In multiparae, the latent phase is short (about 5 hours) and effacement and dilatation occur simultaneously. Because of variable duration of latent phase, it is difficult to plot the cervical dilatation along the graph. But it has got a distinct advantage to sort out the cases of delay in labor, especially after the latent phase is over **(cervix 4 cm dilated)**. **Dilatation of the cervix at the rate of 1 cm/h in primigravidae and 1.5 cm in multigravidae beyond 4 cm dilatation (active phase of labor) is considered satisfactory**.

STATUS OF THE MEMBRANES: Membranes usually remain intact until full dilatation of the cervix or sometimes even beyond in the second stage. However, it may rupture any time after the onset of labor but before full dilatation of cervix—when it is called **early rupture**. When the membranes rupture before the onset of labor, it is called **premature rupture (p. 369)**. As it has got some influence on the obstetric outcome, it is discussed elsewhere (see Chapter 22).

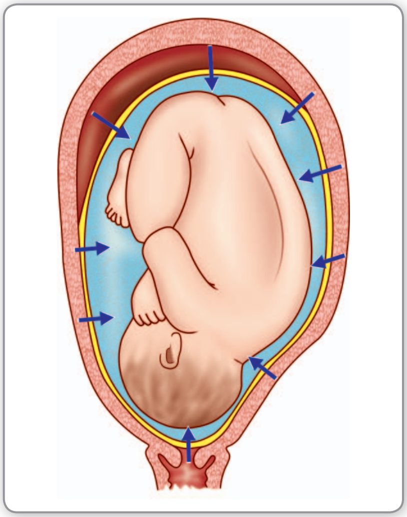


Fig. 13.19: Generalized intra-amniotic pressure with intact membranes. Placental circulation is almost unaffected during uterine contraction

An intact membrane is best felt with fingers during uterine contraction when it becomes tense and bulges out through the cervical opening. In between contractions, the membranes get relaxed and lies in contact with the head. With the rupture of the membranes, variable amounts of liquor escape out through the vagina and often there is acceleration of uterine contractions.

MATERNAL SYSTEM: General condition remains unaffected; although, a feeling of transient fatigue appears following a strong contraction. Pulse rate is increased by 10–15 beats per minute during contraction, which settles down to its previous rate in between contractions. Systolic blood pressure is raised by about 10 mm Hg during contraction. Temperature remains unchanged.

FETAL EFFECT: As long as the membranes are intact, there is hardly any adverse effect on the fetus (Fig. 13.19). However, during contraction, there may be slowing of fetal heart rate by 10–20 beats per minute which soon returns to its normal rate of about 140 per minute as the intensity of contraction diminishes provided the fetus is not compromised.

CLINICAL COURSE OF SECOND STAGE OF LABOR

Second stage begins with full dilatation of the cervix and ends with expulsion of the fetus.

PAIN: The intensity of the pain increases. The pain comes at intervals of 2–3 minutes and lasts for about 1–1½ minutes. It becomes successive with increasing intensity in the second stage.

BEARING-DOWN EFFORTS: It is the additional voluntary expulsive efforts that appear during the second stage of labor (expulsive phase). It is initiated by nerve reflex (Ferguson Reflex) set up due to stretching of the vagina by the presenting part. In majority, this expulsive effort start spontaneously with full dilatation of the cervix. Along with uterine contraction, the woman is instructed to exert downward pressure as done during straining at stool. **Sustained pushing beyond the uterine contraction is discouraged**. **Premature** (in the first stage) **bearing-down efforts** may suggest uterine dysfunction. There may be slowing of the FHR during pushing and it should come back to normal once the contraction is over.

MEMBRANES STATUS: Membranes may rupture with a gush of liquor per vaginam. Rupture may occasionally be delayed till the head bulges out through the introitus. Rarely, spontaneous rupture may not take place at all, allowing the baby to be “born in a caul”.

DESCENT OF THE FETUS: Features of descent of the fetus are evident from abdominal and vaginal examinations. **Abdominal findings are**—progressive descent of the head, assessed in relation to the brim (Fig. 13.20), rotation of the anterior shoulder to the midline and change in position of the fetal heart rate—shifted downward and medially. **Internal examination reveals descent of the head in relation to ischial spines** and gradual rotation of the head evidenced by position of the sagittal suture, and the occiput in relation to the quadrants of the pelvis (Fig. 13.21).

Abdominal assessment of progressive descent of the head (using fifth formula)

Progressive descent of the head can be usefully assessed abdominally by estimating the number of “fifths” of the head above the pelvic brim (Crichton). The amount of head felt suprapubically in finger breadth is assessed by placing the radial margin of the index finger above the symphysis pubis successively until the groove of the neck is reached. **When one-fifth above, only the sinciput can be felt abdominally and nought-fifth represents a head entirely in the pelvis with no poles felt abdominally (Fig. 13.20)**.

Advantages over “station of the head” in relation to ischial spines

1. It excludes the variability due to caput and molding or by a different depth of the pelvis.
2. The assessment is quantitative and can be easily reproduced.
3. Repeated vaginal examinations are avoided.

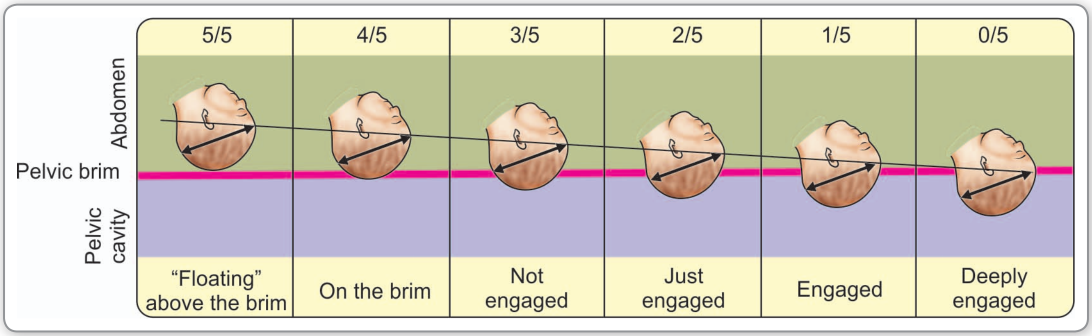


Fig. 13.20: Progressive descent of the head, assessed “in fifths” palpable above the brim (see also Figs 13.18B and C)

VAGINAL SIGNS: As the head descends down, it distends the perineum, the vulval opening looks like a slit through which the scalp hair is visible. During each contraction, the perineum is markedly distended with the overlying skin tense and glistening and the vulval opening becomes circular (expulsive phase). The adjoining anal sphincter is stretched and stool comes out during contraction. The head recedes after the contraction passes off but is held up a little in advance because of retraction. Ultimately, the maximum diameter of the head (biparietal) stretches the vulval outlet and there is no recession even after the contraction passes off. **This is called "crowning" of the head. The head is born by extension**. After a little pause, the mother experiences further pain and bearing-down efforts to expel the shoulders and the trunk. Immediately thereafter, a gush of liquor (hindwaters) follows, often tinged with blood.

MATERNAL SIGNS: There are features of exhaustion. Respiration is, however, slowed down with increased perspiration. During the bearing-down efforts, the face becomes congested with neck veins prominent. Immediately following the expulsion of the fetus, the mother heaves a sigh of relief.

FETAL EFFECTS: Slowing of FHR during contractions is observed, which comes back to normal before the next contraction.

CLINICAL COURSE OF THIRD STAGE OF LABOR

Third stage includes separation, descent and expulsion of the placenta with its membranes.

PAIN: For a short time, the patient experiences no pain. However, intermittent discomfort in the lower abdomen reappears, corresponding with the uterine contractions.

BEFORE SEPARATION: *Per abdomen*—Uterus becomes discoid in shape, firm in feel and non-ballottable. Fundal height reaches slightly below the umbilicus.

Per vaginam: There may be slight trickling of blood. Length of the umbilical cord as visible from outside remains static.

AFTER SEPARATION: It takes about 5 minutes in conventional management for the placenta to separate.

Per abdomen:

1. **Uterus becomes globular, firm, and ballottable.**
2. **The fundal height is slightly raised** as the separated placenta comes down in the lower segment and the contracted uterus rests on top of it.
3. **Slight bulging in the suprapubic region** due to distension of the lower segment by the separated placenta.

Per vaginum:

4. **Slight gush of vaginal bleeding.**
5. **Permanent lengthening of the cord is established.** This can be elicited by pushing down the fundus when a length of cord comes outside the vulva, which remains permanent even after the pressure is released. Alternatively, on suprapubic pressure upward by the fingers, there is no indrawing of the cord and the same lies unchanged outside the vulva.

EXPULSION OF PLACENTA AND MEMBRANES: The expulsion is achieved either by voluntary bearing-down efforts or more commonly aided by manipulative procedure. The afterbirth delivery is soon followed by slight to moderate bleeding amounting to 100–250 mL.

MATERNAL SIGNS: There may be chills and occasional shivering. Slight transient hypotension is not unusual.

PLACE OF DELIVERY

Although in about 85% of cases, the delivery remains uncomplicated and uneventful but in the remaining, unforeseen complications may arise which require urgent and skilled management. Effective antenatal care reduces the hazards of delivery significantly but to get an optimum outcome, adequate supervised

intranatal care is mandatory. Thus ideally, all women should have institutional delivery. The national sociodemographic goals and the Millennium Development Goals (MDG 4 & 5) aim by 2015 to achieve 100% deliveries conducted by skilled birth attendant (SBA) and to reduce maternal mortality ratio below 100 and perinatal death rate below 30 (see p. 683).

In the developed countries, there is a growing demand to have delivery at home (in low risk cases) with minimal intervention (natural birth). Every woman should have one midwife (named midwife) for the **continuity of care during pregnancy and labor, e.g. community-based care**.

Changing child birth stressed the importance of **community-based care** with the integration of community and hospital. There should be agreed protocols for referral and ease of transport between community and hospital.

Screening of high risk mothers (see p. 716) need careful checkup at regular interval. However, in under privileged sector the vast majority are forced to have home delivery either on choice or by compulsion. They are delivered by "*dais*" or even by their relatives. Thus, the teaching hospitals and the district or subdivisional hospitals are mostly being utilized as referral hospitals where most of the neglected cases are rushed late and in a bad shape.

In India, currently, there is significant rise in institutional delivery with the support of Janani Suraksha Yojana (JSY) scheme of National Rural Health Mission (2007).

FLYING SQUAD: The squad consists of a team of experienced obstetrician, anesthetist and nursing staff, equipped with sterilized packs of equipment and containers with stored blood. Ambulance car with the squad is rushed to the spot on call, mostly in the nature of antepartum hemorrhage or postpartum hemorrhage or eclampsia. Manual removal of placenta and resuscitation of the patient at home or during transport to the referral hospital are the common procedures employed.

MANAGEMENT OF NORMAL LABOR

General considerations: Labor events **have got great psychological, emotional and social impact** to the woman and her family. She experiences stress, physical pain and fear of dangers. The caregiver should be tactful, sensitive and respectful to her. The woman is allowed to have her chosen companion (family member). Continuous emotional support during labor may reduce the need for analgesia and decrease the rate of operative delivery. Privacy must be maintained. She is explained about the events from time to time. Comfortable environment, skill and confidence of the caregiver and appropriate support (see p. 607) are all essential so that a woman can give birth with dignity.

Management of normal labor aims at maximal observation with minimal active intervention. **The idea** is to maintain the normalcy and to detect any deviation from the normal at the earliest possible moment.

ANTISEPTICS AND ASEPSIS: Scrupulous surgical cleanliness and asepsis on the part of the patients and the attendants involved in the delivery process are to be maintained.

Patient care: Shaving or hair clipping of the vulva is done. The vulva and the perineum are washed liberally with soap and water and then with 10% Dettol solution or Hibitane (chlorhexidine) 1 in 2,000.

The woman should take a shower or bath, wear laundered gown and stay mobile. Throughout labor she is given continued encouragement and emotional support. **Antiseptic and aseptic precautions are to be taken during vaginal examination and during conduction of delivery**.

VAGINAL EXAMINATION IN LABOR: First vaginal examination should be done by a senior doctor to be more reliable and informative. The examination is done with the patient lying in dorsal position.

PRELIMINARIES: (1) **Toileting**—Hands and forearms should be washed with soap and running water, a scrubbing brush be used for the finger nails. The procedure should take at least 3 minutes.

(2) **Sterile pair of gloves** is donned. (3) **Vulval toileting** is performed. Vulva should once more be swabbed from before backward with antiseptic lotion like 10% Dettol or Hibitane 1 in 2,000. The same solution is poured over the vulva by separating the labia minora by the fingers of left hand. (4) **Gloved middle and index fingers of the right hand** smeared liberally with antiseptic cream like Cetavlon (cetrimide IP 0.5% W/W and hibitane 0.1% W/W) are introduced into the vagina after separating the labia by two fingers of the left hand. (5) Complete examination should be done before fingers are withdrawn. (6) Vaginal examination should be kept as minimum as possible to avoid risks of infection.

The following informations are to be noted and recorded carefully (Partograph—see p. 605):

- **Degree of cervical dilatation in centimeters**. It is marked with a cross (×) on the partograph at 4 cm dilatation. **Alert line** (see p. 465, 606) starts at 4 cm of cervical dilatation and continued to the point of full dilatation (10 cm) at the rate of 1 cm/h. **Action line** (see p. 465, 606) is drawn parallel and 4 hours to the right of the alert line.
- **Degree of effacement** of cervix (see p. 151).
- **Status of membranes and if ruptured—color of the liquor**. Color of the liquor in the partograph is recorded as—I: membranes intact; R: membranes ruptured; C: liquor clear; M: liquor meconium stained; B: liquor blood stained.
- **Presenting part and its position** by noting the fontanels and sagittal suture in relation to the quadrants of the pelvis.
- **Lambda or Posterior fontanel** is recognized by the big "**Y**" shaped three suture lines. **Bregma or anterior fontanel** is recognized by diamond-shaped area and the presence of four suture lines. In case of flexed head occiput is felt at a lower level than the sinciput.
- **Caput or molding of the head** and if present, to note its degree. Molding 1: Sutures apposed; 2: Sutures overlapped but reducible; 3: Sutures overlapped but not reducible.
- **Station of the head** in relation to ischial spines (Fig. 13.21).

  Spines are the most prominent bony projections felt on internal examination and the bispinous diameter is the shortest diameter of the pelvis in transverse plane being 10.5 cm. The level of ischial spines (Fig. 13.21) is the halfway between the pelvic inlet and outlet. This level is known as station zero (0). The levels above and below the spines are divided into fifths to represent centimeters. The station is said to be "O" if the presenting part is at the level of the spines. The station is stated in minus figures, if it is above the spines (–1 cm, –2 cm, –3 cm, –4 cm and –5 cm) and in plus figures if it is below the spines (+1 cm, +2 cm, +3 cm, +4 cm and +5 cm).

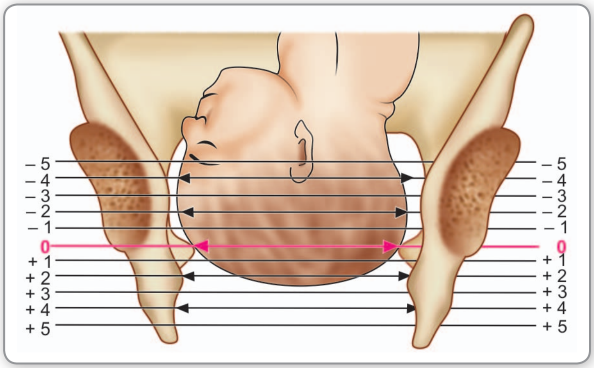


Fig. 13.21: Station of the head in relation to ischial spines

- **Assessment of the pelvis** especially in primigravidae is to be done, and elasticity of the pelvic floor and presence of vulval varicosity, if any, are to be noted.

INDICATIONS OF VAGINAL EXAMINATION:

Whatever aseptic technique is employed, there is always some chance of introducing infection especially after rupture of the membranes. **Hence vaginal examination should be restricted to a minimum**.

— **At the onset of labor:** Confirm the onset of labor, to detect precisely the presenting part and its position. **Pelvic assessment specially in primigravidae should be done during the initial examination**.

- **The progress of labor** can be judged on periodic examinations noting the dilatation of the cervix and descent of the head in relation to the spines (station). Generally, it is done at an interval of 3–4 hours.
- **Following rupture of the membranes** to exclude cord prolapse especially where the head is not yet engaged.
- **Whenever any interference is contemplated**.
- To confirm the actual coincidence of bearing down efforts with complete dilatation of the cervix and **to diagnose precisely the beginning of second stage**.

MANAGEMENT OF THE FIRST STAGE

PRINCIPLES: (1) **Noninterference with watchful expectancy** so as to prepare the patient for **natural birth**. (2) **To monitor carefully** the progress of labor, maternal conditions and fetal behavior so as to detect any intrapartum complication early.

PRELIMINARIES: This consists of basic evaluation of the current clinical condition. Enquiry is to be made about the onset of labor pains or leakage of liquor, if any. Thorough general and obstetrical examinations including vaginal examination are to be carried out and recorded. Records of antenatal visits, investigation reports and any specific treatment given, if available, are to be reviewed.

ACTUAL MANAGEMENT:

■ ***General***—(a) **Antiseptic dressing** is as described before (see p. 155). (b) **Encouragement, emotional support and assurance** are given to keep up the morale. (c) **Constant supervision** is ensured.

Generally, a woman in early normal labor may not be confined to bed. While in bed she may take the position most comfortable to her. She should avoid dorsal supine position to avoid aortocaval compression.

■ ***Bowel***—An enema with soap and water or glycerin suppository is traditionally given in early stage. This may be given if the rectum feels loaded on vaginal examination. But enema neither shortens the duration of labor nor reduces the infection rate.

■ ***Rest and ambulation***—If the membranes are intact, the patient is allowed to walk about. This attitude prevents venacaval compression and encourages descent of the head. Ambulation can reduce the duration of labor, need of analgesia and improve maternal comfort. If, however, labor is monitored electronically or analgesic drug (epidural analgesia) is given, she should be in bed.

■ ***Diet***—There is delayed emptying of the stomach in labor. Low pH of the gastric contents is a real danger if aspirated following general anesthesia when needed unexpectedly (see p. 596). So **food is withheld during active labor**. Fluids in the form of plain water, ice chips or fruit juice may be given in early labor. Intravenous fluid with ringer solution is started where any intervention is anticipated or the patient is under regional anesthesia.

■ ***Bladder care***—Patient is encouraged to pass urine by herself as full bladder often inhibits uterine contraction and may lead to infection. If the woman cannot go to the toilet, she is given a bed pan. Privacy must be maintained and comfort must be ensured. If the patient fails to pass urine especially in late first stage, catheterization is to be done with strict aseptic precautions.

■ ***Relief of pain***—The detail of analgesia in labor is discussed in Chapter 33. For practical purposes, the common analgesic drug used is pethidine 50–100 mg intramuscularly when the pain is well established in the active phase of labor. If necessary, it is repeated after 4 hours. Pethidine is an effective analgesic as well as a sedative. Metoclopramide 10 mg IM is commonly given to combat vomiting due to pethidine. Pethidine crosses the placenta and is a respiratory depressant to the neonate. **The drug should not be given if delivery is anticipated within 2 hours** (For antidote see p. 546).

- **Assessment of progress of labor and partograph recording.**

Pulse is recorded every 30 minutes and is marked with a dot (.) in the partograph. **Blood pressure** is recorded at every 1 hours and is marked with arrows (↕) (see p. 606). **Temperature** is recorded at every 2 hours. **Urine output** is recorded for volume, protein or acetone. **Any drug** (oxytocin or other) when given is recorded in the partograph.

Abdominal palpation—(a) Uterine contractions as regard the frequency, intensity and duration are assessed. **The number of contractions in 10 minutes and duration of each contraction in seconds are recorded in the partograph** (see p. 465, 606). Partograph is charted every half an hour (see p. 606) as: contraction duration less than 20 seconds ⁙ (mild); between 20 and 40 seconds ▨ (moderate) and more than 40 seconds ■ (strong).

(b) *Pelvic grip*: Gradual disappearance of poles of the head (sinciput and occiput) which were felt previously, (usually occur in labor). **Abdominal palpation** for descent of the fetal head in terms of fifths felt above the brim is to be used (Figs 13.18B and C).

(c) Shifting of the maximal intensity of the fetal heart beat downward and medially.

To note the fetal well-being:

- **Fetal heart rate** (FHR) along with its rhythm and intensity should be noted every half hour in the first stage and every 15 minutes in second stage or following rupture of the membranes. **To be of value, the observation should be made immediately following uterine contraction. The count should be made for 60 seconds.** For routine clinical observation, ordinary stethoscope is quite suitable. Doppler ultrasonic cardiography (Dopplex), however, is helpful in the case of obesity and polyhydramnios (see Fig. 42.49). **To avoid confusion of maternal and fetal heart rates, maternal pulse should be counted.** Otherwise maternal tachycardia may be wrongly treated as fetal heart rate. **Normal fetal heart rate ranges from 110 to 150 per minute.**

CONTINUOUS ELECTRONIC FETAL MONITORING (see Fig. 35.4): The device consists of simultaneous recording of fetal heart action by fetal electrocardiography and uterine contraction by tocography (details in page 693). It is commonly used for high risk pregnancies.

Vaginal examination—(a) Dilatation of the cervix in centimeters in relation to hours of labor is a reliable index to note the progress of labor. (b) To note the position of the head and degree of flexion. (c) To note the **station of the head** (degree of descent) in relation to the ischial spines. (d) **Color of the liquor** (clear or meconium stained) if the membranes are ruptured (e) **Degree of molding of the head**—molding occurs first at the junction of occipitoparietal bones and then between the parietal bones (see p. 97). (f) **Caput formation**—progressive increase is more important than its mere presence (see p. 97).

EVIDENCES OF FETAL DISTRESS: (See intrapartum fetal monitoring, p. 692).

TO WATCH THE MATERNAL CONDITION: Routine checkup includes: (a) to record 2 hourly pulse, blood pressure and temperature; (b) to observe the tongue periodically for hydration (see partograph p. 606); (c) to note the urine output, urine for acetone, glucose and (d) IV fluids, drugs.

Evidence of maternal distress are:

- Anxious look with sunken eyes
- Dehydration, dry tongue
- Acetone smell in breath
- Rising pulse rate of 100 per minute or more
- Hot, dry vagina often with offensive discharge
- Scanty high colored urine with presence of acetone

MANAGEMENT OF THE SECOND STAGE

The transition from the first stage to the second stage is evidenced by the following features:

- Increasing intensity of uterine contractions
- Urge to push or defecate with descent of the presenting part
- Complete dilatation of the cervix as evidenced on vaginal examination
- Bearing-down efforts

PRINCIPLES: (1) **To assist** in the natural expulsion of the fetus slowly and steadily, (2) **To prevent** perineal injuries.

GENERAL MEASURES:

— The patient should **be in bed**.

— **Constant supervision is mandatory and the FHR is recorded at every 5 minutes**.

— **To administer inhalation analgesics**, if available, in the form of gas N_2O and O_2 to relieve pain during contractions.

— **Vaginal examination** is done at the beginning of the second stage not only to confirm its onset but to detect any accidental cord prolapse. The position and the station of the head are once more to be reviewed and the progressive descent of the head is ensured.

PREPARATION FOR DELIVERY

— **Position:** Positions of the woman during delivery may be lateral, squatting or partial sitting (45°). **Dorsal position with 15° left lateral tilt** is commonly favored as it avoids aortocaval compression and facilitates pushing effort.

— **The accoucheur scrubs up** and puts on sterile gown, mask and gloves and stands on the right side of the table.

— **Toileting the external genitalia** and inner side of the thighs is done with cotton swabs soaked in Savlon or Dettol solution. One sterile sheet is placed beneath the buttocks of the patient and one over the abdomen. Sterilized leggings are to be used. **Essential aseptic procedures are remembered as three Cs: (a) Clean hands, (b) Clean surface and (c) Clean cutting and ligaturing of the cord.**

— **To catheterize the bladder**, if it is full.

CONDUCTION OF DELIVERY: The assistance required in spontaneous delivery is divided into three phases:

- **Delivery of the head**
- **Delivery of the shoulders**
- **Delivery of the trunk**

■ **Delivery of the head: The principles to be followed are** to maintain flexion of the head, to prevent its early extension and to regulate its slow escape out of the vulval outlet.

▲ The patient is encouraged for the bearing-down efforts during uterine contractions. This facilitates descent of the head.

▲ When the scalp is visible for about 5 cm in diameter, flexion of the head is maintained during contractions. This is achieved by pushing the occiput downward and backward by using thumb and index fingers of the left hand while pressing the perineum by the right palm with a sterile vulval pad. If the patient passes stool, it should be cleaned and the region is washed with antiseptic lotion.

▲ The process is repeated during subsequent contractions until the suboccipu t is placed under the symphysis pubis. At this stage, the maximum diameter of the head (biparietal diameter) stretches the vulval outlet without any recession of the head even after the contraction is over, and it is called "**crowning of the head**" **(Fig. 13.22). The purpose of increasing the flexion of the head is to ensure** that the small suboccipitofrontal diameter 10 cm (4") distends the vulval outlet instead of larger occipitofrontal diameter 11.5 cm (4 1/2") (Fig. 13.23).

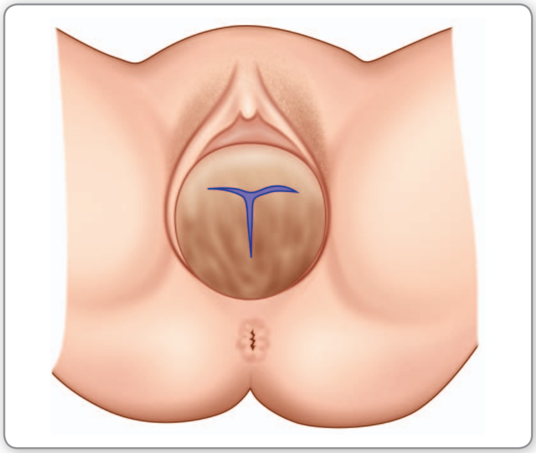


Fig. 13.22: Crowning of the head

- ▲ When the perineum is fully stretched and threatens to tear especially in primigravidae, episiotomy is done at this stage after prior infiltration with 10 mL of 1% lignocaine. **Bulging thinned out perineum is a better criterion** than the visibility of 4–5 cm of scalp to decide the time of performing episiotomy (details in Chapter 37). Episiotomy is done selectively and not as a routine (see p. 647).

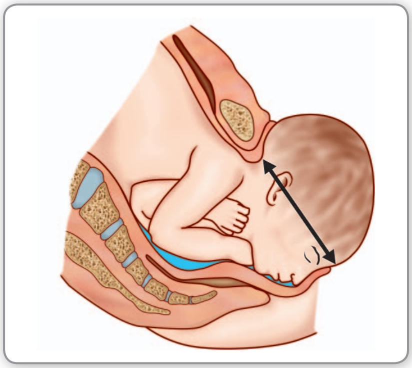


Fig. 13.23: Suboccipitofrontal diameter distending the vulval outlet

- ▲ **Slow delivery of the head in between the contractions is to be regulated**. This is done when the **suboccipitofrontal diameter** emerges out. This is accomplished by pushing the chin with a sterile towel covered fingers of the right hand placed over the anococcygeal region while the left hand exerts pressure on the occiput **(Ritgen's maneuver Fig. 13.24)**. **The forehead, nose, mouth and the chin are thus born successively over the stretched perineum by extension**.

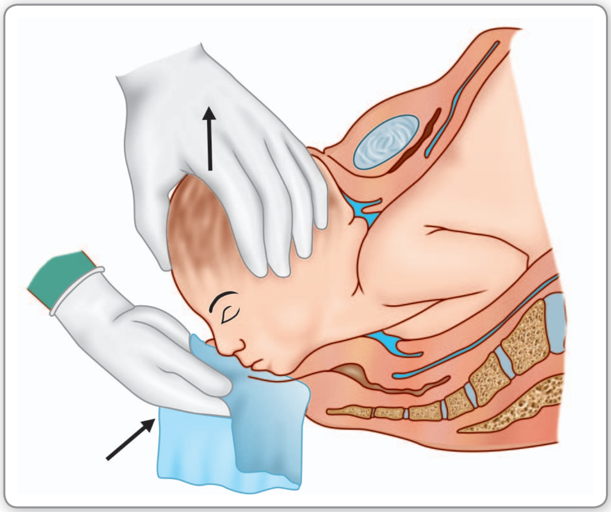
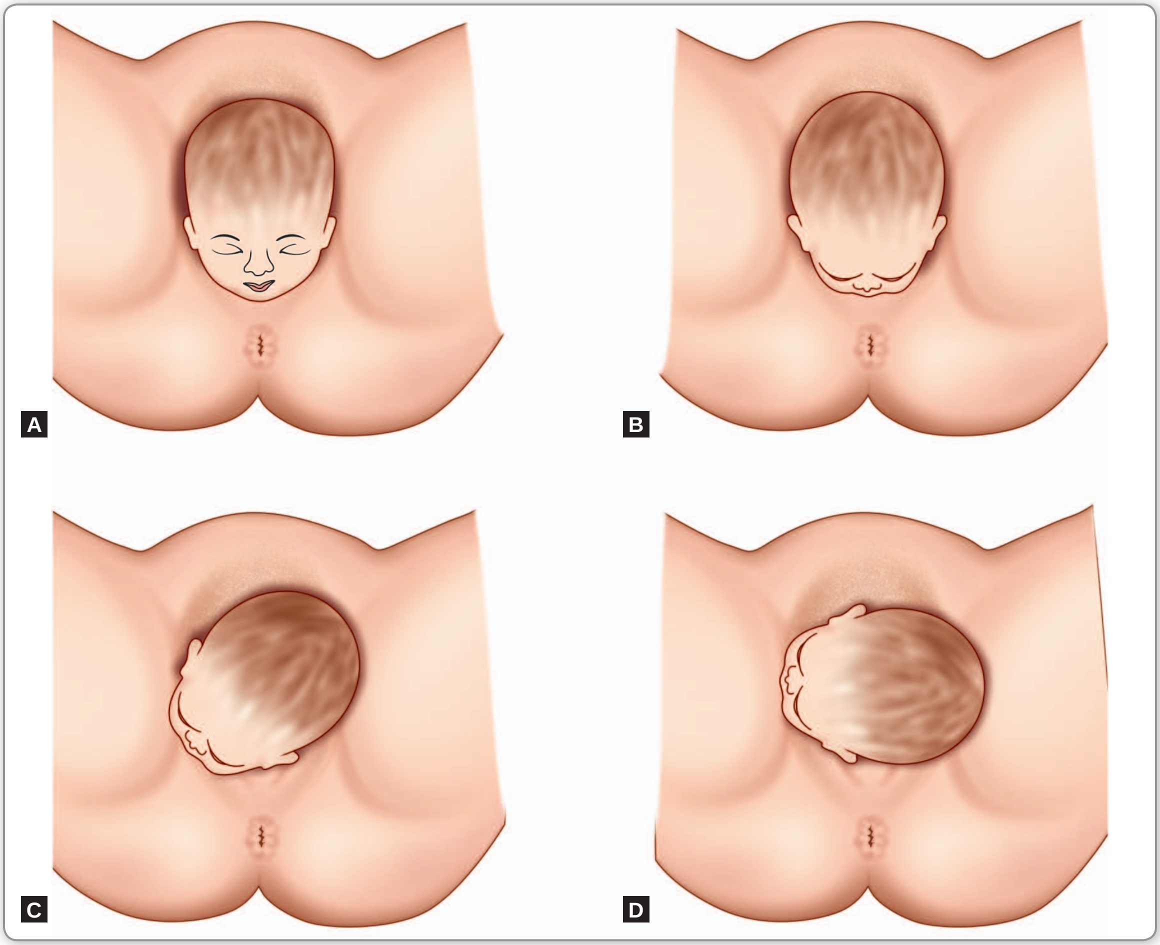


Fig. 13.24: Assisted delivery of the head by extension, exerting an upward pressure to the chin by the right hand placed over the anococcygeal raphe (Ritgen's maneuver)

- ■ **Care following delivery of the head:**
  - — Immediately following delivery of the head, **the mucus and blood in mouth and pharynx are to be wiped** with sterile gauze piece on a little finger. Alternatively, mechanical or electrical sucker may be used. This simple procedure prevents the serious consequence of mucus blocking the air passage during vigorous inspiratory efforts.
  - — **The eyelids are then wiped with sterile dry cotton swabs** using one for each eye starting from the medial to the lateral canthus to minimize contamination of the conjunctival sac.
  - — **The neck is then palpated to exclude the presence of any loop of cord (20–25%)**. If it is found and if loose enough, it should be slipped over the head or over the shoulders as the baby is being born. **But if it is sufficiently tight enough**, it is cut in between two pairs of Kocher's forceps placed 1 inch apart.

- ■ **PREVENTION OF PERINEAL LACERATION:** More attention should be paid not to the perineum but to the controlled delivery of the head.
  - ▲ **Delivery by early extension is to be avoided**. Flexion of the subocciput comes under the symphysis pubis so that lesser suboccipitofrontal 10 cm (4") diameter emerges out of the introitus.
  - ▲ **Spontaneous forcible delivery of the head is to be avoided** by assuring the patient not to bear down during contractions.
  - ▲ **To deliver the head in between contractions**.
  - ▲ **To perform timely episiotomy** (when indicated).
  - ▲ **To take care during delivery of the shoulders** as the wider bisacromial diameter (12 cm) emerges out of the introitus.

- ■ ***Delivery of the shoulders*: Not to be hasty in delivery of the shoulders**. Wait for the uterine contractions to come and for the movements of restitution and external rotation of the head to occur



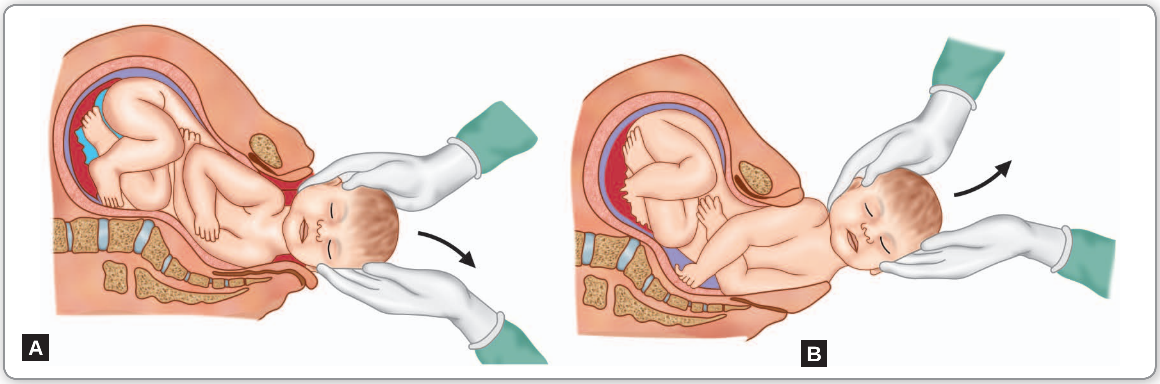
Figs 13.25A to D: (A) Head is born by extension; (B) Head drops down with the face close to the anus; (C) Restitution; (D) External rotation

(Fig. 13.25). This indirectly signifies that the bisacromial diameter is placed in the anteroposterior diameter of the pelvis. During the next contraction, the anterior shoulder is born behind the symphysis. If there is delay, the head is grasped by both hands and is gently drawn posteriorly until the anterior shoulder is released from under the pubis. By drawing the head in upward direction, the posterior shoulder is delivered out of the perineum (Fig. 13.26). **Traction on the head should be gentle** to avoid excessive stretching of the neck causing injury to the brachial plexus, hematoma of the neck or fracture of the clavicle.

- ***Delivery of the trunk*:** After the delivery of the shoulders, the fore finger of each hand are inserted under the axillae and the **trunk is delivered gently by lateral flexion**.

IMMEDIATE CARE OF THE NEWBORN

- **Soon after the delivery of the baby**, it should be placed on a tray covered with clean dry linen with the head slightly downward (15°). It facilitates drainage of the mucus accumulated in the tracheobronchial tree by gravity. The tray is placed between the legs of the mother and **should be at a lower level than the uterus** to facilitate gravitation of blood from the placenta to the infant.



Figs 13.26A and B: Assisted delivery of the shoulders—(A) Anterior shoulder; (B) Posterior shoulder

- **Air passage (oropharynx) should be cleared** of mucus and liquor by gentle suction.
- **Apgar rating** at 1 minute and at 5 minutes is to be recorded.
- **Clamping and ligature of the cord:** The cord is clamped by two Kocher's forceps, the near one is placed 5 cm away from the umbilicus and is cut in between. Two separate cord ligatures are applied with sterile cotton threads 1 cm apart using reef-knot, **the proximal one being placed 2.5 cm away from the navel**. **Squeezing the cord with fingers prior to applying ligatures** or plastic cord clamps (see Fig. 42.30), prevents accidental inclusion of embryonic remnants (see p. 45). **Leaving behind a length of the cord attached to the navel** not only prevents inclusion of the embryonic structure, if present, but also facilitates control of primary hemorrhage due to a slipped ligature. The cord is divided with scissors about 1 cm beyond the ligatures taking aseptic precautions so as to prevent cord sepsis. **Presence of any abnormality** in cord vessels (single umbilical artery) is to be noted. The cut end is then covered with sterile gauze piece after making sure that there is no bleeding. **The purpose of clamping the cord on the maternal end** is to prevent soiling of the bed with blood and to prevent fetal blood loss of the second baby in undiagnosed monozygotic twin.

Delay in clamping for 2–3 minutes or till cessation of the cord pulsation facilitates transfer of 80–100 mL blood from the compressed placenta to a baby when placed below the level of uterus. This is beneficial to a mature baby but may be deleterious to a preterm or a low birthweight baby due to hypervolemia and hyperbilirubinemia. But **early clamping** should be done in cases of Rh-incompatibility (to prevent antibody transfer from the mother to the baby) or babies born asphyxiated or one of a diabetic mother.

Cord is usually clamped after cleaning the airway after about 1–2 minutes of birth. Early clamping reduces the need of phototherapy due to hyperbilirubinemia.

♦ **Quick check is made to** detect any gross abnormality and the baby is wrapped with a dry warm towel. The identification tape is tied both on the wrist of the baby and the mother. Once the management of third stage is over (usually 10–20 minutes), baby is given to the mother or to the nurse.

MANAGEMENT OF THE THIRD STAGE

Third stage is the most crucial stage of labor. Previously uneventful first and second stage can become abnormal within a minute with disastrous consequences.

The principles underlying the management of third stage are **to ensure strict vigilance and to follow the management guidelines strictly in practice** so as to prevent the complications, the important one being postpartum hemorrhage.

STEPS OF MANAGEMENT: Two methods of management are currently in practice.

- **Expectant management**
- **Active management (preferred)**

■ Expectant management (traditional):

In this management, **the placental separation and its descent into the vagina are allowed to occur spontaneously**. Minimal assistance may be given for the placental expulsion if it needed.

- **Constant watch** is mandatory and the patient should not be left alone.
- **If the mother is delivered in the lateral position,** she should be changed to dorsal position to note features of placental separation and to assess the amount of blood loss.
- **A hand is placed over the fundus**—(a) **to recognize** the signs of separation of placenta, (b) to note the state of uterine activity—contraction and relaxation and (c) **to detect**, though rare, cupping of the fundus which is an early evidence of inversion of the uterus.

Desire to fiddle with the fundus or massage the uterus is to be strongly condemned. Placenta is separated within minutes following the birth of the baby. A watchful expectancy can be extended up to 15–20 minutes. In some institutions, "no touch" or "hands off" policy is employed. The patient is expected to expel the placenta within 20 minutes with the aid of gravity.

♦ **Expulsion of the placenta: Only when the features of placental separation and its descent into the lower segment are confirmed,** the patient is asked to bear down simultaneously with the hardening of the uterus. The raised intra-abdominal pressure is often adequate to expel the placenta. If the patient fails to expel, one can wait safely up to 10 minutes if there is no bleeding. As soon as the placenta passes through the introitus, it is grasped by the hands and twisted round and round with gentle traction so that the membranes are stripped intact. If the membranes threaten to tear, they are caught hold of by sponge-holding forceps and in similar twisting movements the rest of the membranes are delivered. **Gentleness, patience and care are prerequisites for** complete delivery of the membranes. If the spontaneous expulsion fails or is not practicable, because of delivery under anesthesia, any one of the following methods can be used to expedite expulsion.

Assisted expulsion: (a) ***Controlled cord traction (modified Brandt-Andrews method)***—The palmar surface of the fingers of the left hand is placed (above the symphysis pubis) approximately at the junction of upper and lower uterine segment (Fig. 13.27). The body of the uterus is pushed upward and backward, toward the umbilicus while by the right hand steady tension (but not too strong traction) is given in downward and backward direction holding the clamp until the placenta comes outside the introitus. It is thus more an uterine elevation which facilitates expulsion of the placenta. **The procedure is to be adopted only when the uterus is hard and contracted**.

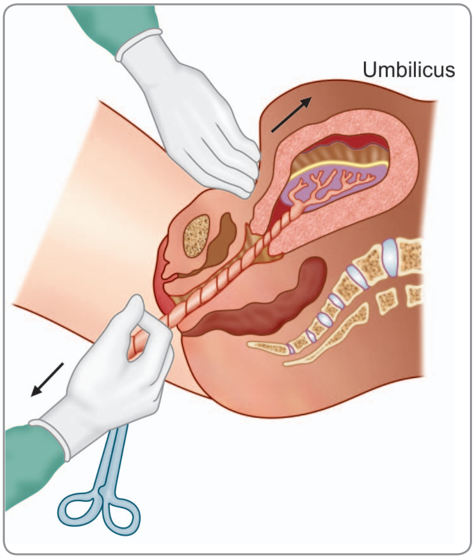


Fig. 13.27: Expression of the placenta by controlled cord traction

(b) ***Fundal pressure*****—The fundus is pushed downward and backward** after placing four fingers behind the fundus and the thumb in front using the uterus as a sort of piston (Fig. 13.28). **Pressure must be given only when the uterus becomes hard.** If it is not, then make it hard by gentle rubbing. The pressure is to be withdrawn as soon as the placenta passes through the introitus. **If the baby is macerated or premature, this method is preferable to cord traction** as the tensile strength of the cord is much reduced in both the instances.

The cord may be accidentally torn which is not likely to cause any problem. The sterile gloved hand should be introduced, and the placenta is to be grasped and extracted.

— **The uterus is massaged** to make it hard, which facilitates expulsion of retained clots if any. Injection of oxytocin (5–10 units) IV slowly/IM or methergine 0.2 mg is given intramuscularly. Oxytocin is more stable and has lesser side effects compared to ergometrine (nausea, vomiting, rise of BP) (see p. 578).

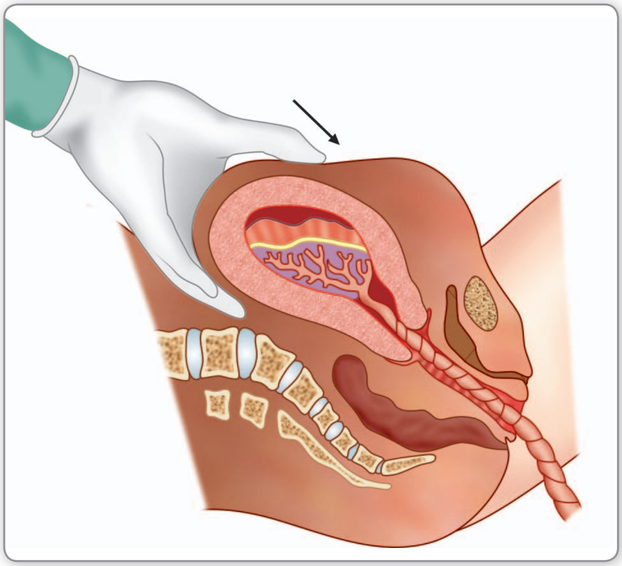


Fig. 13.28: Expression of the placenta by fundal pressure

— **Examination of the placenta membranes and cord:** The placenta is placed on a tray and is washed out in running tap water to remove the blood and clots. The maternal surface is first inspected for its **completeness** and anomalies. **The maternal surface** is covered with grayish decidua (spongy layer of the decidua basalis). Normally the cotyledons are placed in close approximation and any gap indicates a missing cotyledon. **The membranes**—chorion and amnion are to be examined carefully for completeness and presence of abnormal vessels indicative of succenturiate lobe. **The amnion is shiny but the chorion is shaggy**. The cut end of the cord is inspected for number of blood vessels. **Normally, there are two umbilical arteries and one umbilical vein. An oval gap in the chorion with torn ends of blood vessels running up to the margin of the gap indicates a missing succenturiate lobe**. The absence of a cotyledon or evidence of a missing succenturiate lobe or evidence of significant missing membranes demands exploration of the uterus urgently.

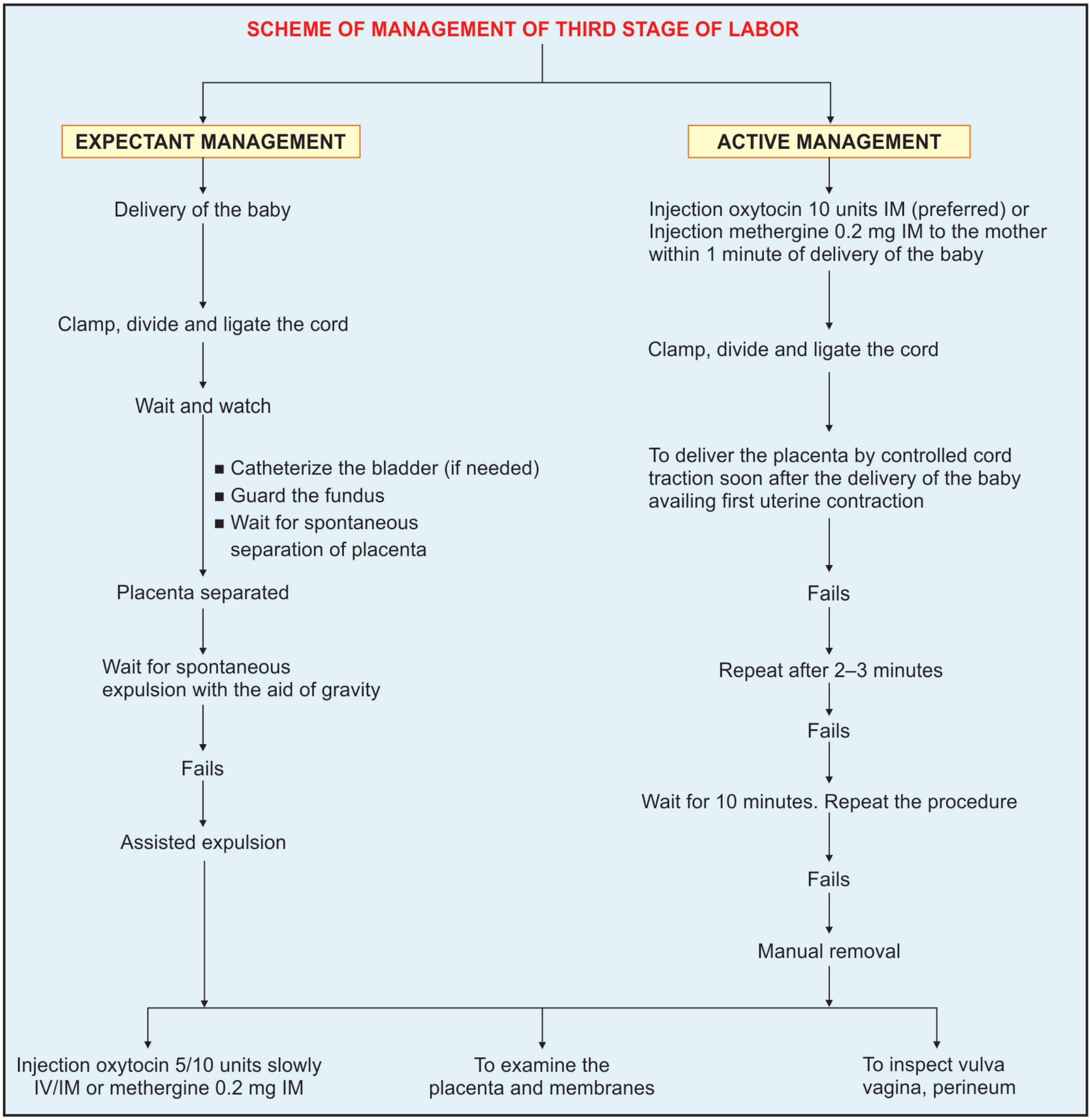
— **Vulva, vagina and perineum are inspected** carefully for injuries and to be repaired, if any. The episiotomy wound is now sutured. The vulva and adjoining part are cleaned with cotton swabs soaked in antiseptic solution. A sterile pad is placed over the vulva.

Active Management of Third Stage of Labor (AMTSL)

The underlying principle in active management is to excite powerful uterine contractions within 1 minute of delivery of the baby (WHO) by giving parenteral oxytocic. This facilitates not only early separation of the placenta but also produces effective uterine contractions following its separation.

The advantages are—(a) to minimize blood loss in third stage approximately to one-fifth and (b) to shorten the duration of third stage to half. **The only disadvantage** is slight increased incidence of retained placenta (1–2%) and consequent increased incidence of manual removal. Of course, accidental administration during delivery of the first baby in undiagnosed twins produces grave danger to the unborn second baby caused by asphyxia due to tetanic contraction of the uterus. **Thus, it is imperative to limit its use in twins only following delivery of the second baby**.

Procedures: Injection oxytocin 10 units IM (preferred) or methergine 0.2 mg IM is given within 1 minute of delivery of the baby (WHO). The placenta is expected to be delivered soon following delivery of the baby. If the placenta is not delivered thereafter, it should be delivered forthwith by controlled cord traction (Brandt-Andrews) technique after clamping the cord while the uterus still remains contracted. If the first attempt fails, another attempt is made after 2–3 minutes failing which another attempt is made at 10 minutes. If this still fails, manual removal is to be done. Oxytocic may be given with crowning of the head, with delivery of the anterior shoulder of the baby or after the delivery of the placenta. **If the administration is mistimed as might happen in a busy labor room, one should not be panicky but conduct the third stage with conventional watchful expectancy**.


SCHEME OF MANAGEMENT OF THIRD STAGE OF LABOR

EXPECTANT MANAGEMENT
Delivery of the baby
Clamp, divide and ligate the cord
Wait and watch
■ Catheterize the bladder (if needed)
■ Guard the fundus
■ Wait for spontaneous separation of placenta
Placenta separated
Wait for spontaneous expulsion with the aid of gravity
Fails
Assisted expulsion
ACTIVE MANAGEMENT
Injection oxytocin 10 units IM (preferred) or Injection methergine 0.2 mg IM to the mother within 1 minute of delivery of the baby
Clamp, divide and ligate the cord
To deliver the placenta by controlled cord traction soon after the delivery of the baby availing first uterine contraction
Fails
Repeat after 2–3 minutes
Fails
Wait for 10 minutes. Repeat the procedure
Fails
Manual removal
Injection oxytocin 5/10 units slowly IV/IM or methergine 0.2 mg IM
To examine the placenta and membranes
To inspect vulva vagina, perineum

Limitation: To be effective, it should be administered in proper time and followed by rapid delivery of the placenta. Thus, it may be an ideal procedure while conducting delivery in an equipped surrounding and the attendant is conversant with the management. Even if it cannot be extended routinely to all cases, **it is certainly of value**, **for cases likely to develop postpartum hemorrhage**. **These are** cases delivered vaginally under anesthesia, anemia, hydramnios, twins, grand multiparae and previous history of PPH. **Methergine should not be used** in cardiac cases or severe preeclampsia, for the risk of precipitating cardiac overload in the former and aggravation of blood pressure in the latter.

FOURTH STAGE OF LABOR: Pulse, blood pressure, tone of the uterus (well retracted) and any abnormal vaginal bleeding are to be watched at least for 1 hour after delivery. When fully satisfied that the general condition is good, pulse and blood pressure are steady, the uterus is well retracted and there is no abnormal vaginal bleeding, the patient is sent to the ward.

KEY POINTS

- **Labor** involves a series of changes in the genital organs associated with regular painful uterine contractions with effacement and dilatation of the cervix. **Delivery** is not synonymous with labor as it can take place without labor. Normal labor should fulfill some defined criteria (see p. 134).
- **Onset of labor** is difficult to understand (see p. 134). Role of estrogen, progesterone, prostaglandins, oxytocin, and the fetus have been explained (see p. 135).
- **Active phase of labor** begins when the cervix is 4 cm dilated. Management of latent phase (observation) and active phase of labor are different.
- **Labor events** are conventionally divided into two phases (latent phase and active phase) and three stages (see p. 137).
- **Main events in the first stage of labor** are: (a) **dilatation** and (b) **effacement** of the cervix (see p. 140). **Second stage** events are (a) **descent** and (b) **delivery** of the fetus. **Second stage** is characterized by two phases: **(a) propulsive and (b) expulsive (bear down)** to deliver the fetus (see p. 143). **Third stage events** are **separation** of placenta and **expulsion** of placenta (see p. 143).
- **Lower uterine segment** is formed mainly during the first stage of labor. Clinical importance of lower uterine segment are many (see p. 141).
- **Mechanism of normal labor** involves a series of movements on the head in the process of adaptation during its passage through the pelvis (see p. 145).
- The principal movements are: Engagement → Descent with increasing flexion of the head → Internal rotation → Crowning → Delivery of the head by extension → Restitution → External rotation → Delivery of the shoulders (anterior first followed by the posterior) → Delivery of the trunk by lateral flexion. **Descent and increasing flexion of the head are the continued process throughout the course of labor** (see p. 146).
- **Diagnosis of labor** (true labor pains) includes regular painful uterine contractions, progressive cervical dilatation and effacement and presence of show (see p. 137).
- **Progressive descent of fetal head** is assessed abdominally in terms of "fifths" (Crichton) (Figs 13.18B, C and 13.20) and also on vaginal examination by noting the station.
- **Successful labor and delivery** is dependent on complex interactions of three variables (three "Ps"): the Power (uterine contractions), the Passenger (fetus), and the Passage (pelvis).
- **Partograph** is used to record labor events (see p. 151, 606). Electronic fetal monitoring is used for high risk cases (see p. 692).

FIRST STAGE OF LABOR:

First stage of labor starts from the onset of true labor pain and **ends with** full dilatation of the cervix. **Its average duration** is about 12 hours in primigravidae and 6 hours in multiparae. **First stage consists** of *latent phase* (up to 4 cm of cervical dilatation) and *active phase* (up to 10 cm). The stage is chiefly concerned with dilatation and effacement of the cervix. **The stage is clinically manifested** by progressive uterine contraction, dilatation and "effacement" of the cervix and ultimate rupture of the membranes. **Maternal and fetal conditions** remain unaffected except during uterine contraction. **Management consists of:** (1) Noninterference with watchful expectancy. (2) Women is given **encouragement, emotional support and adequate pain relief** during the entire course of labor. (3) To monitor carefully the progress of labor, maternal condition and fetal behavior so as to detect any deviation from the normal. (4) **Partograph is maintained** (see p. 606).

SECOND STAGE OF LABOR:

The **second stage of labor starts** from full dilatation of the cervix and **ends with** expulsion of the fetus. **Its average duration** is 2 hours in primigravidae and 30 minutes in multiparae. **The stage concerns** with the descent and delivery of the fetus through the birth canal. **The stage is** clinically manifested by increased frequency and intensity of uterine contractions with appearance of "bearing-down" efforts which result in expulsion of the fetus. The mother may show features of exhaustion. The **principles in management are**: (1) To assist in the natural expulsion of the fetus slowly and steadily. (2) To prevent perineal injuries. During conduction of delivery, head is delivered slowly in between contractions. Flexion is maintained so that smaller diameter of the head stretches the perineum. This, along with timely performed episiotomy (selective), prevents perineal laceration. Shoulders are delivered slowly with next contraction. **Immediate care of the newborn** includes clearing of the air passage and eyes, clamping and ligaturing of the umbilical cord and Apgar scoring.

contd...

contd...

THIRD STAGE OF LABOR:

The third stage begins after the expulsion of the fetus and ends with expulsion of the placenta and membranes. Its **average duration is 15 minutes**. The stage concerns with placental separation and its expulsion. **The separation is** achieved by marked reduction in the uterine surface area of the placental site following delivery due to retraction. **The placenta being** inelastic shears off its attachment through the deep spongy decidual layer. There are **two ways of separation**—central (Schultze) and marginal (Mathews-Duncan). **The bleeding is** controlled by effective myometrial contraction and retraction (living ligature) and by thrombosis. **The expulsion may occur** through "bearing-down" efforts or more commonly with assistance. **The management is** either by employing watchful expectancy or **by active management (WHO)** in cases where oxytocin 10 units IV (slowly) or IM/methergine 0.2 mg IV is administered within 1 minute following the delivery of the baby. The placenta and the membranes should be examined following their expulsion.

FOURTH STAGE OF LABOR:

It is the stage of observation for at least 1 hour after the delivery of the baby, placenta and the membranes to ensure that both the mother and the baby are well.

QUESTIONS

1. What are the different stages of labor? Give an outline of the active management of third stage of labor? What are its advantage? (p. 138, 165)

Write Short Notes on:

A. Normal labor (p. 134)
B. Clinical significance of lower uterine segment (p. 141)
C. Important events in the third stage of labor (p. 143-44)

Chapter 14

Normal Puerperium

DEFINITION: Puerperium is the period following childbirth during which the body tissues, especially the pelvic organs revert back approximately to the prepregnant state both anatomically and physiologically. The retrogressive changes are mostly confined to the reproductive organs with the exception of the mammary glands which in fact show features of activity. **Involution is the process whereby the genital organs revert back approximately to the state as they were before pregnancy**. The woman is termed as a puerpera.

DURATION: Puerperium begins as soon as the placenta is expelled and **lasts for approximately 6 weeks** when the uterus becomes regressed almost to the nonpregnant size. The period is arbitrarily divided into — (a) immediate – within 24 hours, (b) early – up to 7 days and (c) remote – up to 6 weeks. **Similar changes occur following abortion but takes a shorter period for the involution to complete**.

Fourth trimester is the time from delivery until complete physiolgical involution and psychological adjustment.

INVOLUTION OF THE UTERUS

ANATOMICAL CONSIDERATION

Uterus: Immediately following delivery, the uterus becomes firm and retract with alternate hardening and softening. The uterus measures about 20 × 12 × 7.5 cm^3 (length, breadth and thickness) and weighs about 1,000 g (Fig. 14.1). At the end of 6 weeks, its measurement is almost similar to that of the nonpregnant state and weighs about 60 g. The decrease in size of the uterus and cervix has been shown with serial MRI (Fig. 14.2). **The placental site contracts** rapidly presenting a raised surface which measures about 7.5 cm and remains elevated even at 6 weeks when it measures about 1.5 cm.

Lower uterine segment: Immediately following delivery, the lower segment becomes a thin, flabby and collapsed structure. It takes a few weeks to revert back to the normal shape and size of the isthmus, i.e. the part between the body of the uterus and internal os of the cervix.

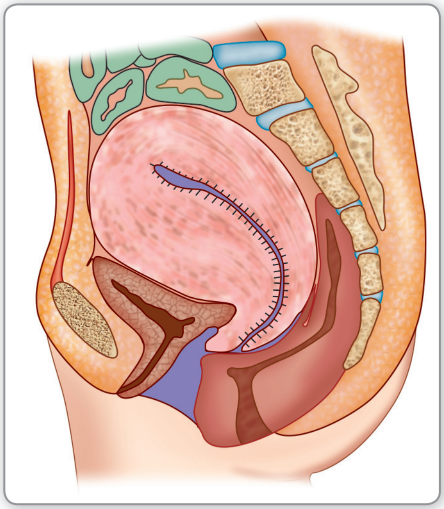


Fig. 14.1: Sagittal section showing uterus 5 days after delivery

Cervix: The cervix contracts slowly; the external os admits two fingers for a few days but by the end of 1st week, narrows down to admit the tip of a finger only. The contour of the cervix takes a longer time to regain (6 weeks) and the external os never reverts back to the nulliparous state.

PHYSIOLOGICAL CONSIDERATION

The physiological process of involution is most marked in the body of the uterus. Changes occur in the following components: (1) **Muscles,** (2) **Blood vessels,** (3) **Endometrium**.

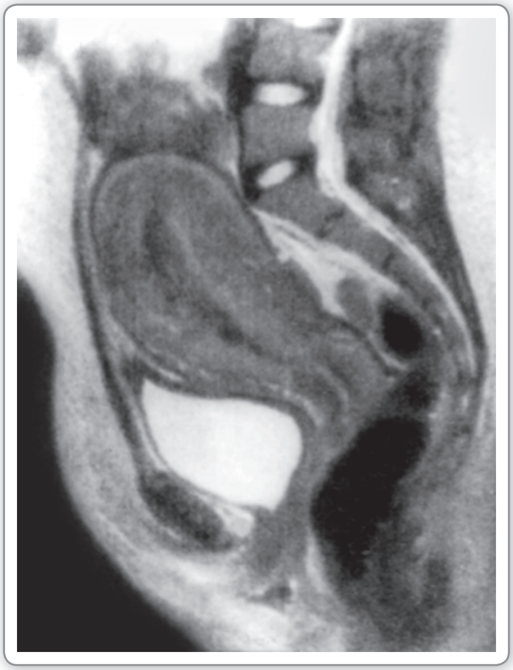


Fig. 14.2: MRI — sagittal view of the uterus and cervix showing large size and thick walls after 6 days of delivery

Muscles: There is marked hypertrophy and hyperplasia of muscle fibers during pregnancy and the individual muscle fiber enlarges to the extent of 10 times in length and 5 times in breadth. **During puerperium, the number of muscle fibers is not decreased, but there is substantial reduction of the myometrial cell size**. Withdrawal of the steroid hormones, estrogen and progesterone, may lead to increase in the activity of the uterine collagenase and the release of proteolytic enzyme. Autolysis of the protoplasm occurs by the proteolytic enzyme with liberation of peptones which enter the bloodstream. These are excreted through the kidneys as urea and creatinine. This explains the increased excretion of the products in the puerperal urine. The connective tissues also undergo the same type of degeneration. **The conditions which favor involution are** — (a) efficacy of the enzymatic action and (b) relative anoxia induced by effective contraction and retraction of the uterus.

Blood vessels: The changes of the blood vessels are pronounced at the placental site. **The arteries are constricted** by contraction of its wall and thickening of the intima followed by thrombosis. During the 1st week, arteries undergo thrombosis, hyalinization and fibrinoid endarteritis. Veins are obliterated by thrombosis, hyalinization and endophlebitis. **New blood vessels grow** inside the thrombi.

Endometrium: Following delivery, the major part of the decidua is cast off with the expulsion of the placenta and the membranes, more at the placental site. The endometrium left behind varies in thickness from 2 mm to 5 mm. The superficial part containing the degenerated decidua, blood cells and bits of fetal membranes becomes necrotic and is cast off in the lochia. **Regeneration starts by 7th day**. **It occurs from the epithelium of the uterine gland mouths and interglandular stromal cells**. **Regeneration of the epithelium is completed by 10th day and the entire endometrium is restored by the day 16, except at the placental site where it takes about 6 weeks**.

CLINICAL ASSESSMENT OF INVOLUTION

The rate of involution of the uterus can be assessed clinically by noting the height of the fundus of the uterus in relation to the symphysis pubis. The measurement should be taken carefully at a fixed time every day, preferably by the same observer. **Bladder must be emptied beforehand** and preferably the bowel too, as the full bladder and the loaded bowel may raise the level of the fundus of the uterus. **The uterus is to be centralized** and with a measuring tape, the fundal height is measured above the symphysis pubis. **Following delivery, the fundus lies about 13.5 cm (5 1/2") above the symphysis pubis. During the first 24 hours, the level remains constant; thereafter, there is**

a steady decrease in height by 1.25 cm (0.5") in 24 hours, so that by the end of 2nd week the uterus becomes a pelvic organ. The rate of involution thereafter slows down until by 6 weeks, the uterus becomes almost normal in size.

The involution may be affected adversely and is called subinvolution. Sometimes, the involution may be continued in women who are lactating so that the uterus may be smaller in size — superinvolution. The uterus, however, returns to normal size if the lactation is withheld.

INVOLUTION OF OTHER PELVIC STRUCTURES

Vagina: The distensible vagina, noticed soon after birth takes a long time (6–10 weeks) to involute. It regains its tone but never to the virginal state. The mucosa remains delicate for the first few weeks and submucous venous congestion persists even longer. It is the reason to withhold surgery on puerperal vagina. Rugae partially reappear at 3rd week but never to the same degree as in prepregnant state. Introitus remains permanently larger than the virginal state. Hymen is lacerated and is represented by nodular tags — the carunculae myrtiformes.

Broad ligaments and round ligaments require considerable time to recover from the stretching and laxation.

Pelvic floor and pelvic fascia take a long time to involute from the stretching effect during parturition.

LOCHIA

It is the vaginal discharge for the first fortnight during puerperium. The discharge originates from the uterine body, cervix and vagina.

Odor and reaction: It has got a peculiar offensive fishy smell. Its reaction is alkaline, tending to become acid toward the end.

Color: Depending upon the variation of the color of the discharge, it is named as: (1) **lochia rubra** (red) 1–4 days, (2) **lochia serosa** (5–9 days) — the color is yellowish or pink or pale brownish, (3) **lochia alba** — (pale white) — 10–15 days.

Composition: Lochia rubra consists of blood, shreds of fetal membranes and decidua, vernix caseosa, lanugo and meconium.

Lochia serosa consists of less RBC but more leukocytes, wound exudate, mucus from the cervix and microorganisms (anaerobic streptococci and staphylococci). **The presence of bacteria is not pathognomonic unless associated with clinical signs of sepsis**.

Lochia alba contains plenty of decidual cells, leukocytes, mucus, cholesterin crystals, fatty and granular epithelial cells and microorganisms.

Amount: The average amount of discharge for the first 5–6 days is estimated to be 250 mL.

Normal duration: The normal duration may extend up to 3 weeks. The red lochia may persist for longer duration especially in women who get up from the bed for the first time in later period. The discharge may be scanty, especially following premature labors or may be excessive in twin delivery or hydramnios.

Clinical importance: The character of the lochial discharge gives useful information about the abnormal puerperal state. **The vulval pads are to be inspected daily to get information of:**

- **Odor:** If malodorous—indicates infection. **Retained plug or cotton piece inside the vagina should be kept in mind**.
- **Amount:** Scanty or absent — signifies infection or lochiometra. If excessive — indicates infection.
- **Color:** Persistence of red color beyond the normal limit signifies subinvolution or retained bits of conceptus.
- **Duration:** Duration of the lochia alba beyond 3 weeks suggests local genital lesion.

GENERAL PHYSIOLOGICAL CHANGES

PULSE: For a few hours after normal delivery, the pulse rate is likely to be raised, which settles down to normal during the second day. However, the pulse rate often rises with after-pain or excitement.

TEMPERATURE: The temperature should not be above 37.2°C (99°F) within the first 24 hours. There may be slight reactionary rise following delivery by 0.5°F but comes down to normal within 12 hours. On the 3rd day, there may be slight rise of temperature due to breast engorgement which should not last for more than 24 hours. **However, genitourinary tract infection should be excluded if there is rise of temperature**.

URINARY TRACT: The bladder mucosa becomes edematous and hyperemic and often shows evidences of submucous extravasation of blood. The bladder capacity is increased. The bladder may be overdistended without any desire to pass urine. **The common urinary problems are**: overdistention, incomplete emptying and presence of residual urine. Urinary stasis is seen in more than 50% of women. **The risk of urinary tract infection is, therefore, high**. Dilated ureters and renal pelvis return to normal size within 8 weeks. There is pronounced diuresis on the 2nd or 3rd day of the puerperium. **Only "clean catch" sample of urine should be collected** and sent for examination and contamination with lochia should be avoided.

GASTROINTESTINAL TRACT: **Increased thirst** in early puerperium is due to loss of fluid during labor, in lochia, diuresis and perspiration. Constipation is a common problem for the following reasons: delayed gastrointestinal motility, mild ileus following delivery, together with perineal discomfort. Some women may have the problem of anal incontinence.

WEIGHT LOSS: In addition to the weight loss (5–6 kg) as a consequence of the expulsion of the fetus, placentae, liquor and blood loss, a further loss of about 2 kg (4.4 lb) occurs during puerperium chiefly caused by diuresis. This weight loss may continue up to 6 months of delivery.

URINARY TRACT AND RENAL FUNCTION: In relation to changes in pregnancy (see p. 63) persistence of urinary stasis in the ureters and bladder is observed even up to 12 weeks postpartum. Glomerular filtration returns to normal by 8 weeks postpartum.

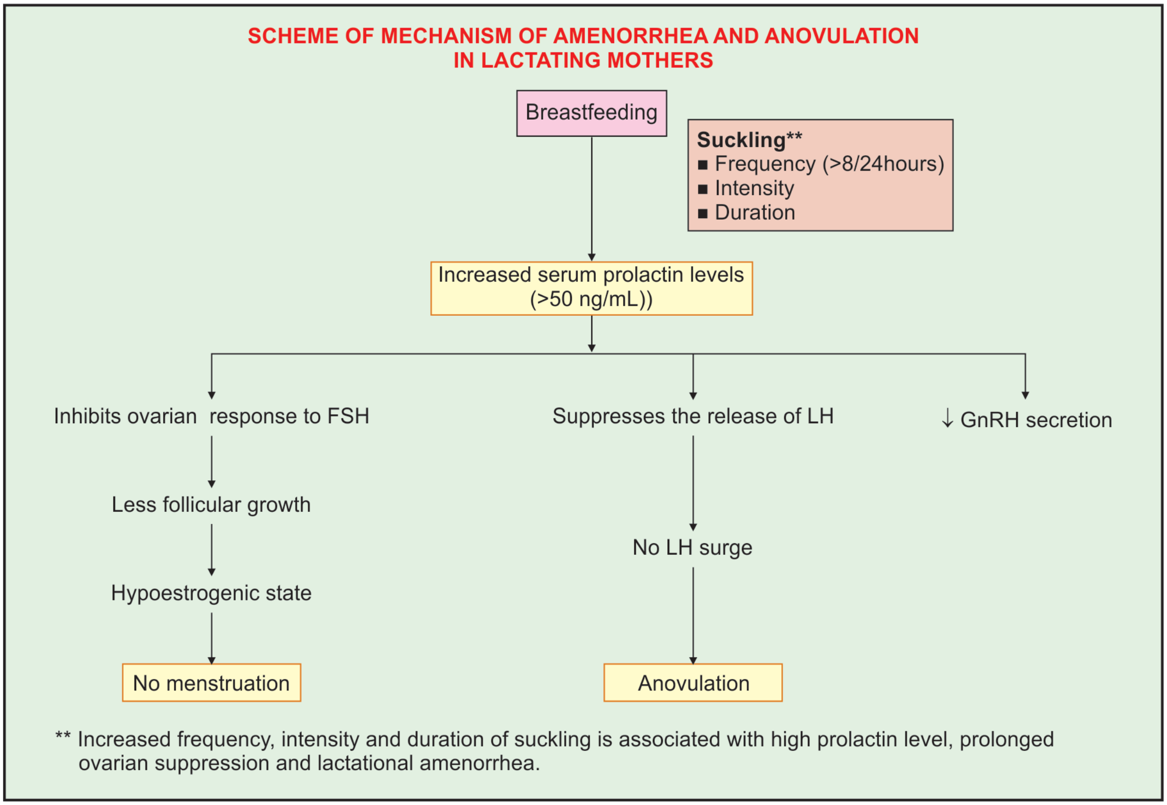
FLUID LOSS: There is a net fluid loss of at least 2 liters during the 1st week and an additional 1.5 liters during the next 5 weeks. **The amount of loss depends** on the amount retained during pregnancy, dehydration during labor and blood loss during delivery. The loss of salt and water are larger in women with preeclampsia and eclampsia.

BLOOD VALUES: Immediately following delivery, there is slight **decrease of blood volume** due to blood loss and dehydration. Blood volume returns to nonpregnant level by the 2nd week. **Cardiac output** rises soon after delivery to about 80% above the prelabor value but slowly returns to normal within 1 week.

RBC volume and hematocrit values returns to normal by 8 weeks postpartum after the hydremia disappears. **Leukocytosis** to the extent of 25,000/mm^3 occurs following delivery probably in response to stress of labor. **Platelet count** decreases soon after the separation of the placenta but secondary elevation occurs, with increase in platelet adhesiveness between 4 and 10 days. **Fibrinogen level** remains high up to the 2nd week of puerperium. A hypercoagulable state persists for 48 hours postpartum and fibrinolytic activity is enhanced in first 4 days. The secondary increase in fibrinogen, factor VIII and platelets in the 1st week increases the risk for thrombosis. The increase in fibrinolytic activity after delivery acts as a protective mechanism.

OVARIAN FUNCTION (MENSTRUATION AND OVULATION): The onset of the first menstrual period following delivery is very variable and depends on lactation. **If woman does not breastfeed her baby**, menstruation returns by 12th week following delivery in 80% of cases. The meantime for onset of first menstruation is 7 – 9 weeks.

In nonlactating mothers, ovulation may occur as early as 4 weeks and **in lactating mothers** about 10 weeks after delivery. Duration of anovulation depends upon the frequency (>8/24 hours), intensity and duration of breastfeeding. The physiological basis of anovulation and amenorrhea is due to elevated levels of serum prolactin associated with suckling. In lactating mothers the mechanism of amenorrhea and anovulation are depicted schematically below. **Women who is exclusively breastfeeding, the**



SCHEME OF MECHANISM OF AMENORRHEA AND ANOVULATION IN LACTATING MOTHERS

Breastfeeding
Suckling**
■ Frequency (>8/24hours)
■ Intensity
■ Duration
Increased serum prolactin levels (>50 ng/mL))
Inhibits ovarian response to FSH
Suppresses the release of LH
↓ GnRH secretion
Less follicular growth
Hypoestrogenic state
No menstruation
No LH surge
Anovulation

** Increased frequency, intensity and duration of suckling is associated with high prolactin level, prolonged ovarian suppression and lactational amenorrhea.

contraceptive protection is about 98% up to 6 months of postpartum. Thus, lactation provides a natural method of contraception (see p. 614). **However, ovulation may precede the first menstrual period in about one-third and it is possible for the patient to become pregnant before she menstruates following her confinement**. Nonlactating mother should use contraceptive measures in **3rd postpartum week** and the lactating mother in **3rd postpartum month**.

THYROID FUNCTION: Thyroid volume regresses gradually to prepregnant state by 12 weeks' time. Thyroid functions return to normal by 4 weeks postpartum. Women on thyroid medications should get their thyroid function checked to readjust the drugs.

LACTATION

For the first 2 days following delivery, no further anatomic changes in the breasts occur. The secretion from the breasts called colostrum, which starts during pregnancy becomes more abundant during the period.

COMPOSITION OF THE COLOSTRUM: It is deep yellow serous fluid, alkaline in reaction. It has got a higher specific gravity; a high protein, vitamin A, sodium and chloride content but has got lower carbohydrate, fat and potassium than the breast milk (Table 14.1). Colostrum and milk contains immunologic components such as immunoglobulin A (IgA), complements, macrophages, lymphocytes, lactoferrin and other enzymes (lactoperoxidase).

Microscopically: It contains fat globules, colostrum corpuscles and acinar epithelial cells. **The colostrum corpuscles** are large polynuclear leukocytes, oval or round in shape containing numerous fat globules.

Table 14.1: Percentage Composition of Colostrum and Breast Milk

	Protein	Fat	Carbohydrate	Water
Colostrum	8.6	2.3	3.2	86
Breast milk	1.2	3.2	7.5	87

Advantages: (1) The antibodies (IgA, IgG, IgM) and humoral factors (lactoferrin) provides immunogical defense to the new born (see p. 520). (2) It has laxative action on the baby because of large fat globules.

PHYSIOLOGY OF LACTATION

Although lactation starts following delivery, the preparation for effective lactation starts during pregnancy. **The physiological basis of lactation is divided into four phases**:

(a) Preparation of breasts (mammogenesis).
(b) Synthesis and secretion from the breast alveoli (lactogenesis).
(c) Ejection of milk (galactokinesis).
(d) Maintenance of lactation (galactopoiesis).

The endocrine control in relation to different phases of lactation has been depicted in Figure 6.2.

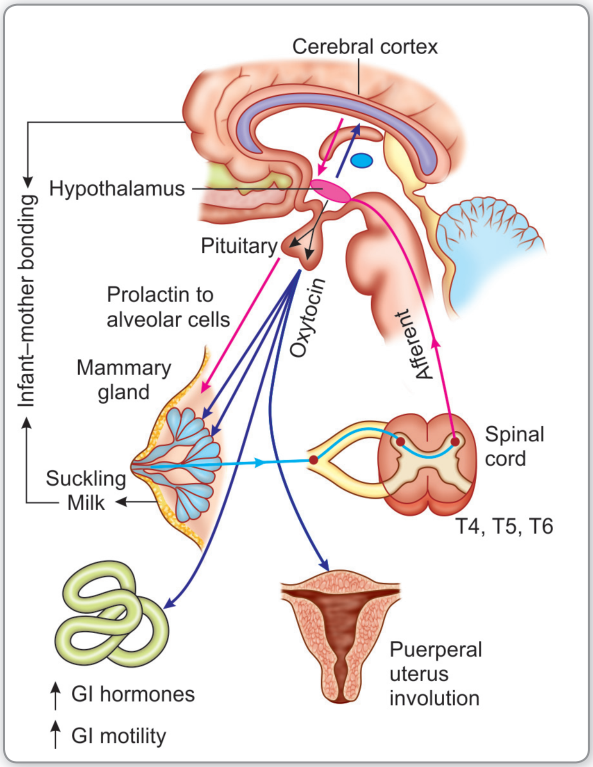


Fig. 14.3: Lactation reflex arc and the role of prolactin and oxytocin

- **Mammogenesis:** Pregnancy is associated with remarkable growth of both ductal and lobuloalveolar systems. **An intact nerve supply is not essential for the growth of mammary glands during pregnancy**.
- **Lactogenesis:** The alveolar cells are the principal sites for production of milk. Though some secretory activity is evident (colostrum) during pregnancy and accelerated following delivery, **milk secretion actually starts on 3rd or 4th postpartum day**. Around this time, the breasts become engorged, tense, tender and feel warm. Inspite of a high prolactin level during pregnancy, milk secretion is kept in abeyance. Probably, steroids — estrogen and progesterone circulating during pregnancy make the breast tissues unresponsive to prolactin. When the estrogen and progesterone are withdrawn following delivery, prolactin begins its milk secretory activity in previously fully developed mammary glands. **Prolactin, insulin, growth hormone and glucocorticoids are the important hormones in this stage**. The secretory activity is also enhanced directly or indirectly by growth hormone, thyroxine and insulin. **For milk secretion to occur, nursing effort is not essential**.
- **Galactokinesis: Oxytocin is the major galactokinetic hormone**. Discharge of milk from the mammary glands depends not only on the suction exerted by the baby during **suckling** but also on the **contractile mechanism** which expresses the milk from the alveoli into the ducts.

  DURING SUCKLING, A CONDITIONED REFLEX IS SET-UP (Fig. 14.3): The ascending tackle impulses from the nipple and areola pass via thoracic sensory (4, 5 and 6) afferent neural arc to the paraventricular and supraoptic nuclei of the hypothalamus to synthesize and transport oxytocin to the posterior pituitary (Fig. 14.3). Oxytocin (efferent arc via blood) is liberated from the posterior pituitary, produces contraction of the myoepithelial cells of the alveoli and the ducts containing the milk. This is the **"milk ejection" or "milk let down" reflex** whereby the milk is forced down into the ampulla of the lactiferous ducts, where from it can be expressed by the mother or sucked out by the baby. Presence of the infant or infant's cry can induce let down without suckling. A sensation of rise of pressure in the breasts by milk experienced by the mother at the beginning of sucking is called "draught". This can also be produced by injection of oxytocin.

  The milk ejection reflex is inhibited by factors such as pain, anxiety, breast engorgement or adverse psychic condition (depression). The ejection reflex may be deficient for several days following initiation of milk secretion and results in breast engorgement.

- **Galactopoiesis**: **Prolactin** appears to be the **single most important** galactopoietic hormone. **For maintenance of effective and continuous lactation, frequency of suckling (>8/24 hours) is essential**. Distension of the alveoli by retained milk is due to failure of suckling. This causes decrease in milk secretion by the alveolar epithelium. Ductal and alveolar distension due to failure of milk transfer (suckling) is a cause of lactation failure. Milk pressure reduces the rate of production and hence periodic breastfeeding is necessary.
- **MILK PRODUCTION:** A healthy mother will produce about 500–800 mL of milk a day to feed her infant. This requires about 700 Kcal/day for the mother, which must be made up from diet or from her body store. For this purpose a store of about 5 kg of fat during pregnancy is essential to make up any nutritional deficit during lactation.

STIMULATION OF LACTATION: Mother is motivated as regard the benefits of breastfeeding (see p. 520) since the **early pregnancy**. No prelacteal feeds (honey, water) are given to the infant. **Following delivery** important steps are: (i) to put the baby to the breast at 2–3 hours interval from the 1st day, (ii) plenty of fluids to drink and (iii) to avoid breast engorgement. **Early (½ – 1 hour) and exclusive breastfeeding in correct position (see p. 521) is encouraged**.

INADEQUATE MILK PRODUCTION (Lactation failure): It may be due to infrequent suckling or due to endogenous suppression of prolactin (ergot preparation, pyridoxin, diuretics or retained placental bits). Pain, anxiety and insecurity may be the hidden reasons. **Unrestricted feeding at short interval (2–3 hours) is helpful**.

DRUGS TO IMPROVE MILK PRODUCTION (Galactogogues): Metoclopramide (10 mg thrice daily) increases milk volume (60–100%) by **increasing prolactin** levels. Sulpiride (dopamine antagonist), domperidone has also been found effective by increasing prolactin levels. Intranasal oxytocin contracts myoepithelial cells and causes milk let down.

Lactation suppression: It may be needed for women who cannot breastfeed for personal or medical reasons. Lactation is suppressed when the baby is born dead or dies in the neonatal period or if breastfeeding is contraindicated. Methods commonly used are: (i) to stop breastfeeding, (ii) to avoid pumping or milk expression, (iii) to wear breast support, (iv) ice packs to prevent engorgement, (v) analgesics (aspirin) to relieve pain and (vi) a tight compression bandage is applied for 2–3 days. The natural inhibition of prolactin secretion will result in breast involution.

Medical methods of suppression with estrogen, androgen or bromocriptine is not advised. The side effects of bromocriptine are: hypotension, rebound secretion, seizures, myocardial infarction and puerperal stroke.

Breast milk for premature infant is beneficial by many ways (psychological, nutritional and immunological). **Metabolic disturbances like azotemia, hyperaminoacidemia and metabolic acidosis are less with breast milk compared to formula**. It gives immunological protection to the premature infant. There are methods for collection (manual expression or electric pumps), and milk preservation.

MANAGEMENT OF NORMAL PUERPERIUM

The principles in management are: (1) To restore the health of the mother. (2) **To prevent infection**. (3) **To take care of the breasts**, including promotion of breastfeeding. (4) **To motivate the mother** for contraception.

IMMEDIATE ATTENTION: Immediately following delivery, the patient should be closely observed as outlined in the management of the fourth stage of labor (see p. 165). She may be given a drink of her choice or something to eat, if she is hungry. **Emotional support is essential**. Usually the first feeling of mother is the sense of happiness and relief, with the birth of a healthy baby. The woman needs emotional support when she suffers from postpartum blues or stress due to newborn's prematurity, illness, congenital malformation or death.

REST AND AMBULANCE: Early ambulation after delivery is beneficial. After a good resting period, the patient becomes fresh and can breastfeed the baby or moves out of bed to go to the toilet. **Early ambulation is encouraged**. **Advantages are**: (1) provides a sense of well-being, (2) bladder complications and constipation are less, (3) facilitates uterine drainage and hastens involution of the uterus and (4) lessens puerperal venous thrombosis and embolism. Following an uncomplicated delivery, climbing stairs, lifting objects, daily household work and cooking may be resumed.

HOSPITAL STAY: Early discharge from the hospital is an almost universal procedure. If adequate supervision by trained health visitors is provided, there is no harm in early discharge. Most women are discharged fit and healthy after 2 days of spontaneous vaginal delivery with proper education and instructions. Early discharge may be done in a few selected women. Some need prolonged hospitalization due to morbidities (infections of urinary tract, or the perineal wound, pain, or breastfeeding problems).

DIET: The patient should be on normal diet of her choice. **If the patient is lactating**, high calories, adequate protein, fat, plenty of fluids, minerals and vitamins are to be given (see p. 112). **However, in nonlactating mothers,** a diet is enough as in nonpregnant woman.

CARE OF THE BLADDER: The patient is encouraged to pass urine following delivery as soon as convenient. At times, **the patient fails to pass urine and the causes are** — (1) unaccustomed position and (2) reflex pain from the perineal injuries. This is common after a difficult labor or a forceps delivery. **If the patient still fails to pass urine, catheterization should be done**. Catheterization is also indicated in case of incomplete emptying of the bladder evidenced by the presence of residual urine of more than 60 mL. ***Continuous drainage* is kept until the bladder tone is regained**. The underlying principle of the bladder care is to ensure adequate drainage of urine so that infection and cystitis are avoided.

CARE OF THE BOWEL: The problem of constipation is much less because of early ambulation and liberalization of the dietary intake. A diet containing sufficient roughage and fluids is enough to move the bowel. If necessary, mild laxative such as isabgol husk two teaspoons may be given at bed time.

SLEEP: The patient is in need of rest, both physical and mental. So she should be protected against worries and undue fatigue. Sleep is ensured providing adequate physical and emotional support. If there is any discomfort, such as after pain or painful piles or engorged breasts, they should be dealt with adequate analgesics (Ibuprofen).

CARE OF THE VULVA AND EPISIOTOMY WOUND: Shortly after delivery, the vulva and buttocks are washed with soap water down over the anus and a sterile pad is applied. The patient should look after personal cleanliness of the vulval region. The perineal wound should be dressed with spirit and antiseptic powder after each act of micturition and defecation or at least twice a day. **The nurse should use sterilized gloves during dressing. Cold (ice) sitz baths** relieve pain by reducing edema and inflammation. It causes vasoconstriction. When the perineal pain is persistent, a vaginal and rectal examination is done to detect any hematoma, wound gaping or infection. For pain Ibuprofen is safe for nursing mothers.

CARE OF THE BREASTS: The nipple should be washed with sterile water before each feeding. It should be cleaned and kept dry after the feeding is over. A nursing brassiere provides comfortable support.

Nipple soreness is avoided by frequent short feedings rather than the prolonged feeding, keeping the nipples clear and dry. Candida infection may be another cause (see p. 523).

Nipple confusion is a situation where the infant accepts the artificial nipple but refuses the mother's nipple. This is avoided by making the mother's nipple more protractile and not offering any supplemental fluids to the infant.

MATERNAL-INFANT BONDING (ROOMING-IN): It starts from first few moments after birth. This is manifested by bonding, kissing, cuddling and gazing at the infant. The baby should be kept in her bed or in a cot besides her bed. This not only establishes the mother-child relationship but the mother is conversant with the art of baby care so that she can take full care of the baby while at home. Baby, friendly hospital initiative (see p. 519) promotes parent-infant bonding, baby rooming with the mother and breastfeeding.

ASEPSIS AND ANTISEPTICS: Asepsis must be maintained especially during the 1st week of puerperium. Liberal use of local antiseptics, aseptic measures during perineal wound dressing, use of clean bed linen and clothings are positive steps. Clean surroundings and limited number of visitors could be of help in reducing nosocomial infection.

IMMUNIZATION: (i) Administration of anti–D–gamma globulin to unimmunized Rh-negative mother bearing Rh-positive baby (see details in Chapter 23). (ii) Women who are susceptible to rubella can be vaccinated safely with live attenuated rubella virus. Mandatory postponement of pregnancy for at least 2 months following vaccination can be easily achieved. (iii) The booster dose of tetanus toxoid, HepB, Tdap, should be given at the time of discharge, if it is not given during pregnancy. All are safe during breastfeeding.

MANAGEMENT OF AILMENTS

- **After pain** — **It is infrequent, spasmodic pain felt in the lower abdomen after delivery** for a variable period of 2–4 days. **Presence of blood clots** or bits of after births lead to hypertonic contractions of the uterus in an attempt to expel them out. This is commonly met in primipara. Pain may also be due to vigorous uterine contraction especially in multipara. The mechanism of pain is similar to cardiac anginal pain induced by ischemia. Both the types are excited during breastfeeding. **The treatment includes** massaging the uterus with expulsion of the clot followed by administration of analgesics (Ibuprofen) and antispasmodics.
- **Pain on the perineum: Never forget to examine the perineum** when analgesic is given to relieve pain. Early detection of vulvo-vaginal hematoma can thus be made. Sitz baths (hot or cold) can give additional pain relief.
- **Correction of anemia**: Majority of the women in the tropics remain in an anemic state following delivery. Supplementary iron therapy (ferrous sulfate 200 mg) is to be given daily for a minimum period of 4–6 weeks.
- **Hypertension** is to be treated until it comes to a normal limit. **Physician should be consulted if proteinuria persists**.

TO MAINTAIN A CHART (Fig.14.4): A progress chart is to be maintained noting the following points: (1) pulse, respiration and temperature recording 6 hourly or at least twice a day, (2) measurement of the height of the uterus above the symphysis pubis once a day in a fixed time with prior evacuation of the bladder and preferably the bowel too, (3) character of the lochia and (4) urination and bowel movement.

POSTPARTUM EXERCISE: The objectives of postpartum exercises are: (1) to improve the muscle tone, which are stretched during pregnancy and labor especially the abdominal and perineal muscles and (2) to educate about correct posture to be attained when the patient is getting up from her bed. This also includes the correct principle of lifting and working positions during day-to-day activities.

Advantages gained thereby are: (1) to minimize the risk of puerperal venous thrombosis by promoting arterial circulation and preventing venous stasis, (2) to prevent backache and (3) to prevent genital prolapse and stress incontinence of urine.

PROCEDURE: (1) Initially, she is taught breathing exercise and leg movements lying in bed. (2) Gradually, she is instructed to tone up the abdominal and perineal muscles and to correct the postural defects. These can be taught well by a trained physiotherapist. The exercise should be continued for at least 3 months. **The common exercises prescribed are: (a) To tone up the pelvic floor muscles:** The patient is asked to contract the pelvic muscles in a manner to withhold the act of defecation or urination and then to relax. The process is to be repeated as often as possible each day. **(b) To tone up the abdominal muscles:** The patient is to lie in dorsal position with the knees bent and the feet flat on

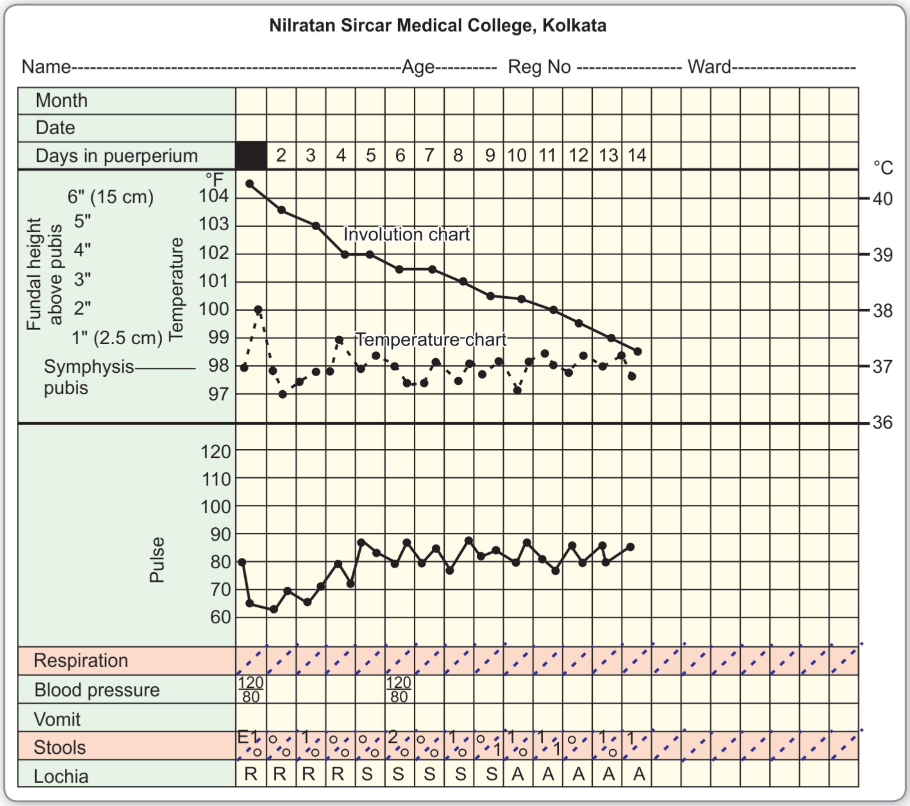


Fig. 14.4: A composite chart to note the important clinical events in the puerperium

the bed. The abdominal muscles are contracted and relaxed alternately and the process is to be repeated several times a day. **(c) To tone up the back muscles:** The patient is to lie on her face with the arms by her side. The head and the shoulders are slowly moved up and down. The procedure is to be repeated 3–4 times a day and gradually increased each day.

Physical activity should be resumed without delay. **Sexual activity** may be resumed (after 6 weeks) when the perineum is comfortable and bleeding has stopped. Some women may get "flaring response" of some autoimmune disorders due to rebound effect of the immune suppression during pregnancy (see p. 719).

CHECK-UP AND ADVICE ON DISCHARGE: A thorough check-up of the mother and the baby is mandatory prior to discharge of the patient from the hospital. Discharge certificate should have all the important informations regarding mother and baby.

Advice includes: (1) Measures to improve her general health. Continuance of supplementary iron therapy, (2) postnatal exercises, (3) procedures for a gradual return to day-to-day activities, (4) breastfeeding and care of the newborn, (5) avoidance of intercourse for a reasonable period of 4–6 weeks until lacerations or episiotomy wound are well healed, (6) **family planning advice and guidance — Nonlactating women should practice some form of contraceptive measures after 3 weeks and the lactating women should start 3 months after delivery** and (7) To have postnatal check up after 6 weeks.

The method of contraception will depend upon breastfeeding status, state of health and number of children (see p. 611). **Natural methods** cannot be used until menstrual cycles are regular. Exclusive breastfeeding provides 98% contraceptive protection for 6 months. **Barrier methods** (see p. 611) may be used. **Steroidal contraceptions** — combined preparations are suitable for nonlactating women and should be started 3 weeks after. In **lactating**

women it is avoided due to its suppressive effects (see p. 621, 627). **Progestin only pill** may be a better choice for them. **Other progestins** (DMPA, Levonorgestrel implants (see p. 615) may be used. **IUDs** are also a satisfactory method irrespective of breastfeeding status. **Insertion of IUD** immediately following delivery is currently done. Perforation rates are less. Expulsion rate is slightly high (10–20%). **Sterilization** (puerperal) is suitable for those who have completed their families.

POSTNATAL CARE

Postnatal care includes systematic examination of the mother and the baby and appropriate advice given to the mother during postpartum period. **The first postnatal examination** is done and the advice is given on discharge of the patient from the hospital. This has already been discussed. **The second routine postnatal care is conducted** at the end of 6th week postpartum.

AIMS AND OBJECTIVES:

- To assess the health status of the mother. Medical disorders like diabetes, hypertension, thyroid disorders should be reassessed.
- To detect and treat at the earliest any gynecological condition arising out of obstetric legacy.
- To note the progress of the baby including the immunization schedule for the infant (see p. 526).
- **To impart family planning guidance (discussed above)**.

PROCEDURE: • Examination of the mother • Advice given to the mother • Examination of the baby and advice

Examination of the Mother:

- **Routine examination includes** recording weight, pallor, blood pressure and tone of the abdominal muscles and examination of the breast.
- **Pelvic examination should be done only when indicated**. **The following should be noted:** A cervical smear may be taken for exfoliative cytological examination if this has not been done previously and insertion of intrauterine contraceptive device may be done when desired.
- **Laboratory investigations (e.g. hemoglobin) depending on the clinical need may be advised**.

Examination of the baby: This should be conducted by a **pediatrician**. In this respect, a well attached baby clinic to the postpartum unit is an absolute necessity. The progress of the baby is evaluated and preventive or curative steps are to be taken. Immunization to the baby is started (see p. 526).

Advice given: General — (1) If the patient is in sound health she is allowed to do her usual duties. (2) Postpartum exercises may be continued for another 4–6 weeks. (3) Vaccination MMR, HepB, (4) To evaluate the progress of the baby periodically and to continue breastfeeding for 6 months.

Family planning counseling and guidance — (discussed above and also see p. 609).

Management of ailments: Additional investigation and appropriate therapy is given according to the abnormalities detected during check up. **Management of some common gynecological problems are given below**. Some women need psychological support also (see p. 512).

- **Irregular vaginal bleeding:** It is not uncommon to encounter irregular or at times, heavy bleeding after 4–6 weeks following an uneventful period after delivery. This is usually the first period especially in nonlactating women and simple assurance is enough. Persistence of bleeding dating back from childbirth is likely due to retained bits of conceptus and usually requires ultrasound examination followed by dilatation and curettage operation.
- **Leukorrhea:** Profuse white discharge might be due to ill health, vaginitis, cervicitis or subinvolution. Improvement of the general health and specific therapy cure the condition.
- **Cervical ectopy (erosion)** met during this period without any symptom should not be treated surgically. Hormone-induced ectopy during pregnancy takes a longer time (about 12 weeks) to regress. **Thus, asymptomatic ectopy should be examined again after 6 weeks and if it still persists, cauterization is to be considered.**

- **Backache:** It is mostly due to sacroiliac or lumbosacral strain. **Backache over the sacrum is likely due to pelvic pathology**, but if it is over the lumbar region, it might be due to an orthopedic condition and is often relieved by physiotherapy.
- **Retroversion** seldom produces backache. If associated with subinvolution with symptoms, a pessary is inserted after correcting the position and is to be kept about 2 months.
- **Slight degree of uterine descent** with cystocele, stress incontinence and relaxed perineum are the common findings at this stage. These can be cured by effective pelvic floor exercise. However, **if the prolapse is marked, effective surgery should be done after 3 months**.
- **Urinary and anal incontinence:** The woman is examined for any sphincter injury. Perineal exercises are advised. Women with persistent symptom after 6 months need special investigations and surgical treatment.

QUESTIONS

1. Define puerperium? Discuss in brief the management of normal puerperium? (p. 168 and 174)

Write Short Notes on:

A. Mechanism of amenorrhea and anovulation during lactation (p. 172)

B. Contraceptive advice in puerperium (p. 177)

Chapter 15

Vomiting in Pregnancy

Vomiting is a symptom which may be related to pregnancy or may be a manifestation of some medical-surgical-gynecological complications, which can occur at any time during pregnancy. The former is by far the most common one and is called vomiting of pregnancy. **The causes of vomiting in pregnancy can be classified as follows:**

A. Early Pregnancy	B. Late Pregnancy
▪ Related to pregnancy (vomiting of pregnancy) • Simple vomiting (morning sickness, emesis gravidarum) • Hyperemesis gravidarum (pernicious vomiting) ▪ Associated with pregnancy (see table below)	▪ Related to pregnancy • Continuation or reappearance of simple vomiting of pregnancy • Acute fulminating preeclampsia ▪ Associated with pregnancy (see table below) • Medical-surgical-gynecological causes in early pregnancy • Hiatus hernia

Medical	Surgical	Gynecological
♦ Intestinal infestation	♦ Appendicitis	♦ Twisted ovarian tumor
♦ Urinary tract infection	♦ Peptic ulcer	♦ Red degeneration of fibroid
♦ Hepatitis	♦ Intestinal obstruction	
♦ Ketoacidosis of diabetes	♦ Cholecystitis	
♦ Pyelonephritis, uremia	♦ Pancreatitis	

VOMITING IN PREGNANCY

The vomiting is related to the pregnant state and depending upon the severity, it is classified as: (i) **Simple vomiting of pregnancy or milder type** (ii) **Hyperemesis gravidarum or severe type**.

SIMPLE VOMITING (Syn: morning sickness, emesis gravidarum): The patient complains of nausea and occasional sickness on rising in the morning. Slight vomiting is so common in early pregnancy (about 50%) that it is considered as a symptom of pregnancy. It may, however, occur at other times of the day. The vomitus is small and clear or bile stained. It does not produce any impairment of health or restrict the normal activities of the women. The feature disappears with or without treatment by 12–14th week of pregnancy. High level of serum human chorionic gonadotropin, estrogen and altered immunological states are considered responsible for initiation of the manifestation, which is probably aggravated by the neurogenic factor.

Management: Assurance is important. Taking of dry toast or biscuit and avoidance of fatty and spicy foods are enough to relieve the symptoms in majority. Supplementation with vitamin B1 100 mg daily is helpful. If the simple measures fail, antiemetic drugs — trifluoperazine (Espazine) 1 mg twice daily is quite effective. Promethazine and ondansetron can be used. Patient is advised to take plenty of fluids (2.5 L in 24 hours) and fruit juice.

HYPEREMESIS GRAVIDARUM

DEFINITION: It is a severe type of vomiting of pregnancy which has got deleterious effect on the health of mother and/or incapacitates her in day-to-day activities. The adverse effects of severe vomiting are—dehydration, metabolic acidosis (from starvation) or alkalosis (from loss of hydrochloric acid), electrolyte imbalance (hypokalemia) and weight loss.

INCIDENCE: There has been marked fall in the incidence during the last 30 years. It is now a rarity in hospital practice (less than 1 in 1,000 pregnancies). **The reasons are** — (a) better application of family planning knowledge which reduces the number of unplanned pregnancies, (b) early visit to the antenatal clinic and (c) potent antihistaminic and antiemetic drugs.

ETIOLOGY: The etiology is obscure but the following are the known facts: (1) It is mostly limited to the first trimester; (2) It is more common in first pregnancy, with a tendency to recur again in subsequent pregnancies (15%); (3) Younger age; (4) Low body mass (5) History of motion sickness or migraine; (6) It has got a familial history — mother and sisters also suffer from the same manifestation; (7) It is more prevalent in hydatidiform mole and multiple pregnancy and (8) It is more common in unplanned pregnancies but much less amongst illegitimate ones.

Women with hyperemesis gravidarum, often suffer from transient form of hyperthyroidism (clinical or subclinical).

THEORIES: (1) *Hormonal*: (a) Excess of chorionic gonadotropin or higher biological activity of hCG is associated. This is proved by the frequency of vomiting at the peak level of hCG and also the increased association with hydatidiform mole or multiple pregnancy when the hCG titer is very much raised; (b) High serum level of estrogen and (c) Progesterone excess leading to relaxation of the cardiac sphincter and simultaneous retention of gastric fluids due to impaired gastric motility. Other hormones involved are: thyroxine, prolactin, leptin and adrenocortical hormones.

(2) *Psychogenic*: It probably aggravates the nausea once it begins. But neurogenic element sometimes plays a role, as evidenced by its subsidence after shifting the patient from the home surroundings. Conversion disorder, somatization, excess perception of sensations by the mother are the other theories.

(3) *Dietetic deficiency*: Probably due to low carbohydrate reserve, as it happens after a night without food. Deficiency of vitamin B_6, vitamin B_1 and proteins may be the effects rather than the cause.

(4) *Allergic* or *immunological basis.* (5) *Decreased gastric motility* is found to cause nausea.

Whatever may be the cause of initiation of vomiting, it is probably aggravated by the neurogenic element. Unless it is not quickly rectified, features of dehydration and carbohydrate starvation supervene and a vicious cycle of vomiting appears — vomiting → carbohydrate starvation → ketoacidosis → vomiting.

PATHOLOGY: There are no specific morbid anatomical findings. The changes in the various organs as described by Sheehan are the generalized manifestations of starvation and severe malnutrition.

Liver: Liver enzymes are elevated. There is centrilobular fatty infiltration without necrosis.

Kidneys: Usually normal with occasional findings of fatty change in the cells of first convoluted tubule, which may be related to acidosis.

Heart: A small heart is a constant finding. There may be subendocardial hemorrhage.

Brain: Small hemorrhages in the hypothalamic region giving the manifestation of Wernicke's encephalopathy. The lesion may be related to vitamin B_1 deficiency.

METABOLIC, BIOCHEMICAL AND CIRCULATORY CHANGES: The changes are due to the combined effect of dehydration and starvation consequent upon vomiting.

Metabolic: Inadequate intake of food results in glycogen depletion. For the energy supply, the fat reserve is broken down. Due to low carbohydrate, there is incomplete oxidation of fat and accumulation of ketone bodies in the blood. The acetone is ultimately excreted through the kidneys and in the breath. There is also increase in endogenous tissue protein metabolism resulting in excessive excretion of nonprotein nitrogen in the urine. Water and electrolyte metabolism are seriously affected leading to biochemical and circulatory changes.

Biochemical: Patients develop acidosis (due to starvation) and alkalosis from loss of hydrochloric acid and hyokalemia. Loss of water and salts in the vomitus results in fall in plasma sodium, potassium and chlorides. The urinary chloride may be well below the normal 5 g/L or may even be absent. Hepatic dysfunction results in ketosis with rise in blood urea and uric acid. Patient suffers from hypoglycemia, hypoproteinemia and hypovitaminosis.

Circulatory: There is hemoconcentration leading to rise in hemoglobin percentage, RBC count and hematocrit values. There is slight increase in the white cell count with increase in eosinophils. There is concomitant reduction of extracellular fluid.

CLINICAL COURSE

From the management and prognostic point of view, the cases are grouped into:

- **Early**
- **Late *(moderate to severe)***

The patient is usually a nullipara, in early pregnancy. The onset is insidious.

EARLY: Vomiting occurs throughout the day. Normal day-to-day activities are curtailed. **There is no evidence of dehydration or starvation**.

LATE: (Evidences of dehydration and starvation are present).

Symptoms: Vomiting is increased in frequency with retching. Urine quantity is diminished even to the stage of oliguria. Epigastric pain, constipation may occur. Complications may appear (see below) if not treated.

Signs: **Features of dehydration and ketoacidosis:** Dry coated tongue, sunken eyes, acetone smell in breath, tachycardia, hypotension, rise in temperature may be noted, jaundice is a late feature. Such late cases are rarely seen these days. **Vaginal examination and/or ultrasonography** is done to confirm the diagnosis of pregnancy.

Investigations:

- **Urinalysis:** (1) Quantity—small, (2) Dark color, (3) High specific gravity with acid reaction, (4) Presence of acetone, occasional presence of protein and rarely bile pigments and (5) Diminished or even absence of chloride.
- **Biochemical and circulatory changes:** The changes are mentioned previously. Routine and periodic estimation of the serum electrolytes (sodium, potassium and chloride) is helpful in the management of the case.
- **Serum TSH, T3 and Free T4:** Women may suffer from transient phase of thyroid dysfunction (clinical or subclinical).
- **Ophthalmoscopic examination** is required if the patient is seriously ill. Retinal hemorrhage and detachment of the retina are the most unfavorable signs.
- **ECG** when there is abnormal serum potassium level.

DIAGNOSIS: The pregnancy is to be confirmed first. Thereafter, all the associated causes of vomiting (enumerated before) are to be excluded. ***Ultrasonography*** is useful not only to confirm the pregnancy but also to exclude other, obstetric (hydatidiform mole, multiple pregnancy), gynecological, surgical or medical causes of vomiting (see p. 180).

Differential diagnosis: When vomiting is persistent in spite of usual treatment other causes of severe vomiting (medical or surgical) should be considered (p. 181) and investigated.

COMPLICATIONS: Maternal: The majority of the clinical manifestations are due to the effects of dehydration and starvation with resultant ketoacidosis. Leaving aside those symptomatology, the following complications may occur which are fortunately rare nowadays. (1) Neurologic complications—(a) Wernicke's encephalopathy, beriberi due to thiamine deficiency; (b) Pontine myelinolysis; (c) Peripheral neuritis; (d) Korsakoff's psychosis. (2) Stress ulcer in stomach; (3) Esophageal tear (Mallory-Weiss syndrome); (4) Jaundice, hepatic failure; (5) Convulsions and coma; (6) Hypoprothrombinemia due to vitamin K deficiency and (7) Renal failure.

Effects on the fetus: Fetus usually remains unaffected once the problem is resolved. Fetal risks may be due to low birth weight.

PREVENTION: The only prevention is to impart effective management to correct simple vomiting of pregnancy.

MANAGEMENT

The principles in the management are:

- Maintenance of hydration
- To control vomiting
- To correct the fluids and electrolytes imbalance
- To correct metabolic disturbances (acidosis or alkalosis)
- To prevent the serious complications of severe vomiting
- Care of pregnancy.

Hospitalization: Whenever a patient is diagnosed as a case of hyperemesis gravidarum, she is admitted. Surprisingly, with the same diet and drugs used at home, the patient improves rapidly. The relatives may be too sympathetic or too indifferent.

Fluids: Oral feeding is withheld for at least 24 hours after the cessation of vomiting. During this period, fluid is given through intravenous drip method. **The amount of fluid to be infused in 24 hours is calculated as follows**: The total amount of fluid approximates 3 liters, of which half is 5% dextrose and half is Ringer's solution. Extra amount of crystalloids equal to the amount of vomitus and urine in 24 hours, is to be added. With this regime — dehydration, ketoacidosis, water and electrolyte imbalance are likely to be rectified. Serum electrolyte should be estimated and corrected if there is any abnormality. Enteral nutrition through nasogastric tube may also be given.

Drugs:

(a) **Antiemetic drugs promethazine** (Phenergan) 25 mg or **prochlorperazine** (Stemetil) 5 mg or **triflupromazine** (Siquil) 10 mg may be administered twice or thrice daily intramuscularly. Trifluoperazine (Espazine) 1 mg twice daily intramuscularly is a potent antiemetic therapy. Vitamin B_6 and doxylamine are also safe and effective. **Metoclopramide** stimulates gastric and intestinal motility without stimulating the secretions. It is found useful.

(b) **Hydrocortisone** 100 mg IV in the drip is given in a case with hypotension or in intractable vomiting. Oral method prednisolone is also used in severe cases.

(c) **Nutritional supplementation**— with vitamin B_1 (100 mg daily), vitamin B_6, vitamin C and vitamin B_{12} are given.

Nursing care: **Sympathetic but firm handling** of the patient is essential. Social and psychological support should be extended.

Hyperemesis progress chart is helpful to assess the progress of patient while in hospital. **Daily record** of pulse, temperature, blood pressure at least twice daily, intake-output, **urine** for acetone, protein, bile, blood biochemistry and ECG (when serum potassium is abnormal) are important.

Clinical features of improvement are evidenced by — (a) subsidence of vomiting (b) feeling of hunger (c) better look (d) normalization of blood biochemistry (electrolytes) (e) disappearance of acetone from the breath and urine (f) normal pulse and blood pressure and (g) normal urine output.

Diet: Before the intravenous fluid is omitted, the foods are given orally. At first, dry carbohydrate foods like biscuits, bread and toast are given. Small but frequent feeds are recommended. Gradually full diet is restored.

Termination of pregnancy is rarely indicated. Intractable hyperemesis gravidarum in spite of therapy is rare these days.

Summary

Excessive vomiting of pregnancy incapacitating day-to-day activities and/or deteriorating the health of the mother is called hyperemesis gravidarum. It is rare nowadays (1 in 1,000). It is common in first birth and limited to early pregnancy. **The exact cause is not known** but once vomiting starts, probably neurogenic elements aggravate the state. The morbid pathological changes are due to starvation. **The clinical manifestations are due to the effect of dehydration, starvation and ketoacidosis.** If not rectified promptly, the condition may turn fatal. **Management** consists of hospitalization, sympathetic but firm handling of the patient, maintenance of hydration by IV infusion, antiemetic drugs, correction of electrolyte imbalance and supply of glucose to protect the liver and vitamin supplement. Intractable hyperemesis gravidarum in spite of therapy is rare these days. Termination of pregnancy is rarely indicated.

QUESTIONS

1. Mention the different causes of vomiting in pregnancy? Write in short the different metabolic changes in a woman due to hyperemesis gravidarum? (p. 181–182)
2. Write short notes on complications of hyperemesis gravidarum (p. 183)

Chapter 16

Hemorrhage in Early Pregnancy

The causes of bleeding in early pregnancy are broadly divided into two groups:

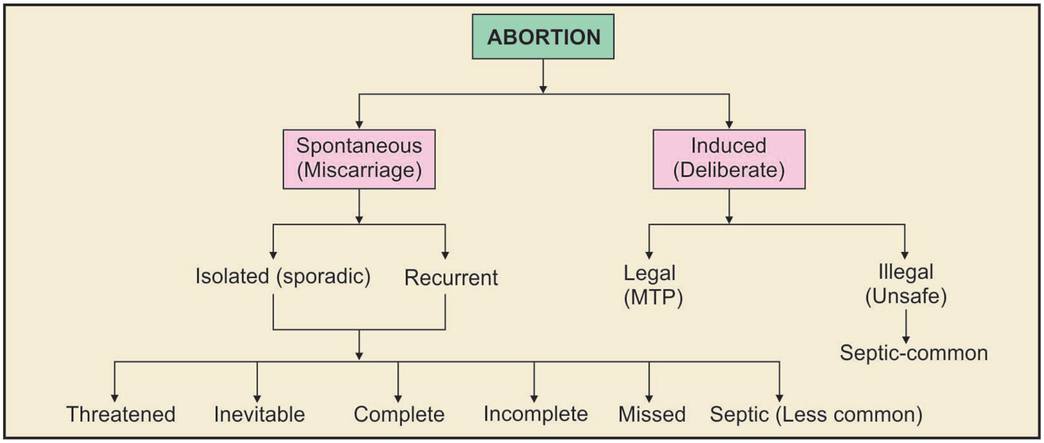
- **Those related to the pregnant state:** This group relates to abortion (95%), ectopic pregnancy, hydatidiform mole and implantation bleeding.
- **Those associated with the pregnant state:** The lesions are unrelated to pregnancy—either pre-existing or aggravated during pregnancy. **Cervical lesions such as** vascular ectopy (erosion), polyp, ruptured varicose veins and malignancy are important causes. These gynecological lesions in relation to pregnancy are discussed in chapter 21.

SPONTANEOUS ABORTION (MISCARRIAGE)

DEFINITION: Abortion is the expulsion or extraction from its mother of an embryo or fetus weighing 500 g or less when it is not capable of independent survival (WHO). This 500 g of fetal development is attained approximately at **22 weeks (154 days) of gestation**. The expelled embryo or fetus is called abortus. The word **miscarriage** is the recommended terminology for spontaneous abortion.

INCIDENCE: The incidence of abortion is difficult to work out but probably 10–20% of all clinical pregnancies end in miscarriage and another optimistic figure of 10% are induced or deliberate. About 75% **miscarriages occur before the 16th week and of these about 80% occur before the 12th week of pregnancy**.

CLASSIFICATION OR VARIETIES:


ABORTION
Spontaneous (Miscarriage)
Induced (Deliberate)
Isolated (sporadic)
Recurrent
Legal (MTP)
Illegal (Unsafe)
Septic-common
Threatened
Inevitable
Complete
Incomplete
Missed
Septic (Less common)

ETIOLOGY

The etiology of miscarriage is often complex and obscure. The following factors (embryonic or parental) are important:

- **Genetic**
- **Endocrine and metabolic**
- **Anatomic**
- **Infection**
- **Immunological**
- **Thrombophilias**
- **Environmental**
- **Others**
- **Unexplained**

GENETIC FACTORS: Majority (50%) of early miscarriages are due to chromosomal abnormality in the conceptus. **Autosomal trisomy** is the commonest (50%) cytogenetic abnormality. Trisomy for every chromosome has been reported. The most common trisomy is trisomy 16 (30%). **Polyploidy** has been observed in about 22% of abortuses. (Polyploidy refers to the presence of three or more multiples of a haploid number of chromosome, e.g. 3n = 69, 4n = 92. Triploidy is more common than tetraploidy). **Monosomy** X (45, X) is the single most common chromosomal abnormality in miscarriages (20%). **Structural chromosomal rearrangements** are observed in 2–4% of abortuses. These include translocation, deletion, inversion and ring formation. **Other chromosomal abnormalities** like mosaic, double trisomy, etc. are found in about 4% of abortuses.

ENDOCRINE AND METABOLIC FACTORS (10–15%): Luteal Phase Defect (LPD) results in early miscarriage as implantation and placentation are not supported adequately. **Deficient progesterone** secretion from corpus luteum or poor endometrial response to progesterone is the cause. **Thyroid abnormalities:** Overt hypothyroidism or hyperthyroidism is associated with increased fetal loss. Thyroid auto-antibodies are often increased. **Diabetes mellitus** when poorly controlled causes increased miscarriage.

ANATOMICAL ABNORMALITIES (3–38%)

- ***Cervico-uterine factors:* These are related mostly to the second trimester abortions.** (1) **Cervical incompetence**, either congenital or acquired is one of the commonest cause of midtrimester and recurrent abortion. (2) **Congenital malformation of the uterus** in the form of bicornuate or septate uterus may be responsible for midtrimester recurrent miscarriages. **Causes of fetal loss are:** (i) reduced intra-uterine volume, (ii) reduced expansile property of the uterus, (iii) reduced placental vascularity when implanted on the septum and (iv) increased uterine irritability and contractility. (3) **Uterine (fibroid)** especially of the submucous variety might be responsible not only for infertility but also for abortion. This is due to distortion or partial obliteration of the uterine cavity. Other causes are: decreased vascularity at the implantation site, red degeneration of fibroid and increased uterine irritability. (4) **Intrauterine adhesions (synechiae)** interfere with implantation, placentation and fetal growth. Depending on the severity of adhesions, e.g. total (Asherman's syndrome), corporal or cervicoisthmic, patient suffers from amenorrhea, hypomenorrhea, infertility or recurrent abortion.

INFECTIONS (5%)—are the accepted causes of late as well as early abortions. Transplacental fetal infections occur with most microorganisms and fetal losses could be caused by any. Infections could be—(i) **Viral:** Rubella, cytomegalovirus, variola, vaccinia or HIV. (ii) **Parasitic**: Toxoplasma, malaria. (iii) **Bacterial**: Ureaplasma, chlamydia, brucella. Spirochetes hardly cause abortion before 20th week because of effective thickness of placental barrier.

IMMUNOLOGICAL DISORDERS (5–10%) (see p. 719)

- **Antiphospholipid antibody syndrome (APAS)**—is due to the presence of antiphospholipid antibodies. These are: lupus anticoagulant (LAC), anticardiolipin antibodies (ACAs) and β-glycoprotein 1 antibodies (β-GP1). Mechanisms of pregnancy loss in women with APAS are: (a) inhibition of trophoblast function and differentiation, (b) activation of complement pathway, (c) release of local inflammatory mediators (cytokines, interleukins) and (d) **thrombosis** of uteroplacental vascular bed. **Ultimate pathology is fetal hypoxia**.
- **Immune factors:** Cytokines are immune molecules. Cytokine response may be either T-helper 1 (Th1) type or T-helper 2 (Th2) type. Th1 response is the production of proinflammatory cytokines [interleukin-2, interferon and tumor necrosis factor (TNF)]. Th2 response is the production of anti-inflammatory cytokines (interleukins -4, -6 and -10). **Successful pregnancy is the result of predominantly Th2 cytokine response. Women with recurrent miscarriage have more Th 1 response**.
- **Autoimmunity: Natural killer (NK) cells** present in peripheral blood and that in the uterus are different functionally. There is no relationship between uNK cell number and future pregnancy outcome though uNK cells help trophoblast invasion, proliferation and angiogenesis. Human leukocyte antigen (HLA) incompatibility between couples or absence of maternal blocking antibodies is not considered as the cause of recurent miscarriage.

MATERNAL MEDICAL ILLNESS (see p. 303): Cyanotic heart disease, hemoglobinopathies are associated with early miscarriage.

PREMATURE RUPTURE OF THE MEMBRANES inevitably leads to abortion.

Paternal factors: Sperm chromosomal anomaly (translocation) can cause miscarriage. Some women who miscarry recurrently may have normal pregnancies following marriage with a different man.

Thrombophilias: Inherited thrombophilia (see p. 319, 399, 508) causes both early and late miscarriages due to intravascular coagulation and thrombosis. **Protein C resistance (factor V Leiden mutation)** is the most common cause. Other conditions are: Protein C deficiency and hyperhomocysteinemia (see p. 319) antithrombin III or prothrombin gene mutation.

ENVIRONMENTAL FACTORS: Conclusions relating to environmental factors are difficult to establish.

- **Cigarette smoking**—increases the risk due to formation of carboxyhemoglobin and decreased oxygen transfer to the fetus. **Alcohol** consumption should be avoided or minimized during pregnancy. X-irradiation and antineoplastic drugs are known to cause abortion. X-ray exposure up to 10 rad is of little risk.
- **Contraceptive agents**—IUD in situ increases the risk whereas oral pills do not.
- **Drugs, chemicals, noxious agents**—anesthetic gases, arsenic, aniline, lead, formaldehyde increase the risk.
- **Miscellaneous**—Exposure to electromagnetic radiation from video display terminals (VDTs) does not increase the risk. Women can use hair dyes, watch television and fly in airlines during pregnancy (see p. 113).

UNEXPLAINED (40–60%): In spite of the numerous factors mentioned, it is indeed difficult, in the majority, to pinpoint the exact cause of miscarriage. Too often, more than one factor is present. However, risk of abortion increases with increased maternal age. About 22% of all pregnancies detected by urinary hCG (peri-implantation) are lost, before the clinical diagnosis.

COMMON CAUSES OF MISCARRIAGE:

First trimester: (1) Genetic factors (50%). (2) Endocrine disorders (LPD, thyroid abnormalities, diabetes). (3) Immunological disorders (autoimmune and alloimmune). (4) Infection. (5) Unexplained.

Second trimester: (1) Anatomic abnormalities—(a) Cervical incompetence (congenital or acquired). (b) Müllerian fusion defects (bicornuate uterus, septate uterus). (c) Uterine synechiae. (d) Uterine fibroid. (2) Maternal medical illness. (3) Unexplained.

MECHANISM OF MISCARRIAGE: In the early weeks, death of the ovum occurs first, followed by its expulsion. In the later weeks, maternal environmental factors are involved leading to expulsion of the fetus which may have signs of life but is too small to survive.

- ***Before 8 weeks:*** The ovum, surrounded by the villi with the decidual coverings, is expelled out intact. Sometimes, the external os fails to dilate so that the entire mass is accommodated in the dilated cervical canal and is called **cervical miscarriage (Fig. 16.1)**.
- ***Between 8 weeks and 14 weeks:*** Expulsion of the fetus commonly occurs leaving behind the placenta and the membranes. A part of it may be partially separated with brisk hemorrhage or remains totally attached to the uterine wall.
- ***Beyond 14th week:*** The process of expulsion is similar to that of a "mini labor". The fetus is expelled first followed by expulsion of the placenta after a varying interval.

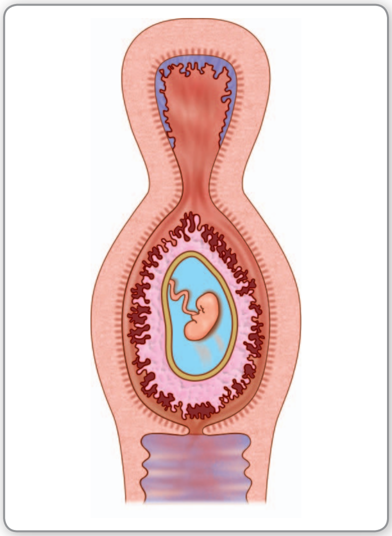


Fig. 16.1: Cervical miscarriage

THREATENED MISCARRIAGE

DEFINITION: It is a clinical entity where the process of miscarriage has started but has not progressed to a state from which recovery is impossible (Fig. 16.2A).

CLINICAL FEATURES:

The patient, having symptoms suggestive of pregnancy, **complains of:**

(1) ***Bleeding per vaginam*** is usually slight and may be brownish or bright red in color. On rare occasion, the bleeding may be brisk, especially in the late second trimester. The bleeding usually stops spontaneously.

(2) ***Pain***: Bleeding is usually painless but there may be mild backache or dull pain in lower abdomen. **Pain appears usually following hemorrhage**.

Pelvic examination should be done as gently as possible. (a) Speculum examination reveals—bleeding if any, escapes through the external os. **Differential diagnosis** includes cervical ectopy, polyps or carcinoma, ectopic pregnancy and molar pregnancy. (b) Digital examination reveals the closed external os (Fig. 16.2A). The uterine size corresponds to the period of amenorrhea. The uterus and cervix feel soft. Pelvic examination is avoided when ultrasonography is available (see below).

INVESTIGATIONS:

Routine investigations include: (1) **Blood**—for hemoglobin, hematocrit, ABO and Rh grouping. Blood transfusion may be required if abortion becomes inevitable and **anti-D gamma globulin** has to be given in Rh-negative nonimmunized women. (2) **Urine** for immunological test of pregnancy is not helpful **as the test remains positive for a variable period even after the fetal death**.

Ultrasonography (TVS) findings may be: (1) A well-formed gestation ring with central echoes from the embryo indicating healthy fetus (see p. 732). (2) Observation of fetal cardiac motion. With this there is 98% chance of continuation of pregnancy. (3) A blighted ovum is evidenced by loss of definition of the gestation sac, smaller mean gestational sac diameter, absent fetal echoes and absent fetal cardiac movements (Fig. 16.1A).

Serum progesterone value of 25 ng/mL or more generally indicates a viable pregnancy in about 95% of cases. Serial serum hCG level is helpful to assess the fetal well-being. Ectopic pregnancy must be ruled out during investigations (see p. 213).

TREATMENT:

Rest: The patient should be in bed for few days until bleeding stops. Prolonged restriction of activity has got no therapeutic value. ***Drugs***: **Relief of pain** may be ensured by diazepam 5 mg tablet twice daily.

There is some evidence that treatment with progesterone improves the outcome. Progesterone induces immunomodulation to shift the Th-1 (proinflammatory response) to Th-2 (antiinflammatory response). Use of hCG is not prefered.

ADVICE ON DISCHARGE: The patient should limit her activities for at least 2 weeks and avoid heavy work. Coitus is avoided during this period. She should be followed up with repeat sonography at 3–4 weeks' time. **The following indicates unfavorable outcome**: falling serum β-hCG, decreasing size of the fetus, irregular shape of the gestational sac or decreasing fetal heart rate (Table 16.1).

PROGNOSIS: The prognosis is very unpredictable. **In isolated spontaneous threatened miscarriage, the following events may occur**: (1) In about two-thirds, the pregnancy continues beyond 28 weeks. (2) In the rest, it terminates either as inevitable or missed miscarriage. **If the pregnancy continues, there is increased frequency of preterm labor, placenta previa, intrauterine growth restriction of the fetus and fetal anomalies**.

Blighted ovum (silent miscarriage Fig. 16.1A): It is a sonographic diagnosis. There is absence of fetal pole in a gestational sac with diameter of 3 cm or more. Uterus is to be evacuated once the diagnosis made.

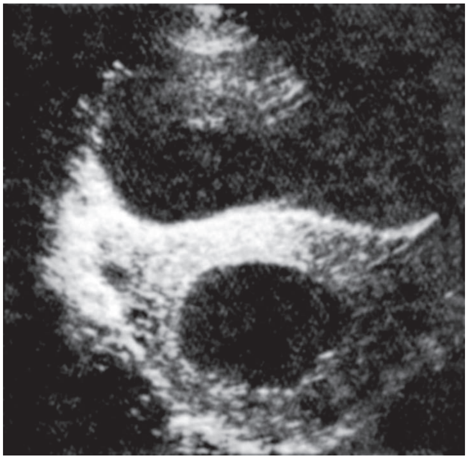


Fig. 16.1A: Ultrasonographic picture of blighted ovum

INEVITABLE MISCARRIAGE

DEFINITION: It is the clinical type of abortion where the changes have progressed to a state from where continuation of pregnancy is impossible.

CLINICAL FEATURES: The patient, having the features of threatened miscarriage, develops the following manifestations: (1) Increased vaginal bleeding. (2) Aggravation of pain in the lower abdomen which may be colicky in nature. (3) **Internal examination reveals dilated internal os of the cervix through which the products of conception are felt (Fig. 16.2B)**. On occasion, the features may develop quickly without prior clinical evidence of threatened miscarriage. In the second trimester, however, it may start with rupture of the membranes or intermittent lower abdominal pain (mini labor).

MANAGEMENT is aimed: (a) to accelerate the process of expulsion. (b) to maintain strict asepsis (p. 642).

General measures: Excessive bleeding should be promptly controlled by administering Methergine 0.2 mg if the cervix is dilated and the size of the uterus is less than 12 weeks. **The blood loss is corrected by intravenous (IV) fluid therapy and blood transfusion**.

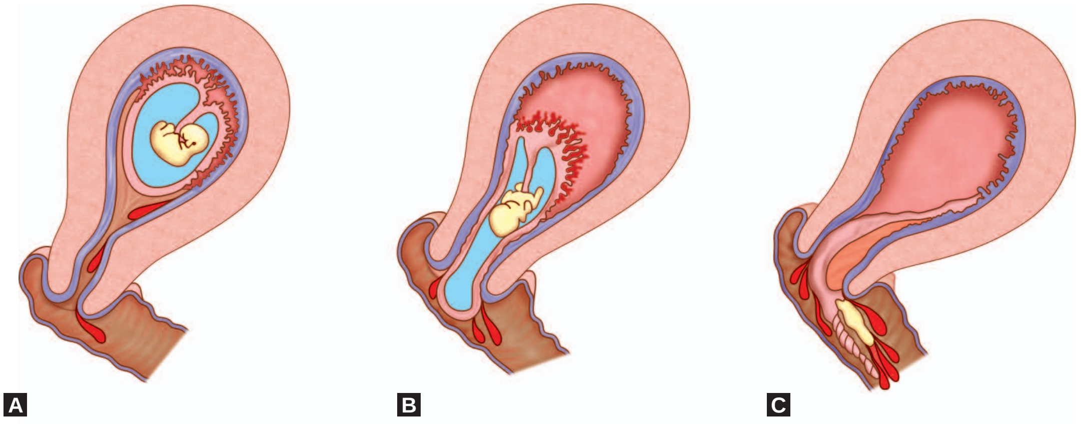
Active Treatment:

- *Before 12 weeks:* (1) Dilatation and evacuation followed by curettage of the uterine cavity by blunt curette using analgesia or under general anesthesia. (2) Alternatively, suction evacuation followed by curettage is done.
- *After 12 weeks:* (1) The uterine contraction is accelerated by oxytocin drip (10 units in 500 mL of normal saline) 40–60 drops per minute. If the fetus is expelled and the placenta is retained, it is removed by ovum forceps, if lying separated. If the placenta is not separated, digital separation followed by its evacuation is to be done under general anesthesia.

COMPLETE MISCARRIAGE

DEFINITION: When the products of conception are expelled *en masse,* **it is called complete miscarriage**.

CLINICAL FEATURES: There is history of expulsion of a fleshy mass per vaginam followed by: (1) Subsidence of abdominal pain. (2) Vaginal bleeding becomes trace or absent. (3) **Internal examination reveals**: (a) Uterus is smaller than the period of amenorrhea and a little firmer. (b) Cervical os is closed (c) Bleeding is trace. (4) Examination of the expelled fleshy mass is found complete. (5) **Ultrasonography (TVS): reveals empty uterine cavity**.



Figs 16.2A to C: (A) Threatened miscarriage; (B) Inevitable miscarriage; (C) Incomplete miscarriage

MANAGEMENT: **Transvaginal sonography** is useful to see that uterine cavity is empty, otherwise evacuation of uterine curettage should be done.

Rh-NEGATIVE WOMEN: A Rh-negative patient without antibody in her system should be protected by anti-D gamma globulin 50 µg or 100 µg intramuscularly in cases of early miscarriage or late miscarriage respectively within 72 hours. However, anti-D may not be required in a case with complete miscarriage before 12 weeks of gestation where no instrumentation has been done.

INCOMPLETE MISCARRIAGE

DEFINITION: **When the entire products of conception are not expelled, instead a part of it is left inside the uterine cavity, it is called incomplete miscarriage**. This is the commonest type met amongst women, hospitalized for miscarriage complications.

CLINICAL FEATURES: History of expulsion of a fleshy mass per vaginam followed by: (1) Continuation of pain in lower abdomen. (2) Persistence of vaginal bleeding. (3) **Internal examination reveals**—(a) uterus smaller than the period of amenorrhea (b) patulous cervical os often admitting tip of the finger and (c) varying amount of bleeding. (4) on examination, the expelled mass is found incomplete (Fig. 16.2C). (5) **Ultrasonography**—reveals echogenic material (products of conception) within the cavity.

COMPLICATIONS: **The retained products may cause:** (a) profuse bleeding (b) sepsis or (c) placental polyp.

MANAGEMENT: ***In recent cases***—evacuation of the retained products of conception (ERCP) is done. **She should be resuscitated before any active treatment is undertaken**.

- ***Early abortion:*** Dilatation and evacuation under analgesia or general anesthesia is to be done. Evacuation of the uterus may be done using MVA also (see p. 753).
- ***Late abortion:*** The uterus is evacuated under general anesthesia and the products are removed by ovum forceps or by blunt curette. In late cases, dilatation and curettage operation is to be done to remove the bits of tissues left behind. **The removed materials are subjected to a histological examination**.

Medical management of incomplete miscarriage may be done. Tablet misoprostol 200 µg is used vaginally every 4 hours. Compared to surgical method, complications (see p. 203) are less with medical method.

MISSED MISCARRIAGE

DEFINITION: **When the fetus is dead and retained inside the uterus for a variable period, it is called missed miscarriage or early fetal demise**.

PATHOLOGY: The causes of prolonged retention of the dead fetus in the uterus are not clear. Beyond 12 weeks, the retained fetus becomes macerated or mummified. The liquor amnii gets absorbed and the placenta becomes pale, thin and may be adherent. Before 12 weeks, the pathological process differs when the ovum is more or less completely surrounded by the chorionic villi.

CARNEOUS MOLE *(Syn: blood mole, fleshy mole)*: It is the pathological variant of missed miscarriage affecting the fetus before 12 weeks. Small repeated hemorrhages in the choriodecidual space disrupt the villi from its attachments. The bleeding is slight, so it does not cause rupture of the decidua capsularis. The clotted blood with the contained ovum is known as a blood mole. By this time, the ovum becomes dead and is either completely absorbed or remains as a rudimentary structure. Gradually, the fluid portion of the blood surrounding the ovum gets absorbed and the wall becomes fleshy, hence the term **fleshy or carneous mole** (Fig. 16.3).

CLINICAL FEATURES: The patient usually presents with features of threatened miscarriage followed by: (1) Persistence of brownish vaginal discharge. (2) Subsidence of pregnancy symptoms.

(3) Retrogression of breast changes. (4) Cessation of uterine growth which in fact becomes smaller in size. (5) Nonaudibility of the fetal heart sound even with Doppler ultrasound if it had been audible before. (6) Cervix feels firm. (7) Immunological test for pregnancy becomes negative. (8) **Realtime ultrasonography** reveals an empty sac early in the pregnancy or the absence of fetal cardiac motion and fetal movements.

COMPLICATIONS: The complications of the missed miscarriage are those mentioned in intrauterine fetal death (see Ch. 21). **Blood coagulation disorders** are less likely to occur in missed miscarriage.

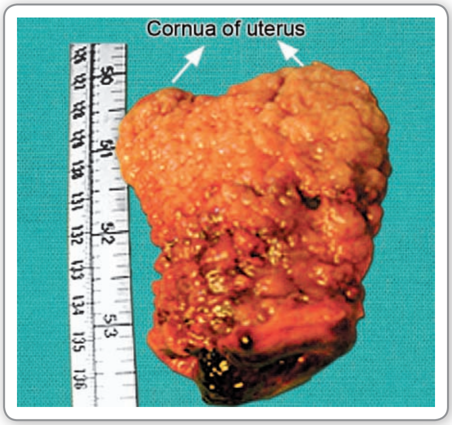


Fig. 16.3: Decidual cast expelled following missed miscarriage (Ref. p. 190)

MANAGEMENT: ♦ **Expectant** ♦ **Medical** ♦ **Surgical**

- ***Uterus is less than 12 weeks:*** (i) **Expectant management**—Many women expel the conceptus spontaneously (Fig. 16.3). (ii) **Medical management**: Prostaglandin E_1 (misoprostol) 800 mg vaginally in the posterior fornix is given and repeated after 24 hours if needed. Expulsion usually occurs within 48 hours. (iii) Suction evacuation or dilatation and evacuation is done either as a definitive treatment or it can be done when the medical method fails. The risk of damage to the uterine walls and brisk hemorrhage during the operation should be kept in mind.
- ***Uterus more than 12 weeks:*** The same principles of the management as advocated in the intrauterine fetal death are to be followed (see Chapter 22). **Induction is done by the following methods:**

  Prostaglandins are more effective than oxytocin in such cases. The methods used are:

  (a) **Prostaglandin E_1 analog (misoprostol)** 200 µg tablet is inserted into the posterior vaginal fornix every 4 hours for a maximum of 5 such.

  (b) **Oxytocin**—10–20 units of oxytocin in 500 mL of normal saline at 30 drops/min is started. If fails, escalating dose of oxytocin to the maximum of 200 mlU/min may be used with monitoring.

  (c) Many patients need surgical evacuation following medical treatment. Following medical treatment, ultrasonography should be done to document empty uterine cavity. Otherwise **evacuation of the retained products of conception (ERPC)** should be done.

  (d) **Dilatation and evacuation** is done once the cervix becomes soft with use of PGE_1. Otherwise cervical canal is dilated using the mechanical dilators or by laminaria tent (see p. 643). Evacuation of the uterine cavity is done thereafter slowly.

SEPTIC ABORTION

DEFINITION: Any abortion associated with clinical evidences of infection of the uterus and its contents is called septic abortion. Although clinical criteria vary, **abortion is usually considered septic when there are**: (1) rise of temperature of at least 100.4°F (38°C) for 24 hours or more, (2) offensive or purulent vaginal discharge and (3) other evidences of pelvic infection such as lower abdominal pain and tenderness.

INCIDENCE: It is difficult to work out the overall incidence of septic abortion. About 10% of abortions requiring admission to hospital are septic. The majority of septic abortions are associated with incomplete abortion. While in the **majority of cases, the infection occurs following illegal induced abortion** but infection can occur even after spontaneous abortion.

MODE OF INFECTION: The microorganisms involved in the sepsis are usually those normally present in the vagina (endogenous). **The microorganisms are**: (a) *Anaerobic*—Bacteroides group *(fragilis)*, anaerobic *Streptococci, Clostridium welchii* and tetanus bacillus. (b) *Aerobic—Escherichia coli* (*E. coli*), *Klebsiella, Staphylococcus, Pseudomonas* and group A beta-hemolytic *Streptococcus* (usually exogenous), methicillin-resistant Staphylococcus aureus (MRSA). **Mixed infection is more common. The increased association of sepsis in unsafe induced abortion is due to the fact that:** (1) proper antiseptic and asepsis are not taken, (2) incomplete evacuation and (3) inadvertent injury to the genital organs and adjacent structures, particularly the bowels.

PATHOLOGY: In the majority (80%), the organisms are of endogenous origin and the infection is localized to the conceptus without any myometrial involvement. In about 15%, the infection either produces localized endomyometritis surrounded by a protective leukocytic barrier, or spreads to the parametrium, tubes, ovaries or pelvic peritoneum. In about 5%, there is generalized peritonitis and/or endotoxic shock.

CLINICAL FEATURES: Depending upon the severity and the extent of infection, the clinical picture varies widely. **History of unsafe termination by an unauthorized person is mostly concealed**.

Clinical Features of Septic Abortion

- The woman looks sick and anxious
- Temperature: >38°C
- Chills and rigors (suggest-bacteremia)
- Persistent tachycardia ≥ 90 bpm (spreading infection)
- Hypothermia (**endotoxic shock**) < 36°C
- Abdominal or chest pain
- Tachypnea (RR) > 20/min
- Impaired mental state
- Diarrhea and/or vomiting
- Renal angle tenderness
- **Pelvic examination**: Offensive, purulent vaginal discharge, uterine tenderness, boggy feel in the POD (pelvic abscess)

CLINICAL GRADING: Grade I: The infection is localized in the uterus. **Grade II:** The infection spreads beyond the uterus to the parametrium, tubes and ovaries or pelvic peritoneum. **Grade III:** Generalized peritonitis and/or endotoxic shock or jaundice or acute renal failure.

Grade I is the commonest and is usually associated with spontaneous abortion. **Grade III is almost always associated with illegal induced abortion**.

INVESTIGATIONS: ***Routine investigations include*:** (1) **Cervical or high vaginal swab is taken prior to internal examination for**—(a) culture in aerobic and anaerobic media to find out the dominant microorganisms, (b) sensitivity of the microorganisms to antibiotics and (c) smear for Gram stain. **Gram-negative organisms are—***E. coli, Pseudomonas, Bacteroides,* etc. **Gram-positive organisms are—***Staphylococci,* anaerobic *Streptococci,* group A beta-hemolytic, *Streptococci, MRSA, Cl. welchii, Cl. tetani,* etc. (2) **Blood for hemoglobin** estimation, total and differential count of white cells, ABO and Rh grouping. (3) **Urine analysis** including culture.

Special investigations—(1) **Ultrasonography** of pelvis and abdomen to detect intrauterine retained products of conception, physometra, foreign body—intrauterine or intra-abdominal, free fluid in the peritoneal cavity or in the pouch of Douglas (pelvic abscess). (2) **Blood—**(a) Culture—if associated with spell of chills and rigors (b) Serum electrolytes, C-reactive protein (CRP), serum lactate—as an adjunct to the management protocol of endotoxic shock. Serum lactate greater than or equal to 4 mmol/L indicates tissue hypoperfusion (c) Coagulation profile. (3) **Plain X-ray—**(a) Abdomen—in suspected cases of bowel injury (b) Chest—for cases with pulmonary complications (atelectasis).

COMPLICATIONS:

Immediate: Most of the fatal complications are associated with illegally induced abortions of **grade III type**.

- **Hemorrhage** related due to abortion process and also due to the injury inflicted during the interference.
- **Injury** may occur to the uterus and also to the adjacent structures particularly the bowels.
- **Spread of infection leads to**: (a) **Generalized peritonitis**—the infection reaches through: (i) the uterine tubes (ii) perforation of the uterus (iii) bursting of the microabscess in the uterine wall and (iv) injury to the gut. (b) **Endotoxic shock**—mostly due to *E. coli* or *Cl. welchii* infection. (c) **Acute renal failure**—multiple factors are involved producing patchy cortical necrosis or acute tubular necrosis. It is common in infection with *Cl. welchii*. **Lungs**: atelectasis, ARDS (see p. 724). (d) **Thrombophlebitis**.

All these lead to increased maternal deaths, the magnitude of which is to the extent of about 20–25% as per hospital statistics.

Remote: The remote complications include: (a) chronic debility, (b) chronic pelvic pain and backache, (c) dyspareunia, (d) ectopic pregnancy, (e) secondary infertility due to tubal blockage and (f) emotional depression.

PREVENTION: (1) To boost up family planning acceptance in order to curb the unwanted pregnancies. (2) Rigid enforcement of legalized abortion in practice and to curb the prevalence of **unsafe abortions**. Education, motivation and extension of the facilities are sine qua non to get the real benefit out of it (see p. 637). (3) To take antiseptic and aseptic precautions either during internal examination or during operation in spontaneous abortion (outlined in p. 155).

MANAGEMENT

GENERAL MANAGEMENT:

- **Hospitalization** is essential for all cases of septic abortion. The patient is kept in isolation.
- **To take high vaginal or cervical swab** for culture, drug sensitivity test and Gram stain.
- **Vaginal examination** is done to note the state of the abortion process and extension of the infection.
- **Overall assessment of the case** and the patient is leveled in accordance with the clinical grading.
- **Investigation protocols** as outlined before are done.
- **Principles of management are:** (a) To control sepsis. (b) To remove the source of infection. (c) To give supportive therapy to bring back the normal homeostatic and cellular metabolism. (d) To assess the response of treatment.

GRADE I

Drugs:

(1) **Antibiotics** (see below).

(2) **Prophylactic antigas gangrene serum** of 8,000 units and 3,000 units of **antitetanus serum** intramuscularly are given if there is a history of interference.

(3) **Analgesics and sedatives**, as required, are to be prescribed.

Blood transfusion is given to improve anemia and body resistance.

Evacuation of the uterus: As abortion is often incomplete, **evacuation should be performed at a convenient time within 24 hours following antibiotic therapy**. Excessive bleeding is, of course, an urgent indication for evacuation. Early emptying not only minimizes the risk of hemorrhage but also removes the nidus of infection. The procedure should be gentle to avoid injury to the uterus.

GRADE II

Drugs: **Antibiotics—**Mixed infections including Gram-positive, Gram-negative and and aerobic and anaerobic organisms are common. Ideal antibiotic regimens should cover all of them.

Antimicrobial therapy: A combination of either piperacillin-tazobactam or carbapenem plus clindamycin (IV) gives broadest range of microbial coverage. Emperical therapy is started first and is changed when culture sensitivity report is available.

(a) **Piperacillin-tazobactam and carbapenems**: Covers most organisms except MRSA, and are not nephrotoxic. Piperacillin-tazobactam does not cover extended spectrum β-lactamase (ESBL) producers.

(b) **Vancomycin** or **teicoplanin** may be added for MRSA resistant to clindamycin.

(c) **Clindamycin**: Covers most streptococci, staphylococci including MRSA and is not nephrotoxic.

(d) **Gentamycin** (3–5 mg/kg—single dose) can be given when renal function is normal.

(e) **Co-amaxiclav**—does not cover MRSA, *Pseudomonas* or ESBL-producing organisms.

(f) **Metronidazole**—covers anaerobes.

Analgesic, **AGS and ATS** are given as in Grade I. **Blood transfusion** is more often needed than in Grade I cases.

Clinical monitoring: To note pulse, respiration, temperature, urinary output and progress of the pain, tenderness and mass in lower abdomen, CVP greater than 8 mm Hg.

Surgery: (1) **Evacuation of the uterus—**Evacuation should be withheld for at least 48 hours when the infection is controlled and is localized, the only exception being excessive bleeding.

(2) **Posterior colpotomy—**When the infection is localized in the pouch of Douglas, pelvic abscess is formed. It is evidenced by spiky rise of temperature, rectal tenesmus (frequent loose stool mixed with mucus) and boggy mass felt through the posterior fornix. Posterior colpotomy and drainage of the pus relieve the symptoms and improve the general outlook of the patient.

Features of Organ Dysfunction

- Peristent hypotension (SBP < 90 mm Hg)
- Oliguria
- Serum creatinine > 44.2 μmol/L
- Coagulation abnormalities (INR > 1.5)
- Thrombocytopenia
- Hyperbilirubinemia
- PaO$_2$: <40 kPa
- Serum lactate : ≥4.0 mmol/L

} Tissue hypoperfusion

Indications for ICU Management (Plaat-2008)

♦ **Cardiovascular**	Persistent hypotension, persistently raised serum lactate (≥4 mmol/L)
♦ **Respiratory**	Pulmonary edema, mechanical ventilation, airway protection
♦ **Renal**	Renal dialysis
♦ **Neurological**	Impaired consciousness
♦ **Miscellaneous**	Multiorgan failure, hypothermia, acidosis

GRADE III

Antibiotics are used as discussed above. **Clinical monitoring** is to be conducted as outlined in Grade II. **Supportive therapy** is directed to treat generalized peritonitis by gastric suction and intravenous crystalloids infusion. Management of endotoxic shock or renal failure, if present, is to be conducted as described in the chapter 38. Features of organ dysfunction should be carefully guarded against. Patient may need intensive care unit management (see above).

Active Surgery: Indications are—(1) Injury to the uterus. (2) Suspected injury to bowel. (3) Presence of foreign body in the abdomen as evidenced by the sonography or X-ray or felt through the fornix on bimanual examination. (4) Unresponsive peritonitis suggestive of collection of pus. (5) Septic shock or oliguria not responding to the conservative treatment. (6) Uterus too big to be safely evacuated per vaginam.

The laparotomy should be done by experienced surgeon with a skilled anesthetist. Removal of the uterus should be done irrespective of parity. **Adnexa** is to be removed or preserved according to the pathology found. Thorough inspection of the gut and omentum for evidence of any injury is mandatory. **Even when nothing is found on laparotomy, simple drainage of the pus is effective**.

UNSAFE ABORTION is defined as the procedure of termination of unwanted pregnancy either by persons lacking the necessary skills or in an environment lacking the minimal standards or both. About 90% of unsafe abortions are in the developing countries comprising 13% of all maternal deaths (WHO 1998). All the complications (see p. 193) are preventable if it is performed in a safe manner with proper **postabortion care services** (see p. 643).

Table 16.1: Types of Miscarriages and the Diagnostic Features

Type	Symptoms	Uterine Size	Cervix (Ext. Os)	Ultrasonography
Threatened	■ Vaginal bleeding present ■ Pelvic pain	■ Corresponds to gestational age	■ Closed	■ Fetus alive ■ Retroplacental hemorrhage +
Inevitable	■ Vaginal bleeding present ■ Pelvic pain	■ Same or smaller	■ Open with palpable conceptus	■ Fetus often dead ■ Retroplacental hemorrhage +
Incomplete	■ Vaginal bleeding (may be heavy)	■ Smaller	■ Open	■ Products of conception partly retained
Complete	■ Vaginal bleeding—trace or absent	■ Smaller	■ Closed	■ Uterine cavity empty
Missed	■ Vaginal bleeding—trace, brownish in color	■ Smaller	■ Closed	■ Blighted ovum or fetus without cardiac activity
Septic	■ Vaginal discharge: purulent, foul smelling with features of sepsis (see p. 192)	■ Variable, may be larger	■ Open	■ Products of conception retained, presence of foreign body (±), free fluid in the peritoneal cavity/ POD

RECURRENT MISCARRIAGE

DEFINITION: Recurrent miscarriage is defined as a sequence of three or more consecutive spontaneous abortion before 20 weeks. Some, however, consider two or more as a standard. It may be primary or secondary (having previous viable birth). A woman procuring three consecutive induced abortions is not a habitual aborter.

INCIDENCE: This distressing problem is affecting approximately 1% of all women of reproductive age. The risk increases with each successive abortion reaching over 30% after three consecutive losses.

ETIOLOGY

The causes of recurrent abortion are complex and most often obscure. More than one factor may operate in a case. Factors may be recurrent or nonrecurrent. There are known specific factors which are responsible for early or late abortion and they are grouped accordingly.

FIRST TRIMESTER ABORTION:

- **Genetic factors (3–5%):** Parental chromosomal abnormalities is a proven cause of recurrent abortion. The **most common abnormality is a balanced translocation**. Risk of miscarriage in couples with a balanced translocation is greater than 25%. However, the chance of a successful pregnancy even without treatment is 40–50%.
- **Endocrine and metabolic:** (1) **Poorly controlled diabetic** patients do have an increased incidence of early pregnancy failure. (2) **Presence of thyroid autoantibodies** is often associated with an increased risk but it is likely that this finding is secondary to a generalized autoimmune abnormality rather than a specific endocrine dysfunction. Thyroid function is usually normal. (3) **Luteal phase defect (LPD)** with less production of progesterone is too often related but whether the diminished progesterone level is the cause or effect is not clear (see p. 186). (4) **Polycystic ovary syndrome (PCOS)—**exact mechanism of increased miscarriage is not known. Besides elevated serum LH levels, the other factors responsible are: insulin resistance, hyperinsulinemia and hyperandrogenemia.
- **Infection:** Infection in the genital tract may be responsible for sporadic spontaneous abortion but its relation to recurrent fetal wastage is inconclusive (see p. 202). Transplacental fetal infection can occur with most microorganisms (see p. 186). Infection with bacterial vaginosis is risk factor.
- **Inherited thrombophilia (see p. 187, 399, 508)** causes both early and late miscarriages due to intravascular (spiral artery), and placental intervillous thrombosis. Protein C resistance (factor V Leiden mutation) is the most common cause. Protein C is the natural inhibitor of coagulation. Other factors are—deficiencies of protein C, S and antithrombin III. Hyperhomocystinemia and prothrombin gene mutation are also the known causes of recurrent miscarriage (see p. 399).
- **Immune factors (10–15%)**

  Autoimmunity—Presence of **autoantibodies** causes rejection of early pregnancy (15%) in the second trimesters mainly. Antibodies responsible are: antinuclear antibodies, anti-DNA antibodies (double or single stranded) and antiphospholipid antibodies (see p. 186, 199). Antiphospholipid antibody-positive women demonstrate a tendency to miscarry at progressively lower gestational ages. **Antiphospholipid antibodies** are: lupus anticoagulant, anticardiolipin antibodies and anti β glycoprotein-I. **Causes of miscarriage** are: (a) inhibition of trophoblast proliteration and function, (b) release of local inflammatory mediators (cytokines) through complement pathway, (c) spiral artery and placental intervillous thrombosis and (d) decidual vasculopathy with fibrinoid necrosis (details see p. 399).

  Alloimmunity—Natural killer (NK) cells are found in peripheral blood (NK) and the uterine mucosa (uNK). These two groups of NK cells are different functionally. There is no relation between peripheral blood NK cells and recurrent miscarriage. uNK cells help trophoblast proliferation, invasion and angiogenesis (see p. 36). However, routine investigation for uNK cells is not recommended.

  Parental human leukocyte antigen (HLA) sharing and absence of maternal blocking and leucocytotoxic antibodies leading to miscarriage is not based on evidence (see p. 635). Parenteral HLA testing is not recommended.
- **Unexplained:** In the majority, the cause remains unknown (see p. 188).

SECOND TRIMESTER MISCARRIAGE:

Anatomic abnormalities are responsible for 10–15% of recurrent abortion. The causes may be **congenital** or **acquired**. Congenital anomalies may be due to defects in the *Müllerian duct* fusion or resorption (e.g. unicornuate, bicornuate, septate or double uterus). Congenital cervical incompetence is rare. **Acquired anomalies** are: intrauterine adhesions, uterine fibroids and endometriosis and **cervical incompetence**. The pathology of abortion has been discussed in p. 186.

Defective Müllerian fusion—such as double uterus, septate or bicornuate uterus (Figs 16.4A and B). The association is about 12% cases of recurrent abortion. Abortions tend to recur beyond 12 weeks and the successive pregnancies are carried longer. Implantation on the septum leads to defective placentation. The diagnosis is confirmed either by hysterography or hysteroscopy combined with laparoscopy in nonpregnant state or during digital exploration following abortion.

Case history (Fig. 16.4B): The patient had three consecutive midtrimester abortions. Hysterography revealed bicornuate uterus. Metroplasty was done. Pregnancy occurred 1 year later, which was delivered by cesarean section at 39 weeks.

OTHER CAUSES OF SECOND TRIMESTER MISCARRIAGE:

- ***Chronic maternal illness***—such as uncontrolled diabetes with arteriosclerotic changes, hemoglobinopathies, chronic renal disease. Inflammatory bowel disease, systemic lupus erythematosus.
- ***Infection***—Syphilis, toxoplasmosis and listeriosis may be responsible in some cases.
- ***Unexplained***.

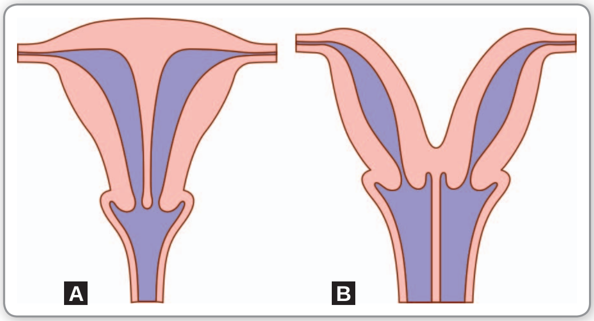


Fig. 16.4A: Diagrammatic representation of—(A) septate and (B) double uterus with double cervix and septate vagina

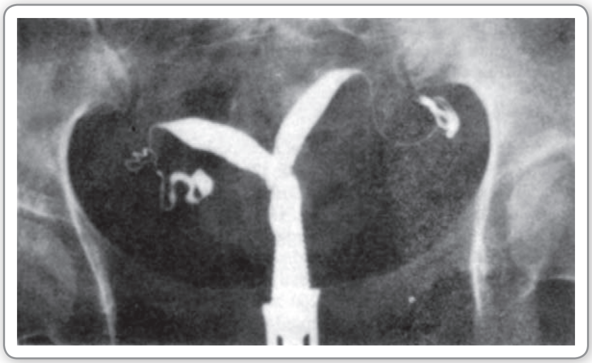


Fig. 16.4B: Hysterosalpingography showing bicornuate uterus

CERVICAL INCOMPETENCE (CERVICAL INSUFFICIENCY)—20%

Causes: The retentive power of the cervix (internal os) may be impaired functionally and/or anatomically due to the following conditions: (a) **Congenital** Uterine anomalies. (b) **Acquired (iatrogenic)**—common, following: (i) D and C operation, (ii) induced abortion by D and E (10%), (iii) vaginal operative delivery through an undilated cervix and (iv) amputation of the cervix or cone biopsy of trachelectomy. (c) **Others**—multiple gestations, prior preterm birth. Cervical incompetence is considered as a biological continuum of spontaneous preterm birth syndrome (see p. 201).

DIAGNOSIS OF CERVICAL INCOMPETENCE:

- **History**—Repeated midtrimester painless cervical dilatation (without apparent cause) and escape of liquor amnii followed by painless expulsion of the products of conception are very much suggestive.
- **Internal examination:** (i) **Interconceptional period—**Bimanual examination reveals presence of unilateral or bilateral tear and/or gaping of the cervix up to the internal os.

INVESTIGATIONS:

Interconceptional period: (i) **Passage number 6–8 Hegar dilator** beyond the internal os without any resistance and pain and absence of internal os snap on its withdrawal specially in premenstrual period indicate incompetence.

(ii) **Premenstrual hysterocervicography** shows funnel-shaped shadow (Fig. 16.5). The internal os is supposed to be tight due to action of progesterone during this phase of cycle. Similar funnel-shaped shadow may be found if hysterography is done in the proliferative phase even with a competent cervix.

(iii) **During pregnancy**—(a) **Clinical (speculum)**: Painless cervical shortening and dilatation. (b) **Sonography**: Short cervix < 25 mm; Funneling of the internal os > 1 cm. (c) **Speculum examination**: Detection of dilatation of internal os with herniation of the membranes (Fig. 16.6).

INVESTIGATIONS FOR RECURRENT MISCARRIAGE

A thorough medical, surgical and obstetric history with meticulous clinical examination should be carried out to find out the possible cause or causes as mentioned previously. Careful history taking should include—(i) The nature of previous abortion process. (ii) Histology of the placenta or karyotyping of the conceptus, if available. (iii) Any chronic illness.

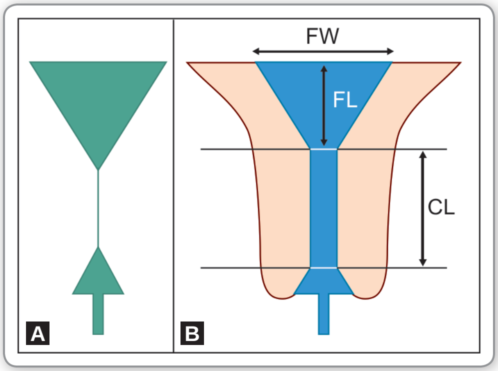


Fig. 16.5A and B: Diagrammatic representation of the hysterographic shadow obtained in the premenstrual phase. (A) A normally competent cervix (B) Incompetent cervix showing funneling. (FW = Funnel width, FL = Funnel length, CL = Cervical length).

Diagnostic tests: (1) Blood-glucose (fasting and postprandial), VDRL, thyroid function test, ABO and Rh grouping (husband and wife), toxoplasma antibodies IgG and IgM. (2) Autoimmune screening—lupus anticoagulant and anticardiolipin antibodies (3) Serum LH on D_2/D_3 of the cycle. (4) Ultrasonography—to detect congenital malformation of uterus, polycystic ovaries and uterine fibroid. (5) Hysterosalpingography in the secretory phase to detect—cervical incompetence, uterine synechiae and uterine malformation. (6) This is supported by hysteroscopy and/or laparoscopy. (7) Karyotyping (husband and wife). (8) Endocervical swab to detect chlamydia, mycoplasma and bacterial vaginosis.

TREATMENT

INTERCONCEPTIONAL PERIOD:

- **To alleviate anxiety and to improve the psychology**—While **counseling the couple**, they should be assured that even after three consecutive miscarriages, the chance of a successful pregnancy is high (70%). However, the success rate depends on the underlying etiology as well as the age of the woman.
- **Hysteroscopic resection** of uterine septa, synechiae and submucous myomas improves the pregnancy outcome. **Uterine unification operation (metroplasty)** is done for cases with bicornuate uterus.
- **Chromosomal anomalies**—If chromosomal abnormality is detected in the couples or in the abortus, genetic counseling is undertaken. Karyotyping of the products of conception from future miscarriage is mandatory. Couples with chromosomal translocations or inversion are counseled for **preimplantation genetic diagnosis (PGD)** (see p. 567) or prenatal diagnosis (cff DNA, amniocentesis or CVS) in subsequent pregnancy or pregnancy with donor gametes (sperm or oocyte).
- **Women with PCOS** are best treated for their insulin resistance, hyperinsulinemia and hyperandrogenemia. Metformin therapy is helpful (see author's Textbook of Gynecology p. 470).
- **Endocrine dysfunction:** Control of diabetes and thyroid disorders is done (see p. 325, 331). Subclinical diabetes and/or thyroid disease may be treated when no other factor is present.
- **Genital tract infections** are treated appropriately following culture of cervical and vaginal discharge. Empirical treatment with doxycycline or erythromycin is cost-effective.

DURING PREGNANCY:

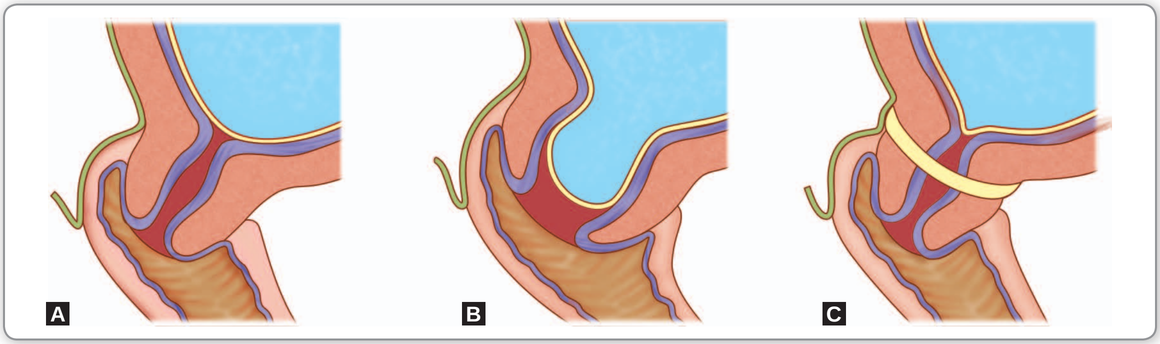
- **Reassurance and tender loving care (TLC)** are very much helpful. Probably this removes the stress and improves uterine blood flow.
- **Ultrasound** should be used at the earliest to detect a viable pregnancy. This will influence further management. If the fetus is viable ultrasonographically at 8–9 weeks, only 2–3% are lost thereafter and similarly fetal loss is only 1% after 16 weeks of viable fetus.
- **Rest—**Patient should take adequate rest and to avoid strenous activities, intercourse and traveling.
- **Progesterone therapy** in cases with luteal phase defect and recurrent miscarriage is given with **natural micronized progesterone** 100 mg daily as vaginal suppository. It is started 2 days after ovulation. Once pregnancy is confirmed, progesterone supplementation is continued until 10–12 weeks of gestation. Progesterone is necessary for successful implantation and continuaion of pregnancy. This is due to its **immunomodulatory role**. It induces pregnancy protective shift from proinflammatory Th1 cytokine response to a more favorable anti-inflammatory Th2 cytokine response. Benefits of hCG therapy in recurrent miscarriage are not effective. hCG stimulates corpus luteum to produce progesterone.
- **Antiphospholipid antibody syndrome (APS):** Women are treated with low-dose aspirin (50 mg/day) and heparin (5,000 units SC twice daily) up to 34 weeks. Unfractionated heparin and low molecular weight heparin (LMWH) are equally effective and safe.
- **Cerclage operation** (Fig. 16.6) for cervical incompetence is to be performed (see below).
- **Chromosomal anomaly—Prenatal diagnosis** by CVS or amniocentesis (see p. 130) is done. Preimplantation genetic diagnosis in blastomere stage is another option (see p. 132). Only then the few balanced embryos are transferred and there is successful pregnancy.
- **Immunotherapy**: Use of paternal cell (leukocytes) immunization, third party donor leukocytes, trophoblast membranes, corticosteroids or IV immunoglobulins does not improve live birth rate. Immunotherapy is no longer used in women with unexplained recurrent miscarriage. It may increase maternal morbidity (anaphylactic shock).
- **Inherited thrombophilias (see p. 399): Antithrombotic therapy improves the pregnancy outcome**. Heparin (5,000 IU SC twice daily) or low molecular weight heparin (enoxaprin) SC once daily (preferred) is effective. Heparin is given up to 34 weeks.
- **Medical complications in pregnancy**: Hemoglobinopathies, SLE, cyanotic heart disease are advised to delay pregnancy until the disease is optimally treated. During pregnancy, specific management is continued (see p. 303).
- **Unexplained**: Despite different investigations, about 40–60% of recurrent miscarriages remain unexplained. However, *'tender loving care'* (TLC) and some supportive therapy improves the pregnancy outcome by 70%.

MANAGEMENT OF CERVICAL INCOMPETENCE

***Cerclage operation*:** Two types of operation are in current use during pregnancy each claiming an equal success rate of about 80–90%. The operations are named after **Shirodkar** (1955) and **McDonald** (1963).

Principle: The procedure reinforces the weak cervix by a nonabsorbable tape, placed around the cervix at the level of internal os.

Time of operation: In a proven case, prophylactic cerclage should be done around 14 weeks of pregnancy or at least 2 weeks earlier than the lowest period of previous wastage, as early as the 10th week. **Emergency (rescue)** cerclage can be done when the cervix is dilated and there is bulging of the membranes. **Case selection**: Cerclage operation is done mainly in cases where careful **history** and



Figs 16.6A to C: Principles of encerclage operation—(A) Competent cervix; (B) Incompetent cervix with herniation of the membranes; (C) Competency restored after encerclage operation

physical examination suggest cervical incompetence **(history indicated cerclage)** as **it remains a diagnosis of exclusion**. Clinical observation is supported with sonographically detected short cervical length (<25 mm), with or without funneling of the internal os (**ultrasound indicated cerclage**). Prior to operation, fetal growth and anomaly (aneuploidy) scan should be done by sonography.

STEPS OF SHIRODKAR'S OPERATION (Fig. 16.7)

Step I: The patient is put under light general anesthesia and placed in lithotomy position with good exposure of the cervix by a posterior vaginal speculum. The lips of the cervix are pulled down by sponge holding forceps or Allis tissue forceps.

Step II: A transverse incision is made anteriorly below the base of the bladder on the vaginal wall and the bladder is pushed up to expose the level of the internal os. A vertical incision is made posteriorly on the cervicovaginal junction.

Step III: The nonabsorbable suture material—Mersilene (Dacron) or Ethibond tape is passed submucously with the help of an aneurysm needle or cervical needle so as to bring the suture ends through the posterior incision.

Step IV: The ends of the tapes are tied up posteriorly by a reef knot. **The bulging membranes, if present, must be gently reduced beforehand into the uterine cavity**. The anterior and posterior incisions are repaired by interrupted stitches using chromic catgut.

McDONALD'S OPERATION (see Fig.16.7)

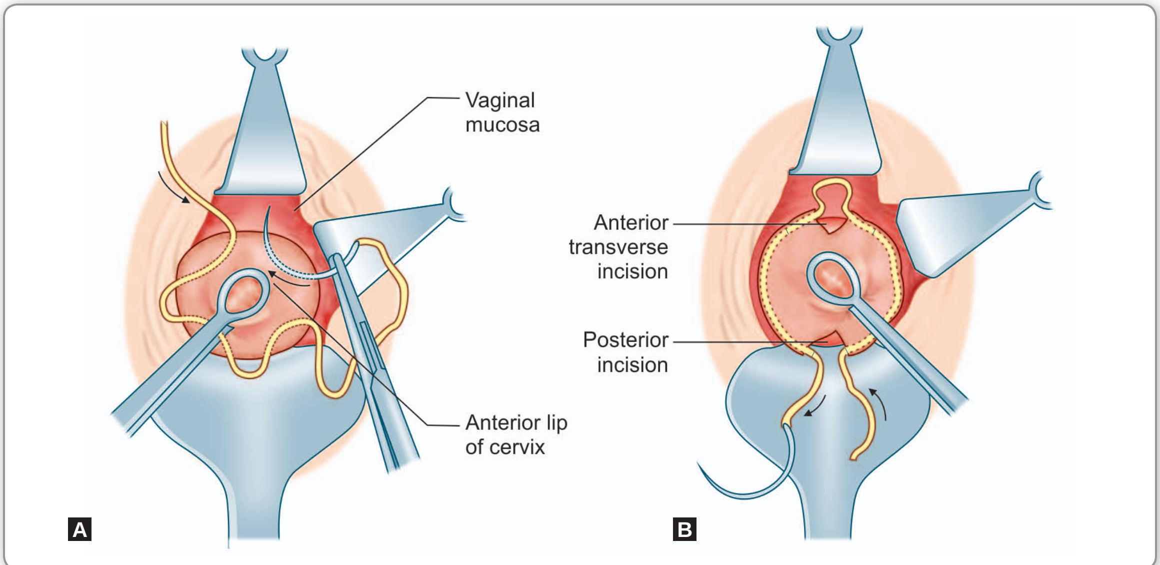
The nonabsorbable suture (Mersilene) material is placed as a purse-string suture as high as possible (level of internal os) at the junction of the rugose vaginal epithelium and the smooth vaginal part of the cervix below the level of the bladder. The suture starts at the anterior wall of the cervix. Taking successive deep bites (4–5 sites), it is carried around the lateral and posterior walls back to the anterior wall again where the two ends of the suture are tied.

The operation is simple having less blood loss, and has got a good success rate. There is less formation of cervical scar and hence less chance of cervical dystocia during labor.

Postoperative care: (1) The patient should be in bed for at least 2–3 days. (2) Weekly injections of 17α-hydroxyprogesterone caproate 500 mg IM are given in women with history of prior preterm delivery. (3) Isoxsuprine (tocolytics) 10 mg tablet may be given thrice daily to avoid uterine irritability.

Advice on discharge: (a) Usual antenatal advice. (b) To avoid intercourse. (c) To avoid rough journey. (d) To report if there is vaginal bleeding or abdominal pain. (e) Periodic ultrasonographic monitoring of the fetus and the cervix.

Removal of stitch: **The stitch should be removed at 37th week** or earlier if labor pain starts or features of abortion appear. If the stitch is not cut in time, uterine rupture or cervical tear may occur. **If the stitch is cut prior to the onset of labor**, it is preferable to cut it in operation theater as there is increased chance of cord prolapse especially in the cases with floating head.



Figs 16.7A and B: Cerclage operation—(A) McDonald's technique; (B) Shirodkar's technique

***Contraindications*:** (i) Intrauterine infection. (ii) Ruptured membranes. (iii) Presence of vaginal bleeding. (iv) Severe uterine irritability. (v) Cervical dilatation greater than 4 cm. (vi) Fetal death or defect.

***Complications*:** (i) Slipping or cutting through the suture. (ii) Chorioamnionitis. (iii) Rupture of the membranes. (iv) Abortion/preterm labor. (v) Cervical lacerations during delivery. (vi) Cervical scarring and dystocia requiring cesarean delivery.

Abdominal cerclage: A Mersilene tape is placed at the level of the isthmus between the uterine wall and the uterine vessels. The tape is tied anteriorly. This is done between 11 weeks and 13 weeks following laparotomy. Disadvantages are: (i) Increased complications during operation. (ii) Subsequent laparotomy for delivery or removal of the tape (if needed). **Indications are**—cases where cervix is hypoplastic or where prior vaginal cerclage has failed. A similar procedure can be done laparoscopically during the nonpregnant state.

Alternative to cervical cerclage—(nonsurgical) may be bed rest alone to avoid pressure on the cervix. Injection of 17α-hydroxyprogesterone caproate 500 mg IM weekly is given as cervical incompetence is considered as a continum of preterm birth syndrome. Use of vaginal pessary, when cervix is found short on ultrasound, is found helpful.

KEY POINTS

- **Cervical incompetence** is primarily a clinical diagnosis. It is manifested with recurrent painless cervical dilatation and spontaneous midtrimester miscarriage. USG diagnosis (during pregnancy) : Short cervix <25 mm length and funneling of the internal Os >1 cm.
- **Cervical incompetence** is the ultimate manifestation of cervical weakness. It may either be due to the primary anatomic defect of the cervix or due to trauma or may be due to the complex pathology of infection or inflammation of decidua/membranes leading to uterine contractions and cervical ripening.
- **A combination of all** the factors may be present in few cases. The pathological process of spontaneous midtrimester loss and preterm birth may be continuous and is known as **spontaneous preterm birth syndrome**.
- **Cervical cerclage** may be done based on the patient's history (history indicated) or repeated ultrasound observation (ultrasound indicated) or on physical examination as an emergency (emergent cerclage).
- **Cervical cerclage** is done at around 14 weeks pregnancy and the stitch is removed generally after 36 completed weeks.
- There are few **contraindications** and **complications of cervical cerclage** operations.

PROGNOSIS OF RECURRENT MISCARRIAGE

The prognosis of recurrent miscarriage is not so gloomy as it was previously thought. The overall risk of recurrent miscarriage is about 25–30% irrespective of the number of previous spontaneous miscarriage. The overall prognosis is good even without therapy. The chance of successful pregnancy is about 70–80% with an effective therapy. **Reassurance and tender loving care are very much helpful**.

INDUCTION OF ABORTION

Deliberate termination of pregnancy either by medical or by surgical method before the viability of the fetus is called induction of abortion. The induced abortion may be legal or illegal (criminal). There are many countries in the globe where the abortion is not yet legalized. In India, the abortion was legalized by "Medical Termination of Pregnancy Act" of 1971, and has been enforced in the year April 1972. The provisions of the act have been revised in 1975.

MEDICAL TERMINATION OF PREGNANCY (MTP)

Since legalization of abortion in India, deliberate induction of abortion by a registered medical practitioner in the interest of mother's health and life is protected under the MTP Act. **The following provisions are laid down**:

- The continuation of pregnancy would involve serious risk of life or grave injury to the physical and mental health of the pregnant woman.
- There is a substantial risk of the child being born with serious physical and mental abnormalities so as to be handicapped in life.
- When the pregnancy is caused by rape, both in cases of major and minor girl and in mentally imbalanced women.
- Pregnancy caused as a result of failure of a contraceptive.

In practice, the following are the indications for termination under the MTP Act:

- ***To save the life of the mother*** (Therapeutic or Medical termination): The indications are limited and scarcely justifiable nowadays except in the following cases: (i) Cardiac diseases (Grade III and IV) with history of decompensation in the previous pregnancy or in between the pregnancies. (ii) Chronic glomerulonephritis. (iii) Malignant hypertension. (iv) Intractable hyperemesis gravidarum. (v) Cervical or breast malignancy. (vi) Diabetes mellitus with retinopathy. (vii) Epilepsy or psychiatric illness with the advice of a psychiatrist.
- ***Social indications:*** **This is almost the sole indication and is covered under the provision "to prevent grave injury to the physical and mental health of the pregnant woman"**. In about 80%, it is limited to parous women having unplanned pregnancy with low socioeconomic status. Pregnancy caused by rape or unwanted pregnancy caused due to failure of any contraceptive device also falls in this category (20%).
- ***Eugenic:*** This is done under the provision of "substantial risk of the child being born with serious physical and mental abnormalities so as to be handicapped in life". **The indication is rare**.
    1. **Structural** (Anencephaly), **chromosomal** (Down's syndrome) or **genetic** (Hemophilia) abnormalities of the fetus.
    2. When the fetus is likely to be deformed due to action of **teratogenic drugs** (warfarin) or **radiation exposure** (>10 rad) in early pregnancy.
    3. **Rubella**, a viral infection affecting in the first trimester, is an indication for termination (see p. 348).

RECOMMENDATIONS

- In the revised rules, a registered medical practitioner is qualified to perform an MTP provided: (a) One has assisted in at least 25 MTP in an authorized center and having a certificate. (b) One has got 6 months house surgeon training in obstetrics and gynecology. (c) One has got diploma or degree in obstetrics and gynecology.
- Termination can only be performed in hospitals, established or maintained by the government or places approved by the government.
- Pregnancy can only be terminated on the written consent of the woman. Husband‘s consent is not required.
- Pregnancy in a minor girl (below the age of 18 years) or lunatic cannot be terminated without written consent of the parents or legal guardian.
- **Termination is permitted up to 20 weeks of pregnancy**. When the pregnancy exceeds 12 weeks, opinion of two medical practitioners is required.
- The abortion has to be performed confidentially and to be reported to the Director of Health Services of the State in the prescribed form.

METHODS OF TERMINATION OF PREGNANCY

First Trimester (Up to 12 Weeks)	Second Trimester (13–20 Weeks)
Medical ■ Mifepristone ■ Mifepristone and misoprostol (PGE$_1$) ■ Methotrexate and misoprostol ■ Tamoxifen and misoprostol **Surgical** ■ Menstrual regulation (see p. 646) ■ Vacuum aspiration (MVA/EVA) (see p. 646) ■ Suction evacuation and/or curettage ■ Dilatation and evacuation: ● Rapid method ● Slow method	♦ **Prostaglandins** PGE$_1$ (misoprostol), 15 methyl PGF$_2\alpha$ (Carboprost), PGE$_2$ (Dinoprostone) and their analogs (used—intravaginally, intramuscularly or intra-amniotically) ♦ **Dilation and evacuation** (13–14 weeks) ♦ **Intrauterine instillation of hyperosmotic solutions** ● Extra-amniotic—ethacridine lactate, Prostaglandins (PGE$_2$, PGF$_2\alpha$) ● Extra-amniotic saline infusion (isotonic) with a transcervical catheter balloon ● Intra-amniotic hypertonic urea (30%), saline (20%) ♦ **Oxytocin infusion**—high dose used along with either of the above two methods ♦ **Hysterotomy** (abdominal)—less commonly done

FIRST TRIMESTER TERMINATION OF PREGNANCY

MEDICAL METHODS OF FIRST TRIMESTER ABORTION: Mifepristone (RU-486) and Misoprostol: Mifepristone an analog of progestin (norethindrone) acts as an antagonist, blocking the effect of natural progesterone. Addition of low-dose prostaglandins (PGE$_1$) improves the efficiency of first trimester abortion. It is effective up to 63 days and is highly successful when used within 49 days of gestation.

PROTOCOL: 200 mg of mifepristone orally is given on day 1. On day 3, misoprostol (PGE$_1$) 400 µg orally or 800 µg vaginally is given. Patient remains in the clinic for 4 hours during which expulsion of the conceptus (95%) often occurs. Patient is reexamined after 10–14 days. Complete abortion is observed in 95%, incomplete in about 2% of cases and about 1% do not respond at all. **Oral mifepristone 200 mg (1 tab) with vaginal misoprostol 800 µg (4 tab, 200 µg each) after 6–48 hours is equally effective**. This combipack (1+4) is approved by DGHS, Government of India for MTP up to 63 days of pregnancy. Medical methods are safe, effective, noninvasive and have minimal or no complications.

Methotrexate and Misoprostol—Methotrexate 50 mg/m^2 IM (before 56 days of gestation) followed by 7 days later misoprostol 800 µg vaginally is highly effective. Misoprostol may have to be repeated after 24 hours if it fails. If the procedure fails, ultrasound examination is done to confirm the failure. Then suction evacuation should be done. **Methotrexate and misoprostol regimen is less expensive but takes longer time than mifepristone and misoprostol**. Misoprostol has less side effects and is stable at room temperative unlike other PGs, which must be refrigerated (see p. 578).

Contraindications—Mifepristone should not be used in women aged over 35 years, heavy smokers and those on long-term corticosteroid.

SURGICAL METHODS OF FIRST TRIMESTER ABORTION:

- **MENSTRUAL REGULATION:** See p. 646.
- **VACUUM ASPIRATION (MVA/EVA)** is done up to 12 weeks with minimal cervical dilatation. It is performed as an outpatient procedure using a plastic disposable cannula (up to 12 mm size) and a 60 mL plastic (double valve) syringe (Fig. 41.20). It is quicker (15 minutes), effective (98–100%), less traumatic and safer than dilatation, evacuation and curettage (details see p. 646).
- **SUCTION EVACUATION AND/OR CURETTAGE:**

This improvised method consists of a suction machine fitted with a cannula either plastic (Karman) or metal available in various sizes. The details of its technique are described in the chapter of operative obstetrics (see p. 645).

Advantages: (1) It is done as an outdoor procedure. (2) Hazards of general anesthesia are absent as it is done, at best, under paracervical block anesthesia. (3) Ideal for termination for therapeutic indications. (4) Blood loss is minimal. (5) Chance of uterine perforation is much less especially with the plastic cannula.

Drawbacks: (1) The method is not suitable with bigger size uterus of more than 10 weeks as chance of retained products is more. (2) Requires electricity to operate and the machine is costly.

- **DILATATION AND EVACUATION:** • **Rapid method** • **Slow method**

- ***Rapid method:*** This can be done as an outdoor procedure with diazepam sedation and paracervical block anesthesia. The details are described in p. 643.

Advantages: (1) As it can be done as an outdoor procedure, the patient can go home after the sedative effect is over. (2) Chance of sepsis is minimal.

Drawbacks: (1) Chance of cervical injury is more. (2) Uterus should not be more than 6–8 weeks of pregnancy. (3) All the drawbacks of D&E (see p. 644).

- ***Slow method:*** Slow dilatation of the cervix is achieved by inserting laminaria tents (hygroscopic osmotic dilators) into the cervical canal (synthetic dilators like Dilapan, Lamicel are also used). This is followed by evacuation of the uterus after 12 hours. Vaginal misoprostol (PGE$_1$) 400 µg 3 hours before surgery is equally effective for cervical ripening. The details are described in p. 646.

Advantages: (1) Chance of cervical injury is minimal. (2) Suitable in cases of therapeutic indications.

Drawbacks: (1) Hospitalization is required at least for 1 day. (2) Chance of introducing sepsis. (3) All the complications of D&E (see p. 644).

MIDTRIMESTER TERMINATION OF PREGNANCY

MEDICAL METHODS:

PROSTAGLANDINS: **Prostaglandins and their analogs** are very much effective. They are used extensively, specially in the second trimester. They act on the cervix and the uterus. The PGE (dinoprostone, sulprostone, gemeprost, misoprostol) and PGF (carboprost) analogs are commonly used. PGEs are preferred as they have more selective action on the myometrium and less side effects.

(i) **Misoprostol (PGE$_1$ analog):** 400–800 µg of misoprostol given vaginally at an interval of 3–4 hours is most effective as the bioavailability is high. Alternatively, first dose of 600 µg misoprostol given vaginally, then 200 μg, orally every 3 hours are also found optimum. This regimen reduces the number of vaginal examinations. Recently 400 μg misoprostol is given sublingually every 3 hours for a maximum of five doses. This regimen has got 100% success in second trimester abortion. The mean induction—abortion interval is 11–12 hours.

(ii) **Mifepristone and prostaglandins**: Mifepristone 200 mg oral, followed 36–48 hours later by misoprostol 800 μg vaginal; then misoprostol 400 μg oral every 3 hours for four doses is used. Success rate of abortion is 97% and median induction delivery interval is 6.5 hours. Pretreatment with mifepristone reduces the induction-abortion interval significantly compared to use of misoprostol alone.

(iii) **Gemeprost (PGE_1 analog):** 1 mg vaginal pessary every 3–6 hours for five doses in 24 hours has got about 90% success. The mean induction-abortion interval was 14–18 hours.

(iv) **Dinoprostone (PGE_2 analog):** 20 mg is used as a vaginal suppository every 3–4 hours (maximum for 4–6 doses). When used along with osmotic dilators, the mean induction to abortion interval is 17 hours. PGE_2 is thermolabile (needs refrigeration) and is expensive.

(v) **Prostaglandin $F_{2\alpha}$ ($PGF_{2\alpha}$)**, carboprost tromethamine—250 μg IM every 3 hours for a maximum ten doses can be used. The success rate is about 90% in 36 hours. Side effects of $PGF_{2\alpha}$ (nausea, vomiting, diarrhea and pain at injection site) are more. It is contraindicated in cases with bronchial asthma.

OXYTOCIN: High-dose oxytocin as a single agent can be used for second trimester abortion. It is effective in 80% of cases. It can be used with intravenous normal saline along with any of the medications used either intra-amniotic or extra-amniotic space in an attempt to augment the abortion process (see below). The drip rate can be increased up to 50 milliunits or more per minute. Currently high dose (up to 300 units in 500 mL of dextrose saline) is favored.

SURGICAL METHODS:

It is difficult to terminate pregnancy in the second trimester with reasonable safety as in first trimester. **The following surgical methods may be employed**.

Between 13 weeks and 15 weeks

- **Dilatation and Evacuation** in the midtrimester is less commonly done. Pregnancies at 13–14 menstrual weeks are evacuated. In all midtrimester abortion, cervical preparation must be used (WHO 1997) to make the process easy and safe. Intracervical tent (Laminaria osmotic dilator), mifepristone or misoprostol are used as the cervical priming agents. *The procedure may need to be performed under ultrasound guidance to reduce the risk of complications (see p. 646).* Simultaneous use of oxytocin infusion is useful.

Between 16 weeks and 20 weeks: INTRAUTERINE INSTILLATION OF HYPERTONIC SOLUTION:

- **Extra-amniotic**
- **Intra-amniotic**

◆ ***Extra-amniotic:*** **Extra-amniotic instillation of 0.1% ethacridine lactate** (estimated amount is 10 mL/week) is done transcervically through a No. 16 Foley's catheter. The catheter is passed up the cervical canal for about 10 cm above the internal os between the membranes and myometrium and the balloon is inflated (10 mL) with saline. It is removed after 4 hours. The success rate is similar to saline instillation but is less hazardous. It can be used in cases contraindicated for saline instillation. Stripping the membranes with liberation of prostaglandins from the decidua and dilatation of the cervix by the catheter are some of the known factors for initiation of the abortion.

◆ Isotonic saline is infused extra-amniotically using a transcervical catheter balloon. Results are similar to that of Foley's catheter use alone.

◆ ***Intra-amniotic***: **Intra-amniotic instillation of hypertonic saline** (20%) is less commonly used now. It is instilled through the abdominal route.

Procedure: Preliminary amniocentesis is done as described in p. 742 by a 15 cm 18-gauge needle. **The amount of saline to be instilled is calculated as number of weeks of gestation multiplied by 10 mL.** The amount is to be infused slowly at the rate of 10 mL/min.

Contraindications: It should not be used in presence of cardiovascular or renal lesion or in severe anemia because of sodium load.

Precautions: (1) To be sure that the needle is in the amniotic cavity evidenced by clear liquor coming out. If there is a bloody tap, the needle should be pushed further or change the direction until clear liquor comes out. If fails, the procedure is to be abandoned. (2) The instillation should be a slow process (10 mL/min). (3) Vital signs should be checked immediately after the instillation and she should be kept at bed rest for at least 1 hour. (4) **To stop the procedure if the untoward symptoms** like acute abdominal pain, headache, thirst or tingling in the fingers appear (feature of intravascular injection of the hypertonic saline). **A rapid infusion of 1,000 mL dextrose in water along with intravenous diuretics is indicated in such cases.** (5) Strict vigilance is taken during and following instillation till expulsion occurs. (6) Routine antibiotic is given such as ampicillin 500 mg thrice daily for 3–5 days.

Mode of action: There is liberation of prostaglandins following necrosis of the amniotic epithelium and the decidua. This in turn excites uterine contraction and results in the expulsion of the fetus.

Success rate: The method is effective in 90–95% cases with induction-abortion interval of about 32 hours. **The method failure (end point) is considered when abortion fails to occur within 48 hours**. If the method fails, some other method may be employed.

Complications: **The complications include—**(a) Minor complaints like fever, headache, nausea, vomiting, abdominal pain. (b) Cervical tear and laceration. (c) Retained products for which exploration has to be done. (d) Infection. (e) Hypernatremia, cardiovascular collapse—due to intravascular injection. (f) Pulmonary and cerebral edema. (g) Renal failure. (h) Disseminated intravascular coagulopathy. The incidence of death rate varies from 0 to 5 per 1,000 instillations.

Intra-amniotic instillation of hyperosmotic urea: Intra-amniotic instillation of 40% urea solution (80 g of urea in 200 mL distilled water) along with syntocinon drip is effective with less complications. Combination of intra-amniotic hyperosmotic urea and 15 methyl $PGF_{2\alpha}$ reduces the induction-abortion interval to 13 hours.

HYSTEROTOMY: The operation is performed through abdominal route. The steps are given in chapter 36.

Indications: (i) Prior failed medical termination of pregnancy (TOP) (ii) Cases where D&E cannot be safely done: (a) fibroid in the lower uterine segment, (b) uterine anomalies, (c) patients with repeated scarred uterus with placenta accreta or percreta. It is less commonly done these days. **The operation should be combined with sterilization operation**.

Hazards: **Immediate:** (i) Hemorrhage and shock. (ii) Anesthetic complications. (iii) Peritonitis. (iv) Intestinal obstruction.

Remote: (1) Menstrual abnormalities. (2) Scar endometriosis (1%). (3) Incisional hernia. (4) If pregnancy occurs, chance of scar rupture.

Rh-NEGATIVE WOMEN: In nonimmunized women, intramuscular administration of 100 μg anti-D immunoglobulin is given within 72 hours of abortion.

COMPLICATIONS OF MTP

There is no universally safe and effective method which is applicable to all cases. However, the complications are much less (5%) if termination is done before 8 weeks by MVA or suction evacuation/curette. The complications are about five times more in midtrimester termination. Use of PG analogs and mifepristone has made second trimester MTP effective and safe. The complications are either related to the methods employed or to the abortion process.

IMMEDIATE: (1) Injury to the cervix (cervical lacerations). (2) Uterine perforation during D&E (see p. 644). (3) Hemorrhage and shock due to trauma, incomplete abortion, atonic uterus or rarely coagulation failure. (4) Thrombosis or embolism. (5) **Postabortal triad** of pain, bleeding and low-grade fever due to retained clots or products. Antibiotics should be continued, may need repeat evacuation. (6) **Related to the methods employed**:

- ***Prostaglandins***—intractable vomiting, diarrhea, fever, uterine pain and cervicouterine injury.
- ***Oxytocin***—water intoxication and rarely convulsions
- ***Hysterotomy*** (p. 647)

- ***Saline***—hypernatremia, pulmonary edema, endotoxic shock, Disseminated intravascular coagulation (DIC), renal failure, cerebral hemorrhage

REMOTE: The complications are grouped into: • ***Gynecological*** • ***Obstetrical***

- ***Gynecological complications include***—(a) menstrual disturbances, (b) chronic pelvic inflammation, (c) infertility due to cornual block, (d) scar endometriosis (1%) and (e) uterine synechiae leading to secondary amenorrhea.
- ***Obstetrical complications include***—(a) recurrent midtrimester abortion due to cervical incompetence, (b) ectopic pregnancy (threefold increase), (c) preterm labor, (d) dysmaturity, (e) increased perinatal loss, (f) rupture uterus, (g) Rh-isoimmunization in Rh-negative women, if not prophylactically protected with immunoglobulin and (h) failed abortion and continued pregnancy.

Failed abortion, continued pregnancy and ectopic pregnancy: Pregnancy may continue following MVA (in spite of histologically proven villi). When no chorionic villi are found on tissue examination, ectopic pregnancy need to be excluded by quantitative serum hCG and vaginal ultrasound. Failed MTP is defined when there is a failure to achieve TOP within 48 hours. Failed second trimester MTP with PG analogs and the rate of live birth is 4–10%.

MORTALITY: ***First trimester:*** The maternal death is lowest (about 0.6/100,000 procedures) in first trimester termination specially with MVA and suction evacuation. Concurrent tubectomy even by abdominal route doubles the mortality rate.

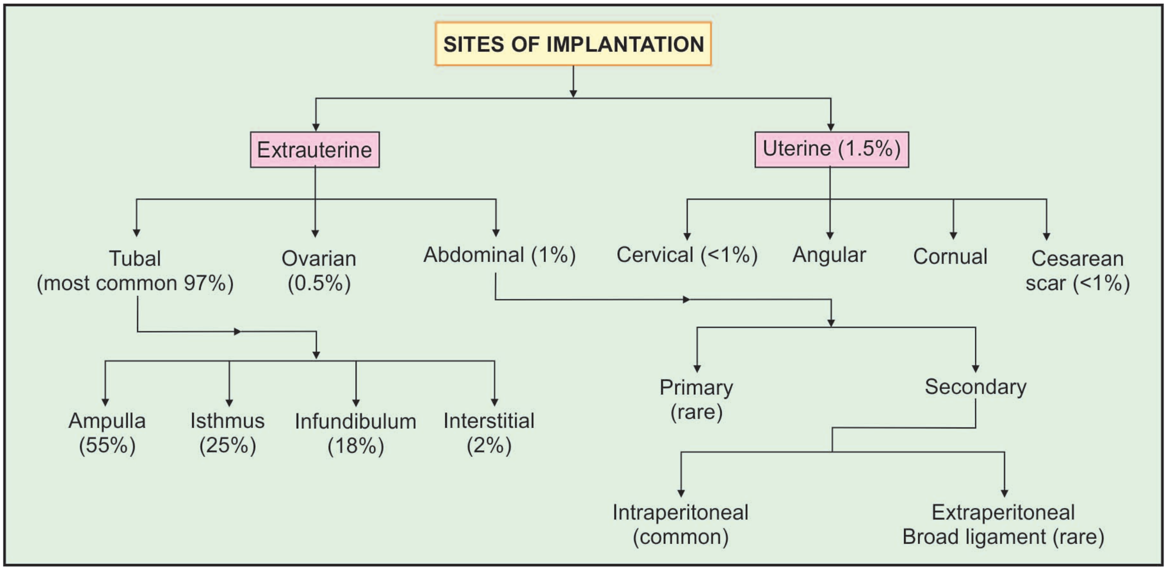
Midtrimester: The mortality rate increases five to six times to that of first trimester. Contrary to the result of the advanced countries, the mortality from saline method has been found much higher in India compared to termination by abdominal hysterotomy with tubectomy.

ECTOPIC PREGNANCY

Ectopic pregnancy still contributes significantly to the cause of maternal mortality and morbidity. While there has been about fourfold increase in incidence over the couple of decades, but the mortality has been slashed down by 80%. **Recognition of high-risk cases (see p. 208), early diagnosis (even before rupture) with the use of TVS, serum β-hCG and laparoscopy have significantly improved the management of ectopic pregnancy**.

DEFINITION: An ectopic pregnancy is one in which the fertilized ovum is implanted and develops outside the normal endometrial cavity.

SITES OF IMPLANTATION:


SITES OF IMPLANTATION
Extrauterine
Uterine (1.5%)
Tubal (most common 97%)
Ovarian (0.5%)
Abdominal (1%)
Cervical (<1%)
Angular
Cornual
Cesarean scar (<1%)
Ampulla (55%)
Isthmus (25%)
Infundibulum (18%)
Interstitial (2%)
Primary (rare)
Secondary
Intraperitoneal (common)
Extraperitoneal Broad ligament (rare)

TUBAL PREGNANCY

FREQUENCY: The incidence has increased. The reasons are: increased prevalence of chronic pelvic inflammatory disease, tubal plastic operations, ovulation induction and IUD use. Secondly early diagnosis helps to detect some cases, that in the past, may have resolved spontaneously. **Early diagnosis and therapy have helped to reduce maternal deaths due to ectopic pregnancy**. The incidence varies from 1 in 300 to 1 in 150 deliveries.

ETIOLOGY:

- **Salpingitis and pelvic inflammatory disease (PID)** increases the risk of ectopic pregnancy by sixfold to tenfold. (a) Loss of cilia of the lining epithelium and impairment of muscular peristalsis. (b) Narrowing of the tubal lumen. (c) Formation of pockets due to adhesions between mucosal folds. (d) Peritubal adhesions resulting in kinking and angulation of the tube. *Chlamydia trachomatis infection* is the most common risk factor. Salpingitis isthmica nodosa also increases the risk.
- **Iatrogenic:**

I. ***Contraception failure:*** Women using any form of contraception have significantly reduced chance of having a ectopic pregnancy. But in selected contraception failure, there is increased incidence of ectopic pregnancy.

(a) **IUD—**It prevents intrauterine pregnancy effectively, tubal implantation to a lesser extent and the ovarian pregnancy not at all. **There is relative increase in tubal pregnancy (7 times more) should pregnancy occur with IUD *in situ*.** CuT 380A and levonorgestrel devices have got the lowest rate of ectopic whereas Progestasert has got the highest one. Whether tubal infection or abnormal tubal mobility in postfertilization period is the cause is not clear.

(b) **Sterilization operation**—There is 15–50% chance of being ectopic if pregnancy occurs. This is due to sterilization failure. The risk is highest following laparoscopic bipolar coagulation.

(c) **Use of progestin only pill** or postcoital estrogen preparations increases the chance of tubal pregnancy probably by impaired tubal motility.

II. ***Tubal surgery***—Tubal reconstructive surgery to improve fertility, increases the risk of tubal pregnancy significantly. Pre-existing tubal pathology, impaired tubal motility, kinking of the tube or terminal stricture are the contributing factors.

III. ***Intrapelvic adhesions*** following pelvic surgery.

Table 16.2: Risk Factors of Ectopic Pregnancy

- History of PID
- History of tubal ligation
- Contraception failure
- Previous ectopic pregnancy
- Tubal reconstructive surgery
- History of infertility
- ART particularly if the tubes are patent but damaged
- IUD use
- Previous induced abortion
- Tubal endometriosis

IV. ***ART***—Tubal pregnancy is increased following ovulation induction and IVF-ET and GIFT procedures. The risk of ectopic is 5–7% and that of heterotypic pregnancy is 1% in contrast to 1 in 5,000 in spontaneous pregnancy.

V. ***Others:***

- **Previous ectopic pregnancy:** There is 10–15% chances of repeat ectopic pregnancy.
- **Prior induced abortion** significantly increases the risk.
- **Developmental defects** of the tube: (a) Elongation. (b) Diverticulum. (c) Accessory ostia.
- **Transperitoneal migration of the ovum**—contralateral presence of corpus luteum is noticed in tubal pregnancy in about 10% cases.

Factors facilitating nidation in the tube: (i) Early resumption of the trophoblastic activity is probably due to premature degeneration of the zona pellucida. (ii) Increased decidual reaction. (iii) Tubal endometriosis.

The reasons for rising incidence of ectopic pregnancies are—(i) Rise in the incidence of STIs and salpingitis. (ii) Rise in the incidence of pregnancy following ART procedures. (iii) Increased tubal surgery (either sterilization or tuboplasty procedure). (iv) Early detection of cases that were otherwise destined to undergo spontaneous absorption.

MORBID ANATOMY

CHANGES IN THE TUBE: **(i)** Implantation in the tube occurs more commonly in intercolumnar fashion, i.e. in between two mucosal folds. **(ii)** Decidual change at the site of implantation is minimal. The muscles undergo limited hyperplasia and hypertrophy but more stretching. Blood vessels are engorged. **(iii)** The blastocyst burrows through the mucous membrane and lies between the lumen and the peritoneal covering—so-called intramuscular implantation. **(iv)** A pseudocapsule is formed consisting of fibrin, lining epithelium and few muscle fibers. **(v)** Blood vessels are eroded by the chorionic villi and blood accumulates in between the blastocyst and the serous coat. **(vi)** The tube on the implantation site is distended and the wall is thinned out. **(vii)** Blood may spill from the fimbriated end and may cause hemoperitoneum. **(viii)** The stretching of the peritoneum over the site of implantation results in episodic pain. Finally, tubal rupture occurs when the muscles and the serosa are maximally stretched and undergo necrosis. **(ix)** Hemoperitoneum is found in all cases of ruptured tubal ectopic pregnancy. **(x)** The trophoblasts of ectopic pregnancy do not usually grow as that of a normal pregnancy. As a result, hCG production is inadequate compared to a normal pregnancy.

CHANGES IN THE UTERUS: Under the influence of estrogen, progesterone from corpus luteum and chorionic gonadotropin, there is varying amount of enlargement of the uterus with increased vascularity. **The decidua develops all the characteristics of intrauterine pregnancy except that it contains no evidence of chorionic villi**. When progesterone level falls due to fall in the level of hCG, endometrial growth is no longer maintained. Endometrium sloughs out causing uterine bleeding. Sometimes entire decidua is expelled as a single piece through the cervix. This is known as **decidual cast** that may be confused with a spontaneous abortion.

MODE OF TERMINATION

Because of the unfavorable environment, early interruption of pregnancy is inevitable within 6–8 weeks. Earliest interruption occurs in the isthmial implantation whereas pregnancy may continue up to 3–4 months in interstitial implantation. However, genuine cases are on record of gestation continuing to term in the Fallopian tube. The modes of termination are as follows:

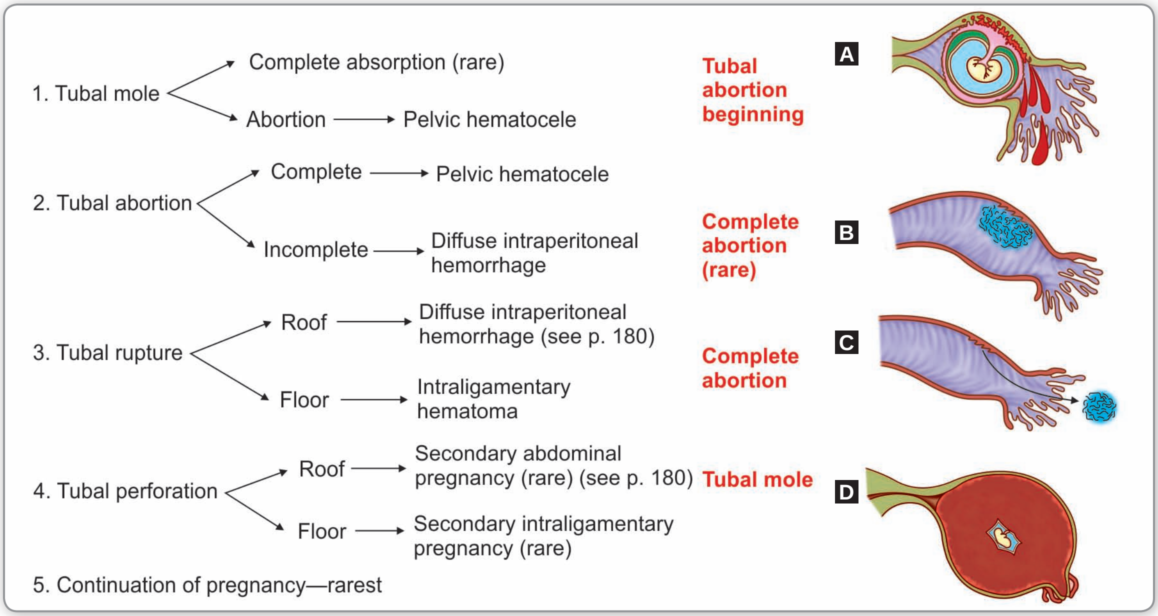
***Tubal mole* (Fig. 16.8):** The formation of the tubal mole is similar to that formed in uterine pregnancy. Repeated small hemorrhages occur in the choriocapsular space, separating the villi from their attachments. The fate of the mole is either—(a) complete absorption or (b) expulsion through the abdominal ostium as tubal abortion with a variable amount of internal hemorrhage. **The encysted blood so collected in the pouch of Douglas is called pelvic hematocele**.

***Tubal abortion* (Fig. 16.8):** This is the **common mode of termination if implantation occurs in the ampulla or infundibulum**. Muscular contraction enhances separation and facilitates its expulsion through the abdominal ostium.

***Tubal rupture*:** Tubal rupture is predominantly **common in isthmic and interstitial implantation**. As the isthmic portion is narrow and the wall is less distensible, the wall may be easily eroded by the chorionic villi. **Isthmic rupture usually occurs at 6–8 weeks, the ampullary one at 8–12 weeks and the interstitial one at about 4 months**.

Depending upon the site of rupture, it is known as: (1) **Intraperitoneal rupture**: This type of rupture is common. The rent is situated on the roof or sides of the tube. The bleeding is intraperitoneal. (2) **Extra-peritoneal rupture** (intraligamentary): This is rare and occurs when the rent lies on the floor of the tube where the broad ligament is attached. **It is commonly met in isthmic implantation**.

***Secondary abdominal pregnancy*: The prerequisites for the continuation of fetal growth outside the tube are:** (1) Perforation of the tubal wall should be a slow process. (2) Amnion must be intact. (3) Placental chorion should escape injury from the rupture. (4) Herniation of the amniotic sac with the living ovum and the placenta should occur through the rent. (5) Placenta gets attached to the


1. Tubal mole
Complete absorption (rare)
Abortion → Pelvic hematocele
2. Tubal abortion
Complete → Pelvic hematocele
Incomplete → Diffuse intraperitoneal hemorrhage
3. Tubal rupture
Roof → Diffuse intraperitoneal hemorrhage (see p. 180)
Floor → Intraligamentary hematoma
4. Tubal perforation
Roof → Secondary abdominal pregnancy (rare) (see p. 180)
Floor → Secondary intraligamentary pregnancy (rare)
5. Continuation of pregnancy—rarest
Tubal abortion beginning
A
Complete abortion (rare)
B
Complete abortion
C
Tubal mole
D

Figs 16.8A to D: Modes of Termination of Tubal Pregnancy

neighboring structures and new vascular connection should be re-established. The fibrin is deposited over the exposed amnion to constitute a secondary amniotic sac. (6) Intestine, omentum and adjacent structures get adherent to the secondary sac.

***Secondary broad ligament pregnancy*:** Rarely pregnancy may continue in the same process as in abdominal pregnancy between the two layers of the peritoneum.

Arias-Stella reaction: This is characterized by a typical adenomatous change of the endometrial glands. Intraluminal budding together with typical cell changes (loss of polarity of cells, pleomorphism, hyperchromatic nuclei, vacuolated cytoplasm and occasional mitosis) are collectively referred to as Arias-Stella reaction. This is strikingly due to progesterone influence. It is present in about 10–15% cases of ectopic pregnancy. **It is not, however, specific for ectopic pregnancy but rather the blightning of conceptus either intrauterine or extrauterine.**

Fate of Secondary Abdominal Pregnancy

- **Death of the blastocyst** → complete absorption
- **Massive intraperitoneal hemorrhage** due to placental separation
- **Infection** → fistulous communication with intestine, bladder, umbilicus
- **Fetal death** → mummification, suppuration, adipocere formation, calcification (*lithopedion*)
- **Continue to term pregnancy** (rare—1.0%) (risk of fetal malformation and deformation)

Source of vaginal bleeding: Levels of progesterone secreted by the corpus luteum fall as there is insufficient level of hCG. Endometrial growth and function is no longer maintained and if then sloughs out. On rare occasion, the bleeding may be due to tubal abortion through the uterine ostium in interstitial pregnancy.

CLINICAL FEATURES OF ECTOPIC PREGNANCY

Very few clinical conditions exhibit so varied features like that of disturbed tubal pregnancy. The clinical types are correlated with the morbid pathological changes in the tube subsequent to implantation and the amount of intraperitoneal bleeding. However, clinically three distinct types are described:

- **Acute**
- **Unruptured**
- **Subacute (chronic or old)**

ACUTE ECTOPIC: An acute ectopic is fortunately less common (about 30%) and it is associated with cases of tubal rupture or tubal abortion with massive intraperitoneal hemorrhage.

Patient profile: (1) The incidence is maximum between the age of 20 years and 30 years, being the maximum period of fertility. (2) The prevalence is mostly limited to nulliparity or following long period of infertility.

Mode of onset: The onset is acute. The patients, however, have got persistent unilateral uneasiness in about one-third of cases before the acute symptoms appear.

Symptoms: The classic triad of symptoms of disturbed tubal pregnancy are: abdominal pain (100%), preceded by amenorrhea (75%) and lastly, appearance of vaginal bleeding (70%).

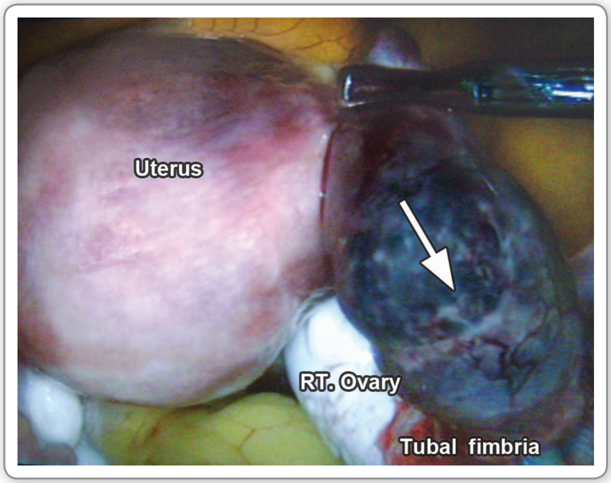


Fig. 16.9: Laparoscopic view of an unruptured tubal ectopic pregnancy (Rt). Hugely dilated ampulla is seen (arrow)

- **Amenorrhea:** Short period of 6–8 weeks (usually); there may be delayed period or history of vaginal spotting. Amenorrhea may be absent even.
- **Abdominal pain** is the most constant feature. It is acute, agonizing or colicky. Otherwise it may be a vague soreness. Pain is located at lower abdomen: unilateral, bilateral or may be generalized. **Shoulder tip pain (25%)** (referred pain due to diaphragmatic irritation from hemoperitoneum) may be present.
- **Vaginal bleeding** may be slight and continuous. Expulsion of decidual cast (5%) may be there (Fig. 16.3) (see p. 191).
- **Vomiting, fainting attack**. Syncopal attack (10%) is due to reflex vasomotor disturbances following peritoneal irritation from hemoperitoneum.

SIGNS:

- **General look (diagnostic):** The patient lies quiet and conscious, perspires and looks blanched.
- **Pallor:** Severe and proportionate to the amount of internal hemorrhage.
- **Features of shock:** Pulse—rapid and feeble, hypotension, extremities—cold clammy.
- **Abdominal examination:** Abdomen (lower abdomen)—tense, tumid, tender. No mass is usually felt, shifting dullness present, bowels may be distended. Muscle guard—usually absent.
- **Pelvic examination** is less informative due to extreme tenderness and it may precipitate more intraperitoneal hemorrhage due to manipulation. **The findings are**: (i) Vaginal mucosa—blanched white. (ii) Uterus seems normal in size or slightly bulky. (iii) Extreme tenderness on fornix palpation or on movement of the cervix. (75%) (iv) No mass is usually felt through the fornix. (v) The uterus floats as if in water. **Caution**: Vaginal examination may precipitate more hemorrhage due to manipulation.

UNRUPTURED TUBAL ECTOPIC PREGNANCY

High degree of suspicion and an ectopic conscious clinician can only diagnose the entity at its prerupture state. There is a high frequency of misdiagnosis and physician delay. **The physician should include**

ectopic pregnancy in the differential diagnosis when a sexually active female has abnormal bleeding and/or abdominal pain. This is especially so when the woman has got some risk factors (see p. 208).

Symptoms: ♦ Presence of delayed period or spotting with features suggestive of pregnancy. ♦ Uneasiness on one side of the flank which is continuous or at times colicky in nature.

Signs: Bimanual examination: (i) Uterus is usually soft showing evidence of early pregnancy. (ii) **A pulsatile small, well-circumscribed tender mass may be felt through one fornix separated from the uterus**. The palpation should be gentle, else rupture may precipitate and massive intraperitoneal hemorrhage when shock and collapse may occur dramatically.

Investigations: With the advent of transvaginal sonography (TVS), highly sensitive radioimmunoassay of β-hCG and laparoscopy (see below), more and more ectopics are now diagnosed in unruptured state.

CHRONIC OR OLD ECTOPIC

Onset: The onset is insidious. The patient had previous attacks of acute pain from which she had recovered or she had chronic features from the beginning.

Symptoms:

- **Amenorrhea:** Short period of 6-8 weeks is usually present.
- **Lower abdominal pain** is present with varying degrees. It starts as an acute one and gradually becomes dull or colicky in nature.
- **Vaginal bleeding** appears sooner or later following the pain. It is scanty, sanguinous or dark-colored and continuous in nature. Expulsion of decidual cast may be present (Fig. 16.3).
- **Other symptoms:** There may be features of bladder irritation—dysuria, frequency or even retention of urine. Rectal tenesmus may appear especially following infected hematocele. Rise of temperature may be due to infection or due to absorption of the products of degenerated blood accumulated in the abdomen.

On examination (signs):

- **The patient looks ill**.
- **Pallor** varying degree is present.
- Pulse persistently high even during rest—a conspicuous finding.
- **Features of shock** are absent.
- **Temperature** may be slightly elevated to 38°C.
- **Abdominal examination:** (i) **Tenderness** and muscle guard on the lower abdomen especially on the affected side are a striking feature. (ii) **A mass** in the lower abdomen may be felt which is irregular and tender. (iii) **Cullen's sign**—Dark bluish discoloration around the umbilicus, if found, suggests intraperitoneal hemorrhage.
- **Bimanual examination is painful and reveals:** (i) Vaginal mucosa—pale. (ii) Uterus seems to be normal in size or bulky, often incorporated in the mass occupying the pelvis. (iii) Extreme tenderness on movement of the cervix. (iv) **An ill-defined, boggy and extremely tender mass is felt through the posterolateral fornix extending to the pouch of Douglas.** The mass may push the uterus to the opposite side.
- **Rectal examination** corroborates the pelvic findings.
- **Examination under anesthesia (EUA)** is helpful to evaluate the pelvic findings but accidental tubal rupture may be provoked during manipulation. Moreover, the diagnostic features of pain on examination and tenderness on moving the cervix (cervical excitation pain) cannot be elicited.

DIAGNOSIS OF ECTOPIC PREGNANCY

ACUTE ECTOPIC: The classic history of acute abdominal catastrophe with fainting attack and collapse associated with features of intra-abdominal hemorrhage in a woman of child-bearing age points to a certain diagnosis of acute ectopic.

No time should be wasted for investigations other than estimation of hemoglobin and blood grouping (ABO and Rh).

Examination under anesthesia: Extreme tenderness on vaginal examination, which is of significance, cannot be elicited by EUA. Moreover, at times, it proves risky as the manipulation may provoke further bleeding. In any case, laparotomy is indicated by its own merit even though it may be proved otherwise.

Differential diagnoses of acute ectopic pregnancy are: (1) acute appendicitis, (2) ruptured corpus luteum. Clinical presentation is similar to ruptured tubal ectopic pregnancy—pregnancy test is negative, (3) twisted ovarian tumor, (4) ruptured chocolate cyst and (5) perforated peptic ulcer. Considering the fact that all the clinical conditions require urgent laparotomy, there is no possibility of acute ectopic being overlooked.

SUBACUTE (CHRONIC) ECTOPIC: It is indeed difficult at times to diagnose old ectopics because of vagaries of clinical features mentioned earlier. **Increased awareness on the part of the clinicians is the sheet anchor in the diagnosis of old ectopic. The confusing features are**: (1) Absence of amenorrhea. (2) Absence of vaginal bleeding. (3) Vaginal bleeding followed by pain. (4) Apparently normal general condition. (5) Presence of bilateral mass on internal examination. (6) Previous history of tubectomy operation or IUD insertion.

Investigations for the diagnosis of tubal ectopic pregnancy

- **Blood examination** should be done as a routine for: (i) Hemoglobin. (ii) ABO and Rh grouping. (iii) Total white cell count and differential count. (iv) Erythrocyte sedimentation rate (ESR). There may be varying degrees of leukocytosis and raised ESR.
- **Culdocentesis** is simple and safe. Where sensitive TVS or laparoscopy is not readily available, culdocentesis is still a diagnostic alternative. Unfortunately negative culdocentesis does not rule out an ectopic pregnancy neither a positive result is very specific. Through an 18-gauge lumbar puncture needle fitted with a syringe, the posterior fornix is punctured to gain access to the pouch of Douglas. Aspiration of nonclotting blood with hematocrit greater than 15% signifies ruptured ectopic pregnancy.
- **Estimation of β-hCG:** Urine pregnancy test—ELISA is sensitive to 10-50 mIU/mL and is positive in 95% of ectopic pregnancies. **A single estimation of β-hCG level either in the serum or in urine confirms pregnancy but cannot determine its location**. The suspicious findings are: (1) Lower concentration of β-hCG compared to normal intrauterine pregnancy (2) Doubling time in plasma fails to occur in 2 days.
- **Sonography:** Transvaginal sonography (TVS) is more informative. **The diagnostic features are**: (1) Absence of intrauterine pregnancy with a positive pregnancy test. (2) Fluid (echogenic) in the pouch of Douglas. (3) Adnexal mass clearly separated from the ovary. (4) Rarely cardiac motion may be seen in an unruptured tubal ectopic pregnancy. **Color Doppler Sonography:** (TV-CDS)—can identify the placental shape (ring-of-fire pattern) and enhanced blood flow pattern outside the uterine cavity.
- **Combination of quantitative β-hCG values and sonography:** TVS provides visualization of a well-formed intrauterine gestational sac as early as 4–5 weeks from the last menstrual period. The lowest level of serum β-hCG at which a gestational sac is consistently visible using TVS (discriminatory zone) is 1,500 IU/L. The corresponding value of serum β-hCG for TAS is 6,000 IU/L. (1) When the β-hCG value is greater than 1,500 IU/L and there is an empty uterine cavity, ectopic pregnancy is more likely. (2) Failure to double the value of β-hCG by 48 hours along with an empty uterus is very much suggestive.
- **Laparoscopy** offers benefit in cases of confusion with other pelvic lesions. It should be employed only when the patient is hemodynamically stable. **Advantages are:** (i) Confirmation of diagnosis. (ii) Removal of the ectopic mass using operative procedures at the same time. (iii) Direct injection of chemotherapeutic agents into the ectopic mass—when medical management is decided. However, laparoscopy runs the risk of false-positive or false-negative diagnosis in 2–5% of cases.
- **Dilatation and curettage**—Identification of decidua without villi structure is very much suggestive. Chorionic villi that float in normal saline as lacy fronds are diagnostic of intrauterine pregnancy.
- **Serum progesterone**—Level greater than 25 ng/mL is suggestive of viable intrauterine pregnancy whereas level less than 5 ng/mL suggests an ectopic or abnormal intrauterine pregnancy.
- **Laparotomy** offers benefit when in doubt. The old axiom, "**open and see**" holds good especially when the patient is hemodynamically unstable. **One should not be ashamed of having a negative abdominal exploration, rather to be disgraced for the mistake in diagnosis with the eventual fatality**.

Differential diagnosis of subacute ectopic pregnancy:

1. *Incomplete abortion:* (i) Bleeding appears prior to pain. (ii) Absence of fainting attack. (iii) Bleeding is bright red and at times profuse. (iv) General condition proportionate with visible blood loss. (v) No features of intraperitoneal bleeding. (vi) Fornix palpation gives negative findings. (vii) D&C relieves the symptoms. (viii) **Histological examination shows presence of villi**. **Persistence of symptoms even after thorough uterine curettage makes one suspicious of ectopic**.

2. *Salpingitis*: (i) Previous history of similar attacks of pain. (ii) The pain is of burning nature. (iii) Amenorrhea is absent although slight bleeding may appear. (iv) Rise of temperature is more. (v) **The patient is tossing in bed**. (vi) **Pallor is absent, rather the face is flushed**. (vii) Pelvic examination reveals—bilateral tenderness or mass (viii) Leukocytosis.

3. *Appendicitis:* (1) Pain and tenderness settles down to the McBurney's point. (2) Muscle guard and rigidity are characteristic. (3) Pelvic findings are negative.

4. *Twisted ovarian tumor:* (1) History of presence of previous swelling. (2) Fainting attack is absent. (3) Vaginal bleeding may appear confusing the picture. (4) General condition remains unaffected. (5) Internal examination reveals a cystic mass felt separated from the uterus.

5. Ruptured chocolate cyst of the ovary.

6. *Ruptured corpus luteum*: Clinical presentation is similar to tubal rupture but pregnancy test is negative.

INTERSTITIAL PREGNANCY (FIG. 16.10)

It is the rarest variety of tubal pregnancy. Because of the thick and vascular musculature of the uterine wall with greater distensibility, the fetus grows dissecting the muscle fibers for a longer period (12-14 weeks) before termination occurs. The usual termination is rupture. **It is associated with massive intraperitoneal hemorrhage due to its combined vascularization by the uterine and ovarian arteries**. On rare occasion, abortion occurs through the uterine cavity.

The diagnosis before rupture is very difficult. Asymmetrical enlargement of the uterus especially detected during active contraction is a conspicuous finding. It is usually confused with lateral flexion of a gravid uterus, pregnancy associated with fibroid or pregnancy in bicornuate uterus or with angular pregnancy. β-hCG, high-resolution sonography and laparoscopy can lead to early diagnosis. However, the diagnosis is revealed on laparotomy following termination as rupture. **Hysterectomy is commonly done**.

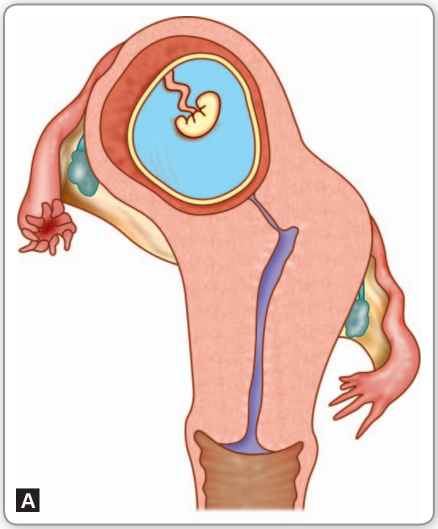


Fig. 16.10A: Interstitial pregnancy

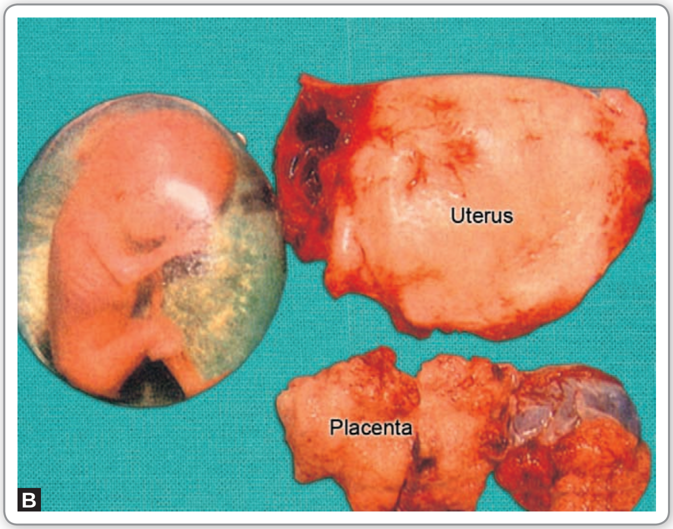


Fig. 16.10B: Ruptured interstitial pregnancy—subtotal hysterectomy done

MANAGEMENT OF ECTOPIC PREGNANCY

Over the past decade, the management of ectopic pregnancy (in uncommon locations) has evolved from a radical operative approach (salpingectomy) to a more conservative surgical or medical treatment. This has been possible due to early diagnosis, advanced laparoscopic techniques and ability to monitor the patient after conservative surgical or medical treatment. **However, the type of treatment must be individualized and depends more on clinical presentation**.

ACUTE

***Principle*: The principle in the management of acute ectopic is resuscitation and laparotomy and not resuscitation followed by laparotomy**.

***Antishock treatment*:** Antishock measures are to be taken energetically **with simultaneous preparation for urgent laparotomy**.

- Ringer's solution (crystalloid) is started, if necessary with venesection.
- Arrangement is made for blood transfusion. **Even if blood is not available, laparotomy is to be done desperately**. When the blood is available, it is better to be transfused after the clamps are placed to occlude the bleeding vessels on laparotomy, as it is of little help to transfuse when the vessels are open.
- After drawing the blood samples for grouping and cross matching, volume replacement with colloids (hemaccel) is to be done.

***Laparotomy*:** Indications of laparotomy are—(i) Patient hemodynamically unstable. (ii) Laparoscopy contraindicated. (iii) Evidence of rupture. **The principle in laparotomy is "quick in quick out"**.

Steps:

- Abdomen is opened by infraumbilical longitudinal incision.
- To grasp the uterus and draw it up under vision.
- The tubes and ovaries of both the sides are quickly inspected to find out the side of rupture.
- **Salpingectomy (Fig. 16.11)** is the definitive surgery. The excised tube should be sent for histological examination.
- **The ipsilateral ovary and its vascular supply is preserved. Oophorectomy is done only if the ovary is damaged beyond salvage or is pathological.**
- **Place of subtotal hysterectomy (Fig. 16.9B):** In interstitial pregnancy, the rupture rent is so big and the general condition is so low that, most often, a quick subtotal hysterectomy is done. However, if the condition permits and the uterine conservation is desirable, resection of the uterus may be attempted.

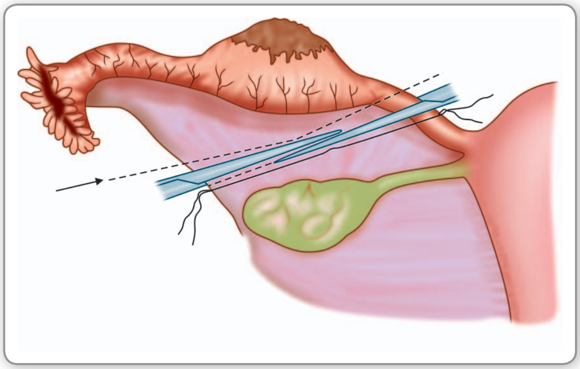


Fig. 16.11: Salpingectomy. Note the placement of the clamps.

Place of auto-transfusion: Its routine use is not advocated because of its adverse reaction. In case, where donated blood is not available, the fresh blood from the peritoneal cavity may be collected for auto-transfusion. The collection is done through strainer consisting of 4-5 layers of sterile gauze pieces into a bottle containing citrate solution (3.8%) in the proportion of five parts of blood to one part of citrate solution.

CHRONIC ECTOPIC: All cases of chronic or suspected ectopic are to be admitted as an emergency. The patient is kept under observation, investigations are done and the patient is put up for **laparotomy** at the earliest convenient time. Usually a pelvic hematocele is found. Blood clots are removed. The affected tube is identified and salpingectomy is commonly done as described previously.

Resumption of ovulation and contraception: About 15% of women ovulate by 19 days and about 25% ovulate by the 30th postoperative day. Contraception should ideally be commenced at the time of hospital discharge.

MANAGEMENT OF UNRUPTURED TUBAL PREGNANCY

- **Expectant**
- **Medical**
- **Surgical**
  - ♦ **Conservative**
  - ♦ **Ablative**

Expectant management: Where only observation is done hoping spontaneous resolution. **Indications are:** (i) Initial serum hCG level less than 1,000 IU/L and the subsequent levels are falling. (ii) Gestation sac size less than 4 cm. (iii) No fetal heart beat on TVS. (v) No evidence of bleeding or rupture on TVS.

Conservative management may be either **medical** or **surgical**. Otherwise **salpingectomy** is done.

The advantages of conservative management are: (1) Significant reduction in operative morbidity, hospital stay as well as cost. (2) Improved chance of subsequent intrauterine pregnancy (3) Less risk of recurrence.

Medical management: Number of chemotherapeutic agents have been used either systemic or direct local (under sonographic or laparoscopic guidance) as medical management of ectopic pregnancy. **The drugs commonly used** for salpingocentesis are: methotrexate, potassium chloride, prostaglandin ($PGF_{2\alpha}$), hyperosmolar glucose or actinomycin. **The patient must be**—(i) Hemodynamically stable. (ii) Serum hCG level should be less than 3,000 IU/L. (iii) Tubal diameter should be less than 4 cm without any fetal cardiac activity. (iv) There should be no intra-abdominal hemorrhage. For systemic therapy, a single dose of methotrexate (MTX) 50 mg/M^2 is given intramuscularly.

Monitoring is done by measuring serum β-hCG on D_4 and D_7. When the decline in hCG between D_4 and D_7 is greater than or equal to 15%, patient is followed up weekly with serum hCG until hCG less than 10 mIU/mL. If the decline is less than 15%, a second dose of MTX 50 mg/M^2 is given on D_7. **Variable dose methotrexate (MTX) includes**: MTX – 1 mg/kg IM on $D_{1,3,5,7}$ and Leucovorin 0.1 mg/kg IM on $D_{2,4,6,8}$. Serum β-hCG is monitored weekly until less than 5.0 mIU/mL.

Conservative Surgery: The procedure can be done either **laparoscopically or by microsurgical laparotomy**.

Indications: (a) Cases not fulfilling the criteria of medical therapy. (b) Cases where β-hCG levels are not decreasing despite medical therapy. (c) persistent fetal cardiac activity.

1. **Linear Salpingostomy**: A longitudinal incision is made on the antimesenteric border directly over the site of ectopic pregnancy. After removing the products (by fingers, scalpel handle or by suction), the incision line is kept open to be healed later on by secondary intention. Hemostasis is achieved by electrocautery or laser (Fig. 16.12).

2. **Linear Salpingotomy**: The procedures are the same as those of salpingostomy. But the incision line is closed in two layers with 7-0 interrupted vicryl sutures. This is not commonly done.

3. **Segmental Resection**: This is of choice in isthmic pregnancy. End-to-end anastomosis can be done immediately or at a later date after appropriate counseling of the patient.

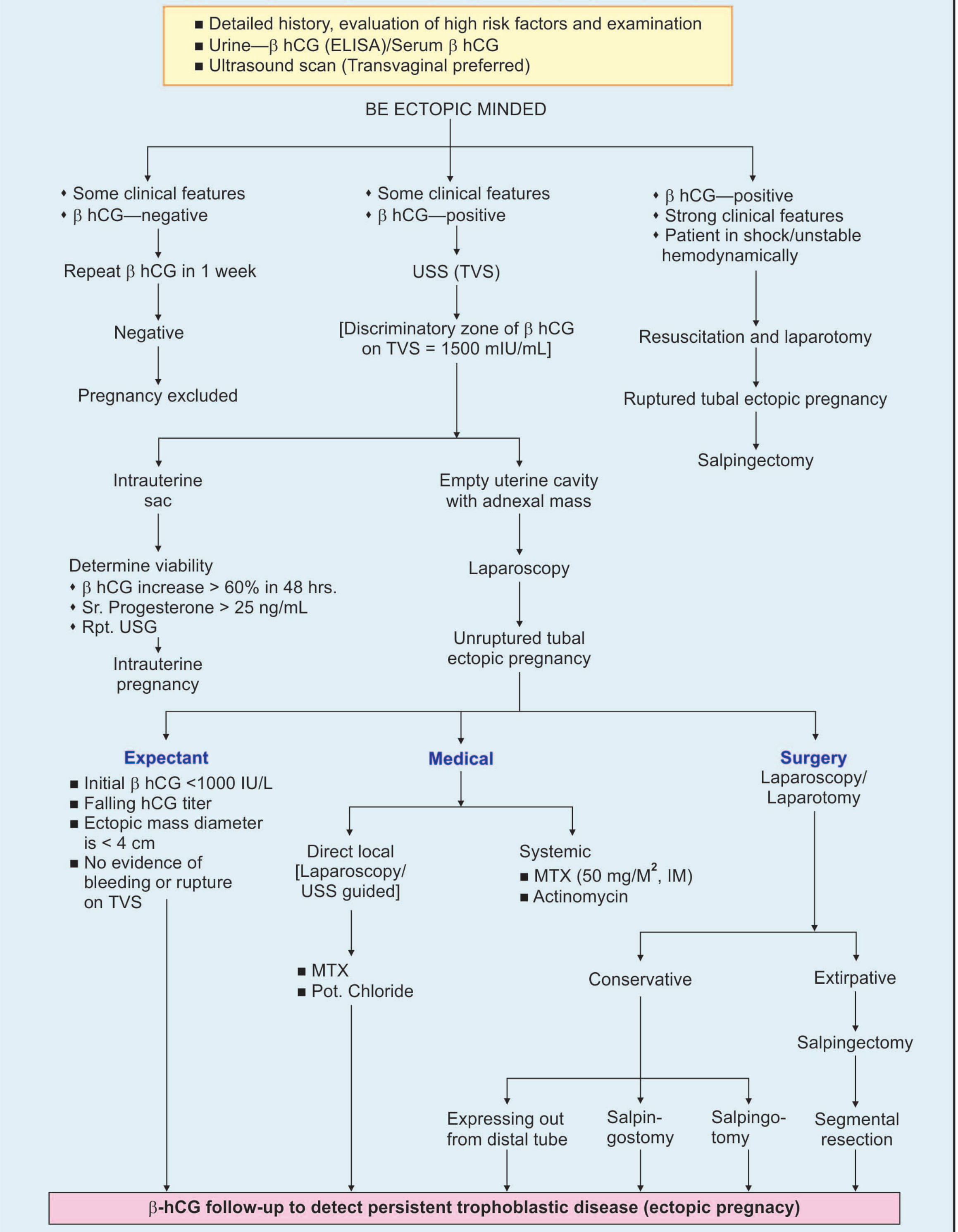
4. **Fimbrial Expression:** This is ideal in cases of distal ampullary (fimbrial) pregnancy and is done digitally.

Salpingectomy is done when (i) whole of the affected tube is damaged, (ii) contralateral tube is normal or (iii) future fertility is not desired.

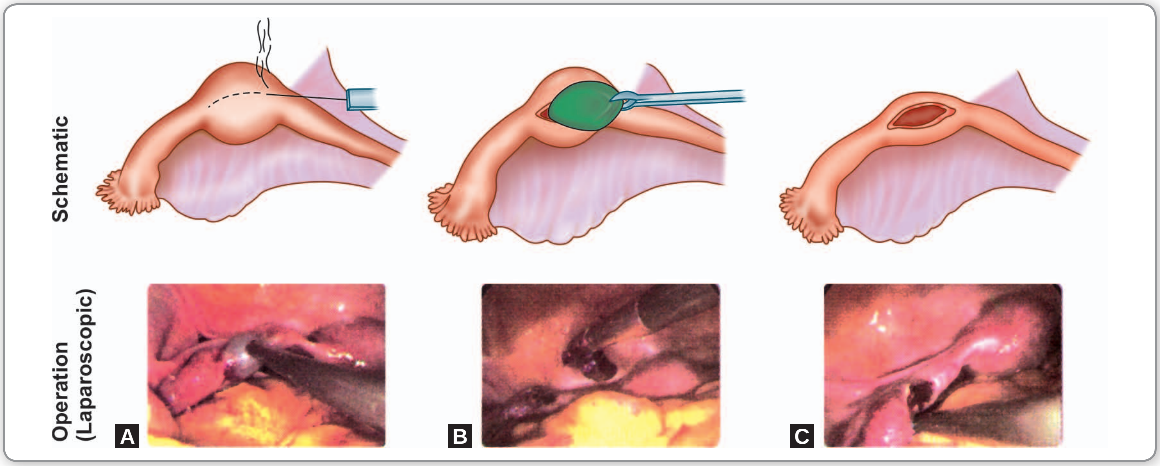
Following conservative surgery or medical treatment, estimation of β-hCG should be done weekly till the value becomes less than 5.0 mlU/mL. Additional monitoring by TVS is preferred. Following laparoscopic salpingostomy, persistent ectopic pregnancy ranges between 4% and 20%.

Persistent ectopic pregnancy is due to incomplete removal of trophoblast. It is high after fimbrial expression and in cases where initial serum β-hCG level is greater than 3,000 IU/L. Prophylactic single dose MTX (1 mg/kg) IM is effective to resolve the problem.

SCHEME OF MANAGEMENT OF TUBAL ECTOPIC PREGNANCY


■ Detailed history, evaluation of high risk factors and examination
■ Urine—β hCG (ELISA)/Serum β hCG
■ Ultrasound scan (Transvaginal preferred)
BE ECTOPIC MINDED
• Some clinical features
• β hCG—negative
Repeat β hCG in 1 week
Negative
Pregnancy excluded
• Some clinical features
• β hCG—positive
USS (TVS)
[Discriminatory zone of β hCG on TVS = 1500 mIU/mL]
• β hCG—positive
• Strong clinical features
• Patient in shock/unstable hemodynamically
Resuscitation and laparotomy
Ruptured tubal ectopic pregnancy
Salpingectomy
Intrauterine sac
Determine viability
• β hCG increase > 60% in 48 hrs.
• Sr. Progesterone > 25 ng/mL
• Rpt. USG
Intrauterine pregnancy
Empty uterine cavity with adnexal mass
Laparoscopy
Unruptured tubal ectopic pregnancy
Expectant
■ Initial β hCG <1000 IU/L
■ Falling hCG titer
■ Ectopic mass diameter is < 4 cm
■ No evidence of bleeding or rupture on TVS
Medical
Direct local [Laparoscopy/ USS guided]
■ MTX
■ Pot. Chloride
Systemic
■ MTX (50 mg/M², IM)
■ Actinomycin
Surgery
Laparoscopy/ Laparotomy
Conservative
Expressing out from distal tube
Salpingostomy
Salpingotomy
Extirpative
Salpingectomy
Segmental resection
β-hCG follow-up to detect persistent trophoblastic disease (ectopic pregnacy)

USS = Ultrasound scan TVS = Transvaginal sonography MTX = Methotrexate PGS = Prostaglandins



Figs 16.12A to C: Laparoscopic linear salpingostomy for unruptured tubal pregnancy—(A) Linear incision on the antimesenteric border (B) Gestation sac is removed (C) Incision margins left unsutured. *Above: Schematic and Below: operation.*

Rh-NEGATIVE WOMEN: In Rh-negative women not yet sensitized to Rh antigen, anti-D gamma globulin— 50 µg (if gestation < 12 weeks) or 300 µg (if > 12 weeks) intramuscularly is administered soon following operation to prevent isoimmunization.

PROGNOSIS OF TUBAL PREGNANCY: Immediate prognosis so far as maternal mortality is concerned has been markedly reduced (0.05%) due to early diagnosis, adequate blood replacement and surgery even in desperately ill patient. An ectopic mother has got every chance of a viable birth in 1 in 3 and a chance of recurrence of ectopic in 1 in 10. Patient is asked to report after she misses her period to confirm and to locate the new pregnancy.

PREVENTION OF RECURRENCE OF TUBAL PREGNANCY: Incidence of subsequent intrauterine pregnancy (IUP) is 60–70%, in women with unruptured tubal ectopic pregnancy treated by conservative surgery.

The incidence of subsequent ectopic pregnancy is about 10–20% and successful conception is about 60%.

Salpingostomy done for unruptured tubal ectopic pregnancy does not increase the risk of ectopic pregnancy compared to salpingectomy. Conservative surgery for unruptured tubal ectopic pregnancy is beneficial.

Future advice: Main concern is the risk of recurrence. Whenever there is amenorrhea, **pregnancy test** is done and if positive, **high resolution TVS** is done to know the site of pregnancy.

ABDOMINAL PREGNANCY

PRIMARY: Primary implantation of the fertilized ovum on the peritoneum is so rare that its existence is questionable. However, the **criteria laid down by Studdiford to diagnose primary abdominal pregnancy are**: (1) Both the tubes and ovaries are normal without evidence of recent injury. (2) Absence of uteroperitoneal fistula. (3) Presence of a pregnancy related exclusively to the peritoneal surface and young enough to eliminate the possibility of secondary implantation following primary nidation in the tube.

SECONDARY: Abdominal pregnancy is almost always secondary, the primary sites being tube, ovary or even the uterus—the conceptus escapes out through the rent in the uterine scar. The morbid pathological process and the fate of the pregnancy have been described previously (see p. 218). The average incidence is about 1 in 3,000 pregnancies. With the use of ART incidence is found rising.

Symptoms: (1) History suggestive of disturbed tubal pregnancy during early months (pain lower abdomen and vaginal bleeding) is often present. (2) Minor ailments of normal uterine pregnancy are often exaggerated such as nausea, vomiting, constipation, pain abdomen and increased fetal movements.

Signs in advanced pregnancy: (1) Uterine contour is not well defined even by massaging the abdominal wall, as the **Braxton-Hicks contraction is absent in abdominal pregnancy**. (2) Fetal parts are felt easily and persistent abnormal attitude and position of the fetus on repeated examination is quite common. While abnormal high position of the fetus is commonly found in intraperitoneal pregnancy, the fetus is lying low in intraligamentary pregnancy.

Internal examination: The uterus is difficult to separate from the abdominal mass. If it does, it is enlarged (12–16 weeks) but the cervix is not typically soft and is usually displaced depending upon the position of the sac.

Imaging Studies:

Sonography: It is commonly performed. Suggestive features are—absence of uterine wall around the fetus, abnormally high position of fetus with abnormal attitude, fetal parts with close approximation to maternal abdominal wall and visualization of the uterus separately. Diagnostic error could be even up to 40%.

Magnetic resonance imaging (MRI) can confirm the diagnosis and may be very accurate. *Computed tomography* is diagnostic and is superior to MRI. CT has the risk of radiation.

X-ray examination: **Straight X-ray reveals**—(a) Abnormally high position of the fetus with absence of outline of uterine shadow. (b) Superimposition of gas shadow on the fetal skeleton. (c) Lateral X-ray on standing position shows superimposition of fetal skeleton shadow with the maternal spinal shadow.

Diagnosis: Its rarity, variegated clinical pictures and not keeping in mind the possibility lead to confusion in diagnosis. **To give a positive diagnosis, the clinician should be conscious of the entity**.

Highly suggestive features: (1) Repeated failure of induction for intrauterine fetal death. (2) During induction of labor by oxytocin, uterine contraction could not be excited. Surest evidence is on laparotomy.

Management: Once the diagnosis is made, the opinion is almost crystallized in favor of **urgent laparotomy irrespective of period of gestation**. **The risks of continuation of pregnancy are:** (1) Catastrophic hemorrhage. (2) Fetal death. (3) Increased fetal malformation. (4) Increased neonatal loss (50%). Thus, continuation of pregnancy for few weeks hoping the baby to become mature enough to survive can only be justified in exceptional circumstances. The patient and her relatives should be explained about the eventuality. **During the period, the patient should be in the hospital**.

Laparotomy: The ideal surgery is to remove the entire sac-fetus, the placenta and the membranes. This may be achieved if the placenta is attached to a removable organ like uterus or broad ligament. If, however, the placenta is attached to vital organs, it is better to take out the fetus and leave behind the placenta and the sac, after tying and cutting the cord flushed with its placental attachment. In such a situation, placental activity is to be monitored by quantitative serum β-hCG level and ultrasound. Complete absorption of the left behind placenta occurs through aseptic autolysis. **Complications** include secondary hemorrhage, intestinal obstruction and infection.

Prognosis: Because of the risk mentioned before maternal mortality is less than 5% but morbidity is high. Perinatal mortality approximates 90%. Fetal malformation could be as high as 50%. Normal infants have been reported in 10% of cases.

OVARIAN PREGNANCY

Spiegelberg's criteria in diagnosis of ovarian pregnancy are—(1) Tube on the affected side must be intact. (2) The gestation sac must be in the position of the ovary. (3) The gestation sac is connected to the uterus by the ovarian ligament. (4) The ovarian tissue must be found on its wall on histological examination.

The embedding may occur intrafollicular or extrafollicular. In either types, rupture is an inevitable phenomenon and **salpingo-oophorectomy is the definite surgery**. Ovarian resection could be done when the diagnosis is made early.

CORNUAL PREGNANCY

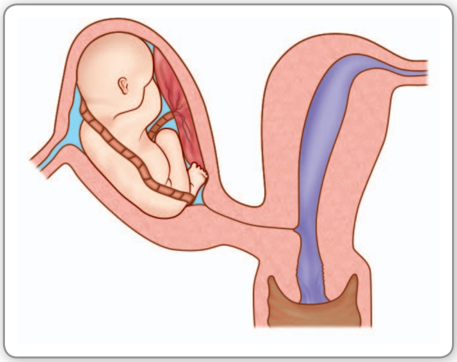


Fig. 16.13: Cornual pregnancy

Pregnancy occurring in rudimentary horn of a bicornuate uterus is called cornual pregnancy (Fig. 16.13). The horn does not usually communicate with the uterine cavity. The impregnation is presumed to occur by a spermatozoa which passes through the normal half of the uterus and tube. It then fertilizes an ovum either in the peritoneal cavity or in the tube connected to the rudimentary horn by transperitoneal migration. The concerned ovum is usually shed from the ovary on the same side of the rudimentary horn.

The general and local reactions are similar to those in the tubal pregnancy. But these are intensified and pregnancy may continue for longer time. **Termination by rupture is inevitable between 12 and 20 weeks with massive intraperitoneal hemorrhage**.

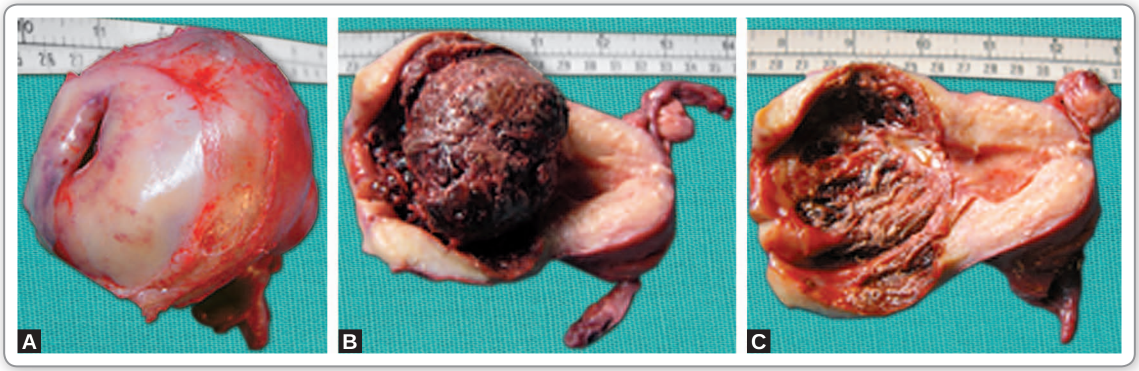
The diagnosis is seldom done before the catastrophe. **The condition is commonly diagnosed as fibroid or ovarian tumor with pregnancy**. Even on laparotomy, the exact position is confused with interstitial pregnancy. **Position of the round ligament which is attached to the sac and the long pedicle by which it is attached to the uterus are the diagnostic points.** Surgery includes removal of the rudimentary horn. If the pedicle is short and the attachment is wide, hysterectomy may have to be done.

CERVICAL PREGNANCY

This is a rare (1 in 16,000 pregnancies) variant of ectopic pregnancy **when the implantation occurs in the cervical canal at or below the internal os**. Erosion of the walls by the trophoblasts occurs resulting in thinning and distension of the canal. The condition is commonly confused with cervical abortion. **In cervical pregnancy, the bleeding is painless and the uterine body lies above the distended cervix**. Intractable bleeding following evacuation or expulsion of the products brings about suspicion. The morbidity and mortality is high because of profuse hemorrhage. **Clinical diagnostic criteria (Rubin–1983) for cervical pregnancy are**—(a) Soft, enlarged cervix equal to or larger than the fundus. (b) Uterine bleeding following amenorrhea, without cramping pain. (c) Products of conception entirely confined within and firmly attached to endocervix. (d) A closed internal cervical os and a partially opened external os (Fig. 16.13). **Sonography** reveals the pregnancy in the cervical canal and an empty uterine cavity. Hysterectomy is often required to stop bleeding. An attempt to preserve the uterus may be made by intracervical plugging. Methotrexate therapy has been considered both systemic and direct local as an alternative or adjunct to hysterectomy. Uterine artery embolization with gelfoam can control hemorrhage. **Confirmation is done by histological evidence of the presence of villi inside the cervical stroma**.

Pregnancy of unknown location: No sign of either intra- or extrauterine pregnancy or retained products of conception in a woman with a positive pregnancy test.

Pregnancy of uncertain viability: Intrauterine gestation sac (<20 mm mean diameter) with no obvious yolk sac or fetus or fetal echo less than 6 mm crown-rump length with no obvious fetal heart activity. In order to confirm or refute viability, a repeat scan at a minimal interval of 1 week is necessary.



Figs 16.14A to C: Cervical pregnancy—hysterectomy done (showing Rubin's diagnostic criteria): (A) Globular cervix with partially opened external os (B) Uterus cut opened through the anterior wall to show the huge hemorrhagic mass occupying the cervical canal with empty uterine cavity (C) When the mass was dissected, the cervical wall showed macroscopic evidence of invasion of trophoblastic tissue (confirmed on histology)

Heterotopic pregnancy: Incidence is about 1 in 8,000 pregnancies at present. It is more common following ART procedures. Intrauterine pregnancy may be coexistent with tubal or rarely with cervical or ovarian pregnancy. Diagnosis is difficult. Absence of vaginal bleeding in the presence of signs and symptoms of an ectopic pregnancy is suspicious. Abnormally rising hCG level and ultrasonography may be helpful.

KEY POINTS

ECTOPIC PREGNANCY

- **An ectopic pregnancy** is one in which the fertilized ovum is implanted and develops outside the normal endometrial cavity.
- **The different sites** of ectopic pregnancy are: tubal (most common), ovarian, abdominal, cervical and others (see p. 207).
- **The common causes** of ectopic pregnancy are: salpingitis, PID, contraception failure (IUCD), tubal ligation, ART procedures and tubal surgery.
- **Presentation** of a women with ectopic pregnancy includes : abdominal pain, amenorrhea and vaginal bleeding.
- **Diagnosis** of ectopic pregnancy is made by: positive hCG (either in urine or serum), transvaginal sonography (no intrauterine pregnancy, fluid in the pouch of Douglas and adnexal mass) and laparoscopy/laparotomy is done for confirmation.
- **Treatment** of ecotopic pregnancy could be surgical or medical (p. 215 and p. 216). Surgery could be done either by laparoscopy (common) or by laparotomy. Either salpigostomy or salpingectomy is done.
- **Ruptured tubal ectopic pregnancy** should be managed by simultaneous resuscitation and laparotomy and it is not resuscitation followed by laparotomy.
- **Unruptured tubal ectopic pregnancy** could be treated medically (see p. 216) with methotrexate.

GESTATIONAL TROPHOBLASTIC DISEASES (GTD)

DEFINITION: Gestational trophoblastic disease (GTD) encompasses a spectrum of proliferative abnormalities of trophoblasts associated with pregnancy. Persistent GTD (persistently raised β-hCG) is referred as gestational trophoblastic neoplasia (GTN).

CLASSIFICATION: Classification and terminology of GTD are extensive and at times confusing. Morphological classification of GTD is less important. Because at present **management is largely medical irrespective of histology.**

Follow-up of patients with GTD again depends on hCG than on histologic diagnosis. Immuno-histochemical and molecular studies are thought to be more important.

The conventional histological classification, includes—hydatidiform mole (complete and partial), invasive mole, choriocarcinoma and placental site trophoblastic tumor (PSTT). Modified WHO (1998) classification on GTD has been given here. Metastatic disease may be—A. Low-risk or B. High-risk group.

Non-gestational trophoblastic disease occurs as a primary choriocarcinoma of the ovary and is probably a teratomatous tumor.

Classification of GTD

- **Hydatidiform mole:** – Complete – Partial
- **Invasive mole**
- **Placental site trophoblastic tumor**
- **Choriocarcinoma**

Non metastatic disease (confined to the uterus)

Metastatic disease:

A. ***Low risk (good prognosis)***
- Disease is present < 4 months duration
- Initial serum hCG level < 40,000 mIU/mL
- Metastasis limited to lung and vagina
- No prior chemotherapy
- No preceding term delivery

B. ***High risk (poor prognosis)***
- Long duration of disease (> 4 months)
- Initial serum hCG > 40,000 mIU/mL
- Brain or liver metastasis
- Failure of prior chemotherapy
- Following term pregnancy
- WHO score > 7

HYDATIDIFORM MOLE *(Syn: Vesicular mole)*

TYPES: ♦ **Complete** ♦ **Incomplete (partial)**

The types are categorized on the basis of gross morphology, histopathology and karyotype (see table p. 230). **However, unless specified, molar pregnancy relates one with complete mole**.

DEFINITION: It is an abnormal condition of the placenta where there are partly degenerative and partly proliferative changes in the young chorionic villi. These result in the formation of clusters of small cysts of varying sizes. Because of its superficial resemblance to hydatid cyst, it is named as hydatidiform mole. **It is best regarded as a benign neoplasia of the chorion with malignant potential**.

INCIDENCE: There is wide range of geographical and ethnic variation of the prevalence of the condition. The molar pregnancy is common in Oriental countries—Philippines, China, Indonesia, Japan, India, Central and Latin America and Africa. **The highest incidence is in Philippines being 1 in 80** pregnancies and lowest in European countries 1 in 752 and USA being about 1 in 2,000. The incidence, **in India, is about 1 in 400**.

ETIOLOGY: The cause is not definitely known, but it appears to be related to the ovular defect as it sometimes affects one ovum of a twin pregnancy. However, the following factors and hypotheses have been forwarded:

— Its prevalence is highest in teenage pregnancies and in those women over 35 years of age.

— The prevalence appears to vary with race and ethnic origin.

— Faulty nutrition caused by inadequate intake of protein, animal fat could partly explain its prevalence in the Oriental Countries. Low dietary intake of carotene is associated with increased risk.

— Disturbed maternal immune mechanisms suggested by—(a) Rise in gamma globulin level in absence of hepatic disease. (b) Increased association with AB blood group which possesses no ABO antibody.

— Cytogenetic abnormality—**In general, complete moles have a 46XX karyotype (85%), the molar chromosomes are derived entirely from the father**. The ovum nucleus may be either absent (empty ovum) or inactivated which has been fertilized by a haploid sperm. It then duplicates its own chromosomes after meiosis. This phenomenon is known as **androgenesis**. Infrequently, the chromosomal pattern may be 46XY or 45X.

— The higher the ratio of paternal: maternal chromosomes, the greater is the molar change. Complete moles show 2:0 paternal/maternal ratio whereas partial mole shows 2:1 ratio.

— History of prior hydatidiform mole increases the chance of recurrence (1–4%).

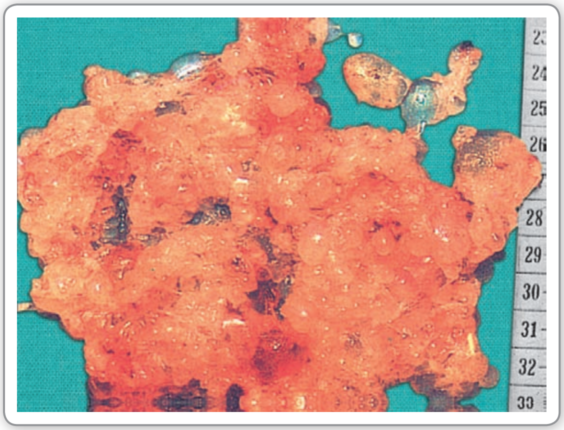


Fig. 16.15: Hydatidiform mole showing clusters of vesicles of varying sizes

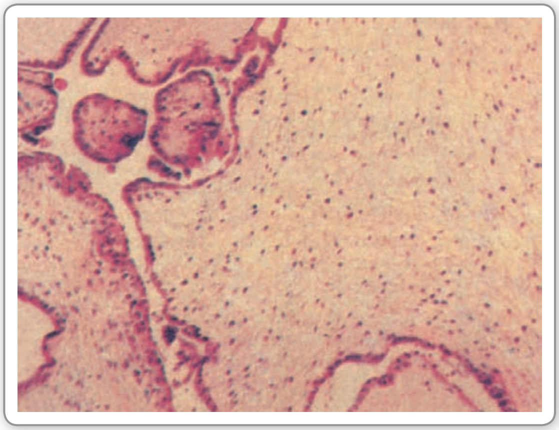


Fig. 16.16: Histological section of hydatidiform mole showing hydropic degeneration of the villous stroma with absence of blood vessels and trophoblastic proliferation (*Dr S K Dutta*)

PATHOLOGY OF HYDATIDIFORM MOLE

It is principally a disease of the chorion. Death of the ovum or failure of the embryo to grow is essential to develop complete (classic) hydatidiform mole. The secretion from the hyperplastic cells and transferred substances from the maternal blood accumulate in the stroma of the villi which are devoid of blood vessels. This results in distension of the villi to form small vesicles. The distension may also be due to edema and liquefaction of the stroma. **Vesicle fluid is interstitial fluid and is almost similar to ascitic or edema fluid, but rich in hCG**.

Naked eye appearance (Fig. 16.15): The mass filling the uterus is made of multiple chains and clusters of cysts of varying sizes. **There is no trace of embryo or the amniotic sac**. Hemorrhage, if occurs, takes place in the decidual space.

Microscopic appearance (Fig. 16.16): **The basic findings are**—(1) There is marked proliferation of the syncytial and cytotrophoblastic epithelium. (2) Marked thinning of the stromal tissue due to hydropic degeneration. (3) There is absence of blood vessels in the villi which seems primary rather than due to pressure atrophy. (4) The villous pattern is distinctly maintained.

Ovarian changes: Bilateral lutein cysts are present in about 50%. These are due to excessive production of chorionic gonadotropin and they are also observed in multiple pregnancy. These regress spontaneously within 2 months after expulsion of mole. **The contained fluid is rich in chorionic gonadotropin**. **It also contains estrogen and progesterone**.

CLINICAL FEATURES

Age and parity: It is prevalent amongst teenaged and elderly patients with high parity. The patient gives history of amenorrhea of 8–12 weeks with initial features suggestive of normal pregnancy but subsequently presents with the following manifestations (often confused with abortion).

SYMPTOMS

— **Vaginal bleeding:** Vaginal bleeding is the commonest presentation (90%). Often the symptoms mimic an incomplete or threatened abortion. The blood may be mixed with a gelatinous fluid from ruptured cysts giving the appearance of discharge "white currant in red currant juice".

- **Varying degree of lower abdominal pain may be due to**—(a) over stretching of the uterus, (b) concealed hemorrhage, (c) rarely perforation of the uterus by the invasive mole, (d) infection or (e) uterine contractions to expel out the contents.
- **Constitutional symptoms:** (a) **The patient becomes sick without any apparent reason**. (b) **Vomiting of pregnancy becomes excessive** to the stage of hyperemesis in 15% cases. It is probably related to excess chorionic gonadotropin. (c) **Breathlessness** due to pulmonary embolization of the trophoblastic cells (2%). (d) **Thyrotoxic features** of tremors or tachycardia are present on occasion (2%). It is probably due to increased chorionic thyrotropin.
- **Expulsion of grape-like vesicles per vaginam is diagnostic of vesicular mole**. Actually, in approximately 50% of cases, the mole is not suspected until it is expelled in part or whole.
- History of quickening is absent.

SIGNS

- Features suggestive of early months of pregnancy are evident.
- **The patient looks more ill** than can be accounted for.
- **Pallor** is present and may be unusually out of proportion to the visible blood loss. This may be due to concealed hemorrhage. It is mostly due to iron deficiency but may be megaloblastic due to folic acid deficiency.
- **Features of preeclampsia** (hypertension, edema and/or proteinuria) are present in about 50%. On rare occasion, convulsion may occur.

Classical Clinical Features of a Complete Mole

- Abnormal vaginal bleeding
- Lower abdominal pain
- Hyperemesis gravidarum
- Features of early onset preeclampsia < 20 weeks
- Uterus > dates (50%)
- Absent fetal parts and FHS
- Expulsion of vesicular tissues (Fig. 16.17)
- Theca lutein cyst of ovaries (25%) > 6 cm
- Hyperthyroidism (rare)
- Serum hCG > 100,000 mIU/mL
- USG: snowstorm appearance

Per abdomen:

- **The size of the uterus** is more than that expected for the period of amenorrhea in 70%, corresponds with the period of amenorrhea in 20% and smaller than the period of amenorrhea in 10%. The frequent finding of undue enlargement of the uterus is due to exuberant growth of the vesicles and the concealed hemorrhage.
- **The feel of the uterus** is firm elastic (doughy). This is due to the absence of the amniotic fluid sac.
- **Fetal parts** are not felt, nor any fetal movements. External ballottement cannot be elicited.
- **Absence of fetal heart sound** which cannot be detected even by the Doppler effect cardioscope.

The negative abdominal signs are of value when these signs should have been present depending on the size of the uterus presented in the particular case.

Vaginal examination:

- **Internal ballottement** cannot be elicited.
- **Unilateral or bilateral enlargement (theca lutein cyst) of the ovary** may be palpable in 25–50% of cases. The enlarged ovary may not be palpable due to the enlarged uterus.
- **Finding of vesicles** in the vaginal discharge is pathognomonic of hydatidiform mole.
- If the cervical os is open, instead of the membranes, blood clot or the vesicles may be felt.

Investigations:

- Full blood count, ABO and Rh grouping.
- Hepatic, renal and thyroid function tests are carried out.

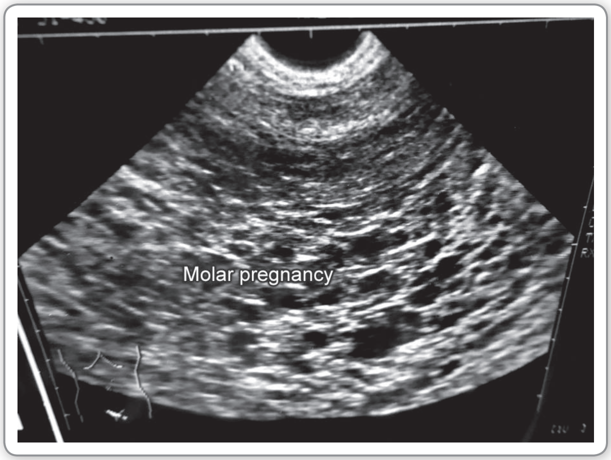


Fig. 16.17: USG showing snow storm appearance of hydatidiform mole

- **Sonography**: Characteristic feature of molar pregnancy is **"snowstorm" appearance (Fig. 16.17)**. Sometimes confusion arises with the missed abortion, partial mole or the degenerated fibroid. Doppler USG, sonography of liver, kidneys and spleen is also carried out.
- **Quantitative estimation of chorionic gonadotropin**: High hCG titer in urine (positive pregnancy test) diluted up to 1 in 200 to 1 in 500 beyond 100 days of gestation is very much suggestive. Rapidly increasing value of serum hCG (hCG > 100,000 mIU/mL), is usual with molar pregnancies. Normal pregnancy value reaches a peak at about 10–14 weeks and rarely it is more than 100,000 mIU/mL. Serum hCG value greater than 2 multiples of the median (MOM) for corresponding gestational age is of value.
- **Plain X-ray abdomen**: If the uterine size is more than 16 weeks, a negative fetal shadow may be of help. **Straight X-ray of the chest should also be carried out as a routine for evidence of pulmonary embolization even in benign mole**.
- **CT and MRI:** Routine use of computed tomography or magnetic resonance imaging for diagnosis is not recommended.
- Definitive diagnosis is made by histological examination of the products of conception (Fig. 16.16).

DIFFERENTIAL DIAGNOSIS: The following conditions are often confused with molar pregnancy: Estimation of serum hCG and ultrasonography are diagnostic.

- **Threatened abortion:** Persistence of dark-colored vaginal bleeding with mistaken date showing disproportionate increase in size of the uterus is quite confusing on clinical examination.
- **Fibroid or ovarian tumor with pregnancy:** Disproportionate enlargement of the uterus is the confusing point.
- **Multiple pregnancy**: Presence of preeclampsia in early months, disproportionate enlargement of the uterus and unusually high hCG titer in the urine are confusing features. **Twin pregnancy** with one normal fetus and placenta and the other with complete mole is differentiated from **partial mole** by cytogenetic and high resolution USG studies.

COMPLICATIONS OF MOLAR PREGNANCY

Immediate: (1) **Hemorrhage and shock—The causes of hemorrhage are:** (a) Separation of the vesicles from its attachment to the decidua. The hemorrhage may be concealed or revealed. (b) Massive intraperitoneal hemorrhage which may be the first feature of a perforating mole. (c) During evacuation of the mole due to—(i) atonic uterus or (ii) uterine injury.

(2) **Sepsis: The increased risk of sepsis is due to:** (a) As there are no protective membranes, the vaginal organisms can creep up into the uterine cavity. (b) Presence of degenerated vesicles, sloughing decidua and old blood favors nidation of bacterial growth. (c) Increased operative interference.

(3) **Perforation of the uterus: The uterus may be injured due to:** (a) Perforating mole—which may produce massive intraperitoneal hemorrhage. (b) During vaginal evacuation especially by conventional (D&E) method or during curettage following suction evacuation.

Risk Factors for Malignant Change

- **Patient's age** ≥ 40 or < 20 years irrespective of parity
- **Parity** ≥ 3. Age is more important than the parity
- **Serum hCG** > 100,000 mIU/mL
- **Uterine size** > 20 weeks
- **Previous history** of molar pregnancy
- **Theca lutein cysts:** large (>6 cm diameter)

(4) **Preeclampsia** with convulsion on rare occasion.

(5) **Acute pulmonary insufficiency due to** pulmonary embolization of the trophoblastic cells with or without villi stroma. Symptoms usually begins within 4–6 hours following evacuation.

(6) **Coagulation failure** due to pulmonary embolization of trophoblastic cells as they cause fibrin and platelets deposition within the vascular tree (see p. 711).

Late: **The development of choriocarcinoma following hydatidiform mole ranges between 2–10%. The known risk factors are recorded in the box above** which are more likely to be associated with the malignant change.

PROGNOSIS: Immediate risk from hemorrhage and sepsis are markedly diminished due to early diagnosis, blood transfusion and treatment. **About 15–20% of complete moles progress to persistent GTD** where there is a plateau or reelevation of the hCG level. In about 5% cases, metastatic disease develops. **The risk of recurrence of hydatidiform mole** in future pregnancy is about 1–4%. However, **chance of fetal malformation is not increased** following chemotherapy. **The improvement and long term prognosis may be attributed to the following factors:** (i) Recognition of high-risk factors related to choriocarcinoma. (ii) Careful follow-up with serum β-hCG. (iii) Use of cytotoxic drug at the optimum time and in the right case.

MANAGEMENT

With the use of ultrasonography and sensitive hCG testing, diagnosis is made early in majority of the cases.

The principles in the management are:

- **Suction evacuation (SE) of the uterus** as early as the diagnosis is made.
- **Supportive therapy**: Correction of anemia and infection, if there is any.
- **Counseling for regular follow-up** (see p. 231).

The patients are grouped into two:

- **Group A:** *The mole is in process of expulsion (Fig. 16.18)—less common.*
- **Group B:** *The uterus remains inert (early diagnosis with ultrasonography).*

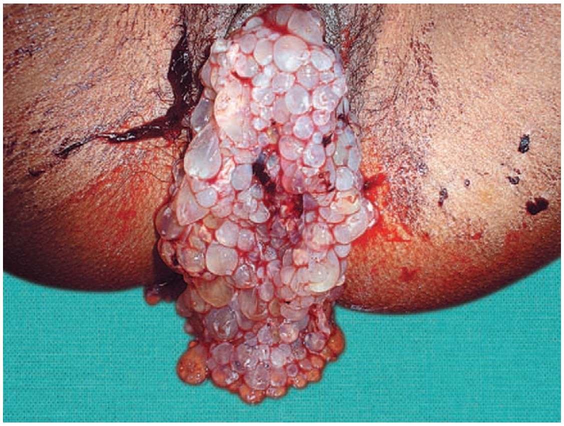


Fig. 16.18: Hydatidiform mole in the process of expulsion showing clusters of vesicles of varying sizes

By courtesy: Dr Subrata Bhattacharya, Silchar

SUPPORTIVE THERAPY: The patient usually presents with variable amount of bleeding and often they are anemic and associated with infection. (i) IV infusion with Ringer's solution is started. (ii) Blood transfusion is given if the patient is anemic. (iii) Parenteral antibiotic is given if there is associated infection. (iv) Blood is kept reserved during the evacuation as there is risk of hemorrhage.

DEFINITIVE MANAGEMENT: Suction evacuation (SE) is the method of treatment. It is safe, rapid and effective in almost all cases. Suction evacuation can safely be done even when the uterus is of 28 weeks of gestation.

- **Group A:** ***Cervix is favorable*** —(a) **The preferred method is suction evacuation**. A negative pressure is applied up to 200–250 mm Hg. The procedure can be performed under diazepam sedation or general anesthesia.

(b) Alternatively, conventional dilatation of the cervix followed by evacuation is done. During evacuation procedure patient should ideally be monitored by pulse oximeter (oxygen saturation). 500 mL Ringer's solution IV infusion is set up. Risk of hemorrhage is high especially when the uterus is large. Senior surgeon should be present during the SE procedure. Use of oxytocin helps the expulsion of moles and reduces blood loss but its routine use is not recommended due to the risks of embolization (see below).

(c) Digital exploration and removal of the mole by ovum forceps under general anesthesia may also be an alternative procedure.

After the evacuation is completed, methergine—0.2 mg is given intramuscularly.

- **Group B: Cervix is tubular and closed—**Prior slow dilatation of the cervix is done by introducing laminaria tent followed by **suction and evacuation**. Alternatively, vaginal misoprostol (PGE_1) 400 µg, 3 hours before surgery may be used.

Complications of vaginal evacuation—**Apart from the injury to the uterus, hemorrhage and shock, there are two more rare but fatal complications**—(1) **Acute pulmonary insufficiency** due to pulmonary embolization of the trophoblastic cells. Symptoms of acute chest pain, tachycardia, tachypnea and dyspnea develop about 4–6 hours following evacuation. Medical induction (oxytocin infusion) before evacuation may increase the risk of pulmonary insufficiency (RCOG). Arterial PO_2 is monitored. Patient may need ventilatory assistance and intensive care unit management. (2) **Thyroid storm**—In presence of hyperthyroid state when evacuation is done under general anesthesia, the acute features such as hyperthermia, delirium, convulsions, coma and cardiovascular collapse develop. The condition can be managed by administration of beta adrenergic blocking agents.

- **Hysterectomy** is indicated in: (i) Patients with age over 35. (ii) Patient completed her family irrespective of age. (iii) Uncontrolled hemorrhage or perforation during surgical evacuation. Hysterectomy reduces the risk of GTN by fivefold.
- **Hysterotomy** is rarely done these days. It may be done in cases with (i) profuse vaginal bleeding, (ii) cervix is unfavorable for immediate vaginal evacuation and (iii) accidental perforation of the uterus during surgical evacuation.

It should be remembered that following hysterectomy, persistent GTD is observed in 3–5% cases. As such, it does not eliminate the necessity of follow-up. The enlarged ovaries (theca lutein cysts) found during operation should be left undisturbed as they will regress following removal of mole. But, if complication arises, like torsion, rupture or infarction, they should be removed. **The uterus following hysterectomy should be sent for histopathological examination**.

Following evacuation Anti–D immunoglobulin should be given to the Rh-negative nonimmunized patient (see p. 389).

PLACE OF CURETTAGE FOLLOWING VAGINAL EVACUATION: Routine curettage is not recommended. It is done in selected cases with persistent vaginal bleeding (persistent GTN). Gentle curettage may be done **5–7 days following evacuation**. At this time the uterine wall gets thicker, firmer and the cavity becomes smaller so that effective curettage can be done without risk of damaging the uterus. The objective of curettage is to remove the necrosed decidua and the attached vesicles so as to accelerate involution and to reduce the irregular bleeding. **The materials should be sent for histology** to note the degree of trophoblastic hyperplasia and to see whether the villous structure is present or not.

FOLLOW-UP: Routine follow-up is mandatory for all cases for at least 1 year. The occurrence of choriocarcinoma is mostly confined to this period. The prime objective is to diagnose persistent trophoblastic disease (20–30%) that is considered malignant. However, hCG levels following evacuation should regress to normal within 3 months time.

***Intervals*:** Initially, the checkup should be **at an interval of one week till the serum hCG level becomes negative**. **This usually happens by 4–8 weeks**. Once negative within 56 days, the patient is followed up at every one month interval for 6 months. Women who undergo chemotherapy should be followed up for one year after hCG has been normal. The patient must not become pregnant during the period of follow-up.

Follow-up protocols: The follow-up protocols include: (i) history and clinical examination and (ii) hCG assay.

Methods employed in each visit: (1) **Enquire** about relevant symptoms like irregular vaginal bleeding, persistent cough, breathlessness or hemoptysis.

(2) **Abdominovaginal examination to note**: (i) involution of the uterus, (ii) ovarian size and (iii) malignant deposit if any, in the anterior vaginal wall. The lutein cysts usually regress within 2 months. Pelvic examination is done after one week of molar evacuation.

(3) **Investigations**: (i) *Detection of hCG in urine or serum*—Urine or serum assays are carried out at every visit. Initially, the less sensitive and less costly immunological test may be carried out until the test becomes negative. Thereafter, it is preferable to use a more sensitive serum hCG level by radioimmunoassay. (ii) *Chest X-ray:* If the pre evacuation chest radiograph shows metastasis, it should be repeated at 4 weeks interval, until remission is confirmed. It is then repeated at 3 months interval during the rest of the follow-up period. When pre-evacuation chest X-ray is normal, it is repeated only when the hCG titer plateaus or rises.

PROPHYLACTIC CHEMOTHERAPY: About 80% of patients undergo spontaneous remission. Sensitive β-hCG assay can identify the rest that develop malignancy. Moreover, the drugs used are toxic. These drugs in young females increase the risk of premature ovarian failure and menopause. So it is not appropriate to treat all patients as a routine prophylactically. However, **it is used with advantages in the following circumstances:**

(i) If the hCG level fails to become normal by the stipulated time (10–12 weeks) or there is reelevation at 4–8 weeks. (ii) Rising β-hCG level after reaching normal levels. (iii) Postevacuation hemorrhage (presence of trophoblastic activity). (iv) Where follow-up facilities are not adequate. (v) Evidences of metastases irrespective of the level of hCG. (vi) In cases, where the malignant sequelae is higher as judged by the risk factors and where proper follow-up facilities are not available. **Prophylactic chemotherapy in such cases is better than no follow-up**. Prophylactic chemotherapy is useful in high-risk group of women as it prevents metastasis and reduces morbidity.

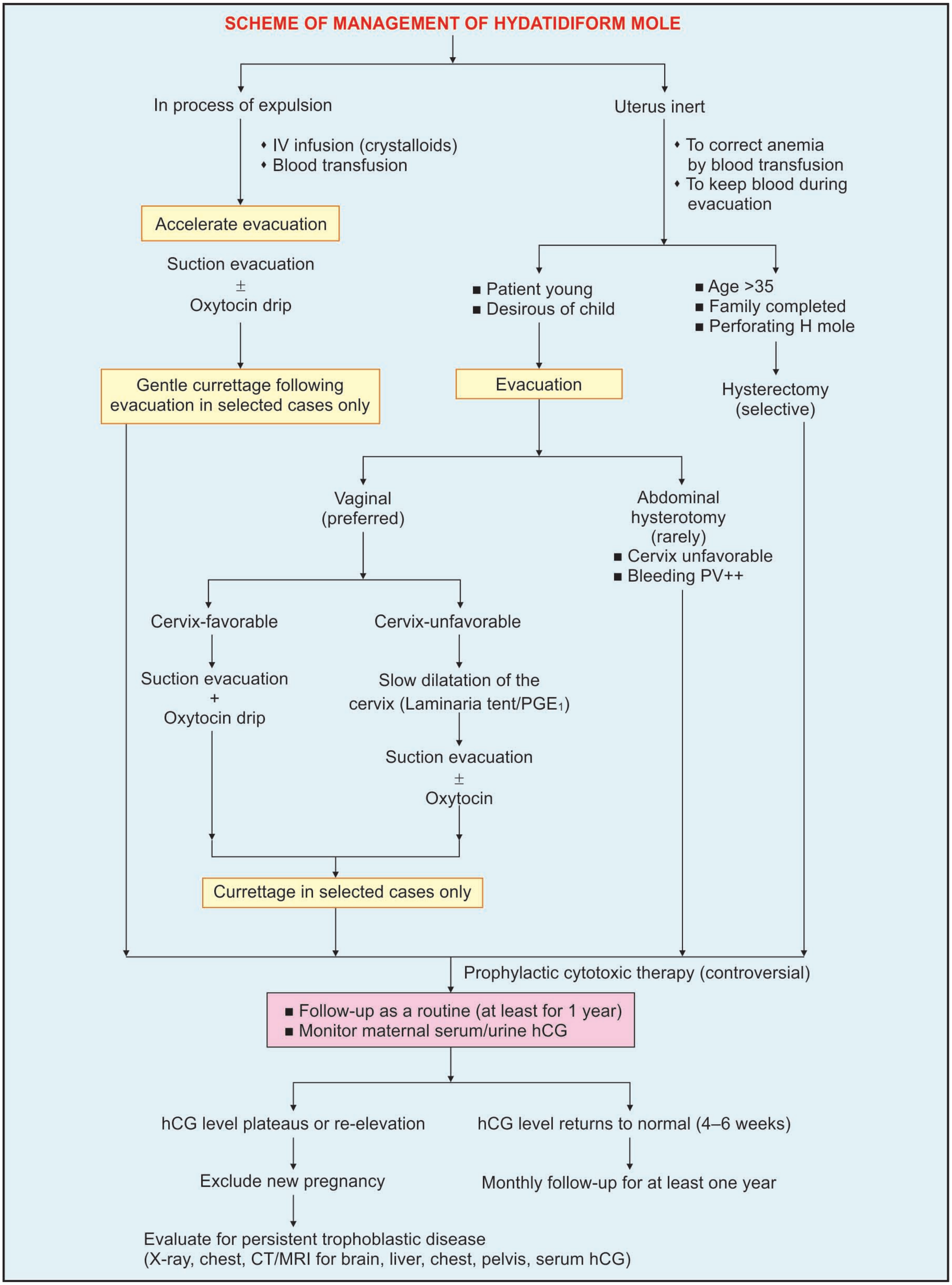
Regimes: **Methotrexate**, 1 mg/kg/day IV or IM is given on days 1, 3, 5 and 7 with folinic acid 0.1 mg/kg IM on days 2, 4, 6 and 8. It is to be repeated every 7 days. A total three courses are given. β-hCG level should decrease by at least 15%, 4–7 days after methotrexate. Alternatively, intravenous actinomycin D 12 µg/kg body weight daily for 5 days may be given. It is less toxic than methotrexate. (see author's Text book of Gynecology, for details of Chemotherapy, Chapter 23).

CONTRACEPTIVE ADVICE: The patient is traditionally advised not to be pregnant for at least one year. A rise in hCG titers might cause confusion between a fresh pregnancy or persistent GTD. However, with vaginal probe ultrasound scan, pregnancy can be diagnosed even as early as 5–6 weeks. **Thus, if the patient so desires, she may be pregnant after a minimum of 6 months, following the negative hCG titer**. But pregnancy is delayed at least up to 1 year for gestational trophoblastic neoplasia and up to 2 years if there is metastasis.

Use of contraception: IUD is contraindicated, because of its frequent association of irregular bleeding—a feature often coexists with choriocarcinoma. **Combined oral** pills can be used after the hCG value has become normal. Injection DMPA can be used safely (see p. 628). **Barrier method** of contraception can also be used. **Surgical sterilization** is another alternative when she has completed her family.

Unfavorable manifestations: (1) Persistent ill health. (2) Irregular vaginal bleeding or continuing amenorrhea. (3) Appearance of respiratory symptoms. (4) Subinvolution. (5) Appearance of secondary metastasis in the vagina. (6) **Chest radiograph showing positive finding of "cannon ball" shadow**. (7) **hCG titers remain elevated** or there is reelevation after a negative report.

hCG levels should be checked 3 weeks after the end of any pregnancy, subsequent to a molar one.


SCHEME OF MANAGEMENT OF HYDATIDIFORM MOLE
In process of expulsion
• IV infusion (crystalloids)
• Blood transfusion
Accelerate evacuation
Suction evacuation ± Oxytocin drip
Gentle currettage following evacuation in selected cases only
Uterus inert
• To correct anemia by blood transfusion
• To keep blood during evacuation
■ Patient young
■ Desirous of child
Evacuation
■ Age >35
■ Family completed
■ Perforating H mole
Hysterectomy (selective)
Vaginal (preferred)
Abdominal hysterotomy (rarely)
■ Cervix unfavorable
■ Bleeding PV++
Cervix-favorable
Suction evacuation + Oxytocin drip
Cervix-unfavorable
Slow dilatation of the cervix (Laminaria tent/PGE1)
Suction evacuation ± Oxytocin
Currettage in selected cases only
Prophylactic cytotoxic therapy (controversial)
■ Follow-up as a routine (at least for 1 year)
■ Monitor maternal serum/urine hCG
hCG level plateaus or re-elevation
Exclude new pregnancy
Evaluate for persistent trophoblastic disease (X-ray, chest, CT/MRI for brain, liver, chest, pelvis, serum hCG)
hCG level returns to normal (4–6 weeks)
Monthly follow-up for at least one year

PARTIAL OR INCOMPLETE MOLE

In partial hydatidiform mole, the affection of the chorionic villi is focal. There is a fetus or at least an amniotic sac.

The karyotype is triploid either 69XXY or 69XYY with one maternal but usually two paternal haploid chromosomes. **Microscopic examination of the dilated chorionic villi shows predominant hyperplasia of the syncytiotrophoblast and presence of fetal blood vessels with fetal red blood cells.** The fetus, if present, dies in early first trimester. Rarely, the baby may be born which is growth retarded with multisystem abnormalities (Fig. 16.19).

The clinical picture does not differ markedly from complete mole and too often confused with threatened or missed abortion (Table 16.3). The hCG titer is not markedly raised. With wider use of sonography, more and more cases are being revealed. In partial mole, uterus is generally not large for dates and malignant potential is very low.

Once the diagnosis is made and the fetus is not alive, termination of pregnancy is to be done. Even if the fetus is alive, the patient should be warned about the risks involved to the fetus if pregnancy is continued.

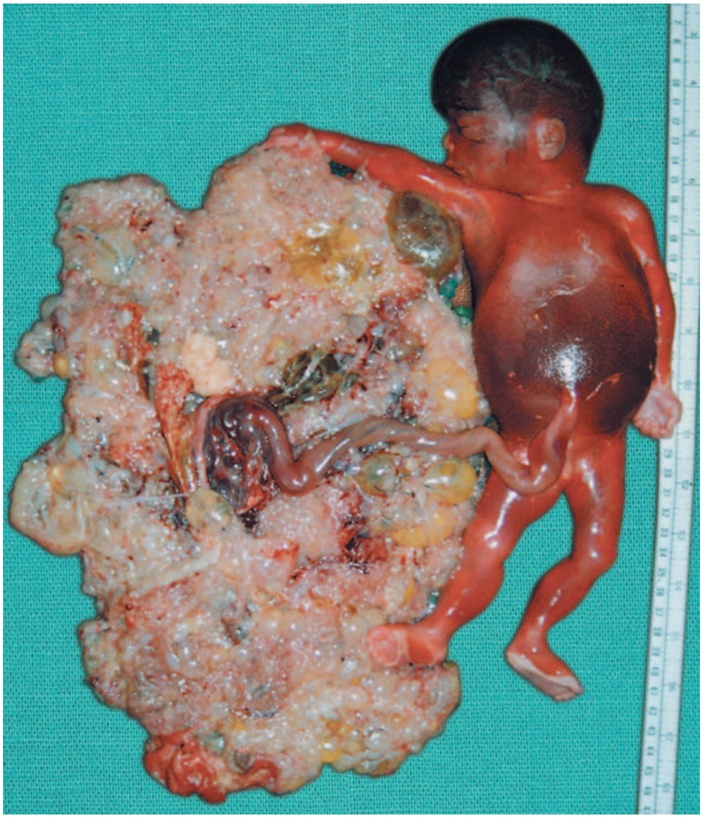
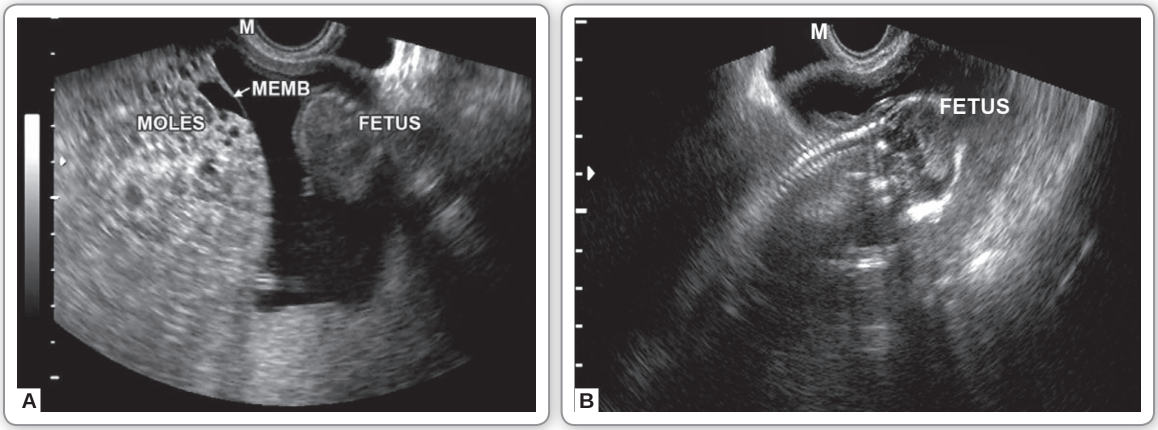


Fig. 16.19: Partial mole with a stillborn baby.
Courtesy: Dr Chandana Das, Professor, NRS Medical College, Kolkata

Post-termination follow-up protocol should be the same as outlined in complete mole. As the chance of malignancy is much less, the follow-up for 3–6 months is to be continued after hCG level returns to normal.

Table 16.3: Important Features of Complete and Partial Moles

Features	Complete Mole	Partial Mole
Embryo/Fetus	Absent	Present
Hydropic degeneration of villi	Pronounced and diffused	Variable and focal
Trophoblast hyperplasia	Diffuse	Focal
Uterine size	More than the date (30–60%)	Less than the date
Theca lutein cysts	Common (25–50%)	Uncommon
Karyotype	46, XX (85%), paternal in origin	Triploid (90%), diploid (10%)
β-hCG	High (>50,000)	Slight elevation (<50,000)
Classic clinical symptoms	Common	Rare
Risk of persistent GTN	20%	<5%
Immunostaining (p57^{KIP2})	Negative	Positive



Figs 16.20A and B: (A) USG plate showing twin pregnancy with coexistence of moles and a fetus (F); (B) Twin pregnancy with molar changes of placenta in one sac (Fig. 16.20A), the other sac with a normal fetus. *By Courtesy: Prof. BN Chakraborty; Director, Institute of Reproductive Medicine, Kolkata*

TWIN PREGNANCY – COEXISTENT MOLAR PREGNANCY AND A NORMAL FETUS: Coexistent molar pregnancy with a normal fetus is relatively rare (1 in 22,000 to 1 in 1,00,000 pregnancies). This patient (Figs 16.20 A&B), age 34-year-old lady, P-0, G-4, A-3, L-0, conceived following IVF and ET. Pregnancy ended in miscarriage at 18 weeks gestation due to the complication of excessive hemorrhage. Medical complications of such a twin pregnancy, including hyperthyroidism, PIH and hemorrhage are increased. These patients have an increased risk of developing postmolar GTN and metastatic disease.

PLACENTAL SITE TROPHOBLASTIC TUMOR (PSTT)

It is a rare histological diagnosis. Syncytiotrophoblastic cells are generally absent. So there is persistent low level of serum or urinary hCG. The tumor arises from the intermediate trophoblasts of the placental bed and is composed mainly of cytotrophoblastic cells. Patient presents with vaginal bleeding. Local invasion into the myometrium and lymphatics occurs. PSTT is not responsive to chemotherapy. Hysterectomy is the preferred treatment.

PERSISTENT GESTATIONAL TROPHOBLASTIC DISEASE

DEFINITION: Persistent GTD is defined where there is persistence of trophoblastic activity as evidenced by clinical, imaging, pathological and/or hormonal study following initial treatment. This may be following treatment of hydatidiform mole, invasive mole, choriocarcinoma or placental site trophoblastic tumor. **A postmolar GTD may be benign or malignant**. **But a GTD after nonmolar pregnancy is always a choriocarcinoma**. Overall incidence of persistent GTN after complete hydatidiform moles is 15–20%.

Approximately, 50% of the cases develop following a hydatidiform mole, 25% following an abortion or ectopic pregnancy and another 25% following normal pregnancy.

DIAGNOSIS: This state is diagnosed during postevacuation follow-up period. **The diagnostic features are:**

- Continued vaginal bleeding.
- Persistent theca lutein cysts.
- Persistently soft and enlarged uterus.
- hCG titers either fail to become negative or remain plateau or there is reelevation after a initial fall by 8 weeks postmolar evacuation. Local or systemic metastases should always be excluded (X-ray chest, CT, MRI of brain and liver). Asymptomatic patients, with a normal chest X-ray, are unlikely to have brain or other visceral metastasis.
- Pathologically this may be due to invasive mole, choriocarcinoma or placental site trophoblastic tumor.

Regardless of the histological diagnosis, the therapeutic approach is almost the same. The prognosis is usually good.

TREATMENT: Patients are classified into low- or high-risk categories (WHO Prognostic Scoring—see Author's Gyne Text p. 364). Low-risk group receives **single-agent chemotherapy** (usually methotrexate). High-risk group receives **combination chemotherapy** [usually etoposide, methotrexate, actinomycin D, cyclophosphamide and vincristine (EMA-CO)].

Hysterectomy—This is justified in women approaching 40 and/or who has completed her family. **Following hysterectomy or chemotherapy, regular follow-up is essential**.

Invasive mole, choriocarcinoma, PSTT and the rest of the gestational trophoblastic neoplasia have been described in author's Textbook of Gynecology (Chapter 23).

KEY POINTS

GESTATIONAL TROPHOBLASTIC NEOPLASIA

Hydatidiform mole:

- **Hydatidifrom mole** or molar pregnancy is a disease of the placenta where there is partly degenerative and partly neoplastic proliferation at the chrionic villi.
- **The patient commonly presents** with abnormal vaginal bleeding, pain abdomen and sometimes expulsion of grape-like vesicular tissues per vagina.
- **Confirmation of diagnosis** is made by sonography showing snowstrom appearance of the uterus and abnormally raised levels of serum hCG is the characteristics (see p. 225). Histology supports the diagnosis.

Complications of molar pregnancy are: hemorrhage, preeclampsia, sepsis, uterine perforation and coagulation failure. Risk of developing choriocarcinoma is about 2–10%.

Management: Suction and Evacuation of the Uterus (S/E) is the method of treatment. Patient may need blood transfusion.

- **Follow-up** of the patient is mandatory. It is done by frequent estimation of serum hCG till it becomes negative.
- The woman should avoid pregnancy for 1 year following treatment of molar pregnancy.

QUESTIONS

1. Enumerate the different types of abortions? How you can differentiate one from the other? (p. 185, 195)
2. Mention the different methods of termination of pregnancy in the first trimester? (p. 203)
3. Outline the management of an ectopic pregnancy? (p. 217)

Write Short Notes on:

A. Causes of recurrent miscarriage (p. 195)
B. Complications of MTP (p. 206)
C. Complications of molar pregnancy (p. 225)

Chapter 17

Multiple Pregnancy, Amniotic Fluid Disorders, Abnormalities of Placenta and Cord

When more than one fetus simultaneously develops in the uterus, it is called multiple pregnancy. Simultaneous development of two fetuses (twins) is the most common; although rare, development of three fetuses (triplets), four fetuses (quadruplets), five fetuses (quintuplets) or six fetuses (sextuplets) may also occur.

TWINS

Simultaneous development of two fetuses in the uterus is the most common variety of multiple pregnancy.

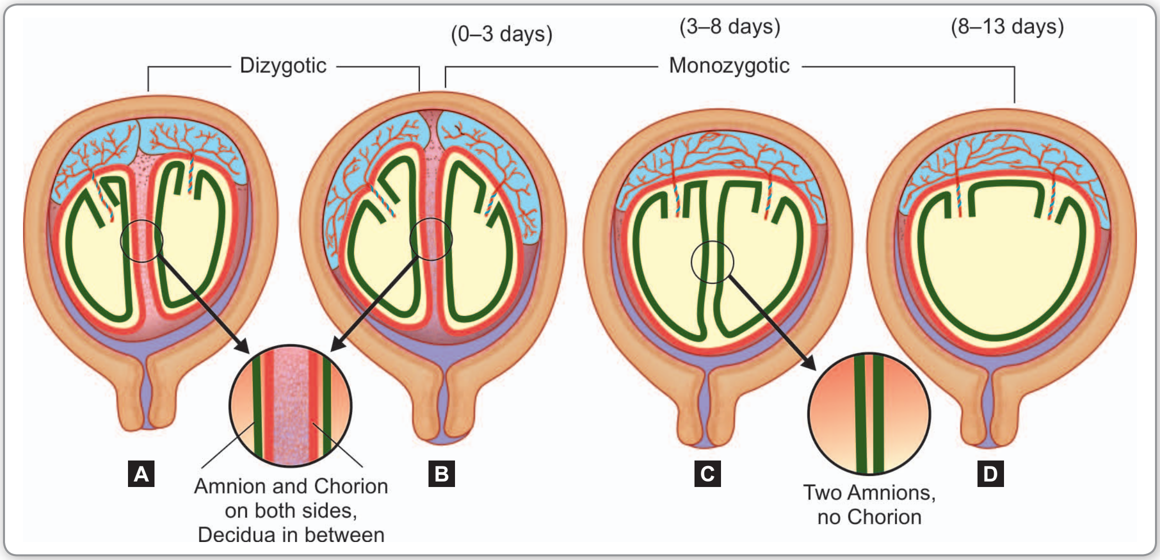
VARIETIES: (1) **Dizygotic (DZ) twins**—It is most common (80%) and results from the fertilization of two ova (2) **Monozygotic (MZ) twins** (20%) results from the fertilization of a single ovum.

GENESIS OF TWINS: ***Dizygotic twins 80%*** *(Syn: fraternal, binovular)* result from fertilization of two ova, most likely ruptured from two distinct Graafian follicles usually of the same or one from each ovary, by two sperms during a single ovarian cycle. Their subsequent implantation and development differ little from those of a single fertilized ovum. The babies bear only fraternal resemblance to each other (that of brothers and sisters from different births) and hence called fraternal twins.

In Monozygotic (MZ) twins 20% *(Syn: identical, uniovular)*, the twinning may occur at different periods after fertilization and this markedly influences the process of implantation and the formation of the fetal membranes.

On rare occasion, the following possibilities may occur:

- *If the division takes place within 72 hours* after fertilization (prior to morula stage) the resulting embryos will have two separate placenta, chorions and amnions (**diamniotic-dichorionic or D/D—30%**).
- *If the division takes place between the fourth and eighth day* after the formation of inner cell mass when chorion has already developed—**diamniotic monochorionic twins** develop (D/M—70–75%).
- *If the division occurs after eighth day of fertilization,* when the amniotic cavity has already formed, a **monoamniotic-monochorionic twin** develops (M/M—1–2%).
- On extremely rare occasions, *division occurs after 2 weeks of the development of embryonic disc resulting in the formation of* **conjoined twin (<1%) called**—Siamese twin. **Four types of fusion may occur:** (i) Thoracopagus (most common), (ii) pyopagus (posterior fusion), (iii) craniopagus (cephalic) and (iv) Ischiopagus (caudal).



Figs 17.1A to D: Twin placenta. **Dizygotic twins** (A, B) have two placentae (D/D): (A) separated and (B) fused without any vascular communications; intervening membranes consist of 4 layers (D/D). **Monozygotic twins;** (B) two placentae (D/D); (C) one placenta with free internal vascular anastomosis; the intervening membranes consist of 2 layers (D/M); (D) same as (C) but without any intervening membranes (M/M)

Zygosity refers to the genetic makeup of twin pregnancy and **chorionicity** refers the placenta's membrane status. **Chorionicity** is determined by the timing of embryo division. Determination of chorionicity is essential as the obstetrical and perinatal outcome depends on it. It is diagnosed reliably by ultrasonography in the first trimester (see p. 237) by counting the number of gestation sacs and evaluating the thickness of the dividing membranes.

Diagnosis of zygosity can be made by: examining fetal genders (different genders = dizygotic), placenta (monochorionic → monozygotic) and by genetic testing.

DETERMINATION OF ZYGOSITY: With the advent of organ transplantation, the identification of the zygosity of the multiple fetuses has assumed much importance (Table 17.1).

- ***Examination of placenta and membranes*:**

Dizygotic twins: (i) There are two placentae, either completely separated or more commonly fused at the margin appearing to be one (9 out of 10). There is no anastomosis between the two fetal vessels. (ii) Each fetus is surrounded by a separate amnion and chorion. (iii) As such, **the intervening membranes consist of four layers**—amnion, chorion, chorion and amnion. In fact in early pregnancy the decidua capsularis of each sac may be identified under the microscope in between the chorionic layers (Fig. 17.1).

Monozygotic twins: (i) The placenta is single. There is varying degree of free anastomosis between the two fetal vessels. (ii) Each fetus is surrounded by a separate amniotic sac with the chorionic layer common to both (diamniotic—monochorionic). (iii) As such **the intervening membranes consist of two layers of amnion only** (Fig. 17.1). However, on rare occasions, the uniovular twins may be diamniotic-dichorionic or monoamniotic-monochorionic.

- ***Sex*:** While twins having opposite sex are almost always dizygotic and twins of the same sex are not always monozygotic but the uniovular twins are always of the same sex.
- ***If the fetuses are of the same sex and have the same genetic features*** (dominant blood group), monozygosity is likely.
- ***A test skin graft***—Acceptance of reciprocal skin graft is almost a certain proof of monozygosity.
- **DNA microprobe technique** is most definitive.
- ***Follow-up study between 2 and 4 years***—showing almost similar physical and behavioral features suggestive of monozygosity.

Table 17.1: Summary of Determination of Zygosity

Zygosity	Placenta	Communicating vessels	Intervening membranes	Sex	Genetic features (dominant blood group) DNA fingerprinting	Skin grafting (Reciprocal)	Follow-up
Monozygotic	One	Present	2 (amnions)	Always identical	Same	Acceptance	Usually identical
Dizygotic	Two (most often fused)	Absent	4 (2 amnions 2 chorions)	May differ	Differ	Rejection	Not identical

INCIDENCE: The incidence varies widely. It is highest in Nigeria being 1 in 20 and lowest in far Eastern countries being 1 in 200 pregnancies. In India, the incidence is about 1 in 80. While the incidence of monozygotic twins remains fairly constant throughout the globe being 1 in 250, it is the dizygotic twins which are responsible for the wide variation of the incidence.

According to Hellin's (1895) rules, the mathematical frequency of multiple birth is, twins 1 in 80 pregnancies, triplets 1 in 80^2, quadruplets 1 in 80^3 and so on.

The actual incidence of multiple pregnancy has increased significantly at present. This is due to early detection by ultrasound as well as increasing use of induction of ovulation and assisted reproductive techniques (ARTs).

ETIOLOGY: The cause of twinning is not known. **The frequency of monozygotic twins remains constant throughout the globe** and is probably related to maternal environmental factors. It is the wide variation in the prevalence of binovular twins which is responsible for the fluctuation in the overall incidence of twins in different populations.

Prevalence of dizygotic twins is related to:

Race: The frequency is highest amongst Negroes, lowest amongst Mongols and intermediate amongst Caucasians.

Hereditary: There is a hereditary predisposition likely to be **more transmitted through the female (maternal side).**

Advancing age of the mother: Increased incidence of twinning is observed with the advancing age of the mother, the **maximum being between the age of 30 and 35 years**. The incidence of twins is markedly reduced thereafter.

Influence of parity: The incidence is increased with increasing parity, especially from **fifth gravida onward**.

Iatrogenic: Drugs used for induction of ovulation may produce multiple fetuses to the extent of 20–40% following gonadotrophin therapy, although to a lesser extent (5–6%) following clomiphene citrate.

- **Superfecundation** is the fertilization of two different ova released in the same cycle, by separate acts of coitus within a short period of time.
- **Superfetation** is the fertilization of two ova released in different menstrual cycles. The nidation and development of **one fetus over another fetus** is theoretically possible until the decidual space is obliterated by 12 weeks of pregnancy.
- **Fetus papyraceous or compressus** is a state which occurs if one of the fetuses dies early. The dead fetus is flattened, mummified and compressed between the membranes of the living fetus and the uterine wall. It may occur in both varieties of twins, but is **more common in monozygotic twins** and is discovered at delivery or earlier by sonography (Fig. 17.2).

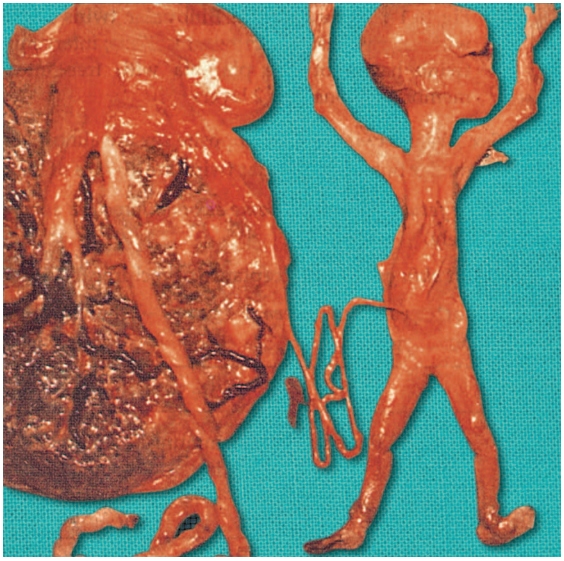


Fig. 17.2: Fetus papyraceous or compressus

- **Fetus acardiacus** occurs only in **monozygotic twins.** Part of one fetus remains amorphous and becomes parasitic without a heart (see p. 238).
- **Hydatidiform mole (from one placenta)** and a normal fetus and placenta (from the other conceptus) have been observed ultrasonographically.
- **Vanishing twin:** Serial ultrasound imaging in multiple pregnancy since early gestation has revealed occasional death of one fetus and continuation of pregnancy with the surviving one. The dead fetus (if within 14 weeks) simply "vanishes" by resorption. The rate of disappearance could be to the extent of 40%.

MATERNAL PHYSIOLOGICAL CHANGES: Multiple pregnancy imposes physical changes on the mother in excess of those seen in singleton pregnancy. (1) There is increase in weight gain and cardiac output. (2) Plasma volume is increased by an addition of 500 mL. There is no corresponding increase in red cell volume resulting in exaggerated hemodilution and anemia. (3) There is increased α-fetoprotein level, tidal volume and glomerular filtration rate.

LIE AND PRESENTATION: The most common lie of the fetuses is longitudinal (90%) but malpresentations are quite common. **The combination of presentation of the fetuses are**—(1) both vertex (50%), (2) first vertex and second breech (30%), (3) first breech and second vertex (10%), (4) both breech (10%), (5) first vertex and second transverse and so on, **but rarest one, being both transverse when the possibility of conjoined twins should be ruled out.**

DIAGNOSIS

HISTORY: (i) History of **ovulation inducing drugs** specially gonadotrophins, for infertility or use of ART. (ii) Family history of twinning (more often present in the maternal side).

SYMPTOMS: Minor ailments of normal pregnancy are often exaggerated. Some of the symptoms are related to the undue enlargement of the uterus: (i) increased nausea and vomiting in early months, (ii) cardiorespiratory embarrassment which is evident in the later months—such as palpitation or shortness of breath, (iii) tendency of swelling of the legs, varicose veins and hemorrhoids is greater, (iv) unusual rate of abdominal enlargement and excessive fetal movements may be noticed by an experienced parous mother.

GENERAL EXAMINATION: (i) Prevalence of anemia is more than in singleton pregnancy. (ii) Unusual weight gain, not explained by preeclampsia or obesity, is an important feature. (iii) Evidence of preeclampsia (25%) is a common association.

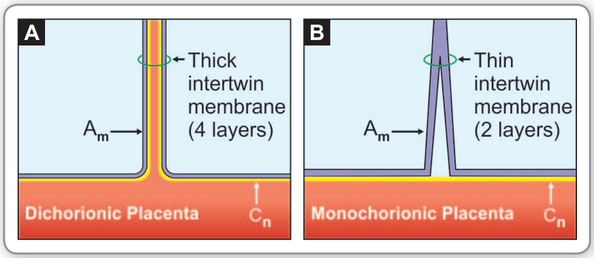

A
Thick intertwin membrane (4 layers)
A_m
Dichorionic Placenta
C_n
B
Thin intertwin membrane (2 layers)
A_m
Monochorionic Placenta
C_n

Fig. 17.3A and B: (A) **"Twin peak"** sign. In dichorionic diamniotic twin gestations, the chorion and amnion for each twin reflect away from the fused placenta to form the **intertwin membrane**. A potential space exists in the intertwin membrane, which is filled by proliferating placental villi giving rise to the **twin peak sign**. Twin peak sign appears as a triangle with the base at the chorionic surface and the apex in the intertwin membrane. (B) **In monochorionic, diamniotic twins**, the intertwin membrane is composed of two amnions only (A_m = Amnion; C_n = Chorion). (Fig. 17.1)

ABDOMINAL EXAMINATION:

Inspection: The elongated shape of a normal pregnant uterus is changed to a more "barrel shape" and the abdomen is unduly enlarged.

Palpation: (i) The height of the uterus is more than the period of amenorrhea. This discrepancy may only become evident from mid-pregnancy onward. (ii) **The girth of the abdomen** at the level of umbilicus is more than the normal average at term (100 cm). (iii) **Fetal bulk seems disproportionately larger** in relation to the size of the fetal head. (iv) **Palpation of too many fetal parts.** (v) **Finding of two fetal heads or three fetal poles** makes the clinical diagnosis almost certain.

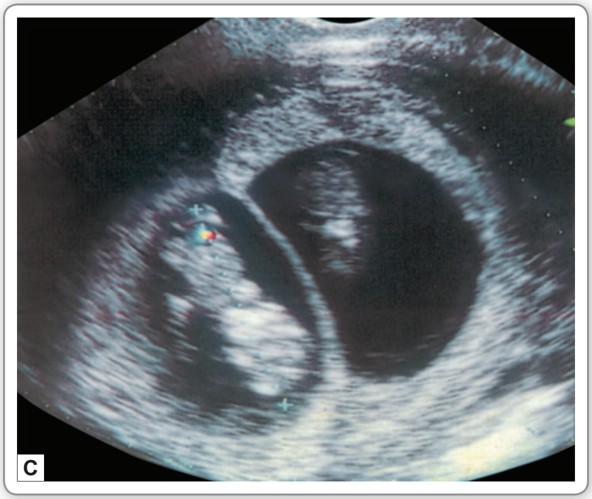


Fig. 17.3C: Color Doppler Scan (TVS) showing twin pregnancy. Thick intertwin membrane **(twin peak sign)** of dichorionic, diamniotic placenta is seen

Auscultation: Simultaneous hearing of **two distinct fetal heart sounds (FHS) located at separate spots with a silent area in between** by two observers, gives a certain clue in the diagnosis of twins, provided the difference in heart rates is at least 10 beats per minute.

The abdominal palpation and auscultation may not be carried out so easily, as described, because of the presence of hydramnios.

INTERNAL EXAMINATION: In some cases, one head is felt deep in the pelvis, while the other one is located by abdominal examination.

On occasions, the clinical methods fail to detect twins prior to the delivery of the first baby.

INVESTIGATIONS

- **Sonography**: In multifetal pregnancy it is done to obtain the following information: (i) confirmation of diagnosis as early as tenth week of pregnancy, (ii) viability of fetuses, vanishing twin in the second trimester, (iii) chorionicity **(lambda or twin peak sign—Figs 17.3A to C)**, (iv) pregnancy dating, (v) fetal anomalies, (vi) fetal growth monitoring (at every 3–4 weeks interval) for *IUGR,* (vii) presentation and lie of the fetuses, (viii) twin transfusion (Doppler studies), (ix) placental localization, (x) amniotic fluid volume.

Chorionicity of the placenta is best diagnosed by ultrasound at 10–13 weeks of gestation. In dichorionic twins there is a **thick septum** between the two gestational sacs. It is best identified at the base of the membrane, where a triangular projection is seen. This is known as **lambda or twin peak sign**. **Presence of lambda or twin peak sign indicates dichorionic placenta (Figs 17.3A to C)**.

Presence of one gestational sac with a thin dividing membrane, and two fetuses, (**"T" sign**) suggests monochorionic diamniotic pregnancy.

- **Biochemical tests:** Maternal serum chorionic gonadotrophin, α-fetoprotein and unconjugated estriol are approximately double than those of singleton pregnancies. But their values cannot diagnose clearly a twin from a single fetus.

DIFFERENTIAL DIAGNOSIS includes: (1) hydramnios, (2) big baby, (3) fibroid or ovarian tumor with pregnancy, (4) ascites with pregnancy.

COMPLICATIONS

■ **MATERNAL:** ▲ **Pregnancy** ▲ **Labor** ▲ **Puerperium** ■ **FETAL:** (p. 239)

MATERNAL — During pregnancy:

Nausea and vomiting occurs with increased frequency and severity.

- **Anemia** is more due to increased iron and folate requirement by the two fetuses. Deficiency of folic acid leads to increased incidence of **megaloblastic** anemia.
- **Preeclampsia (25%)** is increased three times over singleton pregnancy. Exposure to superabundance of chorionic villi is the possible explanation (see p. 256).
- **Hydramnios (10%) is more common in monozygotic twins and usually involves the second sac.** It is perhaps due to increased renal perfusion with consequent increased urinary output which may accompany the hypervolemia in the larger twin.

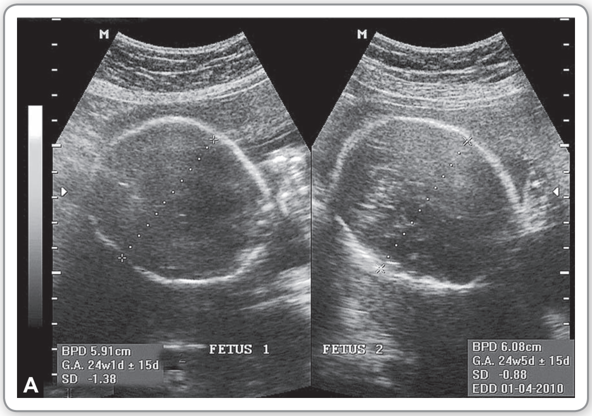


Fig. 17.4A: Ultrasonogram showing two fetal heads. BPDs are measured

- **Antepartum hemorrhage** may occur with slight increased frequency. **The increased incidence of placenta previa** is due to the bigger size of the placenta encroaching on to the lower segment. **The separation of normally situated placenta may be due to** — (i) increased incidence of preeclampsia, (ii) sudden escape of liquor following rupture of the membranes of the hydramniotic sac, (iii) deficiency of folic acid and (iv) following delivery of the first baby due to sudden shrinkage of the uterine wall adjacent to the placental attachment.
- **Malpresentation** is quite common in twins compared to singleton pregnancies. In about 70% cases, the first baby is presented by vertex and in 50%, both presented by vertex. **Malpresentation is thus more common in the second baby**. Fortunately, the babies are usually smaller and do not pose much of a problem.
- **Preterm labor (50%)** frequently occurs and the mean gestational period for twins is 37 weeks. Overdistension of the uterus, hydramnios and premature rupture of the membranes are responsible for preterm labor.
- **Mechanical distress,** such as palpitation, dyspnea, varicosities and hemorrhoids, may be increased compared to a singleton pregnancy.

During Labor

♦ **Early rupture of the membranes and cord prolapse** are likely to be increased due to increased prevalence of malpresentation. Cord prolapse is five times more common than in singleton pregnancy and is more common in relation to the second baby.

♦ **Prolonged labor** though theoretically expected, is practically not met with. This is because of parous women with smaller babies.

♦ **Increased operative interference** is due to high prevalence of malpresentation with its associated complications.

♦ **Bleeding** (intrapartum) following the birth of the first baby may at times be alarming and is due to separation of the placenta following reduction of placental site.

♦ **Postpartum hemorrhage** is the real danger in twins. **It is due to**: (i) atony of the uterine muscle due to overdistension of the uterus, (ii) a longer time taken by the big placenta to separate, (iii) bigger surface area of the placenta exposing more uterine sinuses, (iv) implantation of a part of the placenta in the lower segment which is less retractile.

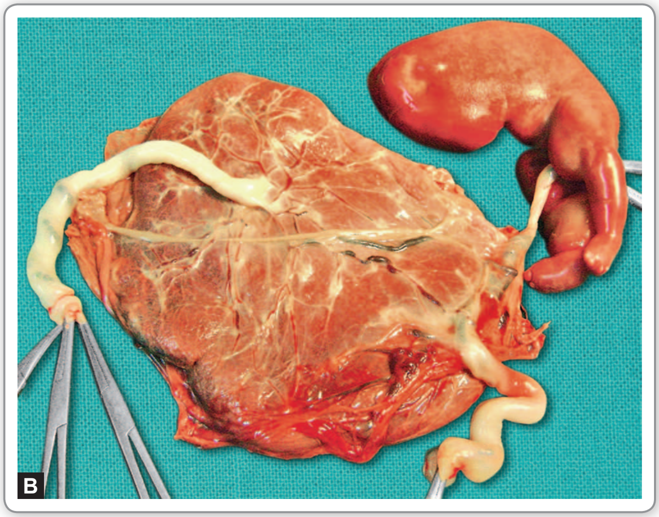


Fig. 17.4B: Acephalus, acardius fetus in a triplet pregnancy. By *courtesy*: Dr S Chaudhury, Assoc. Prof, Dept Obs-Gyn, NRSMC

During puerperium: There is increased incidence of: (1) subinvolution—because of bigger size of the uterus (2) infection because of increased operative interference, preexisting anemia and blood loss during delivery, (3) lactation failure—this is minimized by reassurance and giving her additional support.

Table 17.2: Complications of Multifetal Pregnancy

Maternal	Fetal
■ Nausea, Vomiting ■ Anemia ■ PIH and Preeclampsia ■ Polyhydramnios/ oligohydramios ■ Preterm Labor ■ Malpresentation ■ Antepartum hemorrhage ■ Mechanical distress (dyspnea, palpitation) ■ Prolonged labor ■ Operative interference ■ Postpartum hemorrhage ■ (↑) Postnatal support	■ Abortion ■ Vanishing twin/fetus papyraceous ■ Appearing twin ■ Preterm birth ■ Fetal anomalies ■ Discordant growth ■ Intrauterine death of one fetus ■ Twin transfusion syndrome ■ Cord prolapse ■ Locked twins ■ (↑) Perinatal mortality (complications are more in monozygotic twins, p. 240)

FETAL:

- **Miscarriage rate is increased** especially with **monozygotic twins**.
- **Premature rate (80%)** is very much increased and babies suffer from its hazards (see p. 529).
- **Discordant twin growth (25%)**—Some degree of **discordant growth** is normal in dizygotic twins. Cases of true pathological discordance involve estimated weight difference of 25% or more. This may be due to twin–twin transfusion syndrome, placental insufficiency, IUGR or from structural anomalies occurring in one fetus (Fig. 17.4C).
- **Intrauterine death of one fetus**—is **more in monozygotic one**. If a loss occurs in first trimester, the affected fetus simply "***Vanishes***" by resorption. If the death occurs during second trimester, a *fetus papyraceous* or *compressus* may form. If death occurs late in pregnancy, there may be death of the other fetus in presence of vascular anastomosis (see p. 240) or it may complicate the mother with DIC (rare). **The deaths are due to** cord compression, competition for nourishment or congenital malformation.
- **Appearing twin**—where diagnosis of twin pregnancy is missed on initial USG but diagnosed as twins in a later scan. This is common in monozygotic twins.
- **Fetal anomalies** are increased by 2–4% compared to a singleton pregnancy, **more in monozygotic twins**. They are in the form of anencephaly, hydrocephalus, microcephaly, cardiac anomalies or Down's syndrome.
- **Asphyxia and stillbirth** are more common due to increased prevalence of preeclampsia, malpresentation, placental abruption and increased operative interferences. The second baby is more at risk. *Complications are more in monochorionic twin pregnancies.*

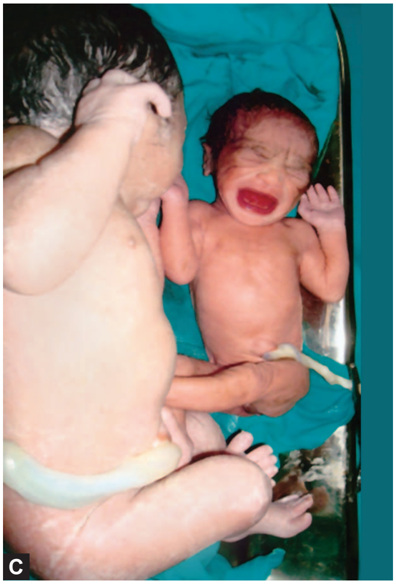


Fig. 17.4C: Discordant growth of the monochorionic twins. Weight of the fetuses were 2.6 kg (left) and 1.3 kg (right).
Courtesy: Dr Meena Jain, JLN Hospital, Bhilai, CG

PROGNOSIS

Maternal mortality is increased in twins than in a singleton pregnancy. **Death is mostly due to hemorrhage (before, during and after delivery)**, **preeclampsia and anemia**. Increased **maternal morbidity** is due to the prevalence of complications and increased operative interference.

Perinatal mortality is markedly **increased mainly due to prematurity**. It is 4–5 times higher than in a singleton pregnancy. It is **extremely high** in monoamniotic monozygotic twins due to cord entanglement. One-third loss is due to stillbirth and two-third due to neonatal death. **During delivery the second baby is more at risk (50%) than the first one due to:** (i) retraction of uterus leading to placental insufficiency, (ii) increased operative interference and (iii) increased incidence of cord prolapse.

Because of increased risk to both the mother and the baby, compared to that of a singleton pregnancy, the **twin pregnancy is considered "high risk" and as such should be delivered in a hospital**.

COMPLICATIONS OF MONOCHORIONIC TWINS

(i) **Twin-twin transfusion syndrome (TTTS)** — It is a clinicopathological state, **exclusively met with in monozygotic twins**, where one twin appears to bleed into the other through some kind of placental vascular anastomosis. Clinical manifestations of twin transfusion syndrome occur when there is hemodynamic imbalance due to unidirectional deep arteriovenous anastomoses. As a result the receptor twin becomes larger with hydramnios, polycythemic, hypertensive and hypervolemic, at the expense of the donor twin which becomes smaller with oligohydramnios, anemic, hypotensive and hypovolemic. The donor twin may appear "stuck" due to severe oligohydramnios. Difference of hemoglobin concentration between the two, usually exceeds 5 g% and estimated fetal weight discrepancy is 25% or more.

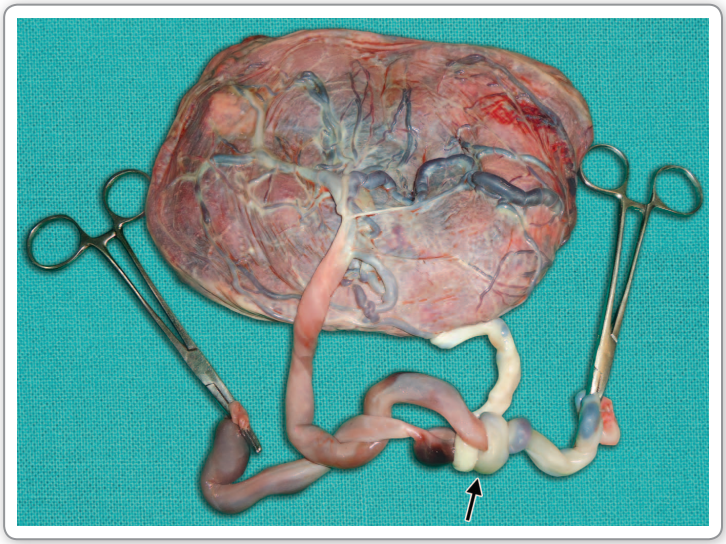


Fig. 17.5: Placenta of monochorionic-monoamniotic twins. One baby died due to cord entanglement (arrow) showing thrombosed cord

Management: Antenatal diagnosis is made by ultrasound with Doppler blood flow study in the placental vascular bed. (a) Repeated amniocentesis to control polyhydramnios in the recipient twin is done. (b) Septostomy (making a hole in the dividing amniotic membrane). (c) Laser photocoagulation to interrupt the anastomotic vessels on the chorionic plate can give some success. (d) Selective reduction (feticide) of one twin is done when survival of both the fetuses is at risk (see p. 245). The smaller twin generally has got better outcome. The plethoric twin runs the risk of congestive cardiac failure and hydrops. Congenital abnormalities (neural tube defects, holoprosencephaly) are high (2–3 times). Perinatal mortality in TTTS is about 70%. (See further, author's "Master Pass in Obstetrics and Gynecology" p. 300).

(ii) **Dead fetus syndrome**—Death of one twin (2–7%) is associated with poor outcome of the co-twin (25%) especially in monochorionic placenta. The surviving twin runs the risk of cerebral palsy, microcephaly, renal cortical necrosis and DIC. This is due to thromboplastin liberated from the dead twin that crosses via placental anastomosis to the living twin.

(iii) **Twin reversed arterial perfusion (TRAP)** is characterized by an "**acardiac perfused twin**" having blood supply from a normal co-twin via large arterio-arterial or vein to vein anastomosis (Fig. 17.4B). In majority the co-twin dies (in the perinatal period) due to high output cardiac failure. The arterial pressure of the donor twin being high, the recipient twin receives the "used" blood from the donor. The perfused twin is often chromosomally abnormal. The anomalous twin may appear as an amorphous mass. Management of TRAP is controversial. Ligation of the umbilical cord of the acardiac twin under fetoscopic guidance has been done.

(iv) **Monoamniocity (2% of all twins)** in monochorionic twins leads to high perinatal mortality due to cord problems (entanglement) (Fig. 17.5). Sulindac, a prostaglandin synthase inhibitor has been used to reduce fetal urine output, creating borderline oligohydramnios and to reduce the excessive movements.

(v) **Conjoined twin** is rare (1.3 per 100,000 births). Perinatal survival depends upon the type of joint (see p. 244 and 473). Major cardiovascular connection leads to high mortality.

ANTENATAL MANAGEMENT

The essence of **successful outcome of a twin pregnancy is to make an early diagnosis**. High index of clinical suspicion and thorough **ultrasound examination** are the keys to the diagnosis. It is useful to make early diagnosis and to detect chorionicity, amniocity, fetal growth pattern and congenital malformations.

ADVICE

- **Diet**: Increased dietary supplement is needed for increased energy supply to the extent of 300 K cal per day, over and above that is needed in a singleton pregnancy. The increased protein demand is to be met with.
- **Increased rest** at home and early cessation of work from 24 weeks onward is advised to prevent preterm labor and other complications.
- **Supplement therapy**: (i) Iron therapy is to be increased to the extent of 100–200 mg per day. (ii) Additional vitamins, calcium and folic acid (5 mg) are to be given, over and above those prescribed for a singleton pregnancy.
- **Interval of antenatal visit** should be more frequent to detect at the earliest, the evidences of anemia, preterm labor or preeclampsia.
- **Fetal surveillance** is maintained by serial sonography at every 3–4 weeks interval or earlier if needed. Assessment of fetal growth, amniotic fluid volume and AFI (see p. 43), non-stress test and Doppler velocimetry (see p. 123) are carried out.

HOSPITALIZATION

- **Routine** hospital admission only for bed rest is not essential. However, bed rest even at home from 24 weeks onward, not only ensures physical and mental rest but also improves uteroplacental circulation. This results in: (i) increased birth weight of the babies, (ii) decreased frequency of preeclampsia, (iii) prolongation of the duration of pregnancy.

To prevent preterm delivery, **routine use of betamimetics or cerclage operation has got no significant benefit**. **Use of corticosteroids to accelerate fetal lung maturation** is given (single dose) to women with preterm labor less than 34 weeks. Twins develop pulmonary maturity 3–4 weeks earlier than singletons.

- **Emergency**: Development of complicating factors necessitates urgent admission irrespective of the period of gestation.

MANAGEMENT DURING LABOR

Place of delivery: As the twin pregnancy is considered a "high risk", the patient should be confined in an equipped hospital preferably having an intensive neonatal care unit.

Vaginal delivery is allowed when both the twins are/or at least the first twin is with vertex presentation.

FIRST STAGE: Usual conduction of the first stage as outlined for a singleton fetus, is to be followed with additional precautions:

- **A skilled obstetrician should be present**. An experienced anesthetist should be made available.
- **Neonatologists (two)** should be present.
- **Presence of ultrasound in the labor ward** is helpful. It makes both the external and internal versions less difficult by visualizing the fetal parts.
- **The patient should be in bed** to prevent early rupture of the membranes.
- **Use of analgesic drugs** is to be limited as the babies are small and rapid delivery may occur. Epidural analgesia is preferred as it facilitates manipulation of second fetus, should it prove necessary.
- **Careful fetal monitoring (preferably electronic)** is to be done (see Ch. 39).
- **Internal examination should be done** soon after the rupture of the membranes to exclude cord prolapse.

- **An intravenous line with** Ringer's solution should be set up for any urgent intravenous therapy, if required.
- **One unit of compatible** and **cross matched blood** should be made readily available.
- **Neonatologist** should be present at the time of delivery.

DELIVERY OF THE FIRST BABY: The delivery should be conducted in the same guidelines as mentioned in normal labor. As the baby is usually small, the delivery does not usually pose any problem. (i) **Liberal episiotomy** under local infiltration with 1% lignocaine. (ii) **Forceps delivery**, if needed, should be done **preferably under pudendal block anesthesia**. General anesthesia is better avoided, as the second baby may be subjected to the effects of prolonged anesthesia. (iii) **Not to give intravenous ergometrine with the delivery of the first baby**. (iv) **Clamp the cord at two places and cut in between**, to prevent exsanguination of the second baby through communicating placental circulation in monozygotic twins (of course, it is an usual procedure even in singleton birth). (v) **At least, 8–10 cm of cord is left behind** for administration of any drug or transfusion, if required. (vi) The baby is handed over to the nurse after labeling it as number 1.

CONDUCTION OF LABOR AFTER THE DELIVERY OF THE FIRST BABY (DELIVERY OF SECOND TWIN)

Principles: The principle is to expedite the delivery of the second baby. The second baby is put under strain due to placental insufficiency caused by uterine retraction following the birth of the first baby.

Steps of management: Step 1: Following the birth of the first baby, **the lie, presentation, size and FHS of the second baby should be ascertained by abdominal examination or if required by real time ultrasound**. A vaginal examination is also to be made not only to confirm the abdominal findings but to note the status of the membranes and **to exclude cord prolapse**, if any.

Lie longitudinal: Step 1: Low rupture of the membranes is done after fixing the presenting part on the brim. Syntocinon may be added to the infusion bottle to achieve this. Internal examination is once more to be done to exclude cord prolapse. More vigilance is employed to watch the fetal condition.

Step 2: If the uterine contraction is poor, 5 units of oxytocin is added to the infusion bottle. The interval between deliveries should ideally be less than 30 minutes.

Step 3: If there is still a delay (say 30 minutes), interference is to be done.

- **Vertex:**
  - *Low down* — Forceps are applied.
  - *High up* — If the first baby is too small and the second one seems bigger, cephalopelvic disproportion should be ruled out. **The possibility of hydrocephalic head should be excluded by ultrasonography**. If these are excluded, internal version followed by breech extraction is performed under general anesthesia. Ventouse may be an effective alternative.
- **Breech:** The delivery should be completed by breech extraction.

Lie transverse: If the lie is transverse, **it should be corrected by external version** into a longitudinal lie preferably cephalic, if fails, podalic. **If the external version fails**, internal version under general anesthesia should be done forthwith. As the fetus is small there is no difficulty in performing internal version and **it is the only accepted indication of internal version in present day obstetric practice** (p. 665).

Indications of urgent delivery of the second baby: (1) Severe (intrapartum) vaginal bleeding, (2) Cord prolapse of the second baby, (3) Inadvertent use of intravenous ergometrine (oxytocics) with the delivery of the first baby, (4) First baby delivered under general anesthesia, (5) Appearance of fetal distress.

Management: In all these conditions, the baby should be delivered quickly. A rational scheme is given below which depends on the lie, presentation and station of the head.

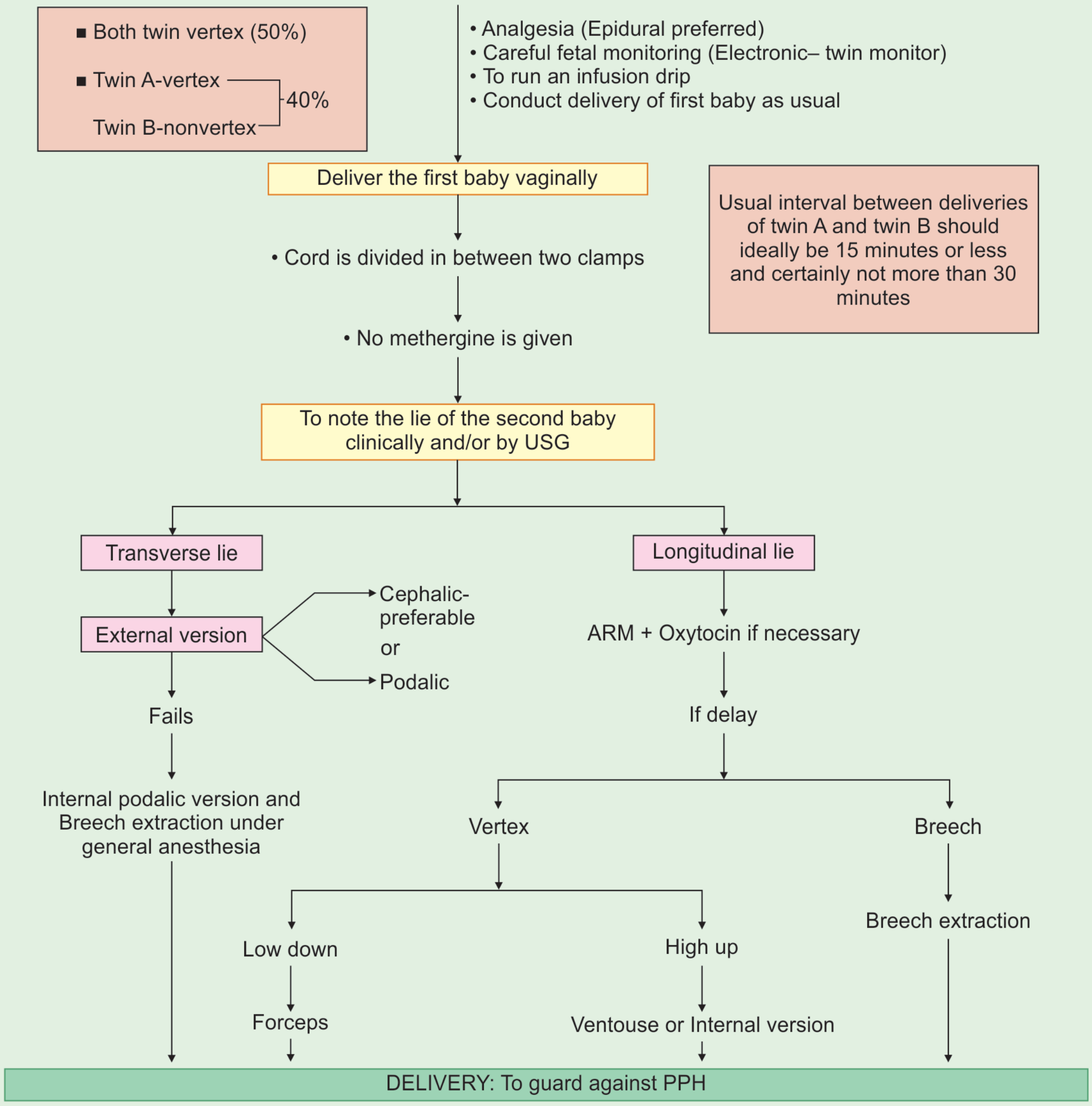
A. Head
  - If low down, delivery by forceps
  - If high up, delivery by internal version under general anesthesia

B. Breech should be delivered by breech extraction

C. Transverse lie—internal version followed by breech extraction under general anesthesia.

If, however, the patient bleeds heavily following the birth of the first baby, immediate low rupture of the membranes usually succeeds in controlling the blood loss.

SCHEME OF MANAGEMENT OF TWINS DURING LABOR


■ Both twin vertex (50%)
■ Twin A-vertex
Twin B-nonvertex
40%
• Analgesia (Epidural preferred)
• Careful fetal monitoring (Electronic– twin monitor)
• To run an infusion drip
• Conduct delivery of first baby as usual
Deliver the first baby vaginally
Usual interval between deliveries of twin A and twin B should ideally be 15 minutes or less and certainly not more than 30 minutes
• Cord is divided in between two clamps
• No methergine is given
To note the lie of the second baby clinically and/or by USG
Transverse lie
Longitudinal lie
External version
Cephalic-preferable
or
Podalic
ARM + Oxytocin if necessary
Fails
If delay
Internal podalic version and Breech extraction under general anesthesia
Vertex
Breech
Low down
High up
Breech extraction
Forceps
Ventouse or Internal version
DELIVERY: To guard against PPH

The indication of cesarean section for the second twin : (I) Larger second twin with noncephalic presentation (ii) Prompt closure of the cervix after the delivery of the first baby (iii) Fetal distress of the second twin.

Delayed delivery of the second twin has been recorded from 21 to 143 days. Delayed delivery may be associated with perinatal death and maternal infection.

MANAGEMENT OF THE THIRD STAGE: The risk of postpartum hemorrhage can be minimized by routine administration of 0.2 mg methergine IV or oxytocin 10 IU IM following the delivery of the second baby. The placenta is to be delivered by controlled cord traction. It is a sound practice to continue the oxytocin drip for at least 1 hour, following the delivery of the second baby. A blood loss of more than average should be immediately replaced by blood transfusion, already kept at hand. **The patient is to be carefully watched for about 2 hours after delivery**. Multiple births put an additional stress and strain on the mother as well as on the family members. *Mother should be given additional support at home to look after both the babies.*

INDICATIONS OF CESAREAN SECTION:

The indications are broadly divided into: • **Obstetric causes** • **For twins**

Obstetric indication: (1) Placenta previa (2) Severe preeclampsia (3) Previous cesarean section (4) Cord prolapse of the first baby (5) Abnormal uterine contractions (6) Contracted pelvis.

For twins: (i) Both the fetuses or even the first fetus with noncephalic (breech or transverse) presentation (ii) Twins with complications: IUGR, conjoined twins (iii) Monoamniotic twins (iv) Monochorionic twins with TTTS (v) Collision of both the heads at brim preventing engagement of either head.

MANAGEMENT OF DIFFICULT CASES OF TWINS

Fortunately, abnormal conditions leading to difficult delivery are extremely rare.

- **Interlocking: The most common one being the after-coming head of the first baby getting locked with the fore-coming head of the second baby.** Vaginal manipulation to separate the chins of the fetuses is done, failing which cesarean section is necessary. Decapitation of the first baby if already dead, pushing up the decapitated head, followed by delivery of the second baby and lastly, delivery of the decapitated head, at least saves one baby.
- **Occasionally, two heads of both vertex twins get locked at the pelvic brim** preventing engagement of either of the head. The possibility should be kept in mind and the diagnosis is confirmed by **intranatal sonography/ radiography**. Disengagement of the higher head can be possible under general anesthesia. If fails, cesarean section is the alternative, for fetal interest.

CONJOINED TWINS (see p. 473): It is extremely rare. Incidence varies from 1:100,000 to 1:50,000 births. In twin pregnancies the incidence is from 1:900 to 1:650 (Fig. 17.6).

Diagnosis: Unfortunately conjoined twins are often diagnosed during delivery when there is obstruction in the second stage. Failure of traction to deliver the first twin in the second stage or inability to move one twin without moving the other suggests conjoined twins. Presence of a bridge of tissue between the fetuses on vaginal examination confirms the diagnosis.

Antenatal diagnosis (see p. 473) is important. Benefits are: (i) Reduces maternal trauma and morbidity (ii) Improves fetal survival (iii) Helps to plan the method of delivery (iv) Allows time to organize the pediatric surgical team. **Management** (see p. 473) depends on (i) Extent and site of union (ii) Possibility of surgical separation and (iii) Size of the fetuses and possibility of survival.

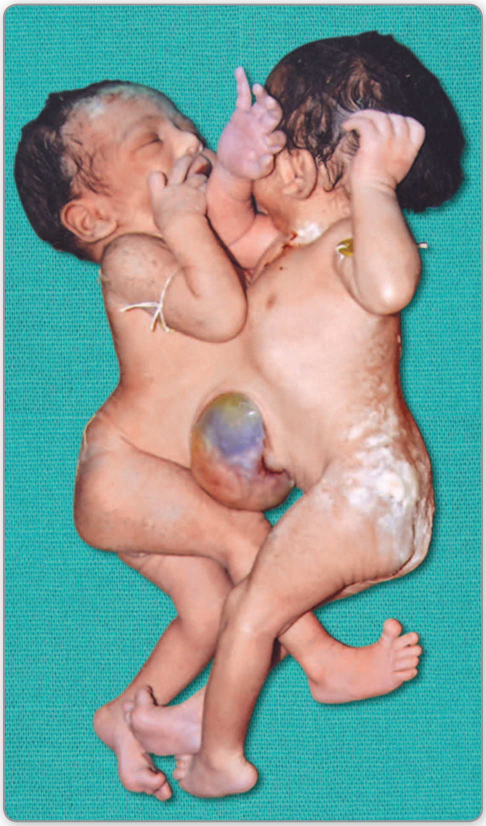


Fig. 17.6: Xiphopagus or omphalopagus fetuses are joined at the anterior abdominal wall from xiphisternum to the level of umbilicus. Congenital malformation (omphalocele) is seen

TRIPLETS, QUADRUPLETS, ETC.

Triplets may develop from fertilization of a single ovum or two or even three ova (Fig. 17.6A); similarly with quadruplets and quintuplets. **Female fetus usually outnumber the male one.** The diagnosis is accidental following sonography, or during births. Clinical course and complications are intensified compared to twins. Perinatal loss is markedly increased due to prematurity. **Preterm delivery is common (50%) and usually delivery occurs by 32–34 weeks (mean 33.5 weeks) time. Discordance of fetal growth** is more common than twins. Perinatal loss is inversely related to birth weight. Management is similar to that outlined in twins. **Average time for delivery in quadruplets is 30–31 weeks**. To improve the fetal salvage, especially in quadruplets, it is advisable to employ liberal cesarean section.

Selective reduction: If there are 4 or more fetuses, selective reduction of the fetuses leaving behind only two is done to improve outcome of the co-fetuses. This can be done by intracardiac injection of potassium chloride between 11 and 13 weeks under ultrasonic guidance. It is done transabdominally. Umbilical cord of the targeted twin is occluded by fetoscopic ligation or by laser or by bipolar coagulation, to protect the co-twin from adverse drug effect. Multiple pregnancy reduction improves perinatal outcome in women with triplets or more.

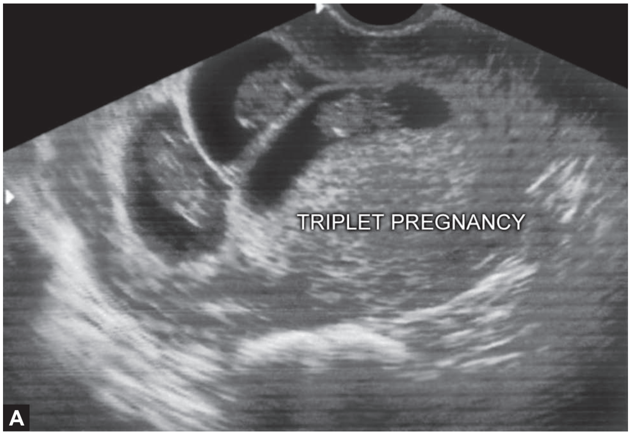


Fig. 17.6A: Ultrasonographic diagnosis of a triplet pregnancy

Selective termination of a fetus with structural or genetic abnormality may be done in a dichorionic multiple pregnancy in the second trimester by intracardiac injection of potassium chloride.

KEY POINTS

- **Twin pregnancy** is a high risk one. Maternal and perinatal morbidity and mortality are significantly high compared to a singleton pregnancy.
- **Diagnosis of chorionicity** is essential in twin pregnancy as the maternal and perinatal outcome depends on it. Chorionicity can be diagnosed by ultrasonography in the first trimester (p. 734).
- **Diagnosis of twin pregnancy** is made provisionally by history evaluation and clinical examination. Confirmation is done by ultrasonography.
- Women with a twin pregnancy should have an **ultrasound examination of 10–13 weeks** of pregnancy. This will help to detect viability, chorionicity and major malformations of the fetus.
- **Ultrasonography** in multiple pregnancy is very informative. Antenatal fetal surveillance is done by serial sonography at every 3–4 weeks interval or even earlier when needed. Sonography is useful in the intrapartum period and for selective fetal reduction and termination.
- **Complications** of twin pregnancy maternal (p. 237) and fetal (p. 239) are significant.
- **Complications of monochorionic twins** are particularly significant (p. 240).
- **Twin pregnancy needs special care** in the antenatal period (maternal nutrition) and hospital admission and supplement therapy (p. 241).
- **Routine hospital admission for bed rest is not essential** (p. 241). To prevent preterm delivery prophylactic tocolytics, cervical cerclage or progesterone supplementation is not recommend.
- **Mode of delivery** in twins depends on fetal presentation, estimated fetal weight and gestational age (p. 241).
- **Vaginal delivery** (trial of labor) following spontaneous onset of labor is often allowed when both the fetuses are in vertex (50%) and also when the first twin is vertex (40%). Cesarean delivery is decided when the first twin is non-vertex or when there is any obstetric indication (p. 242).
- **Management of third stage of labor** should be very prompt and active following delivery of the second twin. Atonic PPH is a major postpartum complication in multiple pregnancy.

AMNIOTIC FLUID DISORDERS

POLYHYDRAMNIOS *(Syn: Hydramnios)*

DEFINITION: Anatomically, polyhydramnios is defined as a state where liquor amnii exceeds 2,000 mL. **Clinical definition** states—the excessive accumulation of liquor amnii causing discomfort to the patient and/or when an imaging help is needed to substantiate the clinical diagnosis of the lie and presentation of the fetus. Sonographic diagnosis is made (see p. 535) when amniotic fluid index (AFI) is more than 24 cm (more than 95th centile for gestational age) and a deepest vertical pocket (DVP) is more than 8 cm.

INCIDENCE: Because of different criteria used in the definition of polyhydramnios, the incidence varies from 1–2% of cases. It is more common in multiparae than primigravidae. While minor degrees of hydramnios are fairly common, hydramnios sufficient to produce clinical symptoms probably occurs in 1 in 1,000 pregnancies.

ETIOLOGY

Because of wide gaps in the knowledge of the origin and excretion of the liquor amnii, the exact cause of excess accumulation of the liquor is still speculative. It may be the result of deficient absorption as well as excessive production of liquor amnii, which may be temporary or permanent. While certain maternal or fetal factors are found to be associated with hydramnios, yet the cause remains unknown in about 60%. The composition of the liquor amnii, however, remains normal.

I. FETAL ANOMALIES: Congenital fetal malformations (structural and chromosomal) are associated with polyhydramnios in about 20% cases.

- **Anencephaly**—Hydramnios is found in association with anencephaly in about 50% cases. The causes of excessive production of liquor amnii may be due to: (a) transudation from the exposed meninges, (b) absence of fetal swallowing reflex and, (c) possible suppression of fetal antidiuretic hormone leading to excessive urination.
- **Open spina bifida**—increased transudation from the meninges.
- **Esophageal or duodenal atresia**—preventing swallowing of the liquor. However, hydramnios is associated only in about 15% cases of esophageal atresia.
- **Facial clefts and neck masses**—by interfering normal swallowing.
- **Hydrops fetalis** due to Rhesus isoimmunization, nonimmune hydrops, cardiothoracic anomalies, fetal cirrhosis and fetal infections with TORCH and parvovirus B19 infection are often associated with hydramnios.
- **Aneuploidy** and genetic syndromes

II. PLACENTA: Chorioangioma of the placenta: Tumor growing from a single villus consisting of hyperplasia of blood vessels and connective tissue results in increased transudation.

III. MULTIPLE PREGNANCY: Multiple pregnancy is about 10 times more common than its overall incidence. **Hydramnios is more common in monozygotic twins, usually affecting the second sac.** In TTTS the recipient twin develops polydramnios.

IV. MATERNAL: (i) **Diabetes**—It is more common in hydramnios. Hydramnios is associated with diabetes in about 30% cases. However, with adequate supervision, the incidence of hydramnios can be lowered. It is presumed that a raised maternal blood sugar → raised fetal blood sugar → fetal diuresis → hydramnios. (ii) **Cardiac or renal disease**—may lead to edema of the placenta leading to increase in transudation.

V. IDIOPATHIC: 50–60%

CLINICAL TYPES: Depending on the rapidity of onset, **hydramnios may be**: (a) ***Chronic*** (most common)—onset is insidious taking few weeks. (b) ***Acute*** (extremely rare)—onset is sudden, within few days or may appear acutely on pre-existing chronic variety. The chronic variety is 10 times more common than the acute one.

Polyhydramnios may be—(a) mild: DVP more than 8–11 cm (b) moderate: DVP: 12–15 cm and (c) severe: DVP more than or equal to 16 cm.

CHRONIC POLYHYDRAMNIOS

In the majority of cases, the accumulation of liquor is gradual and as such, the patient is not very much inconvenienced.

SYMPTOMS: The symptoms are mainly from mechanical causes.

- **Respiratory**—The patient may suffer from dyspnea or even remain in the sitting position for easier breathing.
- **Palpitation**
- **Edema** of the legs, varicosities in the legs or vulva and hemorrhoids.

SIGNS:

- The patient may be in a dyspneic state in the lying down position.
- Evidence of preeclampsia (edema, hypertension and proteinuria) may be present.

ABDOMINAL EXAMINATION

Inspection:

- **Abdomen is markedly enlarged**, looks globular with fullness at the flanks.
- **The skin is** tense, shiny with large striae.

Palpation:

- **Height of the uterus is** more than the period of amenorrhea.
- **Girth of the abdomen** round the umbilicus is more than normal (Fig. 17.7).
- **Fluid thrill** can be elicited in all directions over the uterus.
- **Fetal parts** cannot be well-defined; so also the presentation or the position. External ballottement can be elicited more easily.

Auscultation: Fetal heart sound is not heard distinctly, although its presence can be picked up by Doppler ultrasound.

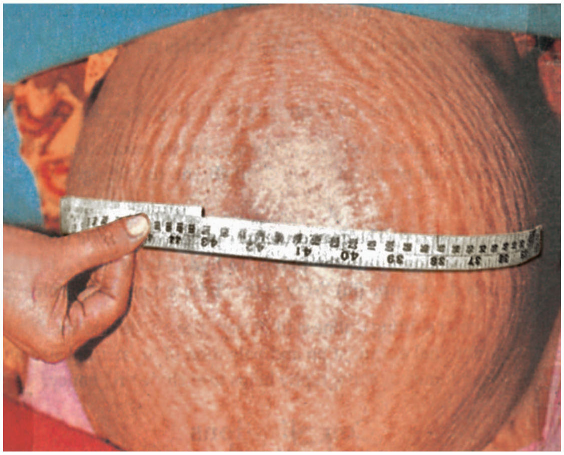


Fig. 17.7: Abdominal girth round the umbilicus is more than normal

INTERNAL EXAMINATION: The cervix is pulled up, may be partially taken up or at times, dilated, to admit a fingertip through which tense bulged membranes can be felt.

INVESTIGATIONS:

- ***Sonography:*** Sonography is helpful (Fig. 3.12)—(1) to detect abnormally large echo-free space between the fetus and the uterine wall (largest vertical pocket more than 8 cm). **Amniotic fluid index (AFI)** is more than 25 cm (p. 535), (2) to exclude multiple fetuses, (3) to note the lie and presentation of the fetus, (4) to diagnose any fetal congenital malformation. (Especially the central nervous system, gastrointestinal system and musculoskeletal system).
- ***Blood***: (1) ABO and Rh grouping — Rhesus isoimmunization may cause hydrops fetalis and fetal ascites. (2) Postprandial sugar and if necessary glucose tolerance test.
- ***Amniotic fluid:*** Estimation of alpha fetoprotein which is markedly elevated in the presence of a fetus with an open neural tube defect.

DIFFERENTIAL DIAGNOSIS: (1) **Twins** (2) **Pregnancy with huge ovarian cyst** (3) **Maternal ascites**.

1. *Twins:* The diagnosis is often confused and difficult because of its association with hydramnios. (i) Abdomen is markedly enlarged, (ii) too many fetal parts, (iii) fluid thrill absent, (iv) straight X-ray or sonography confirms the diagnosis.

2. *Pregnancy with huge ovarian cyst:* (i) The gravid uterus can be felt separate from the cyst, (ii) internal examination shows **the cervix to be pushed down into the pelvis. In hydramnios**, the lower segment has to ride above the pelvic brim, so that the **cervix is drawn up**, (iii) X-ray of the abdomen or sonography is helpful.

3. *Maternal ascites*: (i) Presence of shifting dullness, (ii) resonance on the midline due to floating gut whereas in hydramnios, it becomes dull, (iii) internal examination and palpation of the normal size uterus, if possible, can give the clue, (iv) straight X-ray of the abdomen or sonography helps to exclude pregnancy.

COMPLICATIONS

The complications of hydramnios are grouped into: • **Maternal** • **Fetal**

Maternal: *During pregnancy*—**There is increased incidence of:** (1) **Preeclampsia** (25%) (2) **Malpresentation** and persistence of floating head (3) **Premature rupture of the membranes** (4) **Preterm labor** either spontaneous or induced (5) **Accidental hemorrhage** due to decrease in the surface area of the emptying uterus beneath the placenta, following sudden escape of liquor amnii.

During labor: (1) **Early rupture** of the membranes (2) **Cord prolapse** (3) **Uterine inertia** (4) **Increased operative delivery** due to malpresentation (5) **Retained placenta**, **postpartum hemorrhage and shock**. The postpartum hemorrhage is due to uterine atony.

Puerperium: (1) **Subinvolution** (2) **Increased puerperal morbidity** due to infection resulting from increased operative interference and blood loss.

Fetal: **There is increased perinatal mortality** to the extent of about 50%. **The deaths are mostly due to** prematurity and congenital abnormality (40%). Other contributing factors are cord prolapse, hydrops fetalis, effects of increased operative delivery and accidental hemorrhage.

MANAGEMENT

Recently there has been a falling trend in the incidence of hydramnios of severe magnitude. **The reasons are:** (1) Early detection and control of diabetes. (2) Rhesus isoimmunization is now preventable. (3) Genetic counseling in early months and detection of fetal congenital abnormalities with ultrasound and their termination, reduce their number in late pregnancy.

Treatment of polyhydramnios is usually tailored according to the underlying cause.

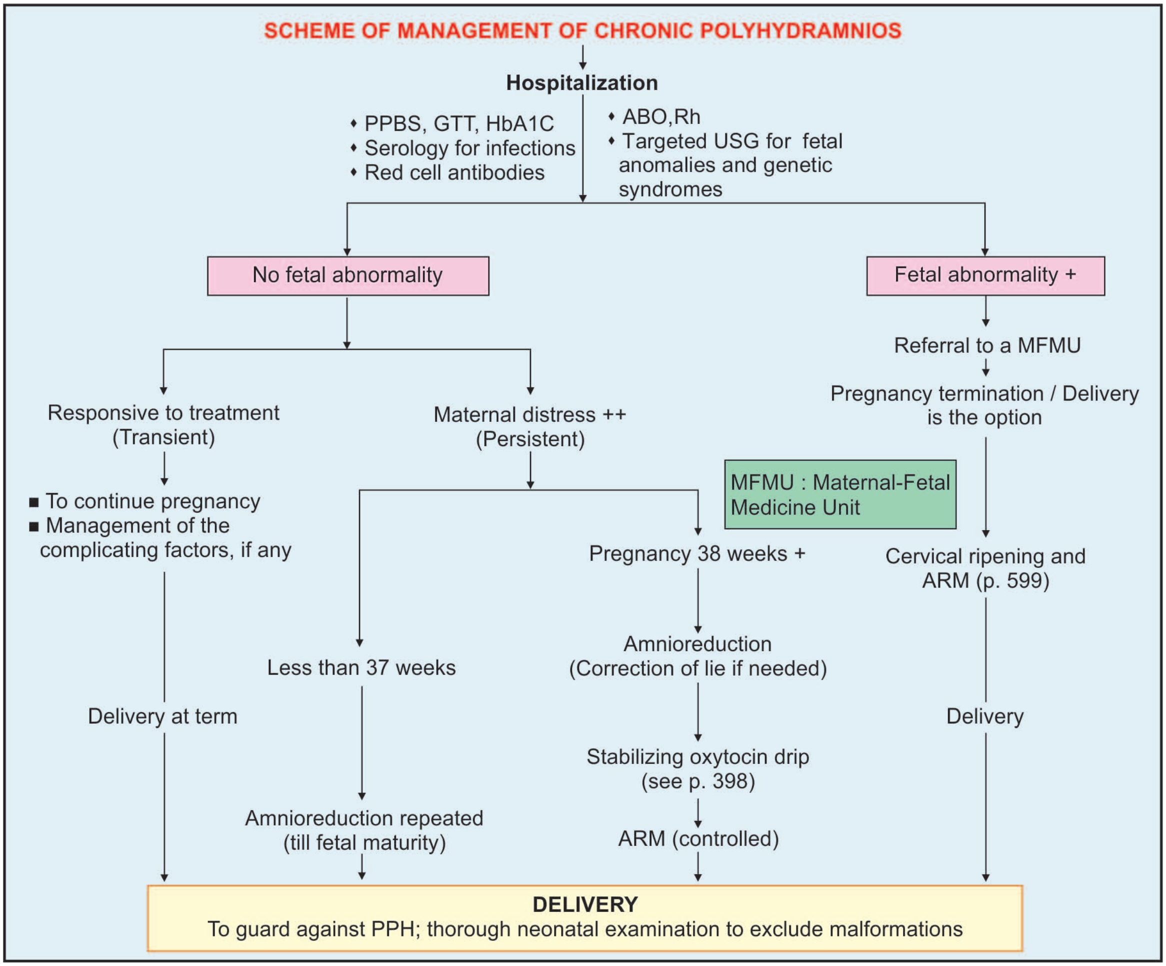
MILD POLYHYDRAMNIOS (DVP: 8–11 cm): It is commonly found in midtrimester and usually requires no treatment, except extra bed rest for a few days. The excess liquor is expected to be diminished as pregnancy advances (transient).

SEVERE POLYHYDRAMNIOS (DVP: ≥16 cm): In view of the risks involved and the high perinatal mortality rate, **the patient should be shifted in a hospital equipped to deal with "high-risk" patients**.

Principles: (1) To relieve the symptoms (2) To find out the cause (3) To avoid and to deal with the complication.

Polyhydramnios may be (a) **transient** where LVP returned to normal with progress of pregnancy or (b) **persistent** cases with persistent polyhydramnios need investigations for congenital fetal anomalies, genetic syndromes and also need close monitoring.

- **Supportive therapy** includes bed rest, if necessary, with a back rest and treatment of the associated conditions like preeclampsia or diabetes on the usual line. The use of diuretic is of little value. Sulindac (COX-2 inhibitor), 200 mg every 12 hours, (under supervision) has been found to be most effective in unexplained cases. It has been found to decrease amniotic fluid as it reduces fetal urine output.


SCHEME OF MANAGEMENT OF CHRONIC POLYHYDRAMNIOS

Hospitalization
• PPBS, GTT, HbA1C
• Serology for infections
• Red cell antibodies
• ABO,Rh
• Targeted USG for fetal anomalies and genetic syndromes
No fetal abnormality
Fetal abnormality +
Referral to a MFMU
Pregnancy termination / Delivery is the option
Responsive to treatment (Transient)
Maternal distress ++ (Persistent)
MFMU : Maternal-Fetal Medicine Unit
■ To continue pregnancy
■ Management of the complicating factors, if any
Pregnancy 38 weeks +
Cervical ripening and ARM (p. 599)
Less than 37 weeks
Amnioreduction (Correction of lie if needed)
Delivery at term
Delivery
Stabilizing oxytocin drip (see p. 398)
Amnioreduction repeated (till fetal maturity)
ARM (controlled)
DELIVERY
To guard against PPH; thorough neonatal examination to exclude malformations

- **Investigations** are done to exclude congenital fetal malformations with the available gadgets and also to detect such complications like diabetes or Rhesus isoimmunization.
- **Further management depends on:** (1) Response to treatment (2) Period of gestation (3) Presence of fetal malformation (4) Associated complicating factors.

***Uncomplicated cases:* (No demonstrable fetal malformation)**

1. *Response to treatment is good:* The pregnancy is to be continued awaiting spontaneous delivery at term.

2. *Unresponsive:* (with maternal distress).

(a) **Pregnancy less than 37 weeks:** An attempt is made to relieve the distress with a hope of continuation of pregnancy by **amniocentesis** (amnio reduction—see p. 741).

Slow decompression is done at the rate of about 500 mL per hour and the amount of fluid to be removed should be sufficient enough to relieve the mechanical distress. Normally amniodrainage is stopped when the AFI is less than 25 cm. **Because of slow decompression, chance of accidental hemorrhage is less** but liquor amnii may again accumulate, for which the procedure may have to be repeated. Amniotic fluid can be tested for fetal lung maturity.

(b) **Pregnancy more than 37 weeks:** Induction of labor is done (see p. 598). **The following procedures may be helpful**.

Amniocentesis → drainage of good amount of liquor → to check the favorable lie and presentation of the fetus → a stabilizing oxytocin infusion is started → low rupture of the membranes is done when the lie becomes stable and the presenting part gets fixed to the pelvis. **This will minimize** sudden decompression with separation of the placenta, change in the lie of the fetus and cord prolapse.

With congenital fetal abnormality: **Referral to a maternal fetal medicine unit should ideally be done. When decision for termination is made, it is to be done irrespective of duration of pregnancy.** Amniocentesis is done to drain good amount of liquor. Thereafter induction by vaginal PGE_2 gel insertion followed by low rupture of membranes is done. **If, accidentally, low rupture of the membranes occurs,** escape of gush of liquor should be immediately controlled by placing the palm over the introitus to avoid accidental hemorrhage. The lie should be checked and if found longitudinal, oxytocin infusion may be started.

DURING LABOR: **Usual management** is followed as outlined in twin pregnancy. **Internal examination** should be done soon after the rupture of the membranes to exclude cord prolapse. If the uterine contraction becomes sluggish, oxytocin infusion may be started, if not contraindicated. **To prevent postpartum hemorrhage**, intravenous methergine 0.2 mg should be given with the delivery of the anterior shoulder. **One must remain vigilant following the birth of the baby for** retained placenta, postpartum hemorrhage and shock. Baby should be thoroughly examined for any congenital anomaly.

ACUTE POLYHYDRAMNIOS

Acute hydramnios is extremely rare. The onset is acute and the fluid accumulates within a few days. **It usually occurs before 20 weeks of pregnancy.** It is usually associated with monozygotic twins with TTTS or chorioangioma of the placenta.

SYMPTOMS: Features of acute abdomen predominate—such as abdominal pain, nausea and vomiting.

SIGNS: (i) **The patient looks ill** (ii) **Absence** of features of shock (iii) **Edema of the legs** or presence of other associated features of preeclampsia (iv) **Abdomen is hugely enlarged** more than the period of amenorrhea; the wall is tense with shiny skin (v) **Fluid thrill** is present (vi) **Fetal parts** cannot be felt nor is the fetal heart sound audible (vii) **Internal examination reveals**—taking up of the cervix or even dilatation of the os through which the bulged membranes are felt (viii) **Sonography** shows multiple fetuses or at times fetal abnormalities.

TREATMENT: Most often, spontaneous abortion occurs. In case with severe TTTS, repetitive amnioreduction until the AFI is normal, may improve the perinatal outcome. Laser ablation may cure the cause of TTTS whereas amnioreduction only treats the symptoms (p. 240).

OLIGOHYDRAMNIOS

(Syn: Oligoamnios)

DEFINITION: It is an extremely rare condition **where the liquor amnii is deficient in amount to the extent of** less than 200 mL at term. **Sonographically, it is defined** when the maximum vertical pocket of liquor is less than <2 cm or when amniotic fluid index (AFI) is less than 5 cm (less than 5 percentile). With AFI less than 5 cm (below 5th percentile) or more than 24 cm (above 95th percentile) was considered abnormal at gestational age, from 28 to 40 weeks. Absence of any measurable pocket of amniotic fluid is defined as Anhydramnios. AFI between 5 and 8 is termed as borderline AFI or borderline Oligohydramnios.

ETIOLOGY

A. Fetal conditions: (i) Fetal chromosomal or structural anomalies (ii) Renal agenesis (iii) Obstructed uropathy (iv) Spontaneous rupture of the membrane (v) Intrauterine infection (vi) Drugs: PG inhibitors, ACE inhibitors (vii) Postmaturity (viii) IUGR (ix) Amnion nodosum (failure of secretion by the cells of the amnion covering the placenta).

B. Maternal conditions: (i) Hypertensive disorders (ii) Uteroplacental insufficiency (iii) Dehydration (iv) Idiopathic.

DIAGNOSIS: (1) Uterine size is much smaller than the period of amenorrhea (2) Less fetal movements (3) The uterus is "full of fetus" because of scanty liquor (4) Malpresentation (breech) is common (5) Evidences of intrauterine growth retardation of the fetus (6) **Sonographic** diagnosis is made when largest liquor pool is less than 2 cm. **Ultrasound** visualization is done following amnioinfusion of 300 mL of warm saline solution (7) Visualization of normal filling and emptying of fetal bladder essentially rules out urinary tract abnormality. (8) **Oligohydramnios with fetal symmetric growth restriction is associated with increased chromosomal abnormality** (see p. 534, 128).

COMPLICATIONS

Fetal: (1) Abortion (2) Deformity due to intra-amniotic adhesions or due to compression. The deformities include alteration in shape of the skull, wry neck, club foot, or even amputation of the limb (3) Fetal pulmonary hypoplasia (may be the cause or effect) (4) Cord compression (5) High fetal mortality.

Maternal: (1) Prolonged labor due to inertia (2) Increased operative interference due to malpresentation. The sum effect may lead to increased maternal morbidity.

TREATMENT: Presence of fetal congenital malformation needs referral to a fetal medicine unit. When decision for delivery is made, it should be done irrespective of the period of gestation. Isolated oligohydramnios in the third trimester with a normal fetus may be managed conservatively. Oral administration of water increases amniotic fluid volume. In labor, cord compression is common. Amnioinfusion (prophylactic or therapeutic) for meconium liquor is found to improve neonatal outcome.

KEY POINTS

- **Amniotic fluid volume** is related to gestational age. Amniotic fluid is dynamic and is replaced every 3 hours (p. 43).
- **Causes of polyhydramnios** (p. 246) and oligohydramnios (p. 250) are many.
- **Oligohydramnios** may be due to pregnancy complications with hypertensive disorders, placental insufficiency or idiopathic.
- **Amniotic fluid volume is estimated** clinically and thorough ultrasound measurements of AFI and DVP.
- **Polyhydramnios** (LVP > 8 cm) and **oligohydramnios** (LVP < 2 cm) is associated with significant perinatal morbidity and mortality.
- **Transient polyhydramnios** has got favorable outcome compared to persistent or worsened group. Persistent polyhydramnios needs investigations for fetal anomalies and genetic syndromes. This group need close monitoring.
- **Amniotic fluid volume (AFV) and AFI** can be increased or decreased by amount of maternal water intake.
- **Complications** of polyhydramnios are grouped into (a) Maternal and (b) Fetal (see p. 248).
- **Complications** of oligohydramnios are mainly fetal (p. 251).
- **Important management issue** in both the groups is to detect any fetal congenital malformation. Referral to a fetal medicine unit should ideally be done when it is present and delivery is planned specially when associated with major structural anomaly or genetic syndromes.

ABNORMALITIES OF PLACENTA AND CORD

There is a marked variation in the morphology including size, shape and weight of the placenta. Variation of the cord is also quite common. Only those of clinical importance are described.

PLACENTA SUCCENTURIATA (Fig. 17.8D, E & F)

Morphology: One (usual) or more small lobes of placenta, size of a cotyledon, may be placed at varying distances from the main placental margin. A leash of vessels connecting the main to the small lobe traverse through the membranes (Fig. 17.8D, E & F). **The accessory lobe is developed from the activated villi on the chorionic laeve**. In cases of absence of communicating blood vessels, it is called **placenta spuria**. The incidence of placenta succenturiata is about 3%.

Diagnosis**:** Diagnosis is made following inspection of the placenta after its expulsion. (1) **With intact lobe**—the features have already been described (2) **With missing lobe**: (a) there is a gap in the chorion and (b) torn ends of blood vessels are found on the margin of the gap.

Clinical significance**: If the succenturiate lobe is retained, following birth of the placenta, it may lead to:** (1) Postpartum hemorrhage which may be primary or secondary (2) Subinvolution (3) Uterine sepsis (4) Polyp formation.

TREATMENT: Whenever the diagnosis of missing lobe is made, exploration of the uterus and removal of the lobe under general anesthesia is to be done.

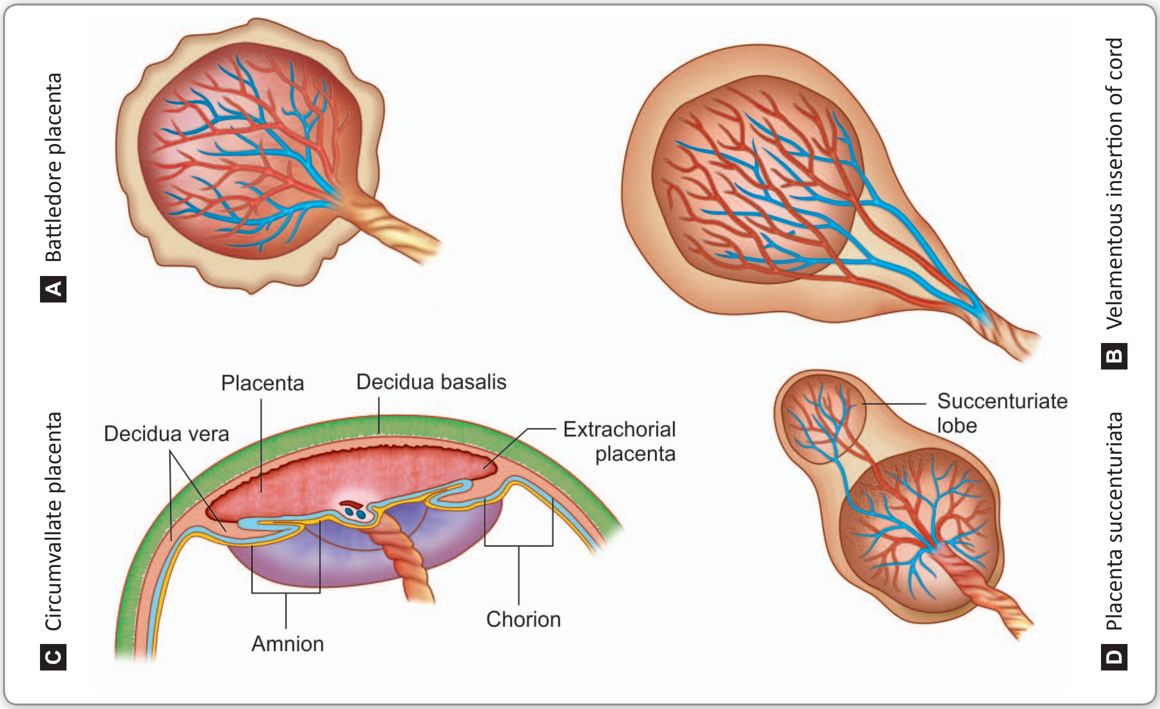
PLACENTA EXTRACHORIALIS

Two types are described: (1) **Circumvallate placenta** (2) **Placenta marginata**

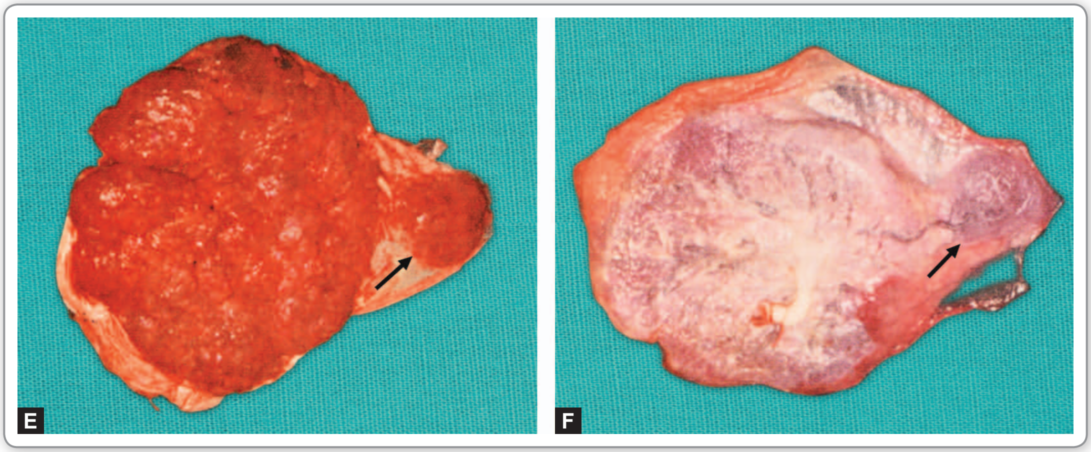
Development: The placenta of such type is **due to the smaller chorionic plate than the basal plate. Recurrent marginal hemorrhage as diagnosed on serial ultrasound is thought to be the cause**. The chorionic plate does not extend to the placental margin. The membranes (amnion and chorion) are folded, rolled back upon itself to form a ring which is reflected centrally. This leaves a rim of bare placental tissue (extrachorial portion—Fig. 17.8C).

Morphology: **Circumvallate placenta** (Fig. 17.8C)—(1) The fetal surface is divided into a central depressed zone surrounded by a thickened white ring which is usually complete. The ring is situated at varying distances from the margin of the placenta. **The ring is composed of** a double fold of amnion and chorion with degenerated decidua (vera) and fibrin in between (2) Vessels radiate from the cord insertion as far as the ring and then disappear from view (3) The peripheral zone outside the ring is thicker and the edge is elevated and rounded (Fig. 17.8C).

Placenta marginata—A thin fibrous ring is present at the margin of the chorionic plate where the fetal vessels appear to terminate.



Figs 17.8A to D: Common types of abnormal placentae of obstetric significance (A, B, C and D)



Figs 17.8E and F: Placenta succenturiate (D, E & F)
(D) = diagrammatic; (E) = maternal surface; (F) = fetal surface with communicating vessels (see arrow).

Clinical significance: There is increased chance of: (1) Abortion (2) Hydrorrhea gravidarum (excessive watery vaginal discharge) (3) Antepartum hemorrhage (4) Growth retardation of the baby (5) Preterm delivery (6) Retained placenta or membranes.

PLACENTA MEMBRANECEA: The placenta is unduly large and thin. The placenta not only develops from the chorion frondosum but also from the chorion laeve so that the whole of the ovum is practically covered by the placenta.

Clinical significance: (1) Encroachment of some part over the lower segment leads to placenta previa. (2) Imperfect separation in the third stage leads to postpartum hemorrhage. (3) Chance of retained placenta is more and manual removal becomes difficult.

CORD ABNORMALITIES

BATTLEDORE PLACENTA: The cord is attached to the margin of the placenta. If associated with low implantation of the placenta, there is chance of cord compression in vaginal delivery leading to fetal anoxia or even death; otherwise, it has got little clinical significance (Fig 17.8A).

VELAMENTOUS PLACENTA: The cord is attached to the membranes. The branching vessels traverse between the membranes for a varying distance before they reach and supply the placenta. If the leash of blood vessels happens to traverse through the membranes overlying the internal os, in front of the presenting part, the condition is called **vasa previa**. Rupture of the membranes involving the overlying vessels leads to vaginal bleeding. As it is entirely fetal blood, this may result in fetal exsanguination and even death (Fig.17.8B).

MANAGEMENT: In the presence of fetal bleeding, urgent delivery is essential either vaginally or by cesarean section. The infant's hemoglobin should be estimated and if necessary, blood transfusion be carried out. If the baby is dead, vaginal delivery is awaited.

ABNORMAL LENGTH: The cord may be unduly long (300 cm) or absent (acordia).

Short cord: The short cord may be true (less than 20 cm or 8") or commonly relative due to entanglement of the cord round any fetal part. In exceptional circumstances, the cord may be absent and the placenta may be attached to the liver as in exomphalos.

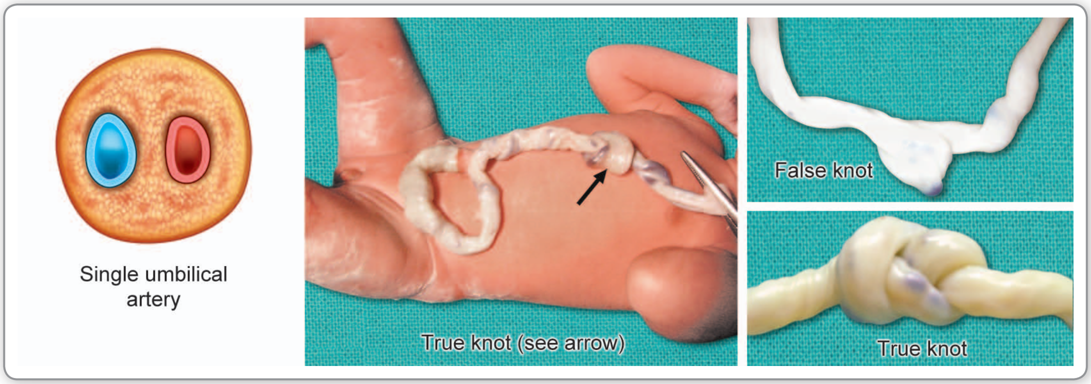


Fig. 17.9: Abnormalities in the umbilical cord

Clinical significance—**In either variety, it may cause**: (1) Failure of external version (2) Prevent descent of the presenting part especially during labor (3) Separation of a normally situated placenta (4) Favor malpresentation (5) Fetal distress in labor.

Long cord: The clinical significance due to the presence of a long cord is that there is an increased chance of: (1) Cord prolapse. (2) Cord entanglement round the neck or the body. The condition may produce sufficient compression on the cord vessels so as to produce fetal distress or rarely death. (3) **True knot is rare**. Even with true knot the fetal vessels are protected from compression, by the Wharton's jelly. **False knots** are the result of accumulation of Wharton's jelly or due to varices (Fig. 17.9).

SINGLE UMBILICAL ARTERY: Single umbilical artery is present in about 1–2% of cases. It may be due to failure of development of one artery or due to its atrophy in later months. It is more common in twins and in babies born of women with diabetes, epilepsy, oligohydramnios, hydramnios, preeclampsia and antepartum hemorrhage. **It is frequently associated with** congenital malformation of the fetus (20–25%). **Renal and genital anomalies, Trisomy 18 are common**. There is increased chance of abortion, fetal aneuploidy, prematurity, IUGR and increased perinatal mortality (Fig. 17.9).

QUESTIONS

Related theory questions (Long & Short), Obstetric Case Discussions, Viva table discussions, Post operative word round discussions, and MCQs are discussed in author's books:

1. **Bedside Clinic and Viva Voce:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

For further reading:

2. **Master pass in Obstetrics and Gynaecology:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

Chapter 18

Hypertensive Disorders in Pregnancy

Hypertension is one of the common medical complications of pregnancy and contributes significantly to maternal and perinatal morbidity and mortality. Hypertension is a sign of an underlying pathology, which may be pre-existing or appears for the first time during pregnancy. The identification of this clinical entity and effective management play a significant role in the outcome of pregnancy, both for the mother and the baby. In developing countries, with inadequately cared pregnancy, this entity on many occasions remains undetected till major complications supervene.

Table 18.1: Classification of Hypertension in Pregnancy (*National High Blood Pressure Education Program 2000 and ACOG-2013*)

Disorder	Definition
Hypertension	BP ≥140/90 mm Hg measured two times with at least 6-hour interval
Proteinuria	Urinary excretion of ≥0.3 g protein/24 hours specimen or 0.1 g/L
Gestational hypertension	BP ≥140/90 mm Hg for the first time in pregnancy after 20 weeks, without proteinuria
Preeclampsia	Gestational hypertension with proteinuria
Eclampsia	Women with preeclampsia complicated with grand mal seizures and/or coma
HELLP syndrome	▪ Hemolysis (H) ▪ Elevated liver enzymes (EL) ▪ Low platelet count (LP)
Chronic hypertension	Known hypertension before pregnancy or hypertension diagnosed first time before 20 weeks of pregnancy
Superimposed preeclampsia or eclampsia	Occurrence of new onset of proteinuria in women with chronic hypertension
Chronic hypertension with superimposed preeclampsia and eclampsia	**The common causes of chronic hypertension:** ▪ Essential hypertension ▪ Chronic renal disease (renovascular) ▪ Coarctation of aorta ▪ Endocrine disorders (diabetes mellitus, pheochromocytoma, thyrotoxicosis ▪ Connective tissue diseases (Lupus erythematosus). **The criteria for diagnosis of superimposed preeclampsia:** ▪ New onset of proteinuria >0.5 g/24 hours specimen ▪ Aggravation of hypertension ▪ Development of HELLP syndrome ▪ Development of headache scotoma, epigastric pain

PREECLAMPSIA

DEFINITION: Preeclampsia is a multisystem disorder of unknown etiology characterized by development of hypertension to the extent of 140/90 mm Hg or more with proteinuria after the 20th week in a previously normotensive and nonproteinuric woman. Some amount of edema is common in a normal pregnancy. Edema has been excluded from the diagnostic criteria unless it is pathological. **The preeclamptic features may appear even before the 20th week as in cases of hydatidiform mole and acute polyhydramnios.**

The term, "**Pregnancy-induced hypertension (PIH)**" is defined as the hypertension that develops as a direct result of the gravid state. It includes—(i) gestational hypertension, (ii) preeclampsia and (iii) eclampsia.

DIAGNOSTIC CRITERIA OF PREECLAMPSIA

Hypertension: **An absolute rise of blood pressure of at least 140/90 mm Hg**, if the previous blood pressure is not known or a rise in systolic pressure of at least 30 mm Hg, or a rise in diastolic pressure of at least 15 mm Hg over the previously known blood pressure is called pregnancy-induced hypertension.

Calculation based on mean arterial pressure (MAP) as advocated by Page

$$\frac{\text{Systolic pressure} + (\text{diastolic pressure} \times 2)}{3} = \text{MAP}$$

A rise of 20 mm Hg MAP over the previous reading, or when the MAP is 105 mm Hg or more should be considered as significant.

Rise of blood pressure should be evident at least on two occasions, at least 6 hours apart. The level is arbitrary and is based on the observation that complications are likely to be more beyond this level. Diastolic blood pressure is noted at the point of disappearance of sounds (Korotkoff – V).

Blood pressure is measured on the right arm with the patient lying on her side at 45° to the horizontal. In outpatient, sitting posture is preferred. In either case, the occluded brachial artery should be kept at the level of the heart.

Edema: Demonstration of **pitting edema over the ankles** after 12 hours bed rest or rapid gain in weight of more than 1 lb a week or more than 5 lb a month in the later months of pregnancy may be the earliest evidence of preeclampsia. However, some amount of edema is common (physiological) in a normal pregnancy.

Proteinuria: Presence of total protein in 24 hours urine of more than 0.3 g or more than or equal to 2+ (1.0 g/L) on at least two random clean-catch urine samples tested more than or equal to 4 hours apart in the absence of urinary tract infection is considered significant.

Test for protein in urine by multiple reagent strip (dipstick) as follows: Trace = 0.1 g/L; 1+ = 0.3 g/L; 2+ = 1.0 g/L; 3+ = 3.0 g/L; 4+ = 10.0 g/L (see p. 743).

INCIDENCE: The incidence of preeclampsia in hospital practice varies widely from 5% to 15%. **The incidence in primigravidae is about 10% and in multigravidae 5%.** Imperfect documentation and lack of uniformity in the diagnostic criteria are the responsible factors in variation of its frequency.

Risk Factors for Preeclampsia	Etiopathological Factors for Preeclampsia
■ **Primigravida**: Young or elderly (first time exposure to chorionic villi) ■ **Family history**: Hypertension, preeclampsia ■ **Placental abnormalities**: • Hyperplacentosis: Excessive exposure to chorionic villi—(molar pregnancy twins, diabetes) • Placental ischemia. ■ **Obesity**: BMI >35 kg/m^2, Insulin resistance. ■ **Pre-existing vascular disease** (p. 255). ■ **New paternity** ■ **Thrombophilias** [antiphospholipid syndrome, protein C, S deficiency, Factor V Leiden (p. 319)].	♦ Failure of trophoblast invasion (abnormal placentation) p. 37 ♦ Vascular endothelial damage ♦ Inflammatory mediators (cytokines) ♦ Immunological intolerance between maternal and fetal tissues (p. 720) • Coagulation abnormalities (p. 711) ♦ Increased oxygen free radicals ♦ Imbalance of angiogenic and antiangiogenic proteins (see p 257) ♦ Genetic predisposition (polygenic disorder) ♦ Dietary deficiency or excess

ETIOPATHOGENESIS OF PREECLAMPSIA

HYPERTENSION: The underlying basic pathology is **endothelial dysfunction** and intense **vasospasm**, affecting almost all the vessels, particularly those of uterus, kidney, placental bed and brain. The basic underlying pathology remains as **endothelial dysfunction** and **vasospasm**. The responsible agent for endothelial dysfunction and vasospasm still has not been isolated precisely, but it seems to be certain humoral in origin. **The following are the considerations:**

- Increased circulating pressor substances (see p. 259).
- Increased sensitivity of the vascular system to normally circulating pressor substances (Genetic).

Trophoblast Invasion and Uterine Vascular Changes: Normally there is invasion of the endovascular trophoblasts into the walls of the the spiral arterioles of the uteroplacental bed. In the **first trimester** (10–12 weeks) endovascular trophoblasts invades **up to decidual segments** and in the **second trimester** (16–18 weeks) another wave of trophoblasts invades **up to the myometrial segments (Fig. 3.7)**. This process replaces the endothelial lining and the muscular arterial wall by fibrinoid formation. The spiral arterioles thereby become distended, tortuous and funnel-shaped. **This physiological change transforms the spiral arterioles into a low resistance, low pressure, high flow system**. In preeclampsia, there is failure of the second wave of endovascular trophoblast migration and there is reduction of blood supply to the fetoplacental unit (p. 37).

In normal pregnancy: (1) Angiotensin-II (part of α_2 globulin) is destroyed by angiotensinase, which is liberated from the placenta. Thus, the blood pressure is stabilized. (2) The vascular system becomes refractory, selectively to pressor agent angiotensin-II. This is probably brought out by **vascular synthesis of prostaglandin I_2 and nitric oxide (NO)** which has got vasodilator effect. The interaction between the two systems stabilizes the blood pressure in normal pregnancy. **Vascular endothelial growth factor (a mitogenic glycoprotein)** — increased VEGF restores the uteroplacental blood flow to normal level.

In preeclampsia: (1) There is an **imbalance in different components of prostaglandins**—relative or absolute deficiency of vasodilator prostaglandin (PGI_2) from vascular endothelium and increased synthesis of thromboxane (TXA_2), a potent vasoconstrictor in platelets. (2) **There is increased vascular sensitivity to the pressor agent angiotensin-II**. Angiotensinase activity is **depressed**, following proteinuria with elimination of α_2 globulin (see scheme for pathophysiology). (3) **Nitric oxide:** It is synthesized in the vascular endothelium and syncytiotrophoblast from L-arginine. **It significantly relaxes vascular smooth muscle, inhibits platelet aggregation and prevents intervillous thrombosis**. Deficiency of nitric oxide contributes to the development of hypertension. (4) **Endothelin-1** is synthesized by endothelial cells, and it **is a potent vasoconstrictor** compared to angiotensin-II. Endothelin-1 also contributes to the cause of hypertension. (5) **Inflammatory mediators**: *Cytokines* [tumor necrosis factor (TNF-α), interleukins (IL-6) and others] derived from activated leukocytes cause endothelial injury (6) **Abnormal lipid metabolism**—results in more oxidative stress. *Lipid peroxides, reactive oxygen species (ROS) and superoxide anion radicals* — cause endothelial injury and dysfunction. Platelet and neutrophil activation, cytokines, superoxide radical production and endothelial damage are in a vicious cycle. (7) Imbalance of angiogenic and antiangiogenic proteins in placental vascular bed—there is overproduction of two antiangiogenic factors from the trophoblastic tissue. These two antiangiogenic factors are: (a) soluble fms-like tyrosine kinase I (SFlt-1) and (b) soluble endoglin. **SFlt-1 binds with VEGF and placental-like growth factor (PLGF) and causes endothelial cell dysfunction.** (8) **Others**—mutation of factor V Leiden increases the risk.

Hence preeclampsia is characterized by endothelial dysfunction and vasospasm. **Endothelial dysfunction** is due to oxidative stress and the inflammatory mediators. **Vasospasm** results from the imbalance of vasodilators (PGI_2, NO) and vasoconstrictors (Angiotensin-II, TXA_2, Endothelin-1). Both are in a vicious cycle.

EDEMA: The cause of excessive accumulation of fluids in the extracellular tissue spaces is not clear. Probable explanations are: Increased oxidative stress → endothelial injury → increased capillary permeability. On this basis, the leaky capillaries and decreased blood osmotic pressure are the probable explanations.

PROTEINURIA: **The probable chain of events is as follows**. Spasm of the afferent glomerular arterioles → anoxic change to the endothelium of the glomerular tuft → glomerular endotheliosis → increased capillary permeability → increased leakage of proteins. Tubular reabsorption is simultaneously depressed. **Albumin constitutes 50–60% and α-globulin constitutes 10–15% of the total proteins excreted in the urine**.

PATHOPHYSIOLOGY

While the question as to why the syndrome occurs still remains unsolved, the pathological changes are well documented, especially in severe preeclampsia or in eclampsia.

Uteroplacental bed: There is increased evidences of premature aging of the placenta. Areas of occasional acute red infarcts and white infarcts are visible on the maternal surface of the placenta.

***Villi*:** Syncitial degeneration, increased syncitial knots, marked proliferation of cytotrophoblast, thickening of the basement layer, and proliferative endarteritis are evident in varying degrees.

In preeclampsia, the normal endovascular invasion of cytotrophoblast into the spiral arteries fails to occur beyond decidua-myometrial junction (see p. 37). As a result, the musculoelastic media in the myometrial segment remains responsive to vasoconstrictor stimuli resulting in decreased blood flow (see Fig. 3.7). There is acute atherosis of spiral arteries with obliteration of lumen.

***Intervillous circulation*:** The blood flow is impaired to the extent of about one-third, secondary to the changes in the maternal blood vessels. This results in placental changes, anatomical and functional, which are responsible for fetal jeopardy.

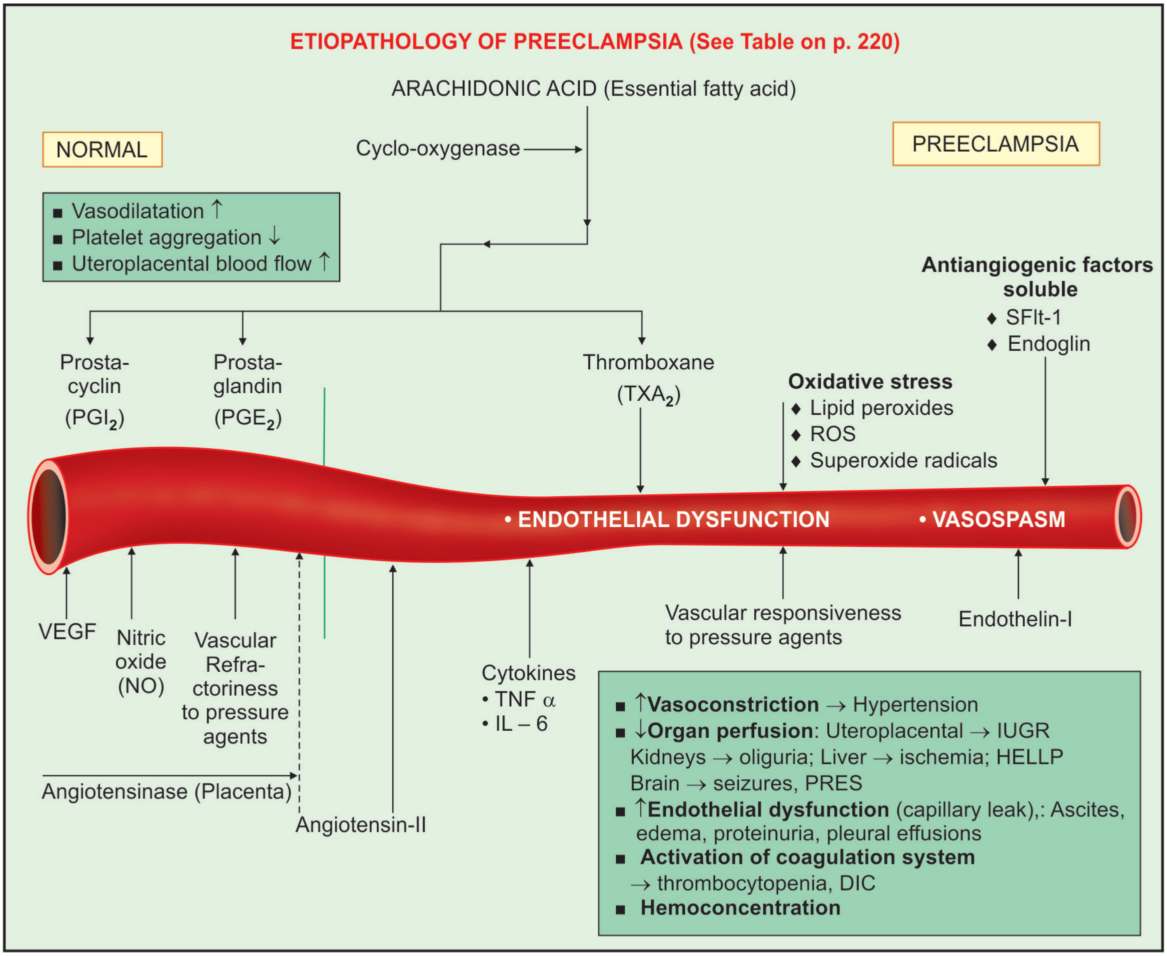
Kidney: The changes are conspicuous in the glomerulus which becomes enlarged **(glomerular endotheliosis)**. Endothelial cells swell up and fibrin-like deposits occur in the basement membrane. The lumen may be occluded. Interstitial cells in between the capillaries proliferate. There is associated spasm of the afferent glomerular arterioles. Patchy areas of damage of the tubular epithelium due to anoxia are evident. **The net effects are** reduced renal blood flow and glomerular filtration rate (25%), and impaired tubular reabsorption or secretory function. Recovery is likely to be complete following delivery. In severe cases, intense anoxia may produce extensive arterial thrombosis leading to *bilateral renal cortical necrosis.*

Blood vessels: There is intense vasospasm. Circulation in the vasa vasorum is impaired leading to damage of the vascular walls, including the endothelial integrity.

Liver: *Periportal hemorrhagic necrosis* of the liver occurs due to thrombosis of the arterioles. The necrosis starts at the periphery of the lobule. There may be *subcapsular hematoma*. Hepatic insufficiency seldom occurs because of the reserve capacity and regenerative ability of liver cells. Liver function tests are specially abnormal in women with HELLP syndrome.

HELLP Syndrome: This is an acronym for Hemolysis (H), Elevated Liver enzymes (EL) and Low Platelet count (LP) (<100,000/mm^3). This is a rare complication of preeclampsia (10–15%). HELLP syndrome may develop even without maternal hypertension. This syndrome is manifested by nausea, vomiting, epigastric or right upper quadrant pain, along with biochemical, and hematological changes. Parenchymal necrosis of liver causes elevation in hepatic enzymes (AST and ALT >70 IU/L, LDH >600 IU/L) and bilirubin (>1.2 mg/dL). There may be subcapsular hematoma formation (which is diagnosed by CT scan) and abnormal peripheral blood smear. Eventually liver may rupture to cause sudden hypotension, due to hemoperitoneum.

Management: Principles of management are same as that of preeclampsia and eclampsia (see p. 264). Antiseizure prophylaxis with magnesium sulfate (see p. 273) is started. Careful assessment of maternal and fetal status followed by delivery is done. Administration of corticosteroids (see p. 367) improves perinatal (↑ pulmonary maturity, ↓ IVH and ↓ necrotizing enterocolitis) and maternal (↑ thrombocyte count, ↑ urinary output) outcome. Cesarean section is the common mode of delivery. Epidural anesthesia can be used safely if the platelet count is >1,00,000/mm^3). Platelet transfusion should be given if the count is <50,000/mm^3. Patient should be managed in an ICU until there is improvement in platelet count, urine output, BP and liver enzymes. Recurrence risk of HELLP syndrome is 3–19%.



ETIOPATHOLOGY OF PREECLAMPSIA (See Table on p. 220)

ARACHIDONIC ACID (Essential fatty acid)
Cyclo-oxygenase
NORMAL
PREECLAMPSIA
■ Vasodilatation ↑
■ Platelet aggregation ↓
■ Uteroplacental blood flow ↑
Antiangiogenic factors soluble
◆ SFlt-1
◆ Endoglin
Prosta-cyclin (PGI2)
Prosta-glandin (PGE2)
Thromboxane (TXA2)
Oxidative stress
◆ Lipid peroxides
◆ ROS
◆ Superoxide radicals
• ENDOTHELIAL DYSFUNCTION
• VASOSPASM
VEGF
Nitric oxide (NO)
Vascular Refra-ctoriness to pressure agents
Vascular responsiveness to pressure agents
Endothelin-I
Cytokines
• TNF α
• IL – 6
Angiotensinase (Placenta)
Angiotensin-II
■ ↑Vasoconstriction → Hypertension
■ ↓Organ perfusion: Uteroplacental → IUGR
Kidneys → oliguria; Liver → ischemia; HELLP
Brain → seizures, PRES
■ ↑Endothelial dysfunction (capillary leak),: Ascites, edema, proteinuria, pleural effusions
■ Activation of coagulation system → thrombocytopenia, DIC
■ Hemoconcentration

Expectant management has been carried out selectively when pregnancy is less than 34 weeks, with bed rest, plasma volume expansion (infusion of 5–25% albumin), antithrombotic agents (dipyridamole), immunosuppressive agents (steroids) and others (fresh frozen plasma). In HELLP syndrome perinatal mortality ranges between 5% and 60% and maternal mortality may be up to 25%. **Complications:** *Maternal*: Abruptio placenta, DIC, acute renal failure, severe ascites, pulmonary edema, pleural effusions, cerebral edema, laryngeal edema, retinal detachment, subcapsular liver hematoma, ARDS, sepsis, and death. *Perinatal*: Morbidity and mortality are significantly increased. This is due to preterm delivery, prematurity, RDS and sepsis (see p. 689).

Brain: Neuroimaging (CT, MRI) studies revealed: hypodense areas in the cortex, cerebral edema, capillary thrombosis, infarction, intraventricular and parenchymal hemorrhages, and necrosis. Clinical manifestations of headache, scotomata, blindness, convulsions are due to PRES (Fig. 18.1).

Posterior reversible encephalopathy syndrome: Posterior (Occipital and Posterior Parietal Lobes) Reversible Encephalopathy Syndrome (PRES) is a transient neuroradiological entity characterized by the features of hypertension, generalized seizures, altered mental status, headache and vision changes. The hallmark of diagnosis is bilateral symmetrical vasogenic edema in the occipital and posterior parietal lobes. It is evident on T2-weighted MRI images.

Underlying pathology is thought to be cerebral edema, and vasospasm of cerebral and retinal vessels. PRES is usually reversible with prompt diagnosis and treatment. Rarely it may lead to cortical infarction and irreversible ischemic damage and even death.

Neuroradiologic imaging should be done in a patient with persistant headache, hypertension, seizures or visual changes (blindness) even in the postpartum period.

Heart: Subendothelial hemorrhages may occur. Focal necrosis and hemorrhage in the myocardium may affect the conducting system leading to heart failure.

Lungs: There is evidence of edema or hemorrhagic bronchopneumonia and ARDS. This is due to low oncotic pressure and leaky capillaries. **Other organs:** Adrenal glands show hemorrhage and necrosis. Stomach shows feature of hemorrhagic gastritis.

Water and electrolyte balance: There is lot of controversy and confusion about the changes in water and electrolyte balance in preeclampsia. The consensus of opinion is varying degree of salt retention. The precise mechanism is not clear. It is not due to increase in aldosterone which, in fact, is diminished in preeclampsia. The retention is likely due to renal vasoconstriction leading to reduction of GFR. The salt retention increases extracellular fluid volume; increases sensitivity to angiotensin-II and may increase peripheral resistance by causing arteriolar endothelial swelling. **The net effect is intravascular dehydration and extravascular overhydration.** Thus, there is hemoconcentration and rise in hematocrit value.

Immediately following delivery, there is rapid mobilization of sodium and water from the extravascular compartment into the intravascular space. If this mechanism fails, the patient may develop shock **(Postpartum vascular collapse)**.

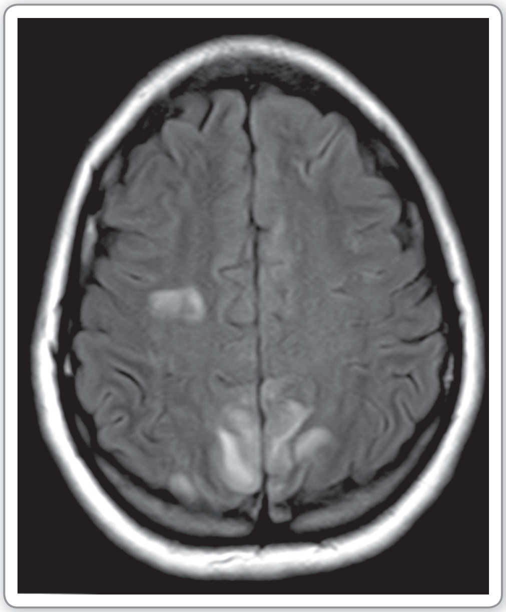


Fig. 18.1: MRI: Neuroradiologic imaging showing multilobe cerebral (occipital lobes) abnormalities. Intense signal lesions on T2 weighted images are seen (From T. Avcher 2013, CMQCC; with permission)

Hematological changes: *Blood volume* — The extent of increase in blood volume in normal pregnancy is not evident in severe preeclampsia. Due to vasospastic state, the intravascular fluid is forced out into the extravascular space. **Thus, there is hemoconcentration with increased hematocrit values**. After delivery, the plasma volume increases with decrease in hemoglobin and hematocrit values.

Erythrocyte destruction — There is evidence of increased erythrocyte destruction following eclampsia with resultant hemoglobinemia and hemoglobinuria.

Coagulation — There is evidence of disseminated intravascular coagulopathy (DIC) affecting widespread organs of the body as opposed to selective DIC only at the placental site in normal pregnancy. The process appears to be initiated by the release of thromboplastin into the circulation. It may arise from the blood platelets as in Shwartzman reaction or from release of trophoblastic fragments into the uterine circulation. **There is reduction of platelets, fibrinogen, antithrombin-III, and plasminogen level in the blood**. The microthrombi affect the arterioles of all the vital organs apart from the placenta to produce specific pathological changes. Degree of **thrombocytopenia** reflects the severity of pathology. Fibronectin, D-dimer and thrombin levels are elevated.

Hormones: There is a conflict regarding the hormonal status in preeclampsia. Estrogen and progesterone levels are lowered, whereas chorionic gonadotropin level may be increased. The serum level of HPL is decreased.

Biochemical values: Nonprotein nitrogen and urea levels of normal pregnancy of 25 mg/dL and 20 mg/dL respectively are maintained in mild preeclampsia. In severe preeclampsia, however, both the levels are increased. **Blood uric acid level is raised** beyond the normal level of 4 mg/dL. Uric acid is secreted by the distal tubules. Raised serum uric acid level indicates renal involvement (increased tubular reabsorption, decreased tubular secretion and/or diminished renal blood flow). High level of serum uric acid is found to correlate with the severity of preeclampsia, volume contraction and fetal jeopardy. Serum creatinine is elevated in severe preeclampsia. *Blood chlorides are increased* due to delay in the excretion of sodium. Serum protein level is lowered, particularly the albumin fraction. **Oxidative stress** (see p. 257): Hyperhomocysteinemia found in preeclampsia causes oxidative stress and endothelial damage.

CLINICAL TYPES

The clinical classification of preeclampsia is arbitrary and is principally dependent on the level of blood pressure for management purpose. But proteinuria is more significant than blood pressure to predict fetal outcome.

- **Mild:** This includes cases of sustained rise of blood pressure of more than 140/90 mm Hg but less than 160 mm Hg systolic or 110 mm Hg diastolic without significant proteinuria.
- **Severe:** (1) A persistent systolic blood pressure above or equal to 160 mm Hg or diastolic pressure above 110 mm Hg. (2) Protein excretion of more than 5 g/24 h (3) Oliguria (<400 mL/24 h). (4) Platelet count less than 100,000/mm^3. (5) HELLP syndrome. (6) Cerebral or visual disturbances. (7) Persistent severe epigastric pain. (8) Retinal hemorrhages, exudates or papilledema. (9) Intrauterine growth restriction of the fetus. (10) Pulmonary edema.

From the prognostic point of view, a diastolic rise of blood pressure is more important than the systolic rise. Moreover, convulsions may occur even with moderate rise of blood pressure; conversely, even with alarming high rise of pressure, the pregnancy may have an uneventful outcome. This calls for a strict vigilance whenever the blood pressure is raised to the preeclamptic level or even before that.

CLINICAL FEATURES

Preeclampsia frequently occurs in primigravidae (70%). It is more often associated with obstetrical–medical complications such as multiple pregnancy, polyhydramnios, pre-existing hypertension, diabetes etc. The clinical manifestations appear usually after the 20th week.

ONSET: The onset is usually insidious and the syndrome runs a slow course. On rare occasion, however, the onset becomes acute and follows a rapid course.

SYMPTOMS: Preeclampsia is principally a syndrome of signs and when symptoms appear, it is usually late.

Mild symptoms: Slight swelling over the ankles which persists on rising from the bed in the morning or tightness of the ring on the finger is the early manifestation of edema due to preeclampsia. Gradually, the swelling may extend to the face, abdominal wall, vulva and even the whole body (see Figs 18.2A & B).

Alarming symptoms: The following are the ominous symptoms, which may be evident either singly or in combination. **These are usually associated with acute onset of the syndrome**. (1) **Headache** — either located over the occipital or frontal region, (2) **Disturbed sleep,** (3) **Diminished urinary output**— Urinary output of less than 400 mL in 24 hours is very ominous, (4) **Epigastric pain**—acute pain in the epigastric region associated with vomiting, at times coffee color, is due to hemorrhagic gastritis or due to

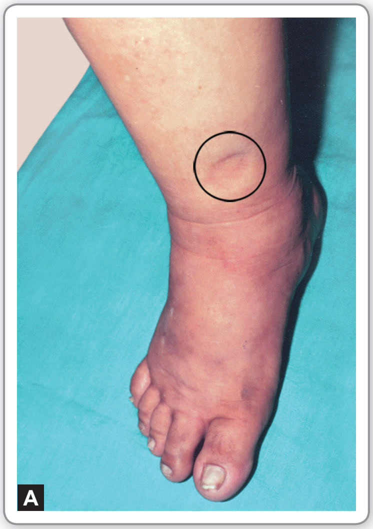


Fig. 18.2A: Marked pedal edema

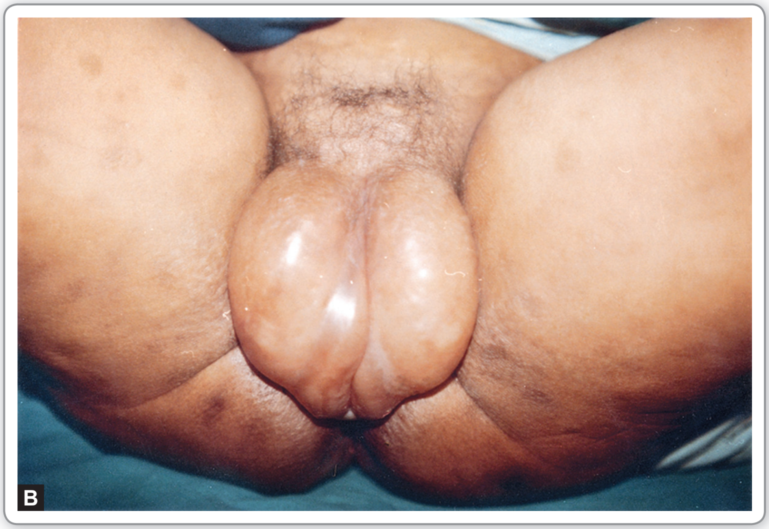


Fig. 18.2B: Marked vulval edema in preeclampsia

subcapsular hemorrhage in the liver, (5) **Eye symptoms**—there may be blurring, scotomata, dimness of vision or at times complete blindness. Vision is usually regained within 4–6 weeks following delivery. The eye symptoms are due to spasm of retinal vessels (retinal infarction), occipital lobe damage (vasogenic edema) or retinal detachment. Reattachment of the retina occurs following subsidence of edema and normalization of blood pressure after delivery.

SIGNS

1. **Abnormal weight gain:** Abnormal weight gain within a short span of time probably appears even before the visible edema. **A rapid gain in weight of more than 5 lb a month or more than 1 lb a week in later months of pregnancy is significant**.
2. **Rise of blood pressure**: The rise of blood pressure is usually insidious but may be abrupt. The diastolic pressure usually tends to rise first followed by the systolic pressure.
3. **Edema:** Visible edema over the ankles on rising from the bed in the morning is pathological. The edema may spread to other parts of the body in uncared cases (Fig. 18.2). Sudden and generalized edema may indicate imminent eclampsia.
4. **There is no manifestation** of chronic cardiovascular or renal pathology.
5. **Pulmonary edema**—due to leaky capillaries and low oncotic pressure.
6. **Abdominal examination** may reveal evidences of chronic placental insufficiency, such as scanty liquor or growth retardation of the fetus.

Thus, the manifestations of preeclampsia usually appear in the following order—rapid gain in weight → visible edema and/or hypertension → proteinuria.

INVESTIGATIONS

- ***Urine:*** Proteinuria is the last feature of preeclampsia to appear. It may be trace or at times copious so that urine becomes solid on boiling (10–15 g/L). There may be few hyaline casts, epithelial cells or even few red cells. 24 hours urine collection for protein measurement is done (see above).
- ***Ophthalmoscopic examination***: In severe cases there may be retinal edema, constriction of the arterioles, alteration of normal ratio of vein: arteriole diameter from 3 : 2 to 3 : 1 and nicking of the veins where crossed by the arterioles. There may be hemorrhage.
- ***Blood values***: The blood changes are not specific and often inconsistent. A serum uric acid level **(biochemical marker of preeclampsia)** of more than 4.5 mg/dL indicates the presence of preeclampsia. Blood urea level remains normal or slightly raised. Serum creatinine level may be more than 1 mg/dL. There may be thrombocytopenia and abnormal coagulation profile of varying degrees. Hepatic enzyme levels may be increased.
- ***Antenatal fetal monitoring***: Antenatal fetal well-being assessment is done by clinical examination, daily fetal kick count, ultrasonography for fetal growth and liquor pockets, cardiotocography, umbilical artery flow velocimetry and biophysical profile (see Chapter 11).

COURSE OF THE DISEASE: Preeclampsia is usually insidious in onset and runs a slow course. Rarely, the onset may be acute and follows a rapid course of events. **The following course of events may occur:**

If detected early: With prompt and effective treatment the preeclamptic features may subside completely.

If left untreated and uncared for: (a) **The preeclamptic features remain stationary** at varying degrees till delivery. (b) **Aggravation of the preeclamptic features** with appearance of symptoms of acute fulminating preeclampsia as mentioned earlier. This happens mostly in cases with acute onset. (c) **Eclampsia** – It may occur following acute fulminating preeclampsia or bypassing it. In fact, eclampsia can occur even with a blood pressure of 140/90 mm Hg. (d) **Spontaneous remission** of the preeclamptic features—a rare and fortunate event.

COMPLICATIONS OF PREECLAMPSIA

The complications are more likely to occur if the patients are left untreated and uncared for.

- **Immediate:**
  - **Maternal**
  - **Fetal**
- **Remote**

IMMEDIATE: Maternal

- *During pregnancy:* (a) **Eclampsia** (2%) — more in acute than in subacute cases, (b) Accidental hemorrhage, (c) Oliguria and anuria, (d) Dimness of vision and even blindness, (e) Preterm labor, (f) HELLP syndrome (see p. 258), (g) Cerebral hemorrhage, (h) Acute respiratory distress syndrome (ARDS)
- *During labor:* (a) **Eclampsia,** (b) Postpartum hemorrhage — may be related with coagulation failure
- *Puerperium:* (a) **Eclampsia** — usually occurs within 48 hours, (b) **Shock** — puerperal vasomotor collapse is associated with reduced concentration of sodium and chloride due to sudden fall in corticosteroid level, (c) **Sepsis** — due to increased incidence of induction, operative interference, and low vitality.

Fetal: The fetal risk is related to the severity of preeclampsia, duration of the disease and degree of proteinuria. **The following hazards may occur**. (a) Intrauterine death—due to spasm of uteroplacental circulation leading to accidental hemorrhage or acute red infarction, (b) Intrauterine growth restriction—due to chronic placental insufficiency, (c) Asphyxia, (d) Prematurity—either due to spontaneous preterm onset of labor or due to preterm induction.

REMOTE

- *Residual hypertension:* It may persist even after 6 months following delivery in about 50% cases. It is more related to familial diathesis and underlying thrombophilias (protein C, protein S deficiency, antiphospholipid syndrome) (see p. 399). Microvascular dysfunction due to insulin resistance is also there.
- *Recurrent preeclampsia:* There is 25% chance of preeclampsia to recur in subsequent pregnancies. **This too is related with familial diathesis, personal predisposition with underlying thrombophilias** (see p. 508).
- *Chronic renal disease*: There is high incidence of glomerulonephritis in women with preeclampsia remote from term. This is more likely due to preexistent underlying renal disease.
- Risk of placental abruption for those women with preeclampsia ranges from 5% to 20% and women with HELLP syndrome, the risk of preeclampsia in subsequent pregnancy is about 20%.

PROGNOSIS: The prognosis of preeclampsia depends on the period of gestation, severity of disease and response to treatment.

IMMEDIATE: **If the preeclampsia is detected early**, with prompt and effective treatment the preeclamptic features subside completely and the prognosis is not unfavorable, both for the mother and the baby. However, if the cases are left uncared for or with cases of acute onset, serious complications are likely to occur. In such conditions, both the mother and the baby are in danger.

Maternal mortality: **Increased maternal deaths are mainly related to** eclampsia, accidental hemorrhage, acute renal failure, pulmonary edema, disseminated intravascular coagulopathy and HELLP syndrome. Though mortality has been reduced significantly in the advanced countries, it still remains high in the developing world.

Perinatal mortality: Although the maternal mortality has been reduced significantly, the perinatal mortality still remains very high even in the developed countries (7–10%). In developing countries, the perinatal mortality remains to the extent of about 20%, about 50% of which being stillborn.

PREDICTION AND PREVENTION OF PREECLAMPSIA: Preeclampsia is not a totally preventable disease. **However, some specific "high risk" factors leading to preeclampsia may be identified in an individual**.

SCREENING TESTS FOR PREDICTION AND PREVENTION OF PREECLAMPSIA

More than 100 clinical, biophysical and biochemical tests have been tried to predict the onset of the disease. Unfortunately, none can provide convincing evidence of any clinical use.

- **Doppler ultrasound** of high resistance index in the uterine artery, second trimester is associated with sixfold increase in rate of preeclampsia (see p. 123). **Presence of diastolic notch** at 24 weeks' gestation in the uterine artery can predict the possible development of preeclampsia.
- **Development of renal dysfunction:** Rise in the level of serum uric acid (hyperuricemia p. 347) and appearance of microalbuinuria are observed to be the predictors of preeclampsia.
- **Absence of end-diastolic frequencies** or reverse diastolic flow patterns in the umbilical artery usually indicates poor perinatal outcome.
- **Average mean arterial pressure (MAP)** in second trimester $\geq$ 90 mm Hg may predict the onset.
- **Maternal serum level of SFlt-1** is increased in women with preeclampsia.
- **Fetal DNA**—Detection of free fetal DNA (ff DNA) in maternal plasma in early pregnancy may be predictive of preeclampsia.
- **Proteomics, metabolomics and transcryptomic markers** are currently being studied. These are the cellular protein metabolites obtained from the serum or urine. These are thought to be useful as the predictors of preeclampsia.
- **Roll over test:** This screening test is done between 28 and 32 weeks. Blood pressure is measured with the patient on her side first and then the patient is asked to roll on her back to check the blood pressure once again. An increase of 20 mm Hg in diastolic pressure from side to back position indicates a positive "roll over test". About 33% of women with positive "roll over test" developed hypertension later. A negative test is of value.

PROPHYLACTIC MEASURES FOR PREVENTION OF PREECLAMPSIA

- **Regular antenatal check up** for early detection of rapid gain in weight or a tendency of rising blood pressure especially the diastolic one.
- **Antithrombotic agents:** *Low-dose aspirin* 60 mg daily beginning early in pregnancy in potentially high risk patients is given. It selectively reduces platelet thromboxane production. Aspirin in low doses is known to inhibit cyclo-oxygenase in platelets thereby preventing the formation of thromboxane A_2 without interfering with prostacyclin generation.
- **Heparin or low-molecular-weight** heparin is useful in women with thrombophilia and with high risk pregnancy.
- **Calcium supplementation** (2 g/day) reduces the risk of gestational hypertension.
- **Antioxidants**, vitamins E and C and nutritional supplementation with magnesium, zinc, fish oil and low-salt diet have been tried but are of limited benefit.
- **Balanced diet** rich in protein may reduce the risk.

MANAGEMENT OF PREECLAMPSIA

So long as the etiology of preeclampsia remains obscure, **the treatment is mostly empirical and symptomatic**. While measures are directed to relieve edema and hypertension, there is no specific therapy for proteinuria which automatically subsides with the control of hypertension.

Objectives are: (1) To stabilize hypertension and to prevent its progression to severe preeclampsia. (2) To prevent the complications (see p. 263). (3) To prevent eclampsia. (4) Delivery of a healthy baby in optimal time. (5) Restoration of the health of the mother in puerperium.

Hospital or home treatment: Ideally, all patients of preeclampsia are to be admitted in the hospital for effective supervision and treatment. **There is no place of domiciliary treatment in an established**

case of preeclampsia. However, in some centers **cases of preeclampsia are managed in the day care unit** (see p. 726). In the developing countries where the prevalence of preeclampsia is more and hospital facilities are meagre, there is no alternative but to put the uncomplicated mild preeclampsias in domiciliary treatment regime. **Rest, high-protein diet** are prescribed and the patient is investigated and checked. If the treatment fails to improve the patient is to be admitted. **It is essential that she should be warned against the ominous symptoms,** such as headache, visual disturbances, vomiting, epigastric pain or scanty urine.

HOSPITAL MANAGEMENT

Rest: Admission in hospital and rest is helpful for continued evaluation and treatment of the patient. While in bed patient should be in left-lateral position as much as possible, to lessen the effects of vena caval compression. **Rest** — (1) increases renal blood flow → diuresis, (2) increases uterine blood flow → improves placental perfusion, and (3) reduces the blood pressure. However completed bed rest is not essential.

Diet: **The diet should contain adequate amount of daily protein (about 100 g)**. Usual salt intake is permitted. Fluids need not be restricted. Total calorie approximate 1,600 cal/day.

Diuretics: The diuretics should not be used injudiciously, as they cause harm to the baby by diminishing placental perfusion and by electrolyte imbalance. **The compelling reasons for its use are**—(1) Cardiac failure, (2) Pulmonary edema, (3) Along with selective antihypertensive drug therapy (diazoxide group) where blood pressure reduction is associated with fluid retention, (4) Massive edema, not relieved by rest and producing discomfort to the patient. The most potent diuretic commonly used is furosemide (Lasix) 40 mg, given orally after breakfast for 5 days in a week. In acute condition, intravenous route is preferred (details in Chapter 34).

Antihypertensives: Antihypertensive drugs have limited value in controlling blood pressure due to preeclampsia. **The indications are:** (1) Persistent rise of blood pressure especially where the diastolic pressure is over 110 mm Hg. The use is more urgent if associated with proteinuria. (2) In severe preeclampsia, to bring down the blood pressure during pregnancy and labor, the **common oral drugs** have been discussed later (see p. 581). Drug selection depends on its local availability and experience of use.

Table 18.2: Commonly Used Drugs in the Management of Preeclampsia

Drug	Mode of Action	Dose
Methyl-dopa	Central and peripheral antiadrenergic action	250–500 mg tid or qid
Labetalol	Adrenoceptor antagonist (α and β blockers)	100 mg tid or qid
Nifedipine	Calcium channel blocker	10–20 mg bid
Hydralazine	Vascular smooth muscle relaxant	10–25 mg bid

Hypertensive crisis: Any of the following drugs (Table 18.1) can be used when the BP is ≥160/110 mm Hg or the mean arterial pressure (MAP) is ≥125 mm Hg:

Table 18.3: Commonly Used Drugs in the Management of Hypertensive Crisis

Drug	Onset of Action	Dose Schedule	Maximum Dose	Maintenance Dose
Labetalol *	5 min	10–20 mg IV every 10 minute	300 mg IV	40 mg/h
Hydralazine	10 min	5 mg IV every 30 minute	30 mg IV	10 mg/h
Nifedipine	10 min	10–20 mg oral, can be repeated in 30 minute	240 mg/24 hr	4–6 hours interval
Nitroglycerin	0.5–5 min	5 μg/minute IV	Short-term therapy only when the other drugs have failed (see p. 583)	
Sodium nitroprusside		0.25–5 μg/kg/minute IV		

* To avoid labetalol in women having asthma or cardiac failure.

Progress chart: The effect of treatment should be evaluated by maintaining a chart which **records the following:** (1) **Daily clinical evaluation for any symptoms** (e.g. headache, right upper quadrant or epigastric pain, visual disturbances, oliguria). (2) **Blood pressure:** at least four times a day. (3) **State of edema and daily weight record.** (4) **Fluid intake and urinary output.** (5) **Urine examination** for protein daily and if present, to estimate its amount in 24 hours urine. (6) **Blood** for hematocrit, platelet count, uric acid, creatinine and liver enzymes, coagulation profile at least once a week. (7) **Ophthalmoscopic examination** on admission and to be repeated, if necessary. (8) **Fetal well-being** assessment (as mentioned in Chapter 11).

Favorable signs: In favorable cases, there is fall of blood pressure and weight with subsidence of edema. Urinary ouput increases with diminishing proteinuria, if previously present.

DURATION OF TREATMENT: The definitive treatment of preeclampsia is termination of pregnancy (delivery). As such, the aim of the above treatment is to continue the pregnancy, if possible, without affecting the maternal prognosis until the fetus becomes mature enough to survive in extrauterine environment (≥37 weeks). **Thus, the duration of treatment depends on** — (1) severity of preeclampsia, (2) duration of pregnancy, and (3) response to treatment, and (4) condition of the cervix.

Depending on the response to the treatment, the patients are grouped into the following:

(a) **Preeclamptic features subside and hypertension is mild**.

(b) **Partial control** of the preeclamptic features but the blood pressure maintains a steady high level.

(c) **Persistently increasing BP to severe level**, despite the use of antihypertensive and/or addition of grave features such as tachycardia more than 100 bpm, restlessness, headache, epigastric pain, oliguria, blurring of vision, HELLP syndrome or SaO_2 less than 95%.

Complete remission of all signs and symptoms is uncommon until after delivery and the underlying disease pathology persists.

Group A: If the duration of pregnancy is remote from term, the patient may be discharged with advice to attend the antenatal clinic after 1 week. **These women are not cured as majority (90%) develop recurrence**. If the patient is near term, she should be kept for a few days till completion of 37th week. Thereafter, decision is to be taken either to deliver her or to wait for spontaneous onset of labor by the due date. **It is not wise to allow the pregnancy to continue beyond the expected date**.

Group B: If the pregnancy is beyond 37 completed weeks, delivery is to be considered without delay. **If less than 37 weeks**, expectant treatment may be extended judiciously at least up to 34 weeks. **Careful maternal and fetal well-being are to be monitored during the period** (see Chapter 11).

Group C: The couple is counseled. **Termination of pregnancy (delivery) is considered irrespective of duration of gestation**. Seizure prophylaxis (magnesium sulfate) should be started (see p. 258). **Steroid therapy is considered if the duration of pregnancy is less than 34 weeks. It prevents neonatal RDS, IVH (see p. 367) and maternal thrombocytopenia**.

METHODS OF DELIVERY: ♦ **Induction of labor** ♦ **Cesarean section**

- **Induction of labor (see p. 598)**

Indications: It is indeed difficult to lay down hard and fast rules for the indications for induction. (1) **Aggravation of the preeclamptic features** in spite of medical treatment and/or appearance of newer symptoms such as epigastric pain. (2) **Hypertension persists** in spite of medical treatment with pregnancy reaching 37 weeks or more. (3) **Acute fulminating preeclampsia** irrespective of the period of gestation (see p. 267). (4) Tendency of pregnancy **to overrun the expected date**.

Methods: **If the cervix is ripe**, surgical induction by low rupture of the membranes is the method of choice. Oxytocin infusion may be added. **If the cervix is unripe**, prostaglandin (PGE_2) gel 500 µg intracervical or 1–2 mg in the posterior fornix is inserted to make the cervix ripe when low rupture of the membranes can be performed. In severe preeclampsia, antihypertensive drugs should be used during induction.

- **Cesarean section**

Indications: (1) **When an urgent termination is indicated and the cervix is unfavorable** (unripe and closed). (2) **Severe preeclampsia** with a tendency of prolonged induction—delivery interval. (3) **Associated complicating factors,** such as elderly primigravidae, contracted pelvis, malpresentation, etc.

The operation should be done by an experienced surgeon with the help of an expert anesthetist. **Epidural anesthesia is preferred,** unless there is coagulopathy.

MANAGEMENT DURING LABOR: Blood pressure tends to rise during labor and convulsions may occur due to stress hormones (intrapartum eclampsia). The patient **should be in bed**. **Antihypertensive drugs** are given if the blood pressure becomes high. **Blood pressure and urinary output are to be noted** frequently so as to detect imminent eclampsia. Prophylactic $MgSO_4$ is started when systolic BP ≥160 diastolic ≥110, MAP ≥125 mm Hg. **Careful monitoring of the fetal well-being** is mandatory.

Labor duration is curtailed by low rupture of the membranes in the first stage; and forceps or ventouse in second stage. **Intravenous ergometrine following the delivery of the anterior shoulder is withheld** as it may cause further rise of blood pressure. However, there is no contraindication of syntocinon IM or slow IV and to keep the patient under close observation for several hours.

PUERPERIUM: The patient is to be watched closely for at least 48 hours, the period during which convulsions usually occur. Antihypertensive drug treatment should be continued if the BP is high (systolic ≥150 mm Hg or diastolic ≥100 mm Hg). Oral nifedipine 10 mg at every 6 hours is given until BP remains below the hypertensive levels for at least 48 hours. Oral furosemide 20 mg a day for 5 days is also found to improve recovery and to reduce the need of antihypertensive drugs in severe preeclampsia. Magnesium sulfate (for at least 24 hours) and antihypertensive drugs may be needed in women with severe hypertension and symptoms of acute fulminant preeclampsia during the postpartum period. The patient is to be kept in the hospital, till the blood pressure is brought down to a safe level and proteinuria disappears. In breastfeeding women, labetalol, nifedipine or enalapril may used on discharge. Methyldopa is avoided due to the risks of postpartum depression.

ACUTE FULMINANT PREECLAMPSIA

(Synonym: Preeclamptic State)

It is a clinical entity where the onset of the preeclamptic manifestations is acute, occurring *de novo* or there is rapid deterioration in an established case of preeclampsia with severe hypertension over a short period of time. There is a constant threat of convulsion, cerebral hemorrhage, cardiac failure or placental abruption. **All the features of severe preeclampsia are intensified** (see p. 268).

TREATMENT: Detected at home, the patient should be **adequately sedated** by midazolam 1–2 mg IV, may be repeated in 5–10 minutes time or diazepam 10 mg IV (slow). **She should be shifted** as gently as possible, with an attendant doctor or a midwife, to tackle the fit, if it occurs during the journey to the hospital.

In the hospital, the patient is to be kept in eclampsia room under close supervision. **Prophylactic anticonvulsant therapy is to be instituted urgently**. Administration of magnesium sulfate either IM or IV regimen in a dose schedule as mentioned in the treatment of eclampsia is recommended (MAGPIE Trial-2002). The blood pressure is to be stabilized by antihypertensive drugs given parenterally (p. 265). First-line antihypertensives are: a) Labetalol (IV) or b) Hydralazine (IV). Response to treatment should be watched carefully noting frequently the blood pressure, urinary output, proteinuria and hematological parameters (see Table 18.3).

Obstetric management: **As there is a constant threat of eclampsia, maternal interest should always be considered.** In cases with pregnancy beyond 37th completed weeks or where the condition fails to improve within a reasonable period (say 6–8 hours), delivery should be seriously considered irrespective of period of gestation (see table below). Termination is done either by low rupture of the membranes aided by oxytocin infusion or by Cesarean section depending upon the severity of the condition and state of the cervix. PGE$_2$ gel may help in cervical ripening. Corticosteroid is given if pregnancy is < 34 weeks.

Monitoring of A Patient with Severe Preeclampsia on MgSO$_4$ (CMQCC)

Monitoring Parameters	Time Interval
▪ Pulse, Blood Pressure, Respiratory Rate, SaO$_2$	Every 10–30 minutes
▪ Temperature, Lung sounds	Every 2 hours
▪ Deep tendon reflexes, level of consciousness, assessment of headache, visual disturbances, epigastric pain	Every 4 hours
▪ Intake and output record	Intake IV crystalloids (N. Saline), Colloids (albumin, blood), Total ≤ 125 mL/h
▪ Fetal well-being (Ante- and Intrapartum)	Continuous EFM

Indications of Delivery without Delay in Severe Preeclampsia

- Persistent symptoms of Severe Preeclampsia
- Pulmonary edema/hypoxia (PaO$_2$ <95%)
- Hepatocellular injury: Increased AST, ALT (× twice the normal)
- Oliguria <500 mL/24h
- Abnormal coagulation profile
- FGR with nonreassuring fetal status
- Eclampsia

According to gestational age, preeclampsia may be: A. Early: <34 weeks and B. Late >34 weeks

ECLAMPSIA

The term eclampsia is derived from a Greek word, meaning "like a flash of lightening". It may occur quite abruptly, without any warning manifestations. In majority (over 80%), however, the disease is preceded by features of severe preeclampsia.

Preeclampsia when complicated with grandmal seizures (generalized tonic-clonic convulsions) and/or coma is called eclampsia. Thus, it may occur in patients with preeclampsia or in patients who have preeclampsia superimposed on essential hypertension or chronic nephritis.

INCIDENCE: The incidence varies widely from country to country and even between different zones of the same country. While in the developed countries, its prevalence is far and few but in the developing ones, particularly in the rural areas, it is still high and contributes significantly to the maternal deaths. The hospital incidence in India ranges from 1 in 500 to 1 in 30. It is more common in primigravidae (75%), five times more common in twins than in singleton pregnancies and occurs between the 36th week and term in more than 50% cases.

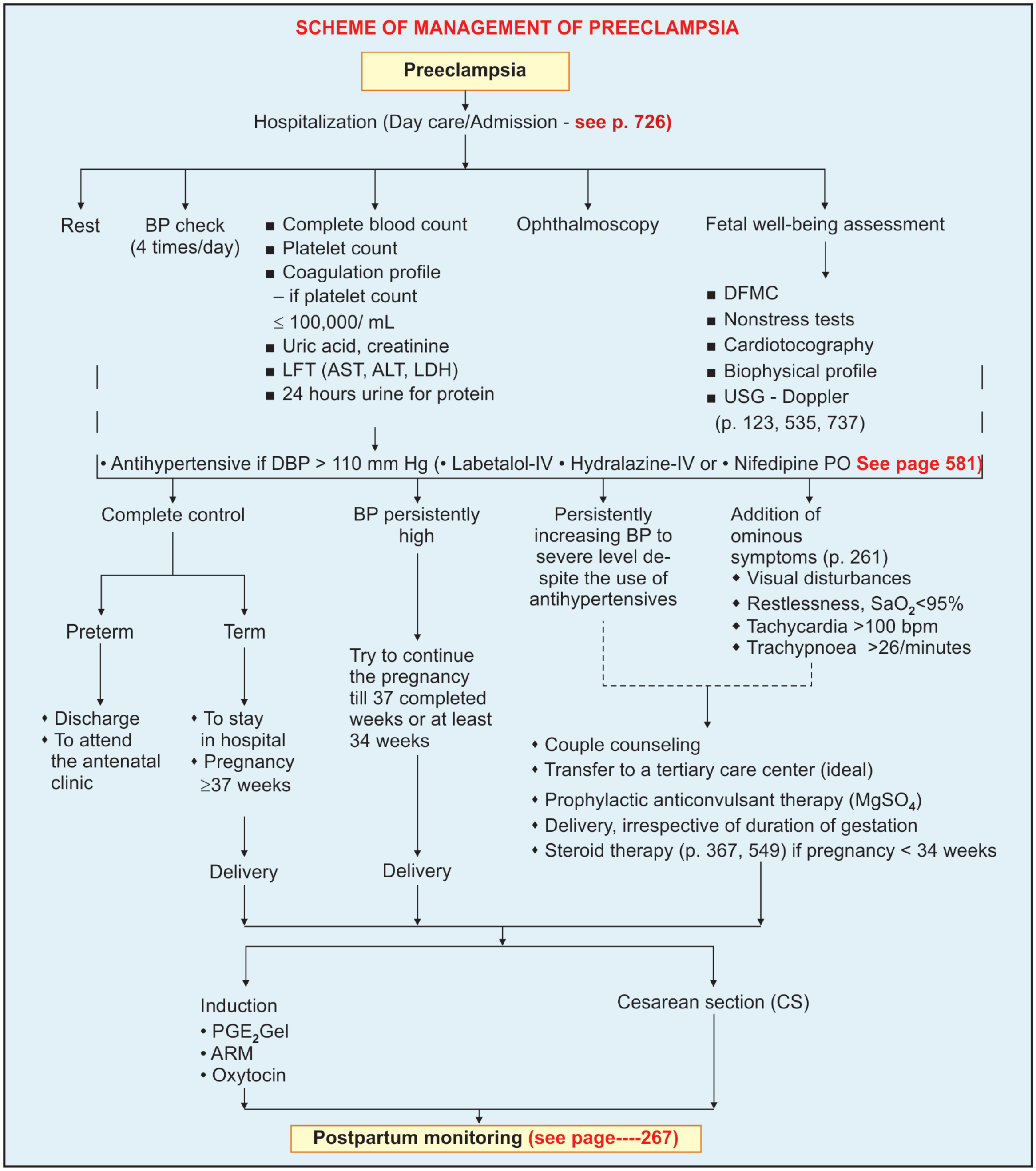
PATHOPHYSIOLOGY: Since eclampsia is a severe form of preeclampsia, the histopathological and biochemical changes are similar; although, intensified than those of preeclampsia as already described.

CAUSE OF CONVULSION: The cause of cerebral irritation leading to convulsion is not clear. The irritation may be provoked by: (1) **Anoxia** — spasm of the cerebral vessels → increased cerebral vascular resistance → fall in cerebral oxygen consumption → anoxia, (2) **Cerebral edema** — may contribute to irritation, (3) **Cerebral dysrhythmia** — increases following anoxia or edema. **There is excessive release of excitatory neurotransmitters (glutamate)**.

ONSET OF FITS: Fits occur more commonly in the third trimester (> 50%). On rare occasions, convulsion may occur in early months as in hydatidiform mole.

— **Antepartum (50%):** Fits occur before the onset of labor. More often, labor starts soon after and at times, it is impossible to differentiate it from intrapartum ones.

— **Intrapartum (30%):** Fits occur for the first time during labor.



SCHEME OF MANAGEMENT OF PREECLAMPSIA

Preeclampsia
Hospitalization (Day care/Admission - see p. 726)
Rest
BP check (4 times/day)
■ Complete blood count
■ Platelet count
■ Coagulation profile – if platelet count ≤ 100,000/ mL
■ Uric acid, creatinine
■ LFT (AST, ALT, LDH)
■ 24 hours urine for protein
Ophthalmoscopy
Fetal well-being assessment
■ DFMC
■ Nonstress tests
■ Cardiotocography
■ Biophysical profile
■ USG - Doppler (p. 123, 535, 737)
• Antihypertensive if DBP > 110 mm Hg (• Labetalol-IV • Hydralazine-IV or • Nifedipine PO See page 581)
Complete control
Preterm
Term
• Discharge
• To attend the antenatal clinic
• To stay in hospital
• Pregnancy ≥37 weeks
Delivery
BP persistently high
Try to continue the pregnancy till 37 completed weeks or at least 34 weeks
Delivery
Persistently increasing BP to severe level despite the use of antihypertensives
Addition of ominous symptoms (p. 261)
• Visual disturbances
• Restlessness, SaO2<95%
• Tachycardia >100 bpm
• Trachypnoea >26/minutes
• Couple counseling
• Transfer to a tertiary care center (ideal)
• Prophylactic anticonvulsant therapy (MgSO4)
• Delivery, irrespective of duration of gestation
• Steroid therapy (p. 367, 549) if pregnancy < 34 weeks
Induction
• PGE2Gel
• ARM
• Oxytocin
Cesarean section (CS)
Postpartum monitoring (see page----267)

— **Postpartum (20%)**: Fits occur for the first time in puerperium, usually within 48–72 hours of delivery. Fits occurring beyond 48 hours but less than 4 weeks after delivery is accepted as **late postpartum eclampsia**.

— **Intercurrent (Antenatal)**: When the patient becomes conscious after recovery from convulsions and the pregnancy continues beyond 48 hours. The time limit is arbitrary as a period of 7–10 days has also been mentioned.

Cerebral pathology includes cortical or subcortical edema, infarction and hemorrhage. The neurological abnormalities are often due to hypoxia, ischemia or edema. Several neurodiagnostic tests, e.g. EEG, CAT, cerebral Doppler Velocimetry, MRI, MRI angiography reveal presence of edema and infarction. Findings are similar to those as seen in hypertensive encephalopathy. Cerebral imaging is indicated when there is focal neurologic deficits, prolonged coma, or atypical presentation for eclampsia.

CLINICAL FEATURES OF ECLAMPSIA

Except on rare occasions, an eclamptic patient always shows previous manifestations of acute fulminating preeclampsia — called premonitory symptoms (mentioned earlier).

Eclamptic convulsion or fit: The fits are epileptiform and consist of four stages.

- **Premonitory stage:** The patient becomes unconscious. There is twitching of the muscles of the face, tongue, and limbs. Eyeballs roll or are turned to one side and become fixed. This stage lasts for about 30 seconds.
- **Tonic stage:** The whole body goes into a tonic spasm — the trunk-opisthotonus, limbs are flexed and hands clenched. Respiration ceases and the tongue protrudes between the teeth. Cyanosis appears. Eyeballs become fixed. This stage lasts for about 30 seconds.
- **Clonic stage:** All the voluntary muscles undergo alternate contraction and relaxation. The twitchings start in the face then involve one side of the extremities and ultimately the whole body is involved in the convulsion. Biting of the tongue occurs (Fig. 18.3). Breathing is stertorous and blood stained frothy secretions fill the mouth; cyanosis gradually disappears. This stage lasts for 1–4 minutes.
- **Stage of coma:** Following the fit, the patient passes on to the stage of coma. It may last for a brief period or in others deep coma persists till another convulsion. On occasion, the patient appears to be in a confused state following the fit and fails to remember the happenings. Rarely, the coma occurs without prior convulsion.

The fits are usually multiple, recurring at varying intervals. **When it occurs in quick succession, it is called status eclampticus**. Following the convulsions, temperature usually rises; pulse and respiration rates are increased and so also the blood pressure. The urinary output is markedly diminished; proteinuria is pronounced, and the blood uric acid is raised.

DIFFERENTIAL DIAGNOSIS: The diseases, which are associated with convulsions and/or coma are to be born in mind while arriving at the diagnosis of eclampsia. Such diseases are: (1) Epilepsy, (2) Hysteria, (3) Encephalitis, (4) Meningitis, (5) Puerperal cerebral thrombosis, (6) Poisoning, (7) Cerebral malaria in tropics, and (8) Intracranial tumors. Absence of previous history of convulsion with presence of edema, hypertension and proteinuria along with fits or coma during pregnancy or soon after, points to the diagnosis of eclampsia. **In doubtful cases, it is desirable to place the patient in the obstetric unit for observation until the final diagnosis is made**.

Table 18.4: Organ Dysfunction in Preeclampsia and Eclampsia

Cardiovascular	Hematological	Renal	Central Nervous System	Hepatic
■ Generalized vasospasm ■ ↑ Peripheral vascular resistance ■ ↓ CVP ■ ↓ Pulmonary wedge pressure	■ ↓ Plasma volume ■ Hemoconcentration ■ Coagulation disorder ■ ↑ Blood viscosity	■ ↓ GFR ■ ↓ Renal plasma flow ■ ↑ Serum uric acid	■ Cerebral edema ■ Cerebral hemorrhage ■ Posterior (parietal and occipital lobe) reversible encephalopathy syndrome (PRES) ■ Basal ganglia, brainstem lesion (rare)	■ Liver cell damage ■ Periportal necrosis ■ Subcapsular hematoma

Table 18.5: Maternal Complications of Eclampsia

- **Injuries:** Tongue bite, injuries due to fall from bed, bed sore.
- **Pulmonary complications:**
  - Edema—due to leaky blood capillaries
  - Pneumonia—due to aspiration, hypostatic or infective
  - Adult respiratory distress syndrome (p. 274)
  - Embolism
- **Hyperpyrexia**
- **Cardiac**—Acute left ventricular failure
- **Cardiomyopathy**
- **Renal** failure
- **Hepatic**—necrosis, Subcapsular hematoma
- **Cerebral:** Edema (vasogenic) hemorrhage
- **Neurological** deficits
- **Disturbed vision**: Due to retinal detachment or occipital lobe ischemia.
- **Hematological**
  - Thrombocytopenia
  - Disseminated intravascular Coagulopathy
- **Postpartum**
  - Shock
  - Sepsis
  - Psychosis

PROGNOSIS

MATERNAL: **Immediate: Once the convulsion occurs, the prognosis becomes uncertain. Prognosis depends on many factors and the ominous features are:** (1) Long interval between the onset of fit and commencement of treatment (late referral). (2) Antepartum eclampsia especially with long delivery interval. (3) Number of fits more than ten. (4) Coma in between fits. (5) Temperature over 102°F with pulse rate above 120/minute. (6) Blood pressure over 200 mm Hg systolic. (7) Oliguria (< 400 mL/24 hours) with proteinuria > 5 g/24 hours. (8) Nonresponse to treatment. (9) Jaundice.

Mortality: Maternal mortality in eclampsia is very high in India and varies from 2% to 30%, much more in rural-based hospital than in the urban counterpart. However, if treated early and adequately, the mortality should be even less than 2%.

Causes of maternal deaths: (1) Cardiac failure. (2) Pulmonary edema. (3) Aspiration and/or septic pneumonia. (4) Cerebral hemorrhage. (5) Acute renal failure. (6) Cardiopulmonary arrest. (7) Adult respiratory distress syndrome (ARDS). (8) Pulmonary embolism. (9) Postpartum shock. (10) Puerperal sepsis. Maternal complications are higher in antepartum eclampsia.

Remote: If the patient recovers from acute illness, she is likely to recover rapidly within 2–3 weeks. Recurrence of eclampsia in subsequent pregnancies is uncommon; although, chance of preeclampsia is about 30%.

FETAL: The perinatal mortality is very high to the extent of about 30–50%. **The causes are:** (1) **Prematurity** — spontaneous or induced, (2) **Intrauterine asphyxia** due to placental insufficiency arising out of infarction, retroplacental hemorrhage and spasm of uteroplacental vasculature, (3) **Effects of the drugs** used to control convulsions, (4) **Trauma** during operative delivery.

MANAGEMENT

PREDICTION AND PREVENTION: In majority of cases, eclampsia is preceded by severe preeclampsia. **Thus the prevention of eclampsia rests on** early detection and effective institutional treatment with judicious termination of pregnancy **during preeclampsia**. However, eclampsia can occur bypassing the preeclamptic state and as such, it is not always a preventable condition. Eclampsia may present in atypical ways; hence, it is at times difficult to predict. Use of antihypertensive drugs, prophylactic anticonvulsant therapy and timely delivery are important steps. Close monitoring during labor and 24 hours' postpartum is also important in prevention of eclampsia. **Magpie trial (2002)** showed prophylactic use of magnesium sulfate lowers the risk of eclampsia. Unfortunately, 30–85% of cases of eclampsia remained unpreventable.

FIRST AID TREATMENT OUTSIDE THE HOSPITAL: The patient, either at home or in the peripheral health centers should be shifted urgently to the tertiary referral care hospitals. **There is no place of**

continuing the treatment in such places. Transport of an eclamptic patient to a tertiary care center is important. Such a patient needs neonatal and obstetric intensive care management. **Important steps in transport are:** ● All maternal records and a detailed summary should be sent with the patient ● BP should be stabilized and convulsions should be arrested ● Magnesium sulfate [4 g IV loading dose with 10 g IM (see p. 584)] is given. Labetalol (see p. 582) 20 mg IV is given to control hypertension. Diuretic is given if there is pulmonary edema. Diazepam, if used, should be given 5 mg slowly over 1 minute period to avoid apnea or cardiac arrest ● One medical personnel or a trained midwife should accompany the patient in the ambulance equipped to prevent injury, recurrent fits and to clear air passage.

HOSPITAL—The Principles of Management are:

- Maintain: airway, breathing and circulation
- Oxygen administration 8–10 L/minute
- Arrest convulsions (see below)
- Ventilatory support (if needed)
- Prevention of injury
- Hemodynamic stabilization (control BP)
- Organize investigations (see p. 262)
- Deliver by 6–8 hours
- Prevention of complications (see p. 271)
- Postpartum care (intensive)

GENERAL MANAGEMENT (MEDICAL AND NURSING)

♦ **Supportive care:** (i) to prevent serious maternal injury from fall, (ii) prevent aspiration, (iii) to maintain airway and (iv) to ensure oxygenation.

Patient is kept in a railed cot and a tongue blade is inserted between the teeth. She is kept in the lateral decubitus position to avoid aspiration. Vomitus and oral secretions are removed by frequent suctioning, oxygenation is maintained through a face mask (8–10 L/minute) to prevent respiratory acidosis. Oxygenation is monitored using a transcutaneous pulse oximeter. Arterial blood gas analysis is needed when O_2 saturation falls below 92%. Sodium bicarbonate is given when the pH is below 7.10. The patient should have a doctor or at least a trained midwife for constant supervision.

♦ **Detailed history is to be taken** from the relatives, relevant to the diagnosis of eclampsia, duration of pregnancy, number of fits and nature of medication administered outside.

♦ **Examination: Once the patient is stabilized**, a thorough but quick general, abdominal and vaginal examinations are made. A self-retaining catheter is introduced and the urine is tested for protein. **The continuous drainage facilitates** measurement of the urinary output and periodic urine analysis.

♦ **Monitoring: Half hourly pulse, respiration rate and blood pressure** are recorded. Hourly urinary output is to be noted. If undelivered, the uterus should be palpated at regular intervals to detect the progress of labour and the fetal heart rate is to be monitored. **Immediately after a convulsion, fetal bradycardia is common** (see p. 692).

♦ **Fluid balance:** Crystalloid solution (Ringer's solution) is started as a first choice. **Total fluids should not exceed the previous 24 hours urinary output plus 1000 mL** (insensible loss through lungs and skin). Normally, it should not exceed 2 litres in 24 hours. Infusion of balanced salt solution should be at the rate of 1 mL/kg/h. In preeclampsia–eclampsia although there is hypovolemia, the tissues are over loaded. An excess of dextrose or crystalline solutions should not be used as it will aggravate the tissue overload leading to pulmonary edema and adult respiratory distress syndrome. Colloids (albumin or Haemaccel) remain in the vascular tree and they withdraw fluids from the interstitial space. Unless used carefully, they can lead to circulatory overload. CVP monitoring is needed for a patient with severe hypertension and reduced urine output. In preeclampsia, eclampsia, both the PCWP and CVP appear to be in the low to normal range. Invasive hemodynamic monitoring is rarely indicated.

♦ **Antibiotic:** To prevent infection, **Ceftriaxone 1 g IV** twice daily is given.

SPECIFIC MANAGEMENT: Anticonvulsant regime: The aim is to control the fits and to prevent its recurrence. In areas where eclampsia is frequently encountered, it is obvious that the obstetric care is inadequate. In such circumstances any complicated regime is unlikely to give good result.

- **Magnesium sulfate is the drug of choice.** It acts as a membrane stabilizer and neuroprotector. It reduces motor endplate sensitivity to acetylcholine. Magnesium also blocks neuronal calcium influx. It induces cerebral vasodilatation, dilates uterine arteries, increases production of endothelial prostacyclin and inhibits platelet activation (see p. 583). It has no detrimental effects on the neonate within therapeutic level. It has got excellent result with maternal mortality of 3%. It does not control hypertension.

Medical Management of Eclampsia—Immediate Measures

- Call for extra help (Communication)
- To put patient in left lateral recumbent position
- Maintain oral airway
- O_2 inhalation – nonbreather mask; 10 L/minute
- Commence IV lines; 1 or 2 wide bore cannulas
- Foley catheter with urometer
- To monitor O_2 saturation; pulse oximeter ($SPO_2 > 95\%$)
- Control of seizures: $MgSO_4$, (IV/IM regimens)
- To monitor vitals; fetal status and magnesium toxicity
- Control of hypertension: Labetalol, Hydralazine
- Fluids: Crystalloids (saline) or colloids (albumin/blood) ≤ 125 mL/h
- Suction: oropharyngeal
- Diuretics: pulmonary edema
- Investigations to organize: Blood: CBC, AST, ALT, LDH, Creatinine, Uric Acid, Urine analysis - protein
- *For OBST Management see p. 275*

Table 18.6: Regimens of $MgSO_4$ for the Management of Severe Preeclampsia and Eclampsia

Regimen	Loading Dose	Maintenance Dose
Intramuscular (Pritchard)	4 g (20% solution) IV over 3–5 minute followed by 10 g (50%), deep IM (5 gm in each buttock)	5 g (50%) IM 4 hourly in alternate buttock
Intravenous (Zuspan or Sibai)	4–6 g IV slow over 15–20 minute	1–2 g/h IV infusion

Repeat injections are given only if the knee jerks are present, urine output exceeds 30 mL/h and the respiration rate is more than 12/minute. **The therapeutic level of serum magnesium is 4–7 mEQ/L.** Serum magnesium levels may be monitored in selected cases (renal insufficiency, absent deep tendon reflexes). To control fits, optimum serum magnesium level is 4.8–8.4 mg/dL (4-7 mEq/L) to be maintained. Magnesium toxicity and serum Mg level is seen as: (a) loss of deep tendon reflexes > 7 mEq/L; (b) respiratory depression more than 10 mEq/L and (c) cardiac arrest more than 25 mEq/L.

Magnesium sulfate is continued for 24 hours after the last seizure or delivery whichever is later. For **recurrence of fits**, further 2 g IV bolus is given over 5 minute in the above regimens. If the patient seizes, despite magnesium therapy, midazolam 1–2 mg IV is given (and may be repeated in 5–10 minutes time).

Monitoring of Magnesium Toxicity

- Loss of deep tendon reflexes
- Decreased respiratory rate (<12 per minute)
- Urine output (< 30 mL/h)
- Chest pain, heart block

Management for Magnesium Toxicity

- Stop magnesium therapy
- Estimation of serum magnesium and creatinine levels
- Injection calcium gluconate 10 mL (10% solution), IV

Other regimens are: (1) Lytic cocktail (Menon 1961) using chlorpromazine, promethazine and pethidine. (2) Diazepam (Lean) and (3) Phenytoin. **Compared to other regimes, magnesium sulfate has got the following benefits:** (i) it controls fits effectively without any depression effect to the mother or the infant. (ii) reduced risk of recurrent convulsions (9%) (iii) significantly reduced maternal death rate (3%) and (iv) reduced perinatal mortality rate.

Antihypertensives and diuretics: Inspite of anticonvulsant regime, if the blood pressure remains more than 160/110 mm Hg, antihypertensive drugs should be administered. First line of antihypertensive drugs are: labetalol and hydralazine (ACOG-2011). Target level of BP is SBP; 140–160 mm Hg and DBP: 90–100 mm Hg. Labetalol 20 mg IV is given. Repeat doses may be needed after an interval of 10 minute. Alternatively hydralazine 5 or 10 mg IV is given. Repeat dose may be needed if no response occurs after 20 minutes time.

Presence of pulmonary edema requires diuretics. In such cases, the potent one (furosemide) should be administered in doses of 20–40 mg intravenously and to be repeated at intervals.

Management during fit: (a) In the premonitory stage, a mouth gag is placed in between the teeth to prevent tongue bite and should be removed after the clonic phase is over. (b) The air passage is to be cleared off the mucus with a mucus sucker. The patient's head is to be turned to one side and the pillow is taken off. Raising the foot end of the bed, facilitates postural drainage of the upper respiratory tract. (c) Oxygen is given until cyanosis disappears.

Status eclampticus: **Thiopentone sodium 0.5 g** dissolved in 20 mL of 5% dextrose is given intravenously very slowly. The procedure should be supervised by an expert anesthetist. **If the procedure fails**, use of complete anesthesia, muscle relaxant and assisted ventilation may be employed. **In unresponsive cases**, cesarean section in ideal surroundings may be a lifesaving attempt.

INDICATIONS OF INTUBATION

- Patient remains unconscious in post seizure period
- Seizures not controlled
- Signs of aspiration
- Persistent hypoxia

Treatment of complications: Prophylactic use of antibiotics markedly reduces the complications like pulmonary and puerperal infection.

Pulmonary edema: Furosemide 40 mg IV followed by 20 g of mannitol IV reduces pulmonary edema and also prevents adult respiratory distress syndrome. **Pulse oximeter** is very useful to monitor such a patient. Aspiration of the mucus from the tracheobronchial tree by a suction apparatus is done.

Heart failure: Oxygen inhalation, parenteral lasix and digitalis are used. For details see p. 278.

Anuria: The treatment should be in the line as formulated in the chapter of anuria (Chapter 39). Dopamine infusion (1 µg/kg) is given with oliguria when CVP is above 8 mm Hg. It is often surprising that urine output returns to normal following delivery.

Hyperpyrexia: It is difficult to bring down the temperature as it is central in origin. However, cold sponging and antipyretics may be tried.

Psychosis: Chlorpromazine or Eskazine (trifluoperazine) is quite effective.

Intensive care monitoring: Patient with multiple organ dysfunction needs to be admitted in an intensive care unit. **Multidisciplinary approach:** obstetrician, obstetric nurse, anesthesiologist, neonatologist, intensive care unit team should be involved. Cardiac, renal or pulmonary complications are managed

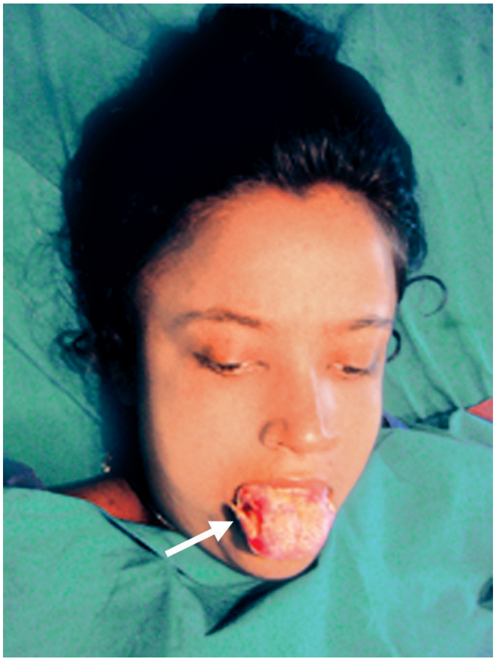


Fig. 18.3: Extensive tongue bite injury following an eclamptic convulsion. It usually occurs in *clonic stage*

effectively. Use of blood gas analyzer (to detect hypoxia and acidosis), pulse oximeter and central venous pressure monitoring should be done depending on individual case (see Chapter 39). A deeply unconscious patient with raised intracranial pressure needs steroid and or diuretic therapy. Neuroradiologic imaging is strongly advised in the postpartum period for cases with neurologic symptoms and focal deficit.

OBSTETRIC MANAGEMENT: During pregnancy: In majority of cases with antepartum eclampsia, labor starts soon after convulsions. But when labor fails to start, the management depends on—(i) whether the fits are controlled or not and (ii) the maturity of the fetus. The decision to deliver is made once the woman is stable.

- **Fits controlled:**

— *Baby mature:* **Delivery should be done**. (a) If the cervix is favorable and there is no contraindication of vaginal delivery, surgical induction by low rupture of the membranes is done. Oxytocin drip may be added, if needed. (b) When the cervix is unfavorable, cervical ripening with PGE_2 gel or pessary could be achieved before ARM. (c) If the cervix is unfavorable and/or there is obstetric contraindication of vaginal delivery, cesarean section is done.

— *Baby premature* (<37 weeks): Delivery is recommended in a set up with neonatal intensive care unit (NICU). The underlying disease process of preeclampsia and eclampsia persists until the woman delivers. At times the disease process may flare up. Moreover, there lies the risk of recurrent convulsions and IUFD. Steroid therapy is given when pregnancy is less than 34 weeks (p. 367). **Conservative management** at very early pregnancy may improve perinatal outcome but this must be carefully balanced with maternal well-being (RCOG-2006).

— *Baby dead:* The preeclamptic process gradually subsides and eventually expulsion of the fetus occurs. Otherwise medical method of induction is started (see p. 598).

- **Fits not controlled:** If the fits are not controlled with anticonvulsant within a reasonable period (6–8 hours), termination of pregnancy should be done. If vaginal examination indicates a quick response to induction, low rupture of the membranes is done. Oxytocin infusion may be added. The uterus responds well to oxytocin in such cases. In presence of unfavorable factors, cesarean section gives a quick response.

During labor: In the absence of any contraindication to vaginal delivery, as soon as the labor is well established, low rupture of the membranes is to be done to accelerate the labor. The dose schedule of antihypertensive and anticonvulsant drugs may be increased to quieten the patient. Second stage should be curtailed by forceps, ventouse or craniotomy, if the baby is dead. Prophylactic intravenous ergometrine or syntometrine following the delivery of the anterior shoulder should not be given as it may produce further rise of blood pressure. Instead, 10 units of oxytocin IM or IV slowly should be given. One should remain vigilant about postpartum hemorrhage and shock.

Indications of cesarean section: (i) Uncontrolled fits in spite of therapy. (ii) Unconscious patient and poor prospect of vaginal delivery. (iii) Obstetric indications (malpresentation).

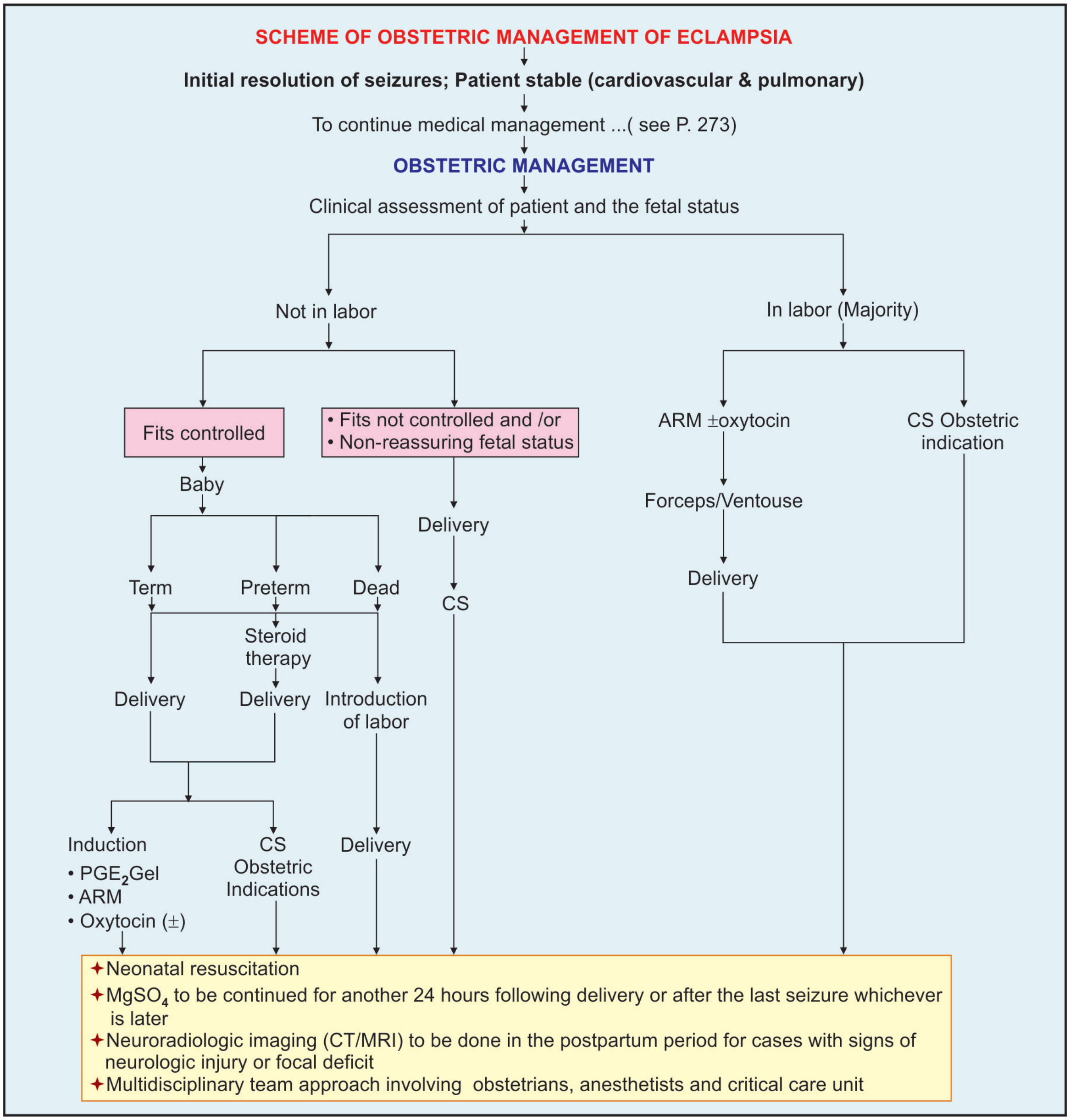
Follow-up and prognosis: Patient should be followed up in the postnatal clinic by 6 weeks time. Persistence of hypertension, proteinuria and abnormal blood biochemistry necessitates further investigation and consultation with a physician. Further pregnancy should be deferred till they are controlled.

Recurrence risk varies between 2% and 25%. The risk of preeclampsia and eclampsia to the daughter of an eclampsia patient is about 25% and 3%, respectively.

Atypical preeclampsia is defined as the development of preeclampsia (even eclampsia) without fulfilling the standard definition or criteria (hypertension or proteinuria). The common presentations are:

♦ Early onset preeclampsia/eclampsia less than 20 weeks ♦ Late postpartum preeclampsia, eclampsia more than 48 hours postpartum ♦ Women with gestational hypertension or gestational proteinuria presenting with symptoms of (a) Preeclampsia (b) Thrombocytopenia (c) Elevated liver enzymes.

Women with atypical preeclampsia and who have other diagnostic criteria of severe preeclampsia should be treaed as if they have severe preeclampsia. Patients are also treated with parenteral $MgSO_4$.


SCHEME OF OBSTETRIC MANAGEMENT OF ECLAMPSIA
Initial resolution of seizures; Patient stable (cardiovascular & pulmonary)
To continue medical management ...(see P. 273)
OBSTETRIC MANAGEMENT
Clinical assessment of patient and the fetal status
Not in labor
In labor (Majority)
Fits controlled
• Fits not controlled and /or
• Non-reassuring fetal status
ARM ±oxytocin
CS Obstetric indication
Baby
Delivery
Forceps/Ventouse
Term
Preterm
Dead
CS
Delivery
Steroid therapy
Delivery
Delivery
Introduction of labor
Induction
• PGE2Gel
• ARM
• Oxytocin (±)
CS Obstetric Indications
Delivery
✦Neonatal resuscitation
✦MgSO4 to be continued for another 24 hours following delivery or after the last seizure whichever is later
✦Neuroradiologic imaging (CT/MRI) to be done in the postpartum period for cases with signs of neurologic injury or focal deficit
✦Multidisciplinary team approach involving obstetrians, anesthetists and critical care unit

GESTATIONAL HYPERTENSION

A sustained rise of blood pressure to 140/90 mm Hg or more on at least two occasions 4 or more hours apart beyond the 20th week of pregnancy or within the first 48 hours of delivery in a previously normotensive woman is called gestational hypertension. It is associated with a much higher incidence of essential hypertension in later life than preeclampsia. Both, thus appear to be two phases of the same disorder. **It should fulfill the following criteria**: (1) Absence of any evidences for the underlying cause of hypertension (2) Generally unassociated with other evidences of preeclampsia (edema or proteinuria). (3) Majority of cases are more than or equal to 37 weeks pregnancy. (4) Generally not associated with hemoconcentration or thrombocytopenia, raised serum uric acid level or hepatic dysfunction. (5) The blood pressure should come down to normal within 12 weeks following delivery.

However, gestational HTN may go to proteinuric phase and may evolve to preeclampsia. It is a retrospective diagnosis.

The hypertensive effect may be a stress response. There are no longer any real differences in management between PE and gestational HTN, in terms of BP management and in the decision to deliver. A case of severe HTN, with appearance of symptoms or abnormal laboratory values, suggests delivery.

Gestational edema is excessive accumulation of fluid with demonstrable pitting edema over the ankles greater than 1 + after 12 hours in bed or gain in weight of 2 kg or more in a week due to influence of pregnancy.

Gestational proteinuria is the presence of protein of more than 0.3 g in the 24 hours urine during or under the influence of pregnancy in the absence of hypertension, edema or renal infection. It may be orthostatic proteinuria.

CHRONIC HYPERTENSION IN PREGNANCY

Chronic hypertensive disease (CHD) is defined as the presence of hypertension of any cause antedating or before the 20th week of pregnancy and its presence beyond the 12 weeks after delivery. The condition poses a difficult problem as regards the diagnosis and management when seen for the first time, beyond the 20th week of pregnancy. Overall incidence is 2–4% of which 90% are due to essential hypertension.

The high risk factors for CHD are: (i) Age (> 40 years), (ii) Duration of hypertension (>15 years), (iii) Level of BP (>160/110 mm of Hg), (iv) Presence of any medical disorder (renovascular), and (v) Presence of thrombophilias. Majority of women with CHD are low risk and have satisfactory maternal and fetal outcome without any antihypertensive therapy.

ESSENTIAL HYPERTENSION IN PREGNANCY

Apart from the specific hypertensive disorder in pregnancy (PIH), essential hypertension is the common hypertensive state in pregnancy. Its incidence varies from 1% to 3%.

DIAGNOSIS: The diagnostic criteria are: (1) Rise of blood pressure to the extent of 140/90 mm Hg or more during pregnancy prior to the 20th week (molar pregnancy excluded), (2) Cardiac enlargement on chest radiograph and ECG, (3) Presence of medical disorders, and (4) Prospective follow-up shows persistent rise of blood pressure even after 42 days following delivery. However, confusion in the diagnosis arises when the case is first seen in later months of pregnancy, especially when the pre-pregnant level of blood pressure remains unknown. **Differential diagnosis with preeclampsia, gestational hypertension and essential hypertension are given below**.

EFFECTS OF PREGNANCY ON THE DISEASE: (1) **There may be a midpregnancy fall of blood pressure in about 50%**. However, the blood pressure tends to rise in the last trimester which may or may not reach its previous level, (2) **In 50%, the blood pressure tends to rise** progressively as pregnancy advances, (3) In about 20%, it is superimposed by preeclampsia evidenced by rise of blood pressure to the extent of 30 mm Hg systolic and 15 mm Hg diastolic associated with edema and/or proteinuria, (4) Rarely, malignant hypertension supervenes, (5) In 30%, there is permanent deterioration of the hypertension following delivery.

EFFECT OF THE DISEASE ON PREGNANCY: Maternal risk: In the milder form, the maternal risk remains unaltered but in the severe form or when superimposed by preeclampsia, the maternal risk is much increased.

Fetal risk: Due to chronic placental insufficiency, the babies are likely to be growth retarded. Preterm birth is high. In the milder form, with the blood pressure less than 160/100 mm Hg, the perinatal loss is

Table 18.7: Differential Features of Preeclampsia with Gestational and Essential Hypertension

Parameters	Preeclampsia	Gestational Hypertension	Essential Hypertension
Age	Mostly young	Young	Usually elderly
Parity	Primigravidae — common	Primigravidae	Multipara — common
Past history	Preeclampsia in previous pregnancy	May be present	Pre-pregnant hypertension present
Family history	May be present	Unrelated	Often present
Onset of hypertension	After 20th week of pregnancy	Usually in third trimester	Before 20th week of pregnancy
Follow-up BP following delivery	Subsides completely	Subsides completely	Persists even after 3 months
Proteinuria	Present	Absent	Usually absent
Eye changes	Usually none. Extreme cases—retinal edema, constriction of arterioles, nicking of the veins	None	Silver wiring of the arterioles. Hypertensive retinopathy
Specific blood values	■ Hemoconcentration + ■ Thrombocytopenia + ■ Serum uric acid > 5 mg/dL ■ Raised liver enzymes	Absent	Not significant

about 10%. **When blood pressure exceeds 160/100 mm Hg, the perinatal loss increases by three to four times and when complicated by preeclampsia, it increases further. Risk of placental abruption is high (0.5–10%).**

MANAGEMENT: The principles of management are: (1) **To stabilize** the blood pressure to below 160/100 mm Hg, (2) **To prevent** superimposition of preeclampsia, (3) **To monitor** the maternal and fetal well-being, (4) **To terminate** the pregnancy at the optimal time.

Preconceptional evaluation and counseling is essential to assess the etiology, severity of hypertension and possible outcome of pregnancy.

GENERAL MANAGEMENT: In mild cases with blood pressure less than 160/100 mm Hg, adequate rest (physical and mental), low-salt diet are all that are needed. The check up should be more frequent 1–2 weeks interval up to 28 weeks and thereafter weekly.

In severe cases or in cases of superimposed preeclampsia, the patients should be hospitalized and are placed in the treatment protocol as described under preeclampsia.

Antihypertensive drugs: Routine use of antihypertensive drug is not favored. **It may lower the blood pressure and thereby benefit the mother but the diminished pressure may reduce the placental perfusion which may be detrimental to the fetus.** Thus, antihypertensive drugs (methyldopa, labetalol, nifedipine or hydralazine) should be used only when the pressure is raised beyond 160/100 mm Hg. (see p. 581), to prevent target organ damage (stroke, renal or cardiac failure). In cases, where these drugs have been used before pregnancy, care should be taken to adjust the dose during pregnancy, especially, during midpregnancy when the blood pressure tends to fall.

OBSTETRIC MANAGEMENT: In mild cases, spontaneous labor is awaited. In severe or complicated cases, the aim is to try to continue the pregnancy to at least 34 weeks otherwise up to the 37th week to attain fetal maturity and then to terminate the pregnancy.

CHRONIC RENAL DISEASES IN PREGNANCY

The overall incidence of chronic renal disease in pregnancy is rare (0.2%).

Features suggestive of chronic renal disease in pregnancy are:

A. Urine on microscopic examination

(i) RBCs more than 1–2/HPF or RBC casts, (ii) Increased number of WBCs or casts

B. Serum levels of uric acid and creatinine raised.

MILDLY COMPROMISED RENAL FUNCTION: (Serum creatinine < 125 µmol/L) is generally not associated with any adverse maternal or fetal outcome. Effect of pregnancy on long-term renal function and development of end-stage renal failure (serum creatinine > 500 µmol/L) or the need of dialysis is very low (5%).

Moderately or severely compromised renal function (High serum creatinine > 125 µmol/L) is associated with adverse pregnancy outcome (50%) as the renal function deteriorates.

Effects of renal disease on pregnancy: Pregnancy outcome depends on the level of (i) hypertension, (ii) proteinuria, and (iii) serum creatinine. Miscarriage, preterm labor, IUGR and IUFD are the known fetal risks. However, with improved pregnancy surveillance and neonatal care, outcome has improved. Superimposed preeclampsia adversely affects the course and perinatal loss may go as high as up to 40–60%.

Effects of pregnancy on renal disease: It depends on the severity of renal disease. When the renal function is mildly compromised (serum creatinine < 125 mmol/L) the risk of end-stage renal failure is low (5%).

On the contrary renal failure may be as high as 10% when renal function is compromised moderately (Sr Cr 125–250 µmol/L) or severely (Sr Cr > 250 µ mol/L). Superimposed preeclampsia worsens the prognosis. The prognosis of membranoproliferative glomerulonephritis (GN), focal glomerulosclerosis or immunonephropathy (IgA) is poor when compared to primary glomerulonephritis.

MANAGEMENT: Pre-pregnancy counseling—Women with severely compromised renal function should be discouraged to become pregnant as the risks of developing end stage renal failure is high.

Antenatal care is done with periodic assessment of renal function. A 24-hour urine collection is done for creatinine clearance and total protein excretion. Antihypertensive therapy is started early (Nifedipine, hydralazine, colinidine or beta blockers) to preserve renal function (see p. 581). Diuretics may be used in cases with massive edema. Regular hemodialysis during pregnancy in a patient with moderate renal compromise may improve the outcome. Sudden volume shift and hypotension should be avoided.

Women planning pregnancy following renal transplant should maintain the following: (a) Plasma creatinine <1.5 mg/dL (<130 µmol/L); (b) Controlled hypertension; (c) No or minimal proteinuria; (d) On maintenance level of immunosuppression; (e) No evidence of graft rejection.

Anemia from chronic renal insufficiency may be treated with recombinant erythroprotein.

Fetal surveillance is maintained more closely. Timing of delivery is decided on individual basis depending upon the control of hypertension, fetal viability and the level of renal function.

KEY POINTS—PREECLAMPSIA AND ECLAMPSIA

- Hypertension in pregnancy is the **most common** medical complication.
- **Preeclampsia** and **eclampsia** are the leading causes of maternal mortality and morbidity in India and worldwide.
- **Preeclampsia** is a syndrome of multiple organ dysfunction (see p. 256). It is peculiar to the pregnant state. It usually manifests for the first time beyond the 20th week and is characterized by the appearance of **hypertension to the extent of 140/90 mm Hg or more and proteinuria with or without pathological edema.**
- **The etiology remains obscure** (see p. 257) but the basic pathology is **endothelial dysfunction and vasospasm.**
- **Etiopathological basis of preeclampsia** (see p. 257) is thought to be an imbalance of vasodilatory factors (PGI_2, PGE_2, VEGF, NO) with that of vasoconstrictors (TXA_2, ROS, Superoxide radicals, lipid peroxides and Endothelin-1) resulting in endothelial dysfunction and vasospasm.
- **Endothelial dysfunction** leads to increased capillary permeability (leak). This is manifested as: ascites, pulmonary edema, proteinuria, pleural effusion and activation of coagulation system (thrombocytopenia, DIC, HELLP syndrome). Others are: visual disturbances, retinal hemorrhage.

contd...

contd...

- **Pregnancy-induced hypertension (PIH)** includes (i) Gestational hypertension, (ii) Preeclampsia, and (iii) Eclampsia.
- **According to gestational age**, preeclampsia may be **A.** Early <34 weeks, and **B.** Late >34 weeks. Late-onset preeclampsia often has favorable outcome.
- **The pathological organ changes** due to vasospasm are more evident in **Uteroplacental bed** → IUGR, placental abruption; **Kidney** → proteinuria, oliguria, renal failure; **Liver** → necrosis, subcapsular hematoma, raised level of AST and ALT; **CNS** → seizures. Posterior Reversible Encephalopathy Syndrome (PRES), involving the parietal and occipital lobes, cortical blindness; (see p. 259).
- **The clinical manifestations are based** on the pathological changes, e.g. Vasoconstriction → hypertension, Diminished organ perfusion → oliguria, IUGR; ↑ Vascular permeability → edema and proteinuria; Endothelial damage → thrombocytopenia and HELLP syndrome (see p. 258).
- **Preeclampsia may be mild or severe** (see p. 260) and the **complications** involve both the mother and the fetus if left untreated (see p. 263).
- **Prediction and prevention** — No screening test is helpful (see p. 264) but presence of high risk factors may help to identify an individual (see p. 256).
- **Management of Eclampsia** and **severe preeclampsia** includes A. Control of fits and B. Control of hypertension. Drug of choice for control of fits is magnesium sulfate (IM/IV regimens). **First line antihypertensives** are (a) Labetalol (b) Hydralazine or (c) Nifedipine. Target levels BP to control is SBP = 140 – 160 mm of Hg and DBP = 90 – 100 mm of Hg
- **Termination of pregnancy (delivery)** is the only definitive treatment for preeclampsia.
- **Acute fulminant preeclampsia** (see p. 267) runs the high risk of eclampsia. Prophylactic anticonvulsant therapy ($MgSO_4$) is recommended (MAGPIE Trial — 2002).
- **Eclampsia** (see p. 268) a complication of preeclampsia is characterized by grandmal seizures. Eclampsia is a significant cause of maternal death.
- **Convulsions in eclampsia** has got four stages (see p. 270): Premonitory stage, Tonic stage, Clonic stage and stage of Coma.
- **Complications of eclampsia** are many (see p. 271). Prevention of eclampsia depends on early detection and management of preeclampsia.
- **Eclampsia should be managed** in a teritary care hospital. Principles of management involves general care of the patient, to arrest convulsions, control of hypertension and to expedite delivery (see p. 271).
- **Women with eclampsia should be delivered** within a period of 6–8 hours even if the fits are not controlled (see p. 276).
- **Gestational hypertension** (see p. 276) is not associated with edema, proteinuria or other hematological changes. Blood pressure usually subsides within 12 weeks following delivery.
- **Preeclampsia** needs to be differentiated (see p. 278) from essential hypertension and chronic renal diseases. Prognostically each has got different pregnancy outcome.
- **Patients with mild preeclampsia** ≥37 weeks or severe preeclampsia ≥34 weeks should be delivered.
- **Patients with severe preeclampsia** of <34 weeks should be delivered if criteria mentioned in table p. 268 are met.
- **Control of BP** should be to a level between 140 mm Hg and 160 mm Hg systolic and between 90 mm Hg and 105 mm Hg diastolic.
- **$MgSO_4$ should be used** in patients with eclampsia, HELLP Syndrome, severe preeclampsia and in patients with unstable mild preeclampsia.
- **Corticosteroids** should be used for cases with preterm delivery and in cases with HELLP syndrome.
- **Patients with gestational hypertension/atypical preeclampsia** and who have other diagnostic criteria of severe preeclampsia should be treated as if they have severe preeclampsia.
- **There are no longer any real differences** in management between PE and gestational hypertension, in terms of BP management and in the decision to deliver. Gestational hypertension may go to proteinuric phase and may evolve to preeclampsia.

QUESTIONS

1. Define preeclampsia? What is severe preeclampsia? Mention the different complications of preeclampsia? (p. 256, 260, 263)
2. A 27-year-old woman primigravida was admitted with hypertension (150/100 mm Hg) and proteinuria at 35 weeks of gestation. Discuss in brief the management for this woman? (p. 264)
3. **Write Short Notes on:**
    A. Complications of eclampsia (p. 271)
    B. Magnesium sulfate (p. 273)
    C. HELLP Syndrome (p. 258)

Related theory questions (Long and Short), Obstetric Case Discussions, Viva table discussions, Postoperative word round discussions, and MCQs are discussed in author's books:

1. **Bedside Clinic and Viva Voce:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

For further reading:

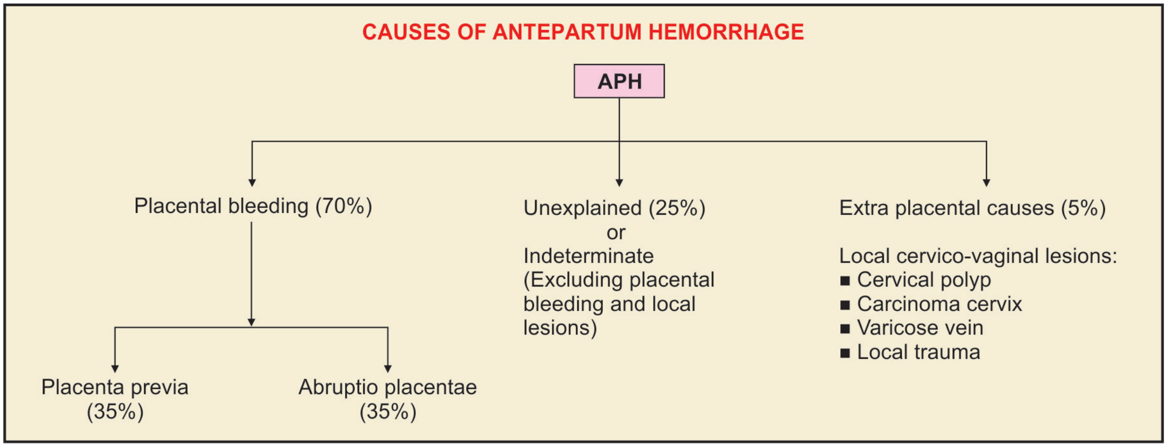
2. **Master pass in Obstetrics and Gynaecology**: 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

Chapter 19

Antepartum Hemorrhage

DEFINITION: It is defined as bleeding from or into the genital tract after the 28th week of pregnancy but before the birth of the baby (the first and second stage of labor are thus included). The 28th week is taken arbitrarily as the lower limit of fetal viability. The incidence is about 3% amongst hospital deliveries.

CAUSES: The causes of antepartum hemorrhage fall into the following categories. The hospital figures do not give a true picture of the incidence of the different varieties. However, on an average, the incidence of placenta previa, abruptio placentae and the indeterminate group is almost the same.


CAUSES OF ANTEPARTUM HEMORRHAGE

APH
Placental bleeding (70%)
Unexplained (25%) or Indeterminate (Excluding placental bleeding and local lesions)
Extra placental causes (5%)
Local cervico-vaginal lesions:
■ Cervical polyp
■ Carcinoma cervix
■ Varicose vein
■ Local trauma
Placenta previa (35%)
Abruptio placentae (35%)

PLACENTA PREVIA

DEFINITION: When the placenta is implanted partially or completely over the lower uterine segment (over and adjacent to the internal os) it is called placenta previa. The term previa (L, in front of) denotes the position of the placenta in relation to the presenting part.

INCIDENCE: About one-third cases of antepartum hemorrhage belong to placenta previa. The incidence of placenta previa ranges from 0.5% to 1% amongst hospital deliveries. In 80% cases, it is found in multiparous women. The incidence is increased beyond the age of 35 years, with high birth order pregnancies and in multiple pregnancy. Increased family planning acceptance with limitation and spacing of birth lowers the incidence of placenta previa.

ETIOLOGY

The exact cause of implantation of the placenta in the lower segment is not known. **The following theories are postulated**.

- **Dropping down theory:** The fertilized ovum drops down and is implanted in the lower segment. Poor decidual reaction in the upper uterine segment may be the cause. Failure of zona pellucida to disappear in time can be a hypothetical possibility. This explains the formation of central placenta previa.
- **Persistence of chorionic activity** in the decidua capsularis and its subsequent development into capsular placenta which comes in contact with decidua vera of the lower segment can explain the formation of lesser degrees of placenta previa.
- **Defective decidua**, results in spreading of the chorionic villi over a wide area in the uterine wall to get nourishment. During this process, not only the placenta becomes membranous but encroaches onto the lower segment. Such a placenta previa may invade the underlying decidua or myometrium to cause placenta accreta, increta or percreta (see p. 486).
- **Big surface area of the placenta** as in twins may encroach onto the lower segment.

The high risk factors for placenta previa are — (a) Multiparity (b) Increased maternal age (> 35 years) (c) History of previous cesarean section or any other scar in the uterus (myomectomy or hysterecotomy) (d) Placental size (mentioned before) and abnormality (succenturiate lobes) (e) Smoking — causes placental hypertrophy to compensate carbon monoxide induced hypoxemia (f) Prior curettage.

PATHOLOGICAL ANATOMY:

Placenta—The placenta may be large and thin. There is often a tongue-shaped extension from the main placental mass. Extensive areas of degeneration with infarction and calcification may be evident. The placenta may be morbidly adherent due to poor decidua formation in the lower segment.

Umbilical cord—The cord may be attached to the margin (battledore) or into the membranes (velamentous). The insertion of the cord may be close to the internal os or the fetal vessels may run across the internal os in velamentous insertion giving rise to **vasa previa,** which may rupture along with rupture of the membranes.

Lower uterine segment—Due to increased vascularity, the lower uterine segment and the cervix becomes soft and more friable.

TYPES OR DEGREES: (Fig. 19.1)—There are four types of placenta previa depending upon the degree of extension of placenta to the lower segment.

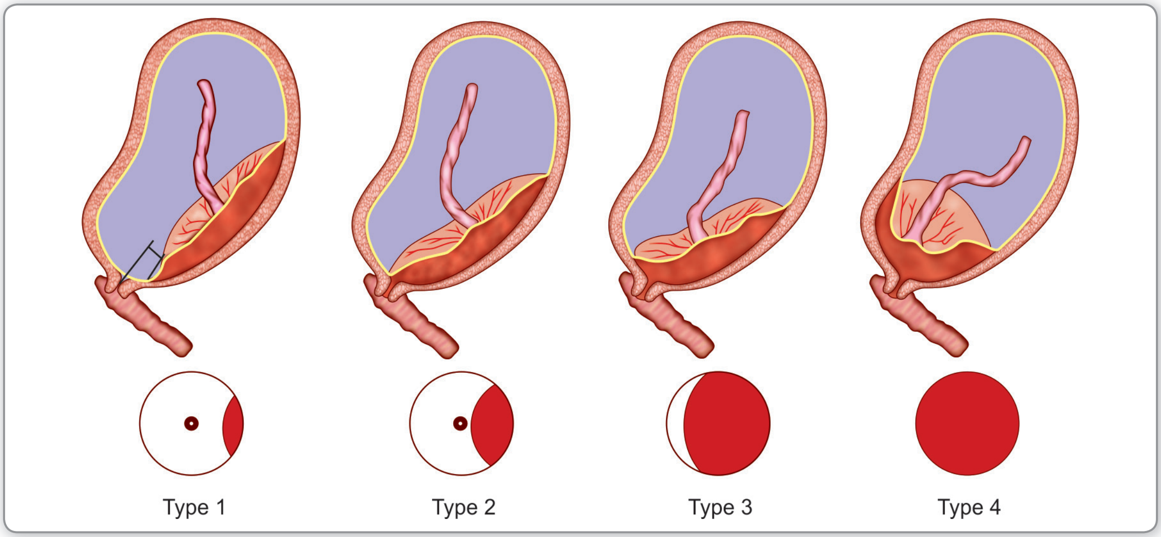


Fig. 19.1: Degrees of placenta previa with findings on ultrasound examination

Type—I (Low-lying): The major part of the placenta is attached to the upper segment and only the lower margin encroaches onto the lower segment but not up to the os.

Type—II (Marginal): The placenta reaches the margin of the internal os but does not cover it.

Type—III (Incomplete or partial central): The placenta covers the internal os partially (covers the internal os when closed but does not entirely do so when fully dilated).

Type—IV (Central or total): The placenta completely covers the internal os even after it is fully dilated.

Currently with ultrasound precision, more accurate placental location is made in relation to the cervical internal os. In the majority, the placenta lies either in the anterior or posterior wall, the latter is more common. Type III and IV constitute about one-third of the cases. **For clinical purpose, the types are graded into mild degree (Type-I and II anterior) and major degree (Type-II posterior, III and IV)**.

Dangerous placenta previa is the name given to the type-II posterior placenta previa (Fig. 19.2). (1) Because of the curved birth canal major thickness of the placenta (about 2.5 cm) overlies the sacral promontory, thereby diminishing the anteroposterior diameter of the inlet and prevents engagement of the presenting part. This hinders effective compression of the separated placenta to stop bleeding. (2) Placenta is more likely to be compressed, if vaginal delivery is allowed. (3) More chance of cord compression or cord prolapse. The last two may produce fetal anoxia or even death.

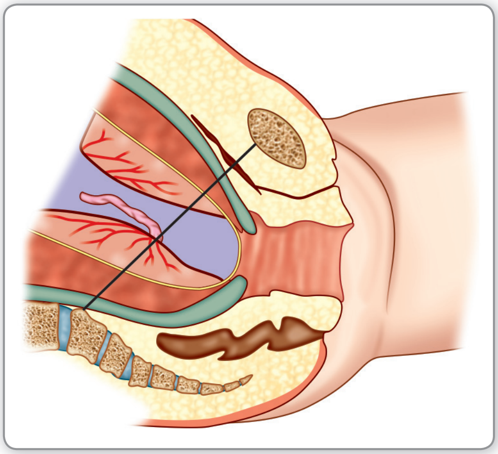


Fig. 19.2: Type II posterior placenta previa. Note the effective reduction of the anteroposterior diameter of the inlet in contrast to type II anterior placenta previa

CAUSE OF BLEEDING: As the placental growth slows down in later months and the lower segment progressively dilates, the inelastic placenta is sheared off the wall of the lower segment. This leads to opening up of uteroplacental vessels and leads to an episode of bleeding. **As it is a physiological phenomenon which leads to the separation of the placenta, the bleeding is said to be inevitable**. However, the separation of the placenta may be provoked by trauma including vaginal examination, coital act, external version or during high rupture of the membranes. **The blood is almost always maternal**, although fetal blood may escape from the torn villi especially when the placenta is separated during trauma.

The mechanisms of spontaneous control of bleeding are: (1) Thrombosis of the open sinuses. (2) Mechanical pressure by the presenting part. (3) Placental infarction.

Placental migration: Ultrasonography at 17 weeks of gestation reveals placenta covering the internal os in about 10% of cases. Repeat ultrasonography at 37 weeks showed no placenta in the lower uterine segment in more than 90% of cases. Lower uterine segment expands from 0.5 cm at 20 weeks to more than 5 cm (10 fold) at term. The term **placental migration** (though misnomer) could be explained in two ways : (i) with the progressive increase in the length of lower uterine segment, the lower placental edge relocates away from the cervical os (ii) due to **trophotropism** (growth of trophoblastic tissue towards the fundus), there is resolution of placenta previa.

CLINICAL FEATURES

SYMPTOMS: The only symptom of placenta previa is vaginal bleeding. The classical features of bleeding in placenta previa are **sudden onset, painless, apparently causeless and recurrent**. In about 5% cases, it occurs for the first time during labor, especially in primigravidae. In about one-third of cases, there is a history of "warning hemorrhage" which is usually slight.

The bleeding is unrelated to activity and often occurs during sleep and the patient becomes frightened on awakening to find herself in a pool of blood. **The bleeding is unassociated with pain unless labor starts simultaneously**. Obvious causes for the placental separation such as trauma or hypertension are usually absent. However, preeclampsia may complicate a case of placenta previa. The first bout of bleeding is usually not alarming but subsequent bouts may be heavier than the previous one due to separation of fresh areas of placenta. In majority of cases, bleeding occurs before 38 weeks and **earlier bleeding is more likely to occur in major degrees**. However, there may not be any bleeding in central placenta previa until labor starts. Asymptomatic cases may be detected by sonography or at the time of cesarean section.

SIGNS: General condition and anemia are proportionate to the visible blood loss. But in the tropics, the picture is often confusing due to preexisting anemia.

Abdominal examination:

- **The size of the uterus** is proportionate to the period of gestation.
- **The uterus feels** relaxed, soft and elastic without any localized area of tenderness.
- **Persistence of malpresentation** like breech or transverse or unstable lie is more frequent. There is also increased frequency of twin pregnancy.
- **The head is floating** in contrast to the period of gestation. Persistent displacement of the fetal head is very suggestive. The head cannot be pushed down into the pelvis.
- **Fetal heart sound** is usually present, unless there is major separation of the placenta with the patient in exsanguinated condition. Slowing of the fetal heart rate on pressing the head down into the pelvis which soon recovers promptly as the pressure is released is suggestive of the presence of low lying placenta especially of posterior type (***Stallworthy's sign***). But this sign is not always significant because it may be due to fetal head compression even in an otherwise normal case.

Vulval inspection: Only inspection is to be done to note whether the bleeding is still occurring or has ceased, character of the blood—bright red or dark colored and the amount of blood loss—to be assessed from the blood-stained clothing. **In placenta previa**, **the blood is bright red** as the bleeding occurs from the separated uteroplacental sinuses close to the cervical opening and escapes out immediately.

Vaginal examination must not be done outside the operation theater in the hospital, as it can provoke further separation of placenta with torrential hemorrhage and may be fatal. It should only be done prior to termination of pregnancy in the operation theater under anesthesia, keeping everything ready for cesarean section.

CONFIRMATION OF DIAGNOSIS

DIAGNOSIS: *Painless and recurrent* vaginal bleeding in the second half of pregnancy should be taken as placenta previa unless proved otherwise. Ultrasonography is the initial procedure either to confirm or to rule out the diagnosis.

I. Localization of Placenta (Placentography)		II. Clinical
■ **Sonography** ● Transabdominal ultrasound (TAS) ● Transvaginal ultrasound (TVS) ● Transperineal ultrasound ● Color Doppler flow study ● 3D Power Doppler study	■ **Magnetic resonance imaging (MRI)** For better diagnosis of ● placenta previa and ● placenta previa accreta	♦ By internal examination (double set up examination) ♦ Direct visualization during cesarean section ♦ Examination of the placenta following vaginal delivery

PLACENTOGRAPHY

- **Sonography:** Sonography is the diagnostic technique of choice (RCOG-2001). It provides the simplest, most precise and safest method of placental localization (Fig.19.3).

In addition, it is helpful for assessing the fetal size and status. It also provides information pertaining to maturity and well being of the fetus for guiding the management.

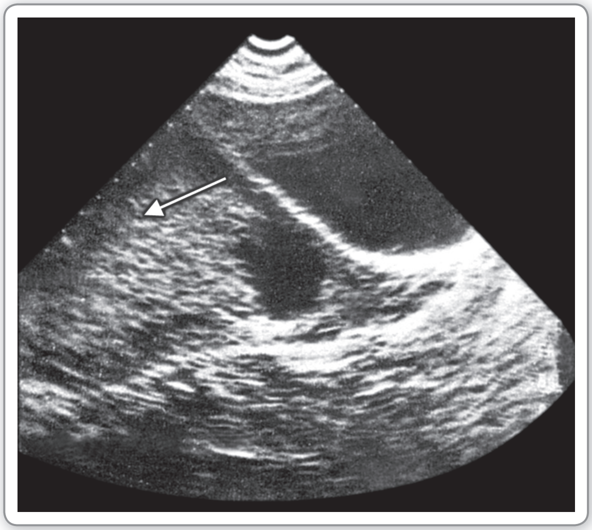


Fig. 19.3: Ultrasonography showing central placenta previa (see arrow)

♦ **Transabdominal (TAS):** The accuracy after 30th week of gestation is about 98%. False positive result may be due to full bladder or myometrial contractions. Poor imaging could be due to maternal obesity and posteriorly situated placenta. The reasons for **poor imaging in a posteriorly situated placenta are**—(a) acoustic shadow from the fetal presenting part may obscure the placental view, (b) there is no anatomical landmark posteriorly (anteriorly uterovesical angle) below which placenta is defined as previa (an arbitary distance of 5 cm from the internal os is considered as lower segment). As such, a positive case should be subjected to repeat scan after emptying the bladder. Cases of placenta previa detected in earlier weeks should be subjected to repeat scan at 36 weeks or earlier for detection of placental migration (see above).

♦ **Transvaginal (TVS):** Transducer is inserted within the vagina without touching the cervix. The probe is very close to the target area and higher frequencies could be used to get a superior resolution. It is safe, obviates the discomfort of full bladder and is more accurate (virtually 100%) than TAS. Complete placenta previa diagnosed in the second trimester will persist into the third trimester in 26% of cases, whereas marginal placenta previa with persist in only 2.5% cases.

♦ **Transperineal (TPS):** This is well accepted by patients. Internal os is visualized in 97–100% of cases.

♦ **Color Doppler:** Diffuse vascular lakes with turbulent flow in the hypoechoic areas near the cervix is consistent with the diagnosis of placenta previa. **Three-dimensional (3-D) Power Doppler is the best**. Hypervascularity at the uterine serosa – bladder junction is diagnostic.

♦ **Magnetic Resonance Imaging (MRI):** It is a noninvasive method without any risk of ionizing radiation. Dark intraplacental bands are seen on T2 weighted images. MRI is better than ultrasonography to diagnose posterior placenta previa and placenta previa accreta. **Limitations of MRI are** more time consuming, lack of portability and the cost.

- **Advantages of Ultrasonography and MRI:** (1) Need of vaginal examination with the risk of hemorrhage is avoided. (2) The need of prolonged and unnecessary hospital stay in patients with clinical diagnosis of APH can be reduced. (3) Diagnosis of placenta previa can be made even before the bleeding starts. (4) Diagnosis of morbid adherent placenta (specially in a woman with placenta previa and prior cesarean delivery) can be made. (5) Plan of delivery can be organized accordingly.

CLINICAL CONFIRMATION

Double set-up examination (vaginal examination): It is less frequently done these days. The indications are: (i) Inconclusive USG report (ii) USG revealed type I placenta or (iii) USG facilities not available. **It is done in the operation theater under anesthesia keeping everything ready for cesarean section**. Palpation of the placenta on the lower segment not only conclusively confirms the clinical diagnosis but also identifies its degree.

Steps: The patient, prepared for cesarean section, is placed on the operation table. An intravenous drip is started with using a wide bore cannula. Blood for transfusion should be available. The patient is put under general anesthesia. All these precautions are taken so that a quick surgery can be performed, if torrential bleeding starts following internal examination. A **Cusco's speculum is introduced** to inspect the cervix and the fornix. After removing the speculum, one finger (index) is gently introduced into the vagina. Firstly, **the fornices are palpated to feel any boggy mass** intervening between the presenting part and the finger, the head being pushed down from above. **If the head can be felt clearly through all the fornices, the finger is then introduced through the cervical canal.** Next one should proceed to feel the cervical canal with great gentleness (Fig.19.1). **The feel of the placenta is firm and tough in contrast to soft and friable feel of blood clot**. Examination should stop if placental edge is felt. Otherwise the lower segment is explored by sweeping the finger 360° around the internal os before declaring that no placental tissue is felt. In that case rupture of the membranes may be done to induce labor depending upon the type (see below).

Visualization of the placental implantation on the lower segment can be confirmed during cesarean section.

Examination of the placenta following vaginal delivery reveals: (a) **A tongue-shaped comparatively thin segment of placental tissue** projecting beyond the main placental mass with evidences of degeneration. (b) **Rent on the membranes** is situated on the margin of the placenta. (c) **Abnormal attachment of the cord** (marginal or membranous) is more common.

DIFFERENTIAL DIAGNOSIS

Placenta previa is at times confused with other causes of bleeding occurring in later months of pregnancy. The most common one from which it has to be differentiated is bleeding from premature separation of a normally situated placenta (**abruptio placentae**). The differentiating features are described in a tabulated form (Table 19.1).

The local cervical lesions (polyps, carcinoma) can easily be differentiated by a speculum examination. However, both the conditions can co-exist. **In circumvallate placenta**, the bleeding is slight and the diagnosis is only made after examining the placenta following delivery (see p. 251).

Table 19.1: Distinguishing Features of Placenta Previa and Abruptio Placentae

Parameters	Placenta Previa	Abruptio Placentae
■ **Clinical features:**		
• Nature of bleeding	(a) Painless, apparently causeless and recurrent (b) Bleeding is always revealed	(a) Painful, often attributed to preeclampsia or trauma and continuous (b) Revealed, concealed or usually mixed
• Character of blood	Bright red	Dark colored
• General condition and anemia	Proportionate to visible blood loss	Out of proportion to the visible blood loss in concealed or mixed variety
• Features of preeclampsia	Not relevant	Present in one-third cases
■ **Abdominal examination:**		
• Height of uterus	Proportionate height to gestational age	May be disproportionately enlarged in concealed type
• Feel of uterus	Soft and relaxed	May be tense, tender and rigid
• Malpresentation	Malpresentation is common. The head is high floating	Unrelated, the head may be engaged
• FHS	Usually present	Usually absent especially in concealed type
■ **Placentography (USG)**	Placenta in lower segment	Placenta in upper segment
■ **Vaginal examination**	Placenta is felt on the lower segment	Placenta is not felt on lower segment. Blood clots should not be confused with placenta

COMPLICATIONS OF PLACENTA PREVIA

MATERNAL: *During pregnancy*— **Antepartum hemorrhage with varying degrees of shock** is an inevitable complication. The first bout of hemorrhage is seldom severe but torrential hemorrhage can easily be provoked by *injudicious internal examination.* Co-existent placental abruption is about 10%.

- **Malpresentation**: There is increased incidence of breech presentation (Fig. 19.4) and transverse lie. The lie often becomes unstable.
- **Premature labor** either spontaneous or induced is common.

Death due to massive hemorrhage during the antepartum, intrapartum or postpartum period. Operative hazards, infection or embolism may also cause death.

During labor

- **Early rupture of the membranes**
- **Cord prolapse** due to abnormal attachment of the cord (Fig. 19.4).
- **Slow dilatation** of the cervix due to the attachment of placenta on the lower segment.
- **Intrapartum hemorrhage** due to further separation of placenta with dilatation of the cervix.
- **Increased incidence of operative interference**.
- **Postpartum hemorrhage is due to:**
  - Imperfect retraction of the lower uterine segment upon which the placenta is implanted (see p. 141).
  - Large surface area of placenta with atonic uterus due to preexisting anemia.
  - Occasional association (15%) of morbidly adherent placenta (placenta accreta, increta, percreta) on the lower segment (see p. 486). **Placenta previa accreta is a serious complication that may cause maternal death**. Often the placenta previa and accreta is managed by hysterectomy (see p. 293).
  - Trauma to the cervix and lower segment because of extreme softness and vascularity.

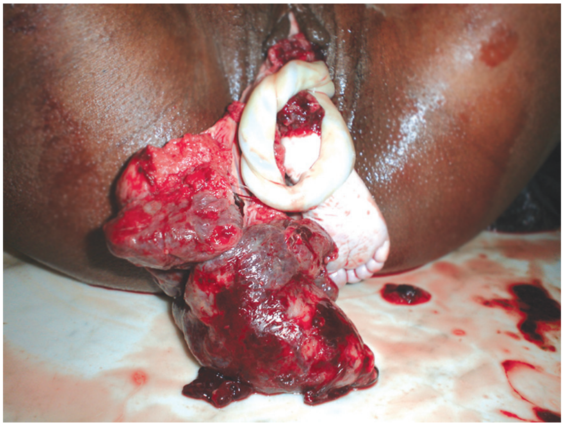


Fig. 19.4: Labor in a woman with placenta previa complicated with prolapse of umbilical cord and footling. *By Courtesy*: *Dr S Bhattacharya, Silchar*

It should be remembered that because of antepartum anemic state, the patient may become shocked with relatively small amount of blood loss.

- **Retained placenta** and increased incidence of manual removal add further hazard to the postpartum shock. Increased incidence of **retained placenta is due to** : (1) Increased surface area and (2) Morbid adhesion. The risk of placenta previa being accreta in a woman with previous one cesarean section is 10–20% and it rises to about 50% with two or more prior cesarean section.

Puerperium: (1) **Sepsis is increased due to**: (a) increased operative interference (b) placental site near to the vagina and (c) anemia and devitalized state of the patient. (2) **Subinvolution** (3) **Embolism**.

FETAL COMPLICATIONS IN PLACENTA PREVIA

- **Low birth weight** babies are quite common (15%) which may be the effect of **preterm labor** either spontaneous or induced. Repeated small bouts of hemorrhage while carrying on the expectant treatment can cause chronic placental insufficiency and **fetal growth restriction**.

- **Asphyxia** is common and it may be the effect of — (a) early separation of placenta (b) compression of the placenta or (c) compression of the cord.
- **Intrauterine death** is more related to severe degree of separation of placenta, with maternal hypovolemia and shock. Deaths are also due to cord accidents (Fig. 19.4).
- **Birth injuries** are more common due to increased operative interference.
- **Congenital malformation** is three times more common in placenta previa.
- **Maternal and fetal morbidity and mortality** from placenta previa are significantly high.

PROGNOSIS

MATERNAL: There has been a substantial **reduction of maternal deaths in placenta previa** throughout the globe. **The contributing factors are**—(a) early diagnosis (diagnosis can even be made prior to bleeding)(b) omission of internal examination outside the hospital (c) free availability of blood transfusion facilities (d) potent antibiotics (e) wider use of cesarean section with expert anesthetist and (f) skill and judgment with which the cases are managed. All these factors have led to reduction of maternal deaths from placenta previa to less than 1% or even to zero in some centers. **But in the developing countries** because of wide gaps in the extension of the medical facilities and also the difference in patients' profile between the urban and the vast rural population, the maternal mortality from placenta previa in hospital statistics ranges from less than 1% to as high as 5%. Significant number of patients are rushed to the referral hospital after repeated bouts of hemorrhage often with history of vaginal examination. Inadequate antenatal care, delay in referral, road and transport difficulties contribute to the poor outcome. **The ultimate causes of death are hemorrhage and shock**. The **morbidity** is somewhat raised due to hemorrhage and operative delivery. **Risk of recurrence** of placenta previa in subsequent pregnancy is about 8 times.

FETAL: The reduction in perinatal deaths is principally due to judicious extension of expectant treatment thereby reducing the loss from prematurity, liberal use of cesarean section which greatly lessens the loss from hypoxia and improvement in the neonatal care unit. But still the perinatal mortality ranges from 10–25%. **The causes of death are**—(a) prematurity (b) asphyxia and (c) congenital malformation.

MANAGEMENT

PREVENTION: Placenta previa is one of the inherent obstetric hazards and in majority the cause is unknown. Thus to minimize the risks, the following guidelines are useful.

- **Adequate antenatal care** to improve the health status of women and correction of anemia.
- **Antenatal diagnosis** of low lying placenta at 20 weeks with routine ultrasound needs repeat ultrasound examination at 34 weeks to confirm the diagnosis.
- **Significance of "warning hemorrhage"** should not be ignored.
- **Color flow Doppler USG** in placenta previa is indicated to detect any placenta accreta. Where this is not possible, such women with an increased risk of placenta accreta, should be managed as if they have placenta accreta until proved otherwise.

AT HOME: (1) The patient is immediately **put to bed**. (2) **To assess the blood loss**—(a) inspection of the clothing soaked with blood (b) to note the pulse, blood pressure and degree of anemia (3) **Quick but gentle abdominal examination** to mark the height of the uterus, to auscultate the fetal heart sound and to note any tenderness on the uterus (4) **Vaginal examination must not be done**. Only inspection is done to see whether the bleeding is present or absent and to put a sterile vulval pad.

TRANSFER TO HOSPITAL: Arrangement is made to shift the patient to an equipped hospital having facilities of blood transfusion, emergency cesarean section and neonatal intensive care unit (NICU). 'Flying Squad' service is ideal for transfer of such type of patients. An intravenous Ringer's solution drip should be started and is kept running during transport. Patient should be accompanied by two or three persons fit for donation of blood, if necessary.

ADMISSION TO HOSPITAL: All cases of APH, even if the bleeding is slight or absent by the time the patient reaches the hospital, **should be admitted**. **The reasons are:** (1) All the cases of APH should be regarded as due to placenta previa unless proved otherwise. (2) The bleeding may recur sooner or later and none can predict when it recurs and how much she will bleed.

TREATMENT ON ADMISSION

- **Immediate attention**
- **Formulation of the line of treatment**

IMMEDIATE ATTENTION: Overall assessment of the case is quickly made as regards: (1) **Amount of the blood loss** — by noting the general condition, pallor, pulse rate and blood pressure. (2) **Blood samples are taken** for group, cross matching and estimation of hemoglobin. (3) **A large-bore IV cannula is sited and an infusion of normal saline** is started and compatible cross matched blood transfusion should be arranged. (4) **Gentle abdominal palpation** to ascertain any uterine tenderness and auscultation to note the fetal heart rate. (5) **Inspection of the vulva** to note the presence of any active bleeding.

Confirmation of diagnosis is made from the history, physical examination and with **sonographic examination**.

FORMULATION OF THE LINE OF TREATMENT:

The definitive treatment depends upon the duration of pregnancy, fetal and maternal status and extent of the hemorrhage.

- **Expectant management**
- **Active (Definite) management**

♦ ***Expectant treatment:***

The policy had been advocated by **Macafee and Johnson** (1945), in an attempt to improve the fetal salvage without increasing undue maternal hazards. **The aim is** to continue pregnancy for fetal maturity without compromising the maternal health.

Vital prerequisites: (1) Availability of **blood for transfusion** whenever required. (2) **Facilities for cesarean section should be available throughout 24 hours,** should it prove necessary.

Selection of cases: **Suitable cases for expectant management are:** (1) Mother is in good health status (hemoglobin $\geq$ 10 g%; hematocrit > 30%). (2) Duration of pregnancy is less than 37 weeks. (3) Active vaginal bleeding is absent. (4) Fetal well-being is assured (CTG and USG).

Conduct of expectant treatment: (1) **Bed rest** with bathroom and toilet privileges. (2) **Investigations**—like hemoglobin estimation, blood grouping and urine for protein are done. (3) **Periodic inspection** of the vulval pads and fetal surveillance with USG at interval of 2–3 weeks (see p. 286). (4) **Supplementary hematinics** should be given and the blood loss is replaced by adequate cross matched blood transfusion, if the patient is anemic. (5) When the patient is allowed out of the bed (2-3 days after the bleeding stops), **a gentle speculum (Cusco's) examination is made** to exclude local cervical and vaginal lesions for bleeding. However, their presence does not negate placenta previa. (6) Use of tocolysis (magnesium sulfate) can be done if vaginal bleeding is associated with uterine contractions. (7) Use of cervical cerclage to reduce bleeding and to prolong pregnancy is not helpful (RCOG 2005). (8) Rh immunoglobin should be given to all Rh negative (unsensitized) women.

Expectant management at Hospital or at Home? Hospital setting is ideal. But considering the cost of prolonged hospitalization and psychological morbidity, home care may be allowed in some. Selected cases are — (a) patient lives close to hospital, (b) 24-hour transportation is available, (c) bed rest assured and (d) patient is well motivated to understand the risks.

Termination of the expectant treatment:The expectant treatment **is carried up to 37 weeks of pregnancy**. By this time, the baby becomes sufficiently mature.

However, preterm delivery may have to be done in conditions, such as: (1) Recurrence of brisk hemorrhage and which is continuing. (2) The fetus is dead. (3) The fetus is found congenitally malformed on investigation. Repeated small bouts of hemorrhage is not an indication for termination of expectant treatment. Replacement of the blood loss can be made by blood transfusion. **However, there is the risk of IUGR**.

Steroid therapy is indicated when the duration of pregnancy is less than 34 weeks. Betamethasone reduces the risk of respiratory distress of the newborn when preterm delivery is considered (see p. 367).

- ***Active (Definite) Management (Delivery):***

The indications of definitive management (delivery) are : (1) Bleeding occurs at or after 37 weeks of pregnancy. (2) Patient is in labor. (3) Patient is in exsanguinated state on admission. (4) Bleeding is continuing and of moderate degree. (5) Baby with nonreassuring cardiac status or dead or known to be congenitally deformed.

A. **Cesarean delivery** is done for all women with sonographic evidence of placenta previa where **placental edge is within 2 cm from the internal os**. It is especially indicated if it is posterior or thick (*RCOG* 2005).

During the recent years, there has been **wider use of cesarean section**, in an attempt not only **to reduce the maternal risk but also to improve the fetal salvage**. Clinical assessment is important to decide the mode of delivery.

B. **Vaginal delivery** may be considered where placenta edge is **clearly 2–3 cm away from the internal cervical os** (based on sonography).

VAGINAL EXAMINATION should be done with a double set up arrangement in the operation theater keeping everything ready for cesarean section (see above).

Contraindications of vaginal examination are: (1) Patient in exsanguinated state. (2) Diagnosed cases of major degree of placenta previa confirmed by ultrasonography (previously mentioned). (3) Associated complicating factors such as malpresentation, elderly primigravidae, pregnancy with history of previous cesarean section, contracted pelvis etc. which themselves are indications for cesarean section.

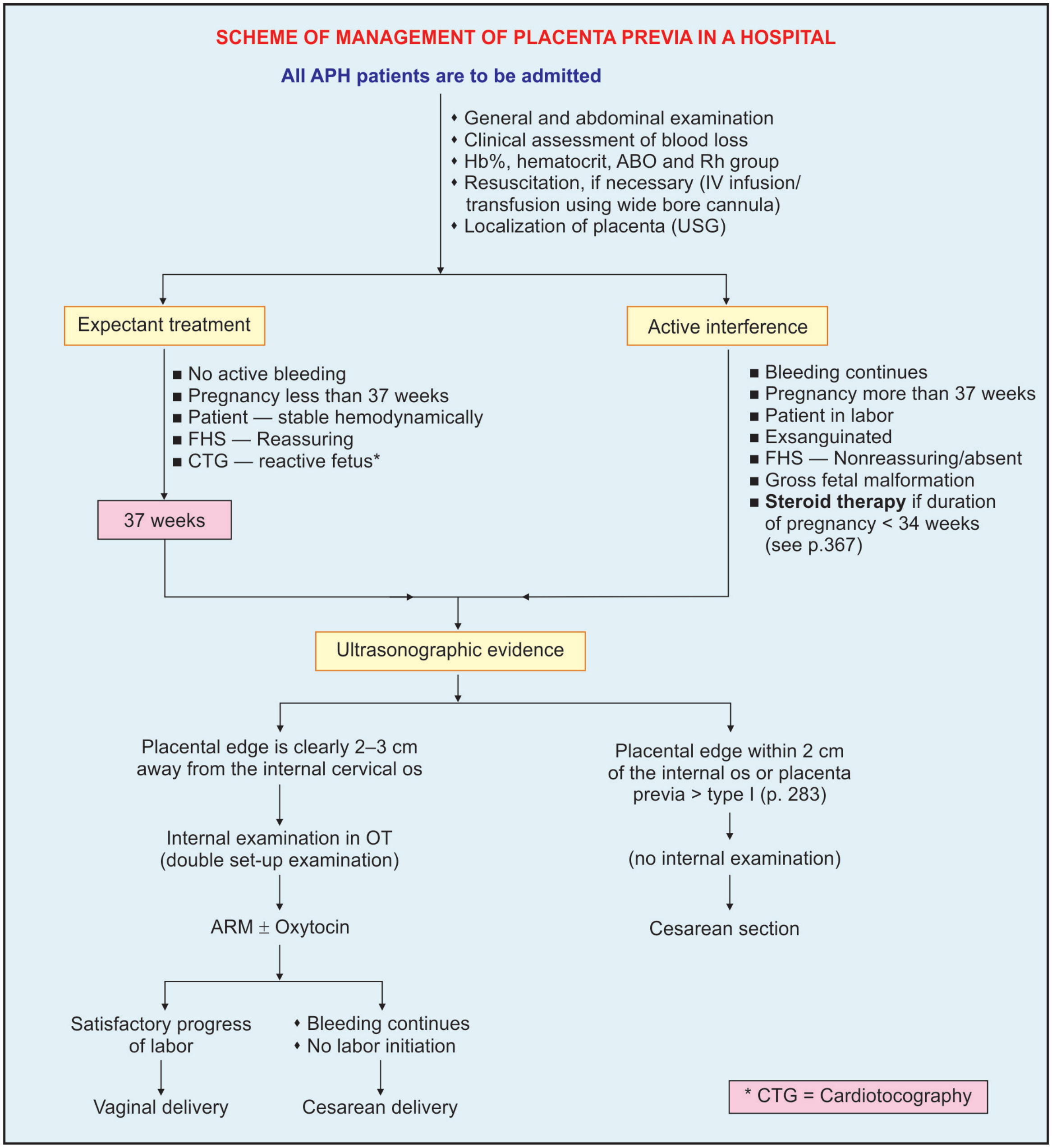
Low rupture of the membranes: The treatment is now crystallized to induce labor by low rupture of the membranes using long Kocher's forceps in lesser degree of placenta previa (Type-I and Type-II anterior). **The finger is reinserted to exclude cord prolapse**. Oxytocin drip may be started, if not contraindicated. **If, amniotomy fails to stop bleeding or fails to initiate labor, cesarean section is performed**.

Precautions during vaginal delivery: (1) All possible steps should be taken to restore the blood volume; (2) Oxytocin 10 IV/IM/methergine 0.2 mg should be given intravenously with the delivery of the baby (see p. 165) to prevent blood loss in third stage. (3) Proper examination of the cervix should be done soon following delivery to detect any evidences of tear. (4) Baby's blood hemoglobin level is to be checked and if necessary arrangements are to be made for blood transfusion.

Placenta accreta is the attachment of placenta directly to the myometrium without any intervening decidua basalis (see p. 486). Important risk factors for placenta accreta are: placenta previa and prior cesarean delivery. Diagnosis is made by 3-D power Doppler and MRI (see p. 486). Management needs cesarean delivery and in many cases peripartum hysterectomy. Interventional radiology and uterine artery embolization can control hemorrhage and avoid hysterectomy.

PRACTICAL GUIDELINES FOR CESAREAN DELIVERY

(1) The operation should be performed by a senior obstetrician with the help of an experienced senior anesthetist. (2) Choice of anesthesia is to be made by the anesthetist. General anaesthesia may be used. (3) If the patient is in hypovolemic state and the bleeding continues, the operation has to be performed immediately along with restorative measures. (4) Blood and blood products should be made available. (5) Counseling and consent for possible other interventions (hysterectomy) should be taken. (6) Multidisciplinary involvement should be made (urologist, transfusion specialist). (7) Availability of a bed in a critical care unit to be ensured. (8) Interventional radiology service is of help, especially in a case with placenta previa and accreta.



SCHEME OF MANAGEMENT OF PLACENTA PREVIA IN A HOSPITAL

All APH patients are to be admitted
• General and abdominal examination
• Clinical assessment of blood loss
• Hb%, hematocrit, ABO and Rh group
• Resuscitation, if necessary (IV infusion/ transfusion using wide bore cannula)
• Localization of placenta (USG)
Expectant treatment
■ No active bleeding
■ Pregnancy less than 37 weeks
■ Patient — stable hemodynamically
■ FHS — Reassuring
■ CTG — reactive fetus*
37 weeks
Active interference
■ Bleeding continues
■ Pregnancy more than 37 weeks
■ Patient in labor
■ Exsanguinated
■ FHS — Nonreassuring/absent
■ Gross fetal malformation
■ Steroid therapy if duration of pregnancy < 34 weeks (see p.367)
Ultrasonographic evidence
Placental edge is clearly 2–3 cm away from the internal cervical os
Internal examination in OT (double set-up examination)
ARM ± Oxytocin
Satisfactory progress of labor
Vaginal delivery
• Bleeding continues
• No labor initiation
Cesarean delivery
Placental edge within 2 cm of the internal os or placenta previa > type I (p. 283)
(no internal examination)
Cesarean section
* CTG = Cardiotocography

Type of incision — lower segment or classical? Ideally, surgeon should make the incision away from the placenta when placenta previa accreta is diagnosed or suspected (RCOG).

1. *Lower segment cesarean section:* (A) Advantages: (1) Conversant technique. (2) The bleeding sinuses at the placental site can be better dealt with under direct vision and as such the decision to preserve or to remove the uterus can easily be made. (3) Placenta accreta, if accidentally met, can also be tackled effectively.

(B) Disadvantages:

1. Engorged vessels on the anterior lower segment (anterior placenta previa) bleed profusely when they are cut.
2. In anteriorly situated placenta, the placenta has to be cut or separated to deliver the baby. This causes massive hemorrhage.
3. Risks of fetal exsanguination with such delivery is a real threat to the baby.

4. Risks of cesarean hysterectomy is high in such a case.
5. Delivery is to be expedited to avoid fetal exsanguination.
6. Umbilical cord should be clamped immediately to prevent neonatal hypovolemia and anemia.
7. The edges of uterine cut margins become so vascular and friable, that the tissues may cut through during suturing.

2. Classical cesarean section: **(A) Advantages:** (1) The operation can be done more quickly. (2) Baby is delivered without disturbing the placenta. (3) There is no risk of fetal exsanguination. (4) Placenta may be left in situ (in case of placenta accreta) if no bleeding and (5) Uterus may be preserved. (6) Reduction of morbidity in terms of hemorrhage, blood transfusion, ITU admission and urological injury.

(B) Disadvantages: (1) The lower segment over which the placenta is implanted cannot be visualized and as such, it is difficult to control bleeding when it is present. (2) All the hazards (immediate and remote) of classical cesarean (see p. 676).

PRACTICAL GUIDE TO LOWER SEGMENT APPROACH FOR PLACENTA PREVIA

- **To make infraumbilical longitudinal incision**.
- To tackle the engorged vessels on the anterior uterine wall — to put two ligatures and to cut in between while making the transverse incision. Some, however, ignore the vessels.
- **To tackle the placenta lying underneath the incision** —Incision should be made away from the placental site. Otherwise the placenta may have to be separated manually to get the lower margin and then membranes are ruptured. Alternatively, the placenta may have to be cut promptly to enter into the amniotic sac to deliver the baby. **In either case, the cord is to be clamped quickly to prevent further fetal exsanguination**. In fact, fetal blood loss starts from the moment the placenta is separated or cut.
- **Once the placenta is separated and delivered**, the lower segment should be inspected for any oozing point which is over sewed, if needed. Aggressive use of oxytocics can control bleeding (see p. 481).
- If hemostasis by sutures fails and the uterus is to be preserved using B-Lynch suture (see p. 482) or **tight intrauterine pack** (tamponade p. 482) to give firm pressure on the oozing area, to bring the plug end through the vagina and suturing the uterine incision over the plug—most often pays good dividend. The plug is removed vaginally after 48 hours.
- **Isthmic-cervical apposition suture** — helps to control bleeding from the lower segment specially in a case with placenta previa or morbid adherent placenta. A suture is passed through the lower flap of the uterine incision (2 cm medial to its lateral border) to the posterior aspect. The same suture is then brought back through (1 cm medial to the first) posterior to the anterior uterine wall and then tied anteriorly. The same procedure is repeated on the other side. The cervical canal is kept patent while tightening the apposition suture. Injury to the bladder and uterus are avoided.
- **Baby's blood hemoglobin level is to be checked at birth** and if necessary, arrangement is made for blood transfusion.

PRACTICAL GUIDELINES TO LOWER SEGMENT APPROACH FOR PLACENTA PREVIA ACCRETA

- Women with anterior placenta previa, implanted at the site of prior hysterotomy or cesarean incision, there is an increased risk of **placenta accreta**. The risk increases with the number of prior cesarean delivery. It increases from 1% with no prior scar to 3% with three prior cesarean scar (see p. 486). This may need hysterectomy.
- Incision is made away from the placenta. Incising the placenta for delivery causes more hemorrhage and may end in hysterectomy (Fig. 19.5).
- Delivering the baby without placental separation may allow conservative management of placenta, if there is no bleeding.

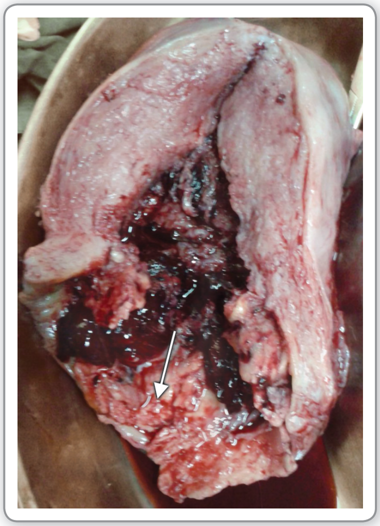


Fig. 19.5: Placenta increta, hysterectomy done due to uncontrolled PPH in a case with prior cesarean delivery. *Courtesy:* Dr Meena Jain, JLN Hospital, Bhilai, CG

- In a case with morbid adherent placenta without bleeding, placenta may be left in situ. The uterus is then closed when preservation of the uterus is desired.
- Any attempt of placental separation in a case with morbid adherent placenta (placenta accreta) should be avoided as it excites massive hemorrhage and risks hysterectomy.
- In presence of bleeding, hysterectomy could be done after closing the uterus without any attempt to separate the placenta. This reduces blood loss.
- B-Lynch suture, isthmic cervical apposition suture, uterine and internal iliac arteries ligation or intervention radiology and uterine artery embolization (see p. 482 and 483) have been done to control hemorrhage and to preserve the uterus.
- A multidisciplinary team approach (urologists, transfusion specialists) should be made.

ABRUPTIO PLACENTAE

(Syn: Accidental Hemorrhage. Premature Separation of Placenta)

DEFINITION: It is one form of antepartum hemorrhage where the bleeding occurs due to premature separation of normally situated placenta. Out of the various nomenclatures, abruptio placentae seems to be appropriate one.

VARIETIES (Figs 19.6A to D):

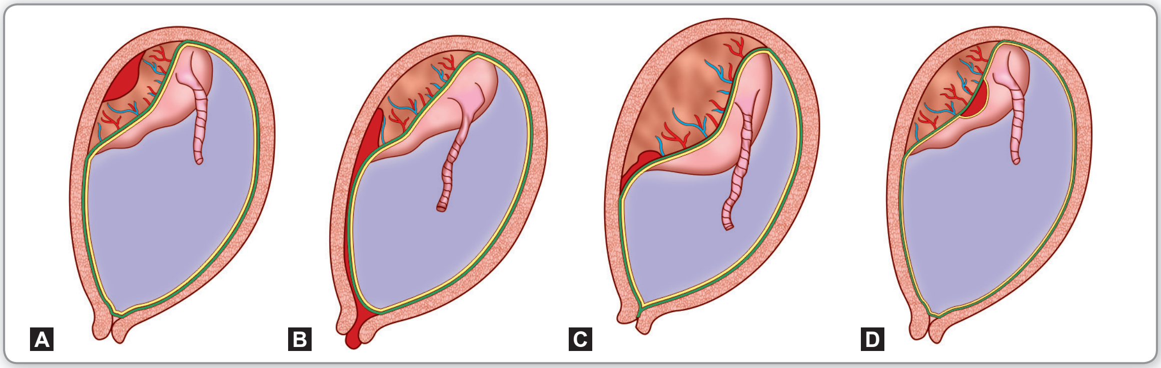
(1) ***Revealed*** : Following separation of the placenta, the blood insinuates downwards between the membranes and the decidua. Ultimately, the blood comes out of the cervical canal to be visible externally. This is the most common type.

(2) ***Concealed*** : The blood collects behind the separated placenta or collected in between the membranes and decidua. The collected blood is prevented from coming out of the cervix by the presenting part which presses on the lower segment. At times, the blood may percolate into the amniotic sac after rupturing the membranes. In any of the circumstances blood is not visible outside. This type is rare.

(3) ***Mixed*** : In this type, some part of the blood collects inside (concealed) and a part is expelled out (revealed). Usually one variety predominates over the other. This is quite common.

Bleeding is almost always maternal. But placental tear may cause fetal bleeding.

Incidence and significance. The overall incidence is about 1 in 200 deliveries. Depending on the extent (partial or complete) and intensity of placental separation, it is a significant cause of **perinatal mortality (15–20%) and maternal mortality (2–5%)**. More and more cases of placental abruption are being diagnosed in the recent years.



Figs 19.6A to D: Varieties of abruptio placentae: (A) Concealed; (B) Revealed; (C) Marginal (subchorionic) and; (D) Preplacental (subamniotic)

ETIOLOGY: The exact cause of separation of a normally situated placenta remains obscure in majority of cases.

Risk factors are : (a) **high birth order** pregnancies with gravida 5 and above — three times more common than in first birth (b) **advancing age** of the mother (c) poor socio-economic condition (d) malnutrition (e) smoking (vasospasm).

Hypertension in pregnancy is the most important predisposing factor. Preeclampsia, gestational hypertension and essential hypertension, all are associated with placental abruption. The association of preeclampsia in abruptio placentae varies from 10% to 50%. **The mechanism of the placental separation in preeclampsia is:** Spasm of the vessels in the utero-placental bed (decidual spiral artery) → anoxic endothelial damage → rupture of vessels or extravasation of blood in the decidua basalis (retroplacental hematoma).

Trauma: Traumatic separation of the placenta usually leads to its marginal separation with escape of blood outside. The trauma may be due to: (i) Attempted external cephalic version specially under anesthesia using great force (ii) Road traffic accidents or blow on the abdomen (iii) Needle puncture at amniocentesis.

Sudden uterine decompression: Sudden decompression of the uterus leads to diminished surface area of the uterus adjacent to the placental attachment and results in separation of the placenta. This may occur following—(a) delivery of the first baby of twins (b) sudden escape of liquor amnii in hydramnios and (c) premature rupture of membranes.

Short cord, either relative or absolute, can bring about placental separation during labor by mechanical pull.

Supine hypotension syndrome: In this condition which occurs in pregnancy there is passive engorgement of the uterine and placental vessels resulting in rupture and extravasation of the blood.

Placental anomaly: Circumvallate placenta (see p. 252).

Sick placenta: Poor placentation, evidenced by abnormal uterine artery Doppler waveforms is associated with placental abruption.

Folic acid deficiency even without evidence of overt megaloblastic erythropoiesis — this has been observed to be associated.

Uterine factor: Placenta implanted over a septum (Septate Uterus) or a submucous fibroid.

Torsion of the uterus leads to increased venous pressure and rupture of the veins with separation of the placenta.

Cocaine abuse is associated with increased risk of transient hypertension, vasospasm and placental abruption.

Thrombophilias (see p. 319, 508) inherited or acquired have been associated with increased risk of placental infarcts or abruption.

Prior abruption: Risk of recurrence for a woman with previous abruption varies between 5% and 17%.

PATHOGENESIS: Depending upon the etiological factors (see above), premature placental separation is initiated by hemorrhage into the decidua basalis. The collected blood (decidual hematoma) at the early phase, hardly produces any morbid pathological changes in the uterine wall or on the placenta. However, depending upon the extent of pathology, there may be degeneration and necrosis of the decidua basalis as well as the placenta adjacent to it.

Rupture of the basal plate may also occur, thus communicating the hematoma with the intervillous space. The decidual hematoma may be small and self limited; the entity is evident only after the expulsion of the placenta (retroplacental hematoma). **The features of retroplacental hematoma are**: (a) Depression found on the maternal surface of the placenta with a clot which may be found firmly attached to the area (b) Areas of infarction with varying degree of organization (Fig. 19.7).

If, however, a major spiral artery ruptures, a big hematoma is formed. As the uterus remains distended by the conceptus, it fails to contract and therefore fails to compress the torn bleeding points.

It has to be remembered that absence of rhythmic uterine contractions plays a significant role for the blood to remain concealed.

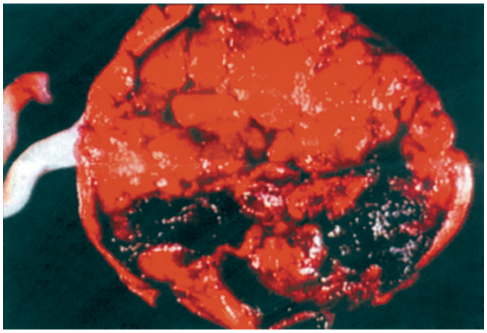


Fig. 19.7: Appearance of a placenta following delivery. Placenta shows adherent clots and depression

Table 19.2: Abruptio Placentae

Clinical Manifestations of Hemorrhage	Ultrasonographic Localization of Hemorrhage
■ Blood may accumulate behind the placenta when it is totally separated from the uterine wall except at the margin (Fig. 19.6A) — **concealed type**. ■ **Blood may dissect downwards** in between the membranes and the uterine wall and ultimately escapes out through the cervix or may be kept concealed by the pressure of the fetal head on the lower uterine segment (Fig. 19.6B) — **revealed type**. ■ **Blood may gain access** to the amniotic cavity after rupturing the membranes (Fig. 19.6D). ■ **Blood** may percolate through the layers of myometrium upto the serous coat – known as **Couvelaire uterus** (Fig. 19.7).	■ **Retroplacental**: Between the placenta and the myometrium (Figs 19.6A and B). ■ **Subchorionic**: Between the placenta and the membranes (Fig. 19.6C). ■ **Pre-placental**: Between the placenta and the amniotic fluid, within amnion and chorion (subamniotic) (Fig. 19.6D). ■ **Fetal prognosis depends upon**: (i) size and (ii) the type of the hematoma. Retroplacental hematoma has got worst prognosis with high fetal mortality (50%). Subchorionic smaller sized hemorrhages have less (10%) fetal mortality. Subamniotic is clinically less significant.

COUVELAIRE UTERUS (uteroplacental apoplexy) (Fig. 19.8): It is a pathological entity first described by Couvelaire and is met with in association with severe form of concealed abruptio placentae. There is massive intravasation of blood into the uterine musculature upto the serous coat. **The condition can only be diagnosed on laparotomy**.

Naked eye features: The uterus is of dark port wine color which may be patchy or diffuse. It tends to occur initially on the cornu before spreading to other areas, more specially over the placental site. Subperitoneal petechial hemorrhages are found under the uterine peritoneum and may extend into the broad ligament. There may be free blood in the peritoneal cavity or broad ligament hematoma.

Microscopic appearance: The uterine muscles over the affected area are necrosed and there is infiltration of blood and fluid in between the muscle bundles. Most of the muscular dissociation occurs in the middle and outer muscle layers. The serosa may split on occasions, to allow the blood to enter the peritoneal cavity. The blood vessels show acute degenerative changes with thrombosis.

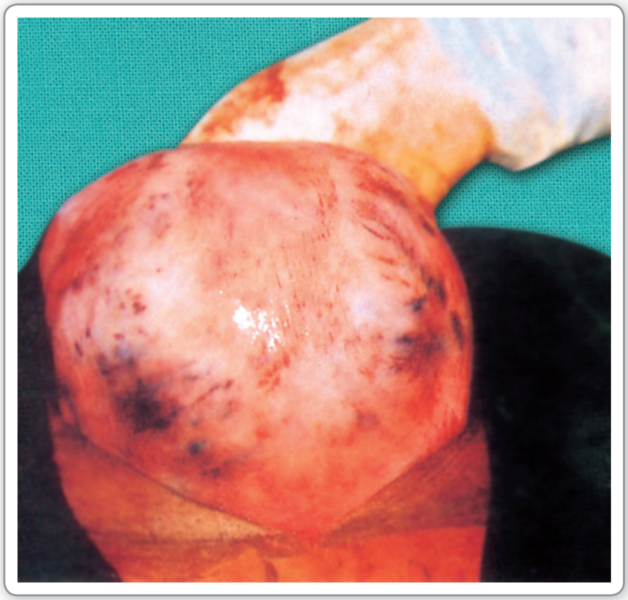


Fig. 19.8: Couvelaire uterus. Uterus was preserved

The myometrial hematoma rarely interferes with uterine contractions following delivery. **Thus, the presence of Couvelaire uterus as observed during cesarean section is not an indication** per se **for hysterectomy.**

CHANGES IN OTHER ORGANS: In the liver, apart from the changes found in preeclampsia, presence of fibrin knots in the hepatic sinusoids is an important finding. **Kidneys** may show acute cortical necrosis or acute tubular necrosis. The precise mechanism is not clear but may be due to intrarenal vasospasm as a consequence of massive hemorrhage. **Shock proteinuria** is probably due to renal anoxia which usually disappears 2 days after delivery, whereas proteinuria due to preeclampsia tends to last longer.

BLOOD COAGULOPATHY: Blood coagulopathy is due to excess consumption of plasma fibrinogen due to disseminated intravascular coagulation and retroplacental bleeding. There is overt hypofibrinogenemia (< 150 mg/dL) and elevated levels of fibrin degradation products and D-dimer (see p. 744).

CLINICAL CLASSIFICATION: Depending upon the degree of placental abruption and its clinical effects, the cases are graded as follows:

- ***Grade—0:*** Clinical features may be absent. The diagnosis is made after inspection of placenta following delivery.
- ***Grade—1 (40%):*** (i) **vaginal bleeding** is slight (ii) **uterus**: irritable, tenderness may be minimal or absent (iii) **maternal BP and fibrinogen** levels unaffected (iv) **FHS is good**.
- ***Grade—2 (45%):*** (i) **vaginal bleeding** mild to moderate (ii) **uterine tenderness is always present** (iii) maternal pulse ↑, BP is maintained (iv) fibrinogen level may be decreased (v) **shock is absent** (vi) **fetal distress or even fetal death occurs**.
- ***Grade—3 (15%):*** (i) **bleeding** is moderate to severe or may be concealed (ii) **uterine tenderness is marked** (iii) shock is pronounced (iv) **fetal death is the rule** (v) associated **coagulation defect or anuria** may complicate.

CLINICAL FEATURES OF ABRUPTIO PLACENTAE

The clinical features depend on: (i) degree of separation of placenta, (ii) speed at which separation occurs and (iii) amount of blood concealed inside the uterine cavity. But they may be very deceptive in posteriorly implanted placenta. The clinical features of the revealed and mixed variety are given in tabulated form (Table 19.3).

DIAGNOSIS: Mainly clinical. Ultrasonography or MRI may be helpful.

ULTRASONOGRAPHY: Early hemorrhage is hyperechoic or isoechoic. Acute hemorrhage is often confused with a fibroid, or a thick placenta. However, even negative findings with USG examination do not exclude placental abruption.

DIFFERENTIAL DIAGNOSIS: (a) *Revealed type*: There may be occasional diagnostic difficulty with placenta previa. The differentiating points have been given previously in tabulated form (Table 19.1). Confusion with the indeterminate causes of APH is difficult to eliminate (b) *Mixed or concealed type*: This variety is often confused with — (i) rupture uterus (ii) rectus sheath hematoma (iii) appendicular or intestinal perforation (iv) twisted ovarian tumor (v) volvulus (vi) acute hydramnios (vii) tonic uterine contraction.

The essential points to arrive at the diagnosis of the concealed variety are: (i) shock out of proportion to external bleeding, (ii) unexplained extreme pallor, (iii) presence of preeclamptic features, (iv) uterus is tense, tender and woody hard, (v) FHS is absent, (vi) diminished urinary output, (vii) presence of blood coagulation disorders.

PROGNOSIS: The prognosis of the mother and the baby depends on the clinical types (revealed, mixed or purely concealed), degree of placental separation, the interval between the separation of the placenta and delivery of the baby and the efficacy of treatment. **Bleeding in placental abruption is almost always maternal**. Fetal bleeding is observed only with traumatic variety of placental abruption.

COMPLICATIONS OF ABRUPTIO PLACENTAE

MATERNAL: *In revealed type*—maternal risk is proportionate to the visible blood loss and maternal death is rare.

In concealed variety—**The following complications** may occur either singly or in combination. (1) **Hemorrhage** which is either totally concealed inside the uterus or more commonly, part is revealed outside. There may be intraperitoneal or broad ligament hematoma. (2) **Shock** may be out of proportion to the blood loss. Release of thromboplastin into the maternal circulation results in DIC or there may be amniotic fluid embolism. (3) **Blood coagulation disorders** (see above). (4) **Oliguria and anuria due to**—(a) hypovolemia (b) serotonin liberated from the damaged uterine muscle producing renal ischemia and (c) acute tubular necrosis. However, a severe case may lead to (d) cortical necrosis and

Table 19.3: Clinical Features of Revealed and Mixed Variety of Abruptio Placentae

Parameters	**Revealed**	**Mixed (Concealed Features Predominate)**
Symptoms	Abdominal discomfort or pain followed by vaginal bleeding (usually slight)	Abdominal acute intenses pain followed by slight vaginal bleeding. The pain becomes continuous
Character of bleeding	Continuous dark color (slight to moderate)	Continuous, dark color (usually slight) or blood stained serous discharge
General condition	Proportionate to the visible blood loss, shock is usually absent.	* Shock may be pronounced which is out of proportion to the visible blood loss.
Pallor	Related with the visible blood loss	Pallor is usually severe and out of proportion to the visible bleeding
Features of preeclampsia	May be absent	Frequent association
Uterine height	Proportionate to the period of gestation.	May be disproportionately enlarged and globular.
Uterine feel	Normal feel with localized tenderness, contractions frequent and local amplitude.	Uterus is tense, tender and rigid
Fetal parts	Can be identified easily	Difficult to make out
FHS	Usually present	Usually absent
Urine output	Normal	Usually diminished
Laboratory Tests:		
Blood: Hb%	Low value proportionate to the blood loss	Markedly lower, out of proportion to the visible blood loss
Coagulation profile	Usually unchanged	Variable changes: ● Clotting time increased (> 6 min) ● Fibrinogen level-low (< 150 mg/dL) ● Platelet count-low ● ↑ partial thromboplastin time ● ↑ FDP and D-dimer
Urine for protein	May be absent	Usually present
Confusion in diagnosis	With placenta previa. As such vaginal examination is withheld unless certain in the diagnosis	With acute obstetrical-gynecological-surgical complications

* **Shock**: Shock is often due to blood loss and hypovolemia or due to coagulopathy. **Mild hemorrhage** (< 15% of the blood volume loss) is generally not associated with any change of vital signs. **Moderate hemorrhage** (15-30% of the blood volume loss) is associated with tachycardia, hypotension, ↓ pulse pressure and mean arterial pressure whereas **severe hemorrhage** (loss > 30-40%) is associated with features of shock.

renal failure. (5) **Postpartum hemorrhage due to** — (a) atony of the uterus and (b) increase in serum FDP (6) **Puerperal sepsis.**

The complicating factors that are responsible for **increased maternal death** varies from 2% to 8%. However, with better understanding in the management of shock, coagulation failure and renal failure, maternal death has been reduced markedly. Some cases who manage to survive may develop features of ischemic pituitary necrosis. **There is failure of lactation (Sheehan's syndrome) later on**.

FETAL: ***In revealed type***, the fetal death is to the extent of 25–30%. ***In concealed type***, however, the fetal death is appreciably high, ranging from 50% to 100%. **The deaths are due to prematurity and anoxia due to placental separation**. With same degree of placental separation, the fetus is put to more risk in abruptio placentae than in placenta previa. This is due to the presence of preexisting placental pathology with poor functional reserve in the former, in contrast to an almost normal placental functions in the latter.

Risk of recurrence in subsequent pregnancy is about 5–20% with high perinatal mortality.

MANAGEMENT OF ABRUPTIO PLACENTAE

Prevention: ***The prevention aims at***—(1) Elimination of the known factors likely to produce placental separation. (2) Correction of anemia during antenatal period so that the patient can withstand blood loss. (3) Prompt detection and institution of the therapy to minimize the grave complications namely shock, blood coagulation disorders and renal failure.

Prevention of known factors likely to cause placental separation are (see p. 294):

- **Early detection and effective therapy** of preeclampsia and other hypertensive disorders of pregnancy.
- **Needle puncture** during amniocentesis should be under ultrasound guidance.
- **Avoidance of trauma**—specially forceful external cephalic version under anesthesia.
- **To avoid sudden decompression of the uterus**— in acute or chronic hydramnios, amniocentesis is preferable to artificial rupture of the membranes.
- **To avoid supine hypotension** the patient is advised to lie in the left lateral position in the later months of pregnancy.
- **Routine administration of folic acid** from the early pregnancy — of doubtful value.

TREATMENT

AT HOME: The patient is to be treated as outlined in placenta previa and arrangement should be made to **shift the patient to an equipped maternity unit as early as possible**.

IN THE HOSPITAL: **Assessment of the case is to be done as regards:** (a) amount of blood loss (b) maturity of the fetus and (c) whether the patient is in labor or not (usually labor starts) (d) presence of any complication and (e) type and grade of placental abruption (see p. 294).

Emergency measures: (i) **blood** is sent for hemoglobin and hematocrit estimation, coagulation profile (fibrinogen level, FDP, prothrombin time, activated partial thromboplastin time and platelets), ABO and Rh grouping and **urine** for detection of protein (ii) **Ringer's solution drip is started** with a wide bore cannula and arrangement for blood transfusion is made for resuscitation. Close monitoring of maternal and fetal condition is done.

Management options are: (a) immediate delivery (b) management of complications if there is any (c) expectant management (rare).

- ♦ ***Definitive treatment (immediate delivery):***
- ■ **The patient is in labor:** Most patients are in labor following a term pregnancy: The labor is accelerated by **low rupture of the membranes**. Rupture of the membranes with escape of liquor amnii accelerates labor and it increases the uterine tone also. Oxytocin drip may be started to accelerate labor when needed.

Vaginal delivery is favored in cases with: (i) limited placental abruption (ii) FHR tracing (see p. 693) is reassuring (iii) facilities for continuous (electronic) fetal monitoring is available (iv) prospect of vaginal delivery is soon or (v) placental abruption with a dead fetus.

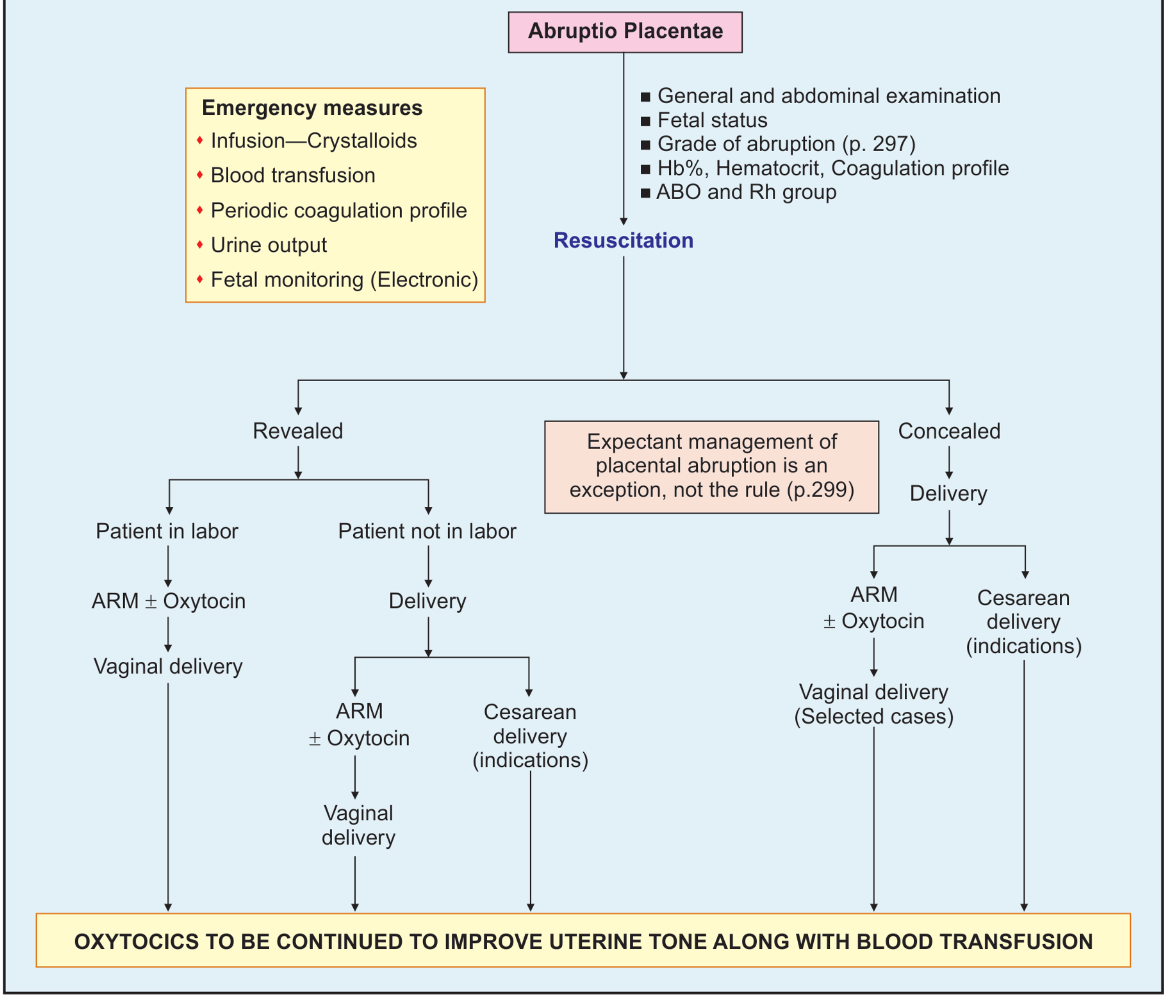
The advantages of amniotomy are: (a) initiates myometrial contraction and labor process (b) expedites delivery (c) better compression of spiral artery to arrest hemorrhage (d) reduces entry of thromboplastin into maternal circulation and thereby (e) reduces the risk of renal cortical necrosis and DIC.

- **The patient is not in labor:** (i) Bleeding continues (ii) > Grade I abruption (see p. 297): **Delivery** either by (A) induction of labor or (B) cesarean section.

(A) **Induction of labor is done by low rupture of membranes**. Oxytocin may be added to expedite delivery. **Labor** usually starts soon in majority of cases and delivery is completed quickly (4–6 hours). Placenta with varying amount of retroplacental clot is expelled most often simultaneously with the delivery of the baby. Inj. oxytocin 10.IU IV (slow) or IM or Inj. methergine 0.2 mg IV is given with the delivery of the baby to minimize postpartum blood loss. Oxytocics should be used to improve the uterine tone along with blood transfusion.

(B) **Cesarean section:** *Indications are* : (a) severe abruption with live fetus (b) amniotomy could not be done (unfavorable cervix) (c) prospect of immediate vaginal delivery despite amniotomy is remote (d) amniotomy failed to control bleeding (e) amniotomy failed to arrest the process of abruption (rising fundal height) (f) appearance of adverse features (fetal distress, falling fibrinogen level, oliguria).

SCHEME OF MANAGEMENT OF ABRUPTIO PLACENTAE


Abruptio Placentae
Emergency measures
Infusion—Crystalloids
Blood transfusion
Periodic coagulation profile
Urine output
Fetal monitoring (Electronic)
General and abdominal examination
Fetal status
Grade of abruption (p. 297)
Hb%, Hematocrit, Coagulation profile
ABO and Rh group
Resuscitation
Revealed
Expectant management of placental abruption is an exception, not the rule (p.299)
Concealed
Delivery
Patient in labor
Patient not in labor
ARM ± Oxytocin
Delivery
ARM ± Oxytocin
Cesarean delivery (indications)
Vaginal delivery
ARM ± Oxytocin
Cesarean delivery (indications)
Vaginal delivery (Selected cases)
Vaginal delivery
OXYTOCICS TO BE CONTINUED TO IMPROVE UTERINE TONE ALONG WITH BLOOD TRANSFUSION

Anesthesia during cesarean section: Regional anesthesia is generally avoided when there is significant hemorrhage. This is to avoid profound and persistent hypotension (see p. 593).

Expectant management in a case of placental abruption is an exception and not the rule. Cases where bleeding is slight and has stopped (Grade I abruption), fetus reactive (CTG) and remote from term, may be considered. The goal of expectant management is to prolong the pregnancy with the hope of improving fetal maturity and survival. Continuous electronic fetal monitoring is maintained. Patient should be observed in the labor ward for 24–48 hours to ensure that no further placental separation is occurring. **Meanwhile betamethasone is given to accelerate fetal lung maturity** (see p. 367) in the event preterm delivery has to be contemplated. Further separation of placenta at any moment may cause fetal death or maternal complications (see below). This is the major risk of conservative management.

Management of complications (see p. 297): The major complications of placental abruption are: (a) hemorrhagic shock. (b) DIC. (c) renal failure (see p. 706) and (d) uterine atony and postpartum hemorrhage.

Hypovolemia should be corrected early. Blood pressure may not be a correct guide to assess shock, as it may be high due to severe degree of vasospasm. Irrespective of the patient's general condition, at least one liter of blood transfusion should be the minimum when the diagnosis of concealed accidental hemorrhage is made. **The best guide to monitor the patient is the use of central venous pressure (CVP), which is maintained at 10 cm of water. Hematocrit should be at least 30% and urinary output ≥ 30 mL/h**.

A. Hemorrhagic shock—Classification of obstetric hemorrhage is based upon volume deficit (see p. 703). Details of management is discussed (see p. 704).

B. DIC—Release of tissue thromboplastin in placental abruption causes consumptive coagulopathy (see p. 711). Diagnosis is based on the coagulation profile assessment. Treatment is to restore the hematologic deficiency (fibrinogen level ≥ 150 mg/dL), 1 unit (500 mL) of fresh blood contains 0.5 mL g of fibrinogen and raises the fibrinogen level by 12.5 mg/dL. Platelet count increases by 10,000–15,000/cu mm to replenish the volume deficit and to arrest the pathologic process (delivery). Details of management is discussed (see p. 715).

Feto-maternal hemorrhage is common with traumatic variety of placental abruption. To combat feto-maternal hemorrhage 300 mcg of **anti-D immunoglobulin** is administered to all Rh-negative women. The amount of fetal to maternal bleed is usually < 15 mL (see p. 387).

INDETERMINATE BLEEDING

The exact cause of vaginal bleeding in late pregnancy is not clearly understood in few cases. The diagnosis of unclassified bleeding should be made after exclusion of placenta previa, placental abruption and local causes (see p. 282). Rupture of vasa previa, marginal sinus hemorrhage, circumvallate placenta, marked decidual reaction on endocervix or excessive show may be a possible cause of such bleeding.

VASA PREVIA: The unsupported umbilical vessels in velamentous placenta, lie below the presenting part and run across the cervical os. These vessels are torn either spontaneously or during rupture of membranes. Color-flow Doppler (TVS) is helpful for antenatal diagnosis. Fetal mortality is high (50%) due to fetal exsanguination. Detection of nucleated red blood cells (Singer's alkali denaturation test) or fetal hemoglobin (Apt test) is diagnostic. Vaginal bleeding is often associated with fetal distress (tachycardia, sinusoidal FHR tracing).

MANAGEMENT: Management depends on fetal gestational age, severity of bleeding, persistence or recurrence of bleeding. Center must be equipped with appropriate neonatal care facilities in view of preterm delivery.

A Considering the risks of bleeding, patient with confirmed vasa previa, needs antenatal admission at 28–32 weeks of gestation. Expectant management can be done in selected cases for fetal lung maturity similar to placenta previa. **Antenatal corticosteroids should be given** (see p. 542).

B Any case with bleeding vasa previa, delivery should be done by category-1 (see p. 671) emergency cesarean section. Intrapartum diagnosis of vasa previa, needs expeditious delivery.

C A case of confirmed vasa previa at term (≥37 weeks) should be delivered by elective cesarean section prior to onset of labor.

D Neonatal blood transfusion may be needed.

SUMMARY OF ANTEPARTUM HEMORRHAGE

- **Hemorrhage is a major cause of** maternal morbidity and mortality throughout the world (p. 684).
- **Antepartum hemorrhage is defined as the bleeding from or within the genital tract** after 28th week of pregnancy but before the birth of the baby. Causes may be placental, extra placental or unexplained (p. 282).
- **Major causes of APH are two:** placenta previa and abruptio placentae.
- **Placenta previa are 4 types (p. 283).** Placentography (USG) confirms the diagnosis (p. 286). Abruptio placentae should be differentiated from placenta previa (p. 287, 298).
- **Placenta previa can be diagnosed by**—(i) ultrasonography (preferred), (ii) clinically, transvaginal ultrasound classify placenta previa: (a) within 2 cm or (b) > 2 cm from the undilated internal cervical os. Vaginal examination for the diagnosis of placenta previa should not be done as it provokes severe hemorrhage (p. 285).
- **Imaging modalities (Doppler USG, MRI)** have reduced the need of double set up examination and the risk of bleeding thereof as they can make improved diagnosis of placenta previa, accreta and abruption.
- **Management of placenta previa (a) Expectant:** pregnancy is preterm (<37 weeks), no active bleeding and the fetus is reactive (CTG), patient is admitted and managed expectantly (b) **Active intervention**: Presence of active bleeding, term pregnancy, patient in labor or with non-reassuring fetal status – Delivery.
- **Maternal complications of placenta previa are:** hemorrhage (antepartum, intrapartum, postpartum), retained placenta (placenta accreta), increased operative delivery and death.
- **Fetal complications of placenta previa are:** Prematurity, asphyxia, IUFD and increased perinatal mortality.

Delivery is planned based on the sonographic location of placenta. Women with placenta previa with placental edge within 2 cm of internal os are delivered by cesarean section. Otherwise vaginal delivery may be allowed.

- **Risk factors for placental abruption are:** increased maternal age, increased parity, hypertension, thrombophilia, rapid uterine decompression (polyhydramnios), trauma or smoking.
- **Placental abruption is diagnosed** mainly clinically and supported by laboratory, USG or MRI.
- **Management of placental abruption** depends on severity of placental abruption, gestational age and condition of the mother and the fetus.
- **Delivery is done in most cases of placental abruption.** Betamethasone is given to accelerate fetal lung maturation. Expectant management of placental abruption is rarely done.

QUESTIONS

Write Short Notes on:

A. Methods of placental localization (p. 285)
B. Complications of placenta previa (p. 288)
C. Complications of abruptio placentae (p. 297)
D. Vasa previa (p. 301)

Related theory questions (Long & Short), Obstetric Case Discussions, Viva table discussions, Post operative word round discussions, and MCQs are discussed in author's books:

Bedside Clinic and Viva Voce: 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

For further reading:

Master pass in Obstetrics and Gynaecology: 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

Chapter 20

Medical and Surgical Illness Complicating Pregnancy

HEMATOLOGICAL DISORDERS IN PREGNANCY

ANEMIA IN PREGNANCY

Anemia is the commonest hematological disorder that may occur in pregnancy, the others being rhesus isoimmunization and blood coagulation disorders.

INCIDENCE: According to the standard laid down by WHO, anemia in pregnancy is present when the hemoglobin concentration in the peripheral blood is 11 g/100 mL or less. During pregnancy plasma volume expands (maximum around 32 weeks) resulting in hemoglobin dilution. For this reason, hemoglobin level below 10 g/dL at any time during pregnancy is considered anemia (WHO, 1993; CDC, 1990). Hb level at or below 9 g/dL requires detailed investigations and appropriate treatment. Adopting this lower level, the incidence of anemia in pregnancy ranges widely from 40% to 80% in the tropics compared to 10% to 20% in the developed countries. **Anemia is responsible for 20% of maternal deaths in the third world countries (p. 685)**.

CLASSIFICATION

The anemia may be classified in various ways. For all practical purposes, a simplified classification is given which is helpful in the management of the cases. Not uncommonly, an atypical form of anemia may be met with and in such cases, the opinion of a hematologist should be sought for.

- **Physiological anemia of pregnancy**
- **Pathological**
- Deficiency anemia (isolated or combined)
  - Iron deficiency
  - Folic acid deficiency
  - Vitamin B_{12} deficiency
  - Protein deficiency
- Hemorrhagic
  - Acute: Following bleeding in early months or APH
  - Chronic: Hookworm infestation, bleeding piles, etc.
- Hereditary
  - Thalassemias
  - Sickle cell hemoglobinopathies
  - Other hemoglobinopathies
  - Hereditary hemolytic anemias (RBC membrane defects, spherocytosis)

- Bone marrow insufficiency—hypoplasia or aplasia due to radiation, drugs (aspirin, indomethacin)
- Anemia of infection (malaria, tuberculosis, kala-azar)
- Chronic disease (renal) or neoplasm
- Hematologic malignancy (leukemias, lymphomas)

However, the obstetricians are more concerned with two common types of anemia—the deficiency anemia and hemorrhagic anemia. For detailed description of other types, any standard book of hematology may be consulted.

CONCEPT OF PHYSIOLOGICAL ANEMIA

Maternal plasma volume increases by about 40–50%. RBC volume increases by 20%. There is relative fall in the level of hemoglobin and hematocrit during pregnancy. All these values return to normal by 6 weeks postpartum. In addition, there is marked demand of extra iron during pregnancy especially in the second half. Even an adequate diet cannot provide the extra demand of iron. Thus, there always remains a physiological iron deficiency state during pregnancy. As a result, there is not only a fall in hemoglobin concentration and hematocrit value in the second half of pregnancy but there is also associated low serum iron, increased iron binding capacity and increased rate of iron absorption as found in iron deficiency anemia.

Table 20.1: Normal Blood Values in Non-pregnant and Pregnant State

Blood Values	Non-pregnant	Second Half Pregnancy
Hemoglobin (Hb)	14.8 gm/100 mL	11–14 gm/100 mL
Red blood cells (RBC)	5 million/cu mm (mm^3)	4–4.5 million/mm^3
Packed cell volume (PCV) (Hematocrit)	39–42 percent	32–36 percent
Mean corpuscular hemoglobin (MCH)	27–32 micromicron (picogram-pg)	26–31 pg
Mean corpuscular volume (MCV)	75–100 cubic micron (μ^3)	75–95 μ^3
Mean corpuscular hemoglobin concentration (MCHC)	32–36 percent	30–35 percent
Serum iron	60–120 µg/100 mL	Slightly lowered (65–75 µg/100 mL)
Total iron binding capacity (TIBC)	300–350 µg/100 mL	Increased (300–400 µg/100 mL)
Saturation percentage (Ratio— Serum iron: TIBC)	30%	Less than 16%
Serum ferritin	20–30 µg/L (mean)	15 mg/L (mean)

Thus, the fall in the hemoglobin concentration during pregnancy is due to combined effect of hemodilution and negative iron balance. The anemia is normocytic and normochromic in type. Normal blood values in non-pregnant and pregnant state are given in the Table 20.1.

CRITERIA OF PHYSIOLOGICAL ANEMIA: The lower limit of physiological anemia during the second half of pregnancy should fulfill the following hematological values: (1) Hb-10 gm% (2) RBC-3.2 million/mm^3 (3) PCV-32%. (4) Peripheral smear showing normal morphology of the RBC with central pallor.

ERYTHROPOIESIS: In adults, erythropoiesis is confined to the bone marrow. Red blood cells are formed through stages of pronormoblasts → normoblasts → reticulocytes → to mature non-nucleated erythrocytes. The average life span of red blood cells is about 120 days after which the RBC degenerate and the hemoglobin is broken down into hemosiderin and bile pigment. For proper erythropoiesis, adequate nutrients are needed. These are minerals, vitamins, proteins and hormones. Inadequate reserve or increased demand or deficient supply of any of the constituents interferes with the normal erythropoiesis.

(A) **Minerals:** (i) Iron is essential element in the synthesis of hemoglobin (ii) Traces of copper and cobalt are also required in the synthesis.

(B) **Vitamins:** The specific vitamins that are required in the maturation process are vitamin B_{12}, folic acid and vitamin C. Folic acid and vitamin B_{12} are essential in the synthesis of nucleoprotein, particularly of erythropoietic cells. Vitamin B_{12} acts at an early stage in the synthesis of RNA but folic acid acts at a later stage in the synthesis of DNA. **Thus, deficiency of Vitamin B_{12} results in defective synthesis of both RNA and DNA, while the deficiency of the folic acid results in defective synthesis of DNA only.** Vitamin C is essential for conversion of folic acid to folinic acid.

(C) **Proteins:** The proteins supply the amino acid for the synthesis of globin moiety.

(D) **Erythropoietin:** The hormone is responsible for increase in red cells volume, by stimulating the stem cells in the bone marrow. Increased secretion of erythropoietin is brought about mostly by placental lactogen and also by progesterone. Erythropoietin is produced by the kidneys (90%) and the liver (10%).

CAUSES OF INCREASED PREVALENCE OF ANEMIA IN TROPICS

Iron deficiency anemia is very much prevalent in the tropics particularly amongst women of child bearing age (75%), especially in the underprivileged sector. In a healthy individual, a **daily intake of dietary iron of 15 mg** can replenish the daily loss of about 1.5 mg of iron assuming an **absorption rate of 10%**. But in the tropical countries especially with low socioeconomic group, **the daily requirement is likely to be more because of the following**.

BEFORE PREGNANCY:

- ***Faulty dietetic habit:*** There is no deficiency of iron in the diet but the diet is rich in carbohydrate. High phosphate and phytic acid help in the formation of insoluble iron phosphate and phytates in the gut, thereby reducing the absorption of iron.

- ***Faulty absorption mechanism:*** Because of high prevalence of intestinal infestation, there is intestinal hurry which reduces the iron absorption. Hypochlorhydria, often associated with malnutrition also hinders absorption.

- ***Iron loss:*** (i) **More iron is lost through sweat** to the extent of 15 mg per month. (ii) **Repeated pregnancies at short intervals** along with a prolonged period of lactation puts a serious strain on the iron reserve. It has been estimated that a normal healthy woman with adequate diet takes about 2 years to replenish about 1,000 mg of iron lost during childbirth and lactation (iii) **Excessive blood loss during menstruation** which is left untreated and uncared for (iv) **Hookworm infestation** with consequent blood depletion to the extent of 0.5–2 mg of iron daily (each worm extracts up to 0.05 mL of blood per day). (v) **Chronic malaria**, chronic blood loss due to **bleeding piles and dysentery** also cause iron deficiency anemia.

DURING PREGNANCY: The woman who has got sufficient iron reserve and is on a balanced diet, is unlikely to develop anemia during pregnancy in spite of an increased demand of iron. But if the iron reserve is inadequate or absent, **the factors which lead to the development of anemia during pregnancy are:**

- ***Increased demands of iron:*** As previously stated (p. 62) the demand of iron during pregnancy is markedly increased. An adequate balanced diet contains not more than 18–20 mg of iron and assuming that the absorption rate is increased by twofold (20%), the demand is hardly fulfilled.

- ***Diminished intake of iron:*** Apart from socioeconomic factors, faulty dietetic habits, loss of appetite and vomiting in pregnancy are responsible factors.

- ***Diminished absorption:*** Acid environment in the duodenum helps iron absorption. On the other hand intake of antacids, H_2 blockers and proton pump inhibitors inhibit iron absorption.

- ***Disturbed metabolism:*** **Presence of infection markedly interferes with the erythropoiesis**; one should not even ignore the presence of **asymptomatic bacteriuria**.

- ***Pre-pregnant health status:*** Majority of the women in the tropics actually start pregnancy on a pre-existing anemic state or at least with inadequate iron reserve. It is the state of the stored iron which largely determines whether or not and how soon a pregnant woman will become anemic.

- ***Excess demand:*** (i) **Multiple pregnancy** increases the iron demand by twofold. (ii) **Women with rapidly recurring pregnancy**, within 2 years following the last delivery, need more iron to replenish deficient iron reserve. (iii) **The demand of iron which accompanies the natural growth before the age of 21**, should not be underestimated, specially where teenage pregnancies are quite prevalent.

IRON DEFICIENCY ANEMIA

CLINICAL FEATURES: The clinical features depend more on the degree of anemia than anything else. In the majority, the patients have got no symptom and the entity is detected accidentally during examination. However, the following features may develop slowly.

Symptoms: (1) Lassitude and fatigue or weakness may be the earliest manifestations. (2) The other features are anorexia and indigestion; palpitation caused by ectopic beats, dyspnea, giddiness and swelling of the legs.

On examination: (1) There is pallor of varying degrees; evidences of glossitis and stomatitis. (2) Edema of the legs may be due to hypoproteinemia or associated preeclampsia. (3) A soft systolic murmur may be heard in the mitral area due to physiological mitral incompetence. (4) Crepitations may be heard at the base of the lungs due to congestion.

INVESTIGATIONS: The patient having a hemoglobin level 9 gm% or less should be subjected to a full hematological investigation. The objectives of investigation are to ascertain:

- **Degree of anemia**
- **Type of anemia**
- **Cause of anemia**

To note the degree of anemia: This requires hematological examination which includes estimation of: (1) Hemoglobin, (2) total red cell count (The red cell count is not of great value unless changed to the extreme) and (3) determination of packed cell volume. All these help not only to identify the physiological anemia of pregnancy but also to note the degree of pathological anemia. **Arbitrary grading of pathological anemia** is done according to the level of hemoglobin: **mild—between 8 gm% and 10 gm%, moderate—less than 8 to 7 gm% and severe—less than 7 gm%**.

To ascertain the type of anemia:

- **Peripheral blood smear:** Examination of a well-prepared peripheral blood smear stained with Leishman stain to study the morphology of the red cells gives a better idea, about the type of anemia. Abundant presence of small pale staining cells with variation in size (anisocytosis) and shape (poikilocytosis) suggest microcytic hypochromic anemia. This is typical in iron deficiency anemia. Reticulocyte count may be slightly raised (Fig. 20.1).

- **Hematological indices:** Calculation of MCHC, MCV and MCH is based on the values of hemoglobin estimation, total red cells count and PCV. MCV and MCH values are not much dependable because of inherent inaccuracy in red cell count which is involved in their calculations. MCHC is the most sensitive index of iron deficiency anemia. It should be remembered that these hematological indices should supplement and not substitute the blood smear examination for correct typing of anemia. **A typical iron deficiency anemia shows the following blood values:** Hemoglobin—less than 10 gm%, red blood cells —less than 4 million/mm^3, PCV—less than 30%, MCHC— less than 30%, MCV— less than 75 μ^3 and MCH—less than 25 pg.

To find out the cause of anemia: Appropriate investigations should be undertaken as per the history and clinical examination to find out the cause of anemia. But the following should be done as a routine.

- **Examination of stool:** This should be done as a routine especially in tropics, to detect helminthic (particularly hookworm) infestation.
- **The urine is examined** for the presence of protein, sugar and pus cells. A "clean catch" mid stream specimen of urine is subjected to culture and colony count. If the counts are over 10^5/mL, it indicates infection.

Bood Values in Iron Deficiency Anemia

- Serum iron is <30 µg/100 mL.
- Total iron binding capacity is raised (>400 mcg/dl)
- Percentage saturation is 10% or less
- Serum ferritin below 30 µg/l (confirms iron deficiency anemia)
- Serum bilirubin is not raised

When definite diagnosis as to the cause of anemia cannot be made with the above, further investigations may be directed according to the clinical findings. This may necessitate X-ray of chest in suspected pulmonary tuberculosis, fractional test meal analysis of gastric juice to find out achlorhydria in pernicious anemia, estimation of serum protein in hypoproteinemia or osmotic fragility in hereditary spherocytosis or hemoglobinopathic disorders.

Place of bone marrow study: **This is not done as a routine but indicated in**: (1) Cases not responding to therapy according to hematological typing. (2) To diagnose hypoplastic anemia. (3) To diagnose kala-azar by detecting L.D. bodies. **In iron deficiency anemia**, the bone marrow is normoblastic in character. There is absence of hemosiderin granules when stained with Prussian blue.

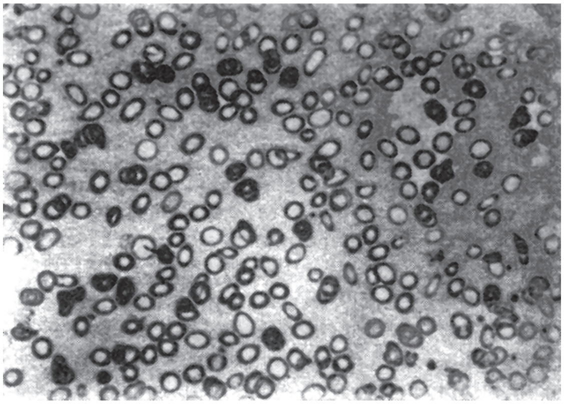


Fig. 20.1: Hypochromic microcytic anemia (*Courtesy: Dr SK Dutta*)

DIFFERENTIAL DIAGNOSIS: All the causes of hypochromic anemia are to be differentiated. Apart from iron deficiency, other causes are: (1) Infection, (2) Nephritis and preeclampsia, (3) Hemoglobinopathies.

COMPLICATIONS OF ANEMIA IN PREGNANCY

DURING PREGNANCY: The following complications are likely to increase: (1) **Preeclampsia** may be related to malnutrition and hypoproteinemia. (2) **Intercurrent infection**—Not only does anemia diminish resistance to infection, but also any pre-existing lesion, if present, will flare up. It should be noted that the infection itself impairs erythropoiesis by bone marrow depression. (3) **Heart failure** at 30–32 weeks of pregnancy. (4) **Preterm labor**.

DURING LABOR: (1) **Uterine inertia** is not a common associate, on the contrary the labor is short because of a small baby and multiparity. (2) **Postpartum hemorrhage is a real threat**. Patient tolerates even a minimal amount of blood loss. (3) **Cardiac failure** may be due to accelerated cardiac output which occurs during labor or immediately following delivery. As the blood in the uterine circulation is squeezed in the general circulation, it puts undue strain on the weak heart already compromised by hypoxia. (4) **Shock—**Even a minor traumatic delivery without bleeding may produce shock or a minor hypoxia during anesthesia which may be lethal.

PUERPERIUM: There is increased chance of: (1) Puerperal sepsis (2) Subinvolution (3) Poor lactation (4) Puerperal venous thrombosis (5) Pulmonary embolism.

Risk periods: The risk periods when the patient may even die suddenly are: (1) At about 30–32 weeks of pregnancy (2) During labor (3) Immediately following delivery (4) Any time in puerperium especially 7–10 days following delivery due to cardiac failure or pulmonary embolism.

EFFECTS ON BABY: Amount of iron transferred to the fetus is unaffected even if the mother suffers from iron deficiency anemia. So the neonate does not suffer from anemia at birth. (1) There is increased incidence of low birth weight babies with its incidental hazards (2) Intrauterine death—due to severe maternal anoxemia. The sum effect is increased perinatal loss.

PROGNOSIS

MATERNAL: If detected early and proper treatment is instituted, anemia improves promptly. At times, there is a tendency for anemia to recur in subsequent pregnancy. In fact, **anemia either directly or indirectly contributes to about 20% of maternal deaths in the third world countries (p. 685)**.

FETAL: If detected early and responsive to treatment, the fetal prognosis is not too bad. In severe and neglected cases, the fetal prognosis is adversely affected by prematurity with its hazards. **Baby born at term, to severely anemic mother will not be anemic at birth, but as there is little or no reserve iron, anemia develops in neonatal periods. Mean cord blood levels of serum iron, ferritin, B_{12} and folate are higher than that of mother. However, total iron binding capacity and serum level of vitamin E are lower than that of mother**.

TREATMENT

PROPHYLACTIC

In the tropics, majority of cases with iron deficiency anemia in pregnancy have a low socioeconomic status. The anemia is either pre-existing or is aggravated during pregnancy.

The prophylaxis includes: **Avoidance of frequent child-births**—a minimum interval between pregnancies, should be at least 2 years, if not three, to replenish the lost iron during childbirth process and lactation. This can be achieved by proper family planning guidance.

- **Supplementary iron therapy:** Only 20% of pregnant women have iron stores of 500 mg which is the minimum essential for pregnancy and about 40% women have virtually no iron stores.

  It has been observed that majority of women during pregnancy have either low or virtually no iron stores. Therefore most women should be given iron supplementation during pregnancy.

  Even with a well-balanced diet, supplementary iron should be a routine after the patient becomes free from nausea of pregnancy. Daily administration of 200 mg of ferrous sulfate (containing 60 mg of elemental iron) along with 1 mg folic acid is a quite effective prophylactic procedure. Tea should be avoided within 1 hour of taking iron tablet.
- **Dietary prescription:** A realistic balanced diet, rich in iron and protein, should be prescribed which should be within the reach of the patient and should be easily digestible. The foods rich in iron are liver, meat, egg, green vegetables, green peas, figs, beans, whole wheat and green plantains, onion stalks, jaggery, etc. Iron utensils should preferably be used for cooking and the water used in rice and vegetable cooking should not be discarded.
- **Adequate treatment** should be instituted to eradicate hookworm infestation, dysentery, malaria, bleeding piles, and urinary tract infection.
- **Early detection of falling hemoglobin level is to be made. Hemoglobin level should be estimated at the first antenatal visit, at the 30th week and finally at 36th week**.

CURATIVE

Anemia is not a disease but a sign of an underlying disorder. **Treatment must be preceded by an accurate diagnosis of the cause of anemia and type of anemia**.

Hospitalization: (1) Ideally all patients having hemoglobin level 9 gm/100 mL or less should be admitted for investigation and treatment. But due to high prevalence of anemia and inadequate hospital beds, **an arbitrary hemoglobin level of 7.5 gm/dL may be considered, when the patient should be hospitalized.** (2) Associated obstetrical-medical complication even with moderate degree of anemia.

General treatment:

- **Diet**: A realistic balanced diet rich in proteins, iron and vitamins and which is easily assimilable is prescribed.
- **To improve the appetite and facilitate digestion**, preparation containing acid pepsin may be given thrice daily after meals.
- **To eradicate** even a minimal septic focus by appropriate antibiotic therapy.
- **Effective therapy to cure** the disease contributing to the cause of anemia.

Specific therapy

The principle is to raise the hemoglobin level as near to normal as possible. Thereafter, an attempt is made to restore the iron reserve at least in part, if possible, before the patient goes in labor.

Choice of therapy depends on: (1) Severity of anemia (2) Duration of pregnancy (time available before delivery). (3) Associated complicating factors.

IRON THERAPY: ♦ **Oral therapy** ♦ **Parenteral therapy**

Oral therapy

Iron is best absorbed in the ferrous form and as such any of the ferrous preparations available either in tablets or capsules may be conveniently prescribed. The preparations available are ferrous gluconate, ferrous fumarate or ferrous succinate. In spite of claims about the superiority of one preparation over the other, ferrous sulfate is widely used. Fersolate tablet contains 325 mg ferrous sulfate which contains 60 mg of elemental iron, trace of copper and manganese. The initial dose is one tablet to be given thrice daily 30 minutes before meals. If larger dose is necessary (maximum six tablets a day), it should be stepped up gradually in 3–4 days. The treatment should be continued till the blood picture becomes normal; **thereafter a maintenance dose of one tablet daily is to be continued for at least 100 days following delivery to replenish the iron stores**.

- *Drawbacks:* (1) **Intolerance—**The intolerance is evidenced by epigastric pain, nausea, vomiting and diarrhea or constipation. It may be related to increased dose of iron or to some preparation. To avoid intolerance, it is preferable to start the therapy with a smaller dose—one tablet daily and then to increase the dose to a maximum three tablets a day. If such a procedure fails to stop the symptoms, an alternate preparation may be prescribed.

  (2) **Unpredictable absorption rate**—Because of various unforeseen factors which are involved in the iron absorption and its utilization, the therapy cannot be instituted confidently when rapid response is needed. Antacids, oxalates and phosphates will reduce absorption while ascorbic acid, lactate and various amino acids increase its absorption.

  (3) **With the therapeutic dose**, the serum iron may be restored but there is difficulty in replenishing the iron store.
- *Response of therapy is evidenced by:* (1) Sense of well-being (2) Increased appetite (3) Improved outlook of the patient (4) Hematological examination: (a) Rise in hemoglobin level, (b) hematocrit value returning to normal, (c) reticulocytosis within 7–10 days. If no significant improvement is evident clinically and hematologically within 3 weeks, diagnostic re-evaluation is needed.

- *Rate of improvement*: The improvement should be evident within 3 weeks of the therapy. After a lapse of few days, the hemoglobin concentration is expected to rise at the rate of about 0.7 gm/100 mL/week.
- *Causes of failure of improvement:* (1) Improper typing of anemia (2) Defective absorption due to associated gastrointestinal disorders (3) The patient fails to take iron (4) Concurrent blood loss as in hookworm infestation or bleeding piles (5) Inhibition of erythropoiesis by infection (6) Co-existent folate deficiency.
- *Contraindications of oral therapy*: The following are the contraindications of oral therapy: (1) Intolerance to oral iron. (2) Severe anemia in advanced pregnancy. Considering the unpredictable absorption and utilization following oral therapy, parenteral therapy is the preferred choice.

Parenteral therapy:

- **Intravenous route:** (i) Repeated injections (ii) Total dose infusion (TDI)
- **Intramuscular route**

Indications of parenteral therapy:

— Contraindications of oral therapy as previously mentioned.

— Patient is not cooperative to take oral iron.

— Cases seen for the first time during the last 8–10 weeks with severe anemia.

The main advantage of parenteral therapy is the certainty of its administration to correct the hemoglobin deficit and to fix up the iron store. The expected rise in hemoglobin concentration after parenteral therapy is 0.7 to 1 g/100 mL/week.

(A) **Intravenous route**:

- **Total dose infusion (TDI):** The deficit of iron is first calculated and the total amount of iron required to correct the deficit is administered by a single sitting intravenous infusion. The compounds used are: **iron (ferrous) sucrose** compound, **sodium ferric gluconate** or **iron dextran**. **Iron sucrose** is safe, effective and has less side effects (ACOG–2008).

Advantages: (1) It eliminates repeated and painful intramuscular injections. (2) The treatment is completed in a day and the patient may be discharged much earlier from the hospital. (3) It is less costly compared to the repeated intramuscular therapy.

Limitations: (1) As the maximum hemoglobin response does not appear before 4 to 9 weeks, the method is unsuitable if at least 4 weeks' time is not available, to raise the hemoglobin to a safe level of 10 gm/dL before delivery. Thus, **it is mostly suitable during 30–36 weeks of pregnancy** where the patient is unwilling or unable to complete the course of intramuscular injections (2) Previous history of reaction to parenteral therapy is contraindicated for its use.

- Sodium ferric gluconate complex 12.5 mg/dose usually one dose/day, usually 8 doses needed (12.5 mg elemental iron/mL).
- **Iron (ferrous) Sucrose:** (20 mg elemental iron/mL) 100 mg/dose, usually one dose daily for 10 days.

Estimation of the total requirement: The manufacturer's information is to follow for dose calculation. One such formula for **iron dextran** is:

- 0.3 × W (100–Hb%) mg of elemental iron. Where W = patient's weight in pounds. Hb% = observed hemoglobin concentration in percentage. Additional 50% is to be added for partial replenishment of the body store iron.

Example (iron dextran): The total elemental iron required in an anemic patient weighing 100 lb with hemoglobin 50% is calculated as follows: $0.3 \times 100\ (100 - 50) = {}^{3}/_{10} \times 100 \times 50 = 1500$ mg. Add 50% = 750 mg. Total elemental iron required 2,250 mg.

Prerequisites: (1) Correct diagnosis of true iron deficiency anemia (2) Adequate supervision (3) Facilities for management of anaphylactic reaction.

Procedures:

- The patient is admitted in the morning for infusion
- The required iron is mixed with 500 mL of 0.9% saline. Manufacturer's guidance in the drug information sheet is to be followed as regard the total dose and duration of therapy
- Precautions like those of blood transfusion are to be taken both prior to and during the infusion process
- The drip rate should be 10 drops per minute during the first 20 minutes and thereafter is increased to 40 drops per minute
- Any adverse reaction like rigor, chest pain or hypotension calls for omission of the drip

(B) **Intramuscular therapy:** The compounds used (with elemental iron/ml) are—

- Iron sucrose (20 mg/ml)
- Iron-dextran (Imferon) (50 mg/ml)
- Sodium ferric gluconate complex 12.5 mg elemental iron/mL

All the above preparations contain different amount of elemental iron in 1 milliliter (see above). Total dose to be administered is calculated as that previously mentioned in intravenous therapy.

Procedure of injections: After an initial test dose of 1 mL, the injections are given daily or on alternate days in doses of 2 mL intramuscularly. **To prevent dark staining of the skin over the injection sites and to minimize pain**, the injections are given with a 2 inch needle deep into the upper outer quadrant of the buttock using a 'Z' technique (pulling the skin and subcutaneous tissues to one side before inserting the needle). An additional precaution is to inject small quantity of air or saline down the needle before withdrawing it. These procedures prevent even a slight drop of the solution to come beneath the skin surface so as to stain it.

Drawbacks: (1) The injections are painful although less with Jectofer. (2) Chance of abscess formation and a discoloration of the skin over the injection sites are real problems especially with Imferon. (3) Reactions are far and few—pyrexia, lymphadenopathy, headache, nausea, vomiting and allergic reactions are infrequently met with.

PLACE OF BLOOD TRANSFUSION: The indication of blood transfusion in anemia during pregnancy is very much limited. **The indications are**:

(1) To correct anemia due to blood loss and to combat postpartum hemorrhage.
(2) Patient with severe anemia seen in later months of pregnancy (beyond 36 weeks)—to improve the anemic state and oxygen carrying capacity of blood before the patient goes into labor. The primary concern is not only to correct anemia but also to make the patient fit to withstand the strain of labor and blood loss following delivery.
(3) Refractory anemia: Anemia not responding to either oral or parenteral therapy in spite of correct typing.
(4) Associated infection

The quality and quantity of blood: The blood to be transfused should be relatively fresh, properly typed, grouped and cross matched. Only packed cells are transfused. The quantity should be between 80 mL and 100 mL at a time. To allow time for circulatory readjustment, transfusion should not be repeated within 24 hours.

Advantages of blood transfusion: (1) Increases oxygen carrying capacity of the blood (2) Hemoglobin from the hemolyzed red cells may be utilized for the formation of new red cells (3) Stimulates erythropoiesis. (4) Supplies the natural constituents of blood like proteins, antibodies, etc. (5) Improvement is expected after 3 days.

Precautions: Utmost precautions are to be taken to minimize reaction and over loading of the heart. (1) Antihistaminic (Phenergan 25 mg) is given intramuscularly (2) Diuretics (Frusemide 20 mg) is given intramuscularly at least 2 hours prior to transfusion to produce negative fluid balance (3) The drip rate should be about 10 drops per minute (4) To observe carefully the pulse, respiration and crepitations in the base of lungs.

Drawbacks: (1) Premature labor may start which is more related to blood reaction. (2) There is increased chance of cardiac failure with pulmonary edema because of overloading of the heart. (3) Features of transfusion reaction, if occur, are often exaggerated.

Exchange transfusion: The place of exchange transfusion is very much limited except in desperate cases. **Its indications are:** (1) Cardiac failure due to severe anemia (2) Cases of severe anemia requiring surgery (3) Severe anemia whatever may be the type (with packed cell volume less than 13%) near term as a safer alternative to simple transfusion. The method is well tolerated and dramatic improvement of the patient‘s outlook occurs within 30 minutes. **The main drawbacks are**: (1) Large quantity of fresh blood is required and that too has to be collected from several donors. (2) Chance of serum hepatitis is more.

Quantity of blood transfused: This is in fact a partial exchange transfusion when 1300 mL of packed cells are transfused under pressure through an antecubital vein with simultaneous withdrawal of 1500 mL of blood from the opposite femoral vein. Thus the patient ends up with a deficit in blood volume although with marked improvement of oxygen carrying capacity of the red cells.

MANAGEMENT DURING LABOR

First stage: The following are the special precautions that are to be taken when an anemic patient goes into labor.

- **The patient should be in bed** and should lie in a position comfortable to her.
- **Arrangements for oxygen inhalation** is to be kept ready to increase the oxygenation of the maternal blood and thus diminish the risk of fetal hypoxia.
- **Strict asepsis** is to be maintained to minimize puerperal infection.

Second stage: Asepsis is maintained. Prophylactic low forceps or vacuum delivery may be done to shorten the duration of second stage. **Intravenous methergine 0.2 mg should be given soon following the delivery of the baby**.

Third stage: **One should be very vigilant during the third stage**. Significant amount of blood loss should be replenished by fresh packed cell transfusion after taking the usual precautions mentioned earlier. **The danger of postpartum overloading of the heart should be avoided**.

PUERPERIUM: (1) Prophylactic antibiotics are given to prevent infection. (2) Predelivery antianemic therapy should be continued till the patient restores her normal clinical and hematological states. **Even in an otherwise normal case, iron therapy should be continued for at least 3 months following delivery**. (3) Patient should be warned of the danger of recurrence in subsequent pregnancies.

MEGALOBLASTIC ANEMIA

In megaloblastic anemia, there is derangement in red cell maturation with the production in the bone marrow of abnormal precursors known as megaloblasts due to impaired DNA synthesis. Thus, it may be regarded as a deficiency disease caused by **lack of either vitamin B_{12} or folate or both**. **Vitamin B_{12} deficiency is rare in pregnancy**. Vit B_{12} is first bound to intrinsic factor which is secreted by the gastric parietal cells. Thereafter it is absorbed in the distal ileum. Megaloblastic anemia in pregnancy is almost always due to folic acid deficiency. The daily requirement of Vit B_{12} in non-pregnant condition is 2 μg and during pregnancy is 3 μg. This amount is met with any diet that contains animal products. Only the strict vegetarians, may need supplementation. Folic acid is a water soluble vitamin. Folate stores are located in the liver. Normal folate store is sufficient for 6 weeks. After 3 weeks of a deficient folate diet, the serum level falls. Two weeks later there is hypersegmentation of neutrophils and after about 17 weeks RBC folate levels drop and then megaloblastic bone marrow develops. The daily folate requirement is about 200 μg during pregnancy. Folate deficiency rarely occurs in the fetus and it is not associated with any significant perinatal morbidity. 1 mg of folic acid is sufficient to prevent and treat folate deficiency.

CAUSES: Common causes of Vit B_{12} deficiency are:

(i) Strict vegetarian diet (ii) Gastritis (iii) Gastrectomy (iv) Ileal bypass (v) Crohn’s disease (vi) Drugs —Cocs, Metformin, proton pump inhibitors (vii) Addisonian pernicious anemia—rare in pregnancy (viii) Megaloblastic anemia of malabsorption syndrome.

Addisonian pernicious anemia is caused by lack of absorption of vitamin B_{12} due to lack of intrinsic factor. It is an extremely rare autoimmune disease in pregnancy because of: (1) Rare occurrence during child-bearing period (usually manifests above 40 years). (2) If occurs early, produces infertility.

For Cobalamin absorption, the requirements are: (i) Stomach acid pepsin (ii) Intrinsic factors secreted by gastric parietal cells (iii) Pancreatic proteases (iv) Intact ileum with mucosal receptors. Because of abundant vitamin B_{12} stores in the body (liver), clinical Vit B_{12} deficiency takes several years to occur. Women following bariatric surgery suffer vitamin B_{12} deficiency. Similarly women (10–30%) taking metformin.

CAUSES OF FOLIC ACID DEFICIENCY IN PREGNANCY

- **Inadequate intake due to:** (a) Nausea, vomiting and loss of appetite. (b) Dietary insufficiency—the main sources of folic acid are green leafy vegetables, cauliflower, spinach, liver, kidney. Excessive cooking destroys much of the folate in food.
- **Increased demand due to:** (a) Increased maternal tissue including red cell volume (b) Growing fetus (c) Multiple pregnancy. Daily requirement of folic acid in a non-pregnant woman is 50–100 µg/day and during pregnancy is increased to 400 µg/day.
- **Diminished absorption:** Intestinal malabsorption syndrome is responsible for its recurrence in subsequent pregnancies.
- **Abnormal demand:** (a) Infection—Infection reduces the life span of the red cells and hence increases the demand of folic acid to replenish the red cells; (b) Hemorrhagic states such as peptic ulcer, hookworm infestation, hemorrhoids and the hemolytic states such as chronic malaria, sickle cell anemia or hemoglobinopathy, lead to increased erythropoiesis and exhaustion of the available supply of folic acid resulting in megaloblastic erythropoiesis.
- **Failure of utilization:** This is associated with anticonvulsant drugs used in epilepsy or with presence of infection.
- **Diminished storage:** This is associated with hepatic disorders, hyperhomocysteinemia and vitamin C deficiency.
- **Iron deficiency anemia:** Correction of anemia by iron therapy alone may unmask the underlying folic acid deficiency state in the following manner. Anemia → depressed bone marrow → iron therapy → hyperplastic marrow → increased need for folic acid → if the extra folic acid is not supplemented, the pre-existing low level of folic acid is further lowered → ineffective erythropoiesis with evidence of megaloblastic features and simultaneous anemic state nonresponsive to simple iron therapy alone. **Thus, when the anemia fails to improve with iron therapy, addition of folic acid should be tried before proceeding for a detailed investigation**.

An elevated homocysteine level is found when serum folate levels are low. Women with high homocysteine levels are at increased risk of preeclampsia, preterm labor and IUGR. Evaluation of methylmalonate and homocysteine levels can be used to differentiate folate from vit B_{12} deficiency. Increased methylmalonate and folate levels indicate vit B_{12} deficiency whereas increased homocysteine and normal methylmalonate indicate folate deficiency. Iron deficiency is common with folic acid deficiency. Absence of reticulocytosis following replacement therapy with folic acid, iron deficiency must be excluded.

INCIDENCE: It is very difficult to estimate the exact incidence, as full hematological or bone marrow study is essential to establish the diagnosis. **The incidence varies from 0.5% to 3%**. It is **more common in multiparae** (5 times more than primigravidae) and in multiple pregnancy (eightfold increase than singleton pregnancy).

CLINICAL FEATURES: (1) The onset is usually insidious and is first revealed in the last trimester or may be acutely manifested in early puerperium (2) Anorexia or protracted vomiting (3) Occasional diarrhea (4) Constitutional symptom like unexplained fever is often associated.

On examination:

(1) Pallor of varying degree (2) Ulceration in the mouth (glossitis) and tongue (one-third cases) (3) Hemorrhagic patches under the skin and conjunctiva (4) Enlarged liver and spleen which may be difficult to palpate due to an enlarged gravid uterus (5) Features of preeclampsia may be present (two and half times increased).

Hematological examination and other blood values:

(1) **Hemoglobin level** is usually below 10 gm% (2) Stained blood film: Presence of any two of the following features in the peripheral blood, but more often seen in buffy coat preparations, are diagnostic:

(a) **Hypersegmentation of the neutrophils** (5 or more lobes), (b) macrocytosis and anisocytosis, (c) giant polymorphs, (d) megaloblasts, (e) Howell-Jolly bodies. (3) **MCV** is more than 100 μ^3. **MCH** is high (more than 33 pg), but **MCHC is normal** (4) **Associated leukopenia** and *thrombocytopenia* (5) **Serum iron** is normal or high and iron binding capacity is low (6) **Red cell folate** is below 3 ng/mL (normal non-pregnant level being 2.8–8 ng/mL) (7) **Serum vitamin B_{12} level** is below 90 pg/mL (normal levels 300 pg/mL) (8) Serum bilirubin—may be raised (9) Bone marrow—shows megaloblastic erythropoiesis.

COMPLICATIONS: The complications of iron deficiency anemia are mentioned before. The following are the special complications in megaloblastic anemia: (1) Miscarriage (2) Dysmaturity (3) Prematurity (4) Abruptio placentae (5) Fetal malformation (cleft lip, cleft palate, neural tube defects). **There is association between preconceptional folate deficiency and neural tube defects**.

PROPHYLACTIC THERAPY: All woman of reproductive age should be given 400 µg of folic acid daily. Additional amount (4 mg) should be given in situations where the demand is high. **Such conditions are**: multiple pregnancy, patient having anticonvulsant therapy, hemoglobinopathies or associated chronic infection or disease. Women, who have infants with neural tube defects, should be given 4 mg of folic acid daily beginning 1 month before conception to about 12 weeks of pregnancy.

CURATIVE: Specific therapy includes—daily administration of folic acid 4 mg orally which should be continued for at least 4 weeks following delivery. Supplementation of 1 mg of folic acid daily along with iron and nutritious diet can improve pregnancy induced megaloblastic anemia by 7–10 days. **Response is evidenced by**—(i) sense of well-being and increased appetite (ii) increase in reticulocyte, leukocyte and thrombocyte count (iii) rise in hemoglobin level. Folic acid should never be given without supplemental iron. Supplementary intramuscular vitamin B_{12} 100 µg daily or on alternate days may be added when response to folic acid alone is not adequate. Ascorbic acid 100 mg tablet thrice daily enhances the action of folic acid by converting it into folinic acid.

DIMORPHIC ANEMIA

This is the most common type of anemia met with in the tropics. It is related to dietary inadequacy or intestinal malabsorption. As such, anemia results from deficiency of both iron and folic acid or vitamin B_{12}. While there is polydeficiency state, the hematological findings or the bone marrow picture usually show predominance of one deficiency. **The red cells** become macrocytic or normocytic and hypochromic or normochromic (Fig. 20.2). Bone marrow picture is predominantly megaloblastic as the folic acid is required for the development of the number of red cell precursors. The treatment consists of prescribing both the iron and folic acid in therapeutic doses.

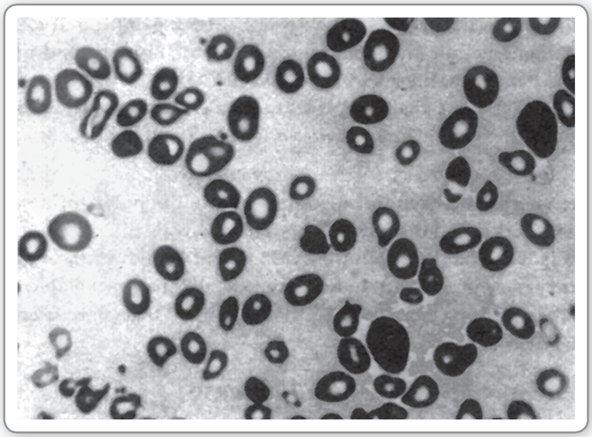
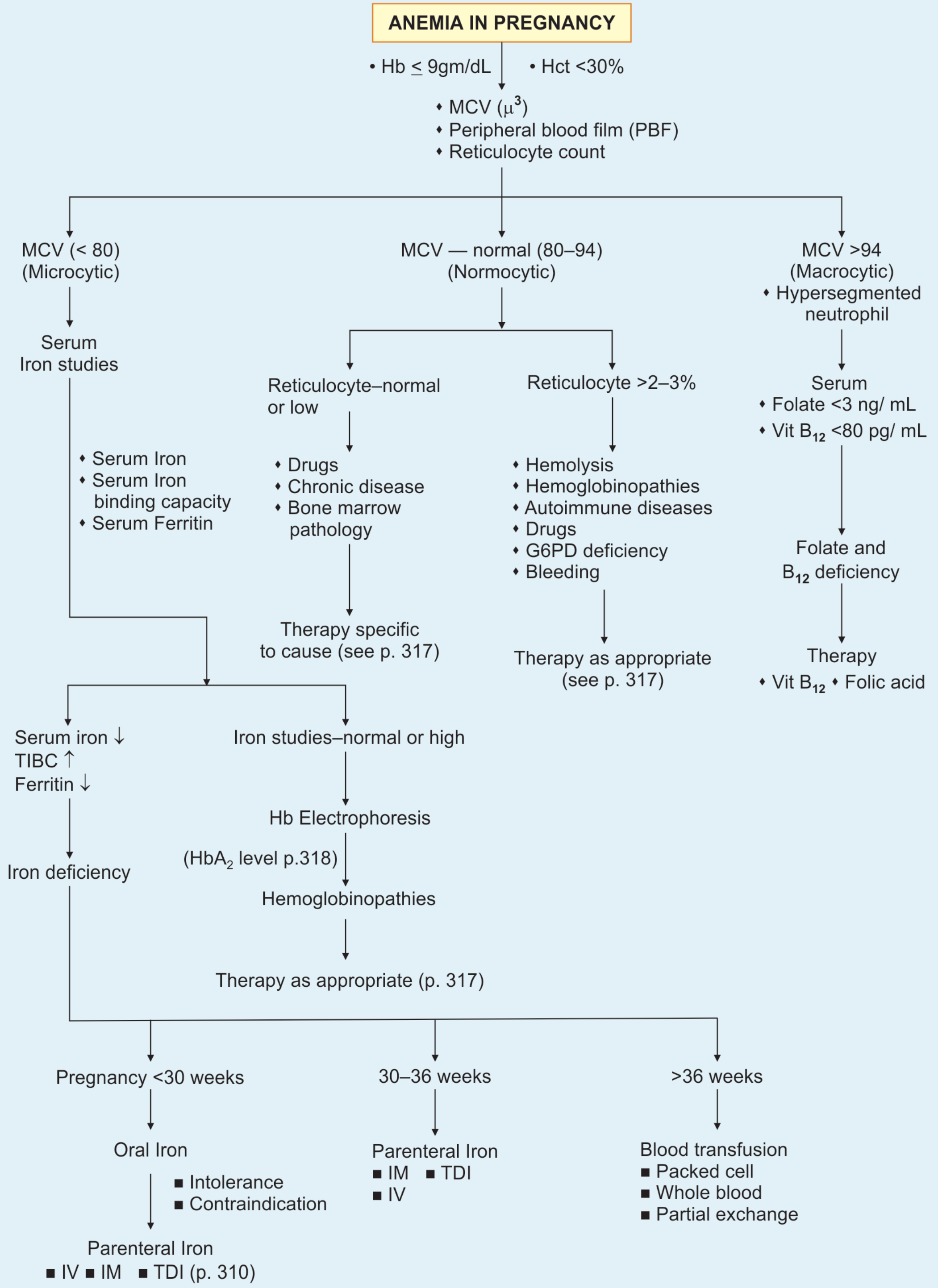


Fig. 20.2: Dimorphic anemia *(Courtesy: Dr SK Dutta)*

APLASTIC ANEMIA

It is rarely seen in pregnancy. There is marked decrease in the marrow stem cells. Exact cause is unknown. It may be immunologically mediated or may be an autosomal recessive inheritance. In about 30% of cases, anemia improves once pregnancy is terminated. **The significant complications** in pregnancy are hemorrhage and infection.

SCHEME OF INVESTIGATIONS AND SPECIFIC THERAPY FOR ANEMIA IN PREGNANCY (Hb $\leq$ 9 gm/dL and Hct < 30%)


ANEMIA IN PREGNANCY
• Hb ≤ 9gm/dL
• Hct <30%
• MCV (μ^3)
• Peripheral blood film (PBF)
• Reticulocyte count
MCV (< 80)
(Microcytic)
MCV — normal (80–94)
(Normocytic)
MCV >94
(Macrocytic)
• Hypersegmented neutrophil
Serum
Iron studies
• Serum Iron
• Serum Iron binding capacity
• Serum Ferritin
Reticulocyte–normal or low
Reticulocyte >2–3%
Serum
• Folate <3 ng/ mL
• Vit B_{12} <80 pg/ mL
• Drugs
• Chronic disease
• Bone marrow pathology
• Hemolysis
• Hemoglobinopathies
• Autoimmune diseases
• Drugs
• G6PD deficiency
• Bleeding
Folate and
B_{12} deficiency
Therapy specific to cause (see p. 317)
Therapy as appropriate (see p. 317)
Therapy
• Vit B_{12} • Folic acid
Serum iron ↓
TIBC ↑
Ferritin ↓
Iron studies–normal or high
Hb Electrophoresis
(HbA_2 level p.318)
Hemoglobinopathies
Iron deficiency
Therapy as appropriate (p. 317)
Pregnancy <30 weeks
30–36 weeks
>36 weeks
Oral Iron
■ Intolerance
■ Contraindication
Parenteral Iron
■ IV ■ IM ■ TDI (p. 310)
Parenteral Iron
■ IM ■ TDI
■ IV
Blood transfusion
■ Packed cell
■ Whole blood
■ Partial exchange

Diagnosis: ***Blood values***—Anemia, leukopenia and thrombocytopenia. **Bone marrow**—markedly hypocellular.

Management: Repeated blood transfusions are given to maintain hematocrit level above 20. Specific therapy may be needed, e.g., **granulocyte transfusion to combat infection** and **platelet transfusion to** control hemorrhage. Glucocorticoid therapy may be helpful in some patients. In a severe case of aplastic anemia, **bone-marrow** or **stem cell transplantation** is effective. Vaginal delivery is always preferred.

Anemia due to chronic diseases, infections or neoplasms is of hypochromic microcytic type. Serum iron level is low, serum ferritin level is usually elevated. Anemia of chronic renal disease is due to deficiency of erythropoietin. **Recombinant erythropoietin** therapy is found effective in cases with chronic renal disease, infection or malignancy.

HEMOGLOBINOPATHIES

Hemoglobin is a conjugated protein which contains a globin fraction bound to 4 heme moieties. There are 4 polypeptide chains within **the globin fraction—**namely alpha, beta, gamma and delta. Composition of a **normal adult hemoglobin** is: HbA ($\alpha_2\ \beta_2$) = 96.98%, HbF ($\alpha_2\ \gamma_2$) = 0.5–0.8% and HbA$_2$ ($\alpha_2\ \delta_2$) = 1.5–3.7% . In normal **fetal hemoglobin**, the beta chains are replaced by two gamma chains ($\alpha_2\ \gamma_2$). The last two varieties constitute not more than 5% in normal human adults.

Hemoglobinopathies are inherited specific biochemical disorders (quantity or quality) within the polypeptide chains of globin fraction. Two common varieties are met. **Sickle cell disease is inherited structural abnormality** involving primarily the β chain of HbA. **Thalassemia is inherited defect in the synthesis and production of globin** in otherwise normal HbA. Homozygous and heterozygous forms occur. In homozygous, the abnormal globin chain is inherited from each parent and in heterozygous, the abnormal globin chain is only inherited from one parent. The latter groups are not anemic but are carriers of the defect.

SICKLE CELL HEMOGLOBINOPATHIES

Sickle cell hemoglobinopathies are hereditary disorders. It is caused by a point mutation in the β globin gene on chromosome II. **This results in substitution of valine for glutamic acid at position 6 of the β-chain of normal hemoglobin.** Gene mutation—when **homozygous** the individual has sickle cell anemia (Hb-SS). She has a small quantity of fetal hemoglobin (HbF) but no HbA. **Heterozygous** individual for sickle cell hemoglobin has sickle cell trait (HbAS). Such an individual has about 55–60% of HbA and 35–40% of HbS. Sickle cells have a life span of 5–10 days compared to normal RBCs of 120 days. The prevalence rate of sickle cell hemoglobinopathies is highest in Africa and ranges from 20% to 50%.

HbC is produced when there is **substitution of lysine for glutamic acid** at the sixth postion of the globin-chain. HbC is less soluble than HbA and can cause hemolytic anemia. Management of these women with HbC is same as that of with women with HbSS. During crises these patients become profoundly thrombocytopenic and there is fall in hematocrit value.

Sickle cell-β-thalassemia—is observed when one β chain gene carries the sickle cell mutation and the other gene is deleted. Pregnancy outcome is similar to sickle cell anemia.

Sickle cell trait: Hb-S comprises 30–40% of the total hemoglobin, the rest being Hb–A, Hb–A$_2$ and Hb–F. If the husband is a carrier, there is 25% chance that the infant will be homozygous sickle cell disease and 50%—sickle cell trait. As such, preconceptional counseling should be done to know whether the husband also carries the trait or not.

There is no special problem so far as reproductive performance is concerned. The patient will require iron supplementation. As the concentration of Hb–S is low, crisis is rare but can occur in extreme hypoxia. Hematuria and urinary infection are quite common.

Sickle cell disease: Homozygous sickle cell disease (Hb–SS) is transmitted equally by males and females. Partner must be tested. Termination of pregnancy is an option if a fetus is diagnosed to have major hemoglobinopathy on prenatal diagnosis by CVS (see p. 129).

Pathophysiology: Red cells with HbS in oxygenated state behave normally but in the deoxygenated state it *aggregates, polymerizes and distort the* red cells to sickle. These sickle shaped cells block the microcirculation due to their rigid structure. This **sickling phenomenon is precipitated** by infection, acidosis, dehydration, hypoxia and cooling. The cells have got shorter life span and are more fragile. Increased destruction leads to hemolysis, anemia and jaundice.

Diagnosis: (a) Refractory hypochromic anemia (b) Identification by sickling test (c) Persistent reticulocytosis (10–20%) (d) High fasting serum iron level (e) Identification of the type of hemoglobinopathies by electrophoresis.

Effects on pregnancy: There is increased incidence of miscarriage (25%), prematurity, IUGR and fetal loss. Perinatal mortality is high. Incidence of **preeclampsia, postpartum hemorrhage and infection** is increased. Increased maternal morbidity is due to infection (UTIs), cerebrovascular accident and sickle cell crisis. Maternal death is increased up to 25% due to pulmonary infarction, acute chest syndrome, congestive heart failure and embolism.

Effects on the disease: There is chance of sickle cell crisis which usually occurs in the last trimester. Two types are met —(1) hemolytic crisis and (2) painful crisis.

Hemolytic crisis: It is due to hemolysis with rapidly developing anemia along with jaundice. There is associated leukocytosis and fever.

Painful (vaso-occlusive) crisis: It is due to vascular occlusion of the various organs by capillary thrombosis resulting in infarction. Organs commonly affected due to vaso-occlusion and infarction are: bones (osteonecrosis), kidney (renal medulla), hepatosplenomegaly, lung (infarction) and heart (failure), neurologic (seizures, stroke) and super added infections are high.

MANAGEMENT

Preconceptional counseling: Prenatal identification (CVS, p. 129) of homozygous state of the disorder is an indication for early termination of the pregnancy, if the parents desire (see below). Management needs multidisciplinary team approach.

During pregnancy: (1) Careful antenatal supervision (2) Air travelling in unpressurized aircraft is to be avoided (3) Prophylactically folic acid 1 mg tablet should be given daily (4) Iron supplementation is reserved only in proven cases of iron deficiency (5) Prophylactic booster or exchange blood transfusion may be given. The objective of transfusion is to keep the hematocrit value above 25%, Hb A > 20% and concentration of Hb–S under 50% (6) Infection (pneumococcal) or appearance of unusual symptoms necessitates hospitalization. Penicillin prophylaxis is given to all patients with SCD as they are at risk of infection with *N. meningitidis, S. pneumoniae* and *H. influenzae* (7) Hydroxy urea is used as a disease modifying drug. It increases HbF, improves red cell hydration and reduces polymerization of HbS and the crises. Hydroxy urea should be stopped at least 3 months before conception as it is teratogenic.

Aggressive iron chelation before conception should be advised in women who are significantly iron overloaded.

Hemopoietic cell (bone marrow/cord blood stem cell) transplantation has been used with success.

Labor and delivery: Vaginal delivery is preferred. (1) The labor is to be conducted as outlined in anemia (2) Continuous oxygen therapy by nasal cannula is done to maintain $PaO_2 > 94\%$ (3) Anoxia is to be avoided during anesthesia. **Epidural anesthesia** is preferred (4) Adequate fluid infusion to avoid dehydration and acidosis (5) Cesarean section is performed for obstetric indication only (6) Routine antibiotic is used in puerperium to prevent infection (7) Women should be given thromboprophylaxis (LMWH) during pregnancy and up to puerperium (8) Cord blood is sent for hemoglobinopathy screening.

Contraception: (1) **Sterilization** should be considered even with low parity because of the short life span of the patient (2) Oral pill is contraindicated as it might aggravate risk of thromboembolism (3) Intrauterine device is avoided for fear of infection (4) **Barrier method of contraceptive is safe and effective**. Progesterone containing contraceptives (POP, injectables) and LNG-IUS are safe and effective. Estrogen containing contraceptions should be used as second-line agents.

THALASSEMIA SYNDROMES

The thalassemia syndromes are the commonly found genetic disorders of the blood. The basic defect is a reduced rate of globin chain synthesis. As a result, the red cells being formed with an inadequate hemoglobin content. There is deficient erythropoiesis, hemolysis and ultimately anemia. **The major syndromes are of two groups—the alpha or beta thalassemia depending on whether the alpha or the beta globin chain synthesis of the adult hemoglobin is depressed.** α and β thalassemia exist in both the homozygous (major) and heterozygous (minor) states. Overall incidence during pregnancy is 1 in 300 to 500.

Alpha thalassemia is distributed amongst South East Asia and China. Alpha thalassemia major is incompatible with life. **α-peptide chain production is controlled by four genes, located on chromosome 16 (two on each copy)**. Depending upon the degree of deficient α-peptide chain synthesis, four clinical types of syndromes have been identified.

A. *Mutation of one gene*—there is no clinical or laboratory abnormalities. Subject remains as a **silent carrier.**

B. *Mutation in two of the four genes*—α-thalassemia minor. It often goes unrecognized and **pregnancy is well tolerated.**

C. *Mutation in three of the four genes*—**hemoglobin H disease.** The patient has some HbA and large percentage of HbH (four β chains) and hemoglobin Bart (four γ chains). The hemoglobin Bart present at birth, is gradually replaced by hemoglobin H. These women suffer from hemolytic anemia. During pregnancy, anemia deteriorates further.

D. *Mutation in all four genes*—**α-thalassemia major.** There is no α globin chain, hemoglobin Bart (four γ chains) and hemoglobin H (four β chains) are formed. The fetus dies either in utero or soon after birth. This is an important cause of non-immune fetal hydrops and perinatal death (p. 571).

Parental diagnosis: All forms of α-thalassemia can be diagnosed by CVS or amniocentesis.

Treatment: *Alpha thalassemia minor*—The reproductive performance in α-thalassemia minor is usually normal. They require oral iron and folate supplementation during pregnancy. If the hemoglobin is low, blood transfusion is indicated. Parenteral iron therapy should never be given.

Beta thalassemia: This entity is predominantly distributed along the Mediterranean coast, South East Asia. Normal adult hemoglobin (HbA) is composed of two α and two β peptide chains ($\alpha_2 \beta_2$). **β chain production is directed by two genes, one on each copy chromosome 11.** More than 150 point mutations in the β-globin gene have been identified. With β-thalassemia, β chain production is decreased and excess of α-chains precipitate to cause red cell membrane damage.

Beta thalassemia major (Cooley anemia)—When mutation affect both the genes. There is red cell destruction as there is no β chain production. Erythropoiesis is ineffective. Such an infant needs repeated blood transfusion to survive. There is **progressive hepatosplenomegaly, impaired growth, anemia, congestive cardiac failure and intercurrent infection.** Chance of survival beyond teens is uncommon. They are often sterile. Problem of iron overload is observed beyond the first decade of life. **Iron chelation therapy** with desferrioxamine and blood transfusion can improve the outcome.

Preconception counseling: Father of the fetus is advised for hemoglobin electrophoresis (if MCV is low). When father has normal hemoglobin—fetus has a 50% chance of β-thalassemia minor and 25% chance of nomal hemoglobin. When father is β-thalassemia minor the risk of fetus being β-thalassemia major is 50%. All forms of β-thalassemia can be detected by CVS or amniocentesis (amniocytes). **Preimplantation blastomere biopsy and DNA study** is possible to select unaffected embryos during in vitro fertilization.

Beta thalassemia minor—when there is mutation of one gene, β peptide chain production is reduced by half. Excess α-chains combine with δ chains producing HbA_2 ($\alpha_2 \delta_2$) or with γ chains producing HbF ($\alpha_2 \gamma_2$). Sickle cell trait may coexist with thalassemia minor.

Hematological findings in thalassemia: (1) There is **low MCV and MCH but normal MCHC** (c.f.—in iron deficiency anemia where all are low). (2) Serum iron and total iron binding capacity are normal or elevated. (3) Hemoglobin electrophoresis shows raised concentration of HbA_2 ($\alpha_2 \delta_2$) to more than 3.5% with normal or raised Hb-F ($\alpha_2 \gamma_2$). (4) Serum bilirubin may be raised to about 2–3 mg%. (5) Usually anemia is mild. The diagnosis is often late when the patient fails to respond to oral or parenteral iron therapy to correct anemia. There is thus chance of hepatic and cardiac hemosiderosis from iron overload.

Treatment: In thalassemia major oral and IV iron therapy is contraindicated. These women need careful monitoring for cardiac, liver, thyroid and parathyroid functions. These organs are affected due to iron overload. Frequent evaluation of fetal well-being is needed (p. 121). **Labor and delivery** management are usual. Patients with thalassemia major are often small in stature, with small pelvis. Cesarean delivery is often needed. Majority of the women tolerate pregnancy well with good maternal and fetal outcome. Oral folic acid supplementation is continued. Oral iron therapy in thalassemia minor is given only when the laboratory diagnosis of iron deficiency is established. Blood transfusion is rarely indicated.

PLATELET DISORDERS

Gestational thrombocytopenia is considered when platelet count is less than 1,50,000/mm^3 (μL). Platelet count of 50,000 to 1,50,000/mm^3 during pregnancy is not associated with any increase in maternal or fetal morbidity. Thrombocytopenia in pregnancy may be due to—(i) defective production (bone marrow pathology) (ii) Sequestration (enlarged spleen) or due to (iii) Accelerated destruction which again may be due to: (a) Non-immunological:

preeclampsia, HELLP syndrome, abruptio placenta, DIC, or (b) Immunological: thrombocytopenic purpura, lupus anticoagulant, SLE, antiphospholipid antibody. **Others**: HIV, folic acid deficiency.

Gestational thrombocytopenia: Is mainly the physiological fall resulting from **hemodilution** of normal pregnancy and increased platelet destruction.

Immune (idiopathic) thrombocytopenic purpura (ITP): Is due to accelerated destruction of antibody coated platelets in the spleen and other reticuloendothelial systems. Antibodies are of IgG, IgM and IgA types. Patients may present with skin bruising. Generally capillary bleeding or purpura occurs when the platelet count falls below 20,000/mm^3. Asymptomatic patient with count more than 50,000/μL with normal bleeding time, generally does not need any treatment.

Fetus and the neonate may be affected due to transplacental carriage of IgG antibodies. Thrombocytopenia in the fetus when severe may cause intracranial hemorrhage especially during labor.

Management: Objective is to maintain platelet count more than 50,000/μL.

During pregnancy: (a) Administer methylprednisolone (1–1.5 mg/kg) or gamma globulin (IVIG)—only if platelet count is < 20,000/mm^3. This will increase the platelet count. (b) Platelet transfusion is indicated when there is clinically significant bleeding. (c) **Splenectomy**—as this may be the site of antibody production or red cell sequestration (c) Platelet transfusion—as a temporary measure. (d) In a patient with thrombotic thrombocytopenic purpura plasma exchange should be done. IVIG prevents platelets destruction and may result in long-term remission.

During labor: Vaginal route is the preferred method as severe thrombocytopenia is rarely encountered.

Thrombophilias: Some proteins normally inhibit blood clotting process and maintain the blood fluidity. Inherited deficiency of such proteins leads to recurrent episodes of thromboembolism or thrombophilias. **The important inhibitory proteins are:** antithrombin III, protein C and protein S. **The risks associated with thrombophilias are**: deep vein thrombosis, pulmonary embolism, increased pregnancy complication like preeclampsia, placental abruption, infarction, IUGR, IUD, thromboembolism and sagittal sinus thrombosis.

Deficiency of these inhibitory proteins is due to mutation of the genes regulating their synthesis. Mutation of factor V Leiden, plasma resistance to activated protein C and hyperhomocysteinemia are also thought to cause thrombophilias. At present routine screening for thrombophilias is not recommended. **Heparin therapy is indicated throughout pregnancy**.

HEART DISEASE IN PREGNANCY

INCIDENCE AND TYPES: The incidence of cardiac lesion is less than 1% amongst hospital deliveries. **The commonest cardiac lesion is of rheumatic origin followed by the congenital ones.** The ratio between the two has fallen over the past two decades from 10: 1 to about 3: 1 or even 1: 1 in advanced countries. Adequate treatment of rheumatic fever by appropriate antibiotics to cope with the group A β-hemolytic streptococcal infection, pari passu with the advancement in cardiac surgery to rectify the congenital heart lesions, are responsible for the change in the profile.

Rheumatic valvular lesion predominantly includes mitral stenosis (80%). Predominant congenital lesions include patent ductus arteriosus, atrial or ventricular septal defect, pulmonary stenosis, coarctation of aorta and Fallot's tetralogy. Rare causes are hypertensive, thyrotoxic, syphilitic or coronary cardiac diseases.

EFFECT OF CARDIOVASCULAR PHYSIOLOGY ON HEART LESION: Marked hemodynamic changes in pregnancy (see p. 60) and cardiac output in particular, have profound effects on heart disease. A normal heart has got enough reserve power so that the extra load can well be tackled. While a damaged heart with good reserve can even withstand the strain but if the reserve is poor, cardiac failure occurs sooner or later. The **cardiac failure occurs** during pregnancy around 30 weeks, during labor and mostly soon following delivery. **Factors responsible for cardiac failure**: (1) Advanced age (2) Cardiac arrhythmias or left ventricular hypertrophy (3) History of previous heart failure (4) Appearance of "risk factors" in pregnancy are: infection, anemia, hypertension, excessive weight gain and multiple pregnancy (5) Inadequate supervision.

EFFECTS OF HEART LESION ON PREGNANCY: There is a tendency of preterm delivery and prematurity. IUGR is quite common in cyanotic heart diseases.

PROGNOSIS

- **Maternal**
- **Fetal**

MATERNAL: The prognosis depends on: (1) Nature of lesion (2) Functional capacity of the heart (3) Quality of medical supervision provided during pregnancy, labor and puerperium (4) Presence of other risk factors mentioned earlier (5) Whether patient has undergone corrective surgery or not.

Maternal mortality is lowest in rheumatic heart lesions and acyanotic group of heart diseases—less than 1%. With elevation of pulmonary vascular resistance especially with cyanotic heart lesions, the mortality may be raised to even 50% (Eisenmenger's syndrome). Most of the **deaths occur due to cardiac failure** and the maximum deaths occur following birth. The **other causes of death are—**(a) pulmonary edema (b) pulmonary embolism (c) active rheumatic carditis (d) subacute bacterial endocarditis and (e) rupture of cerebral aneurysm in coarctation of aorta.

However, with improved medical care, surgical correction of the congenital lesions and better obstetric care, the maternal mortality has been reduced markedly. Pregnancy however, does not affect the long-term survival of a woman with rheumatic heart lesion provided she survives pregnancy itself.

FETAL: In rheumatic heart lesions, the fetal outcome is usually good and in no way different from the patients without any heart lesion. However, in cyanotic group of heart lesion, there is increased fetal loss (45%) due to abortion, IUGR and prematurity. **Fetal congenital cardiac disease is increased by 3–10% if either of the parents have congenital lesions**.

DIAGNOSIS:

Anatomical and Physiological Changes During Pregnancy that Mimic Cardiac Disease

- Hyperdynamic circulation
- Systolic ejection murmur at left sternal border (due to increased blood flow across the aortic and pulmonary valves)
- Dyspnea, decreased exercise tolerance, fatigue, syncope
- Tachycardia, shift of ventricular apex
- Continuous murmur at 2nd to 4th intercostal space—mammary souffle
- Loud first sound with splitting

Symptoms must be carefully assessed and investigations are to be done to confirm the diagnosis

Diagnosis of Heart Disease in Pregnancy

- ***Symptoms:*** Breathlessness, nocturnal cough, syncope, chest pain
- ***Signs:*** Chest murmurs—pansystolic, late systolic, louder ejection systolic or diastolic associated with a thrill.
- **Cardiac enlargement, arrhythmia**
- **Chest radiography (using lead shield):** Cardiomegaly, increased pulmonary vascular markings, enlargement of pulmonary veins.
- **Electrocardiography:** T wave inversion, biatrial enlargement, dysrhythmias
- **Echocardiography (color flow Doppler study):** Structural abnormalities (ASD, VSD), valve anatomy, valve area, function, left ventricular ejection fraction, pulmonary artery systolic pressure
- Cardiac MRI can delineate complex (anatomy when it is not well-evaluated by echocardiography)

New York Heart Association (NYHA) Classification of Heart Disease (Depending Upon the Cardiac Response to Physical Activity)

Grade-I: Uncompromised and no limitation of physical activity

Grade-II: Slightly compromised with slight limitation of physical activity. The patients are comfortable at rest but ordinary physical activity causes discomfort

Grade-III: Markedly compromised with marked limitation of activity. The patients are comfortable at rest but discomfort occurs with less than ordinary activity

Grade-IV: Severely compromised with discomfort even at rest

Limitation: This classification has considered the symptoms only but not the anatomical type and severity of pathology. It does not predict pregnancy outcome

Table 20.2: Risks of Maternal Mortality with Heart Disease (NYHA, 1992)

Cardiac Disease	Mortality (%)
Group 1 (minimal risk): ASD, VSD, PDA, Fallot tetralogy (corrected), Mitral stenosis (NYHA– Grade I and II), bioprosthetic valve	0 – 1
Group 2 (moderate risk): MS (NYHA – III and IV) AS, Marfan syndrome (Normal aorta), Fallot tetralogy (uncorrected), M S with atrial fibrillation, artificial valve	5 – 15
Group 3 (major risk): Pulmonary hypertension, Marfan syndrome (aortic involvement), aortic coarctation with valvular involvement	25 – 50

GENERAL MANAGEMENT

PRINCIPLES:

— Early diagnosis and evaluation of anatomical type and functional grade of the case.

— To detect the high risk factors and to prevent cardiac failure.

— Multidisciplinary team approach (obstetrician, cardiologist and neonatologist) and mandatory hospital delivery.

PLACE OF THERAPEUTIC TERMINATION: Considering high maternal deaths, **absolute indications are** (a) primary pulmonary hypertension (b) Eisenmenger's syndrome and (c) pulmonary veno-occlusive disease.

Relative indications are—(a) Parous woman with grade III and IV cardiac lesions (b) Grade I or II with previous history of cardiac failure in early months or in between pregnancy.

The termination should be done within 12 weeks by suction evacuation (MVA) or by conventional D & E.

ANTENATAL CARE: The patients with heart disease should be supervised in a **tertiary care hospital**. The initial assessment should be made in consultation with a cardiologist. Injection penidure LA-12 (benzathine penicillin) is given at intervals of 4 weeks throughout pregnancy and puerperium to prevent **recurrence of rheumatic fever**. **Counseling** is to be done regarding prognosis and risks.

Special care in each antenatal visit is to detect and to treat the **risk factors that precipitate cardiac failure in pregnancy**. **Risk factors for cardiac failure are:**

- **Infections**—Urinary tract, dental and respiratory tract.
- **Anemia** • **Obesity** • **Hypertension** • **Arrhythmias** • **Hyperthyroidism** • **Drugs**—Betamimetics.
- **Dietary indiscretion:** Excess intake of caffeine, alcohol, high calorie diet, excess salt.

ROLE OF ANTICOAGULANTS:

Anticoagulants are indicated in cases with: (a) Congenital heart disease, (b) pulmonary hypertension, (c) mechanical heart valve, (d) atrial fibrillation. The patient taking warfarin should discontinue it (see p. 510) as soon as pregnancy is diagnosed and to replace it by heparin 5,000 units twice daily subcutaneously up to 12th week. Low molecular weight heparin (LMWH) can also be used. This is then replaced by warfarin tablet 3 mg. daily to be taken at the same time each day and continued up to 36 weeks. Thereafter it is replaced by heparin up to 7 days postpartum. Warfarin is then to be continued.

UFH, LMWH and Warfarin therapy do not contraindicate breast-feeding.

Indications for cardiac surgery in pregnancy are: Failure of medical treatment for: (i) Intolerable symptoms (ii) Intractable cardiac failure

ADMISSION: ***Elective***:

- **Grade–I** : At least 2 weeks prior to the expected date of delivery
- **Grade–II** : At 28th week especially in case of unfavorable social surroundings
- **Grade III and IV** : As soon as pregnancy is diagnosed. **The patient should be kept in the hospital throughout pregnancy**.

Emergency: (1) Deterioration of the functional grading (2) Appearance of dyspnea or cough or basal crepitations or tachyarrhythmias (3) Appearance of any pregnancy complication like anemia, preeclampsia.

MANAGEMENT DURING LABOR

PLACE OF INDUCTION: Most patients with cardiac disease go into spontaneous labor and deliver without any difficulty. However, induction (vaginal PGE_2) may be employed in very selected cases for obstetric indications. One should guard against infection and pulmonary edema due to fluid overload.

LABOR: ***First stage***:

- **Position:** The patient should be in lateral recumbent position to minimize aortocaval compression
- **Oxygen** is to be administered (5–6 L/min) if required
- **Analgesia** in the majority, is best given by epidural
- **Prophylactic antibiotics** against bacterial endocarditis
- **Fluids** should not be infused more than 75 mL/hour to prevent pulmonary edema.
- **Careful watch** of the pulse and respiration rate. If the pulse rate exceeds 110 per minute in between uterine contractions, rapid digitalization is done by intravenous digoxin 0.5 mg.
- **Cardiac monitoring and pulse oximetry** can detect arrhythmias and hypoxemia early.
- **Central venous pressure** monitoring may be needed in selected cases.

Prophylactic antibiotics for bacterial endocarditis: Antibiotic prophylaxis during labor and 48 hours after delivery is considered appropriate. This is to prevent bacterial endocarditis. The recommended regimens include intravenous ampicillin 2 g and gentamicin 1.5 mg/kg (not to exceed 80 mg), at the onset or induction of labor followed by repeat doses 8 hours interval. **High risk patients are:** (a) Structural heart disease (b) Rheumatic heart disease (c) Cyanotic congenital heart disease (d) Presence of dental and respiratory tract infections (e) Hypertrophic cardiac myopathy (f) Prosthetic heart valves (g) Prior history of infective endocarditis (h) Cardiac transplant.

Second stage: No maternal pushing and the tendency to delay in the second stage of labor is to be curtailed by forceps or ventouse under pudendal and/or perineal block anesthesia. Ventouse is preferable to forceps as it can be applied without putting the patient in lithotomy position (raising the legs increases the cardiac load). Intravenous ergometrine with the delivery of the anterior shoulder should be withheld to prevent sudden overloading of the heart by the additional blood squeezed out from the uterus.

Third stage: Conventional management is to be followed. Slight blood loss is not detrimental but if it is in excess, oxytocin can be given by infusion. This may be accompanied by aggressive diuresis by IV frusemide. It is better to administer oxytocin in preference to ergometrine in all cases of heart disease in third stage.

PLACE OF CESAREAN SECTION: In general, there is no indication of cesarean section for heart disease.

CARDIAC INDICATIONS OF CESAREAN DELIVERY:

- Coarctation of aorta
- Aortic dissection or aneurysm
- Aortopathy with aortic root > 4 cm
- Warfarin treatment within 2 weeks

In coarctation of aorta, elective cesarean section is indicated to prevent rupture of the aorta or mycotic cerebral aneurysm. The anesthesia should be given by expert anesthetist using either epidural (preferred) or general anesthesia.

PUERPERIUM:

- The patient is to be observed closely for the first 24 hours. Oxygen is administered. Hourly pulse, BP and respiration are recorded. Diuretic may be used if there is volume overload.
- Breastfeeding is not contraindicated unless there is failure. Anticoagulant therapy is not a contraindication of breastfeeding.

CONTRACEPTION: **Steroidal contraception** is avoided as it may cause thromboembolic phenomenon. **Intrauterine device (copper IUCD or LNG-IUS)** is often avoided for fear of infection though WHO permits its use. **Progestin only pills or parenteral progestins** are safe and effective. They may cause irregular bleeding especially if the patient is anticoagulated. **Barrier method** of contraceptives (condom) is the best. **Sterilization** should be considered with the completion of the family at the end of first week in the puerperium under local anesthesia through abdominal route by minilap technique. If the heart is not well compensated, the husband is advised for vasectomy.

MANAGEMENT OF CARDIAC FAILURE IN PREGNANCY: The principles of management are the same as in nonpregnant state. A cardiologist should be involved. Underlying pathophysiology and the cause of failure must be understood.

- Propped up position ♦ O_2 administration ♦ Monitoring with ECG and pulse oximetry
- Diuretic: Frusemide (Loop) (40–80 mg) IV (anticipatory aggressive diuresis is needed to avoid pulmonary congestion)
- Mechanical ventilation ♦ Injection morphine 15 mg IM
- Digoxin 0.5 mg IM followed by tab digoxin 0.25 mg P.O. (Digoxin crosses the placenta and is excreted in breast milk)
- Dysrhythmias—quinidine or electrical cardioversion
- Tachyarrhythmias—Adenosine (3–12 mg) IV or DC conversion

PREDICTORS OF ADVERSE MATERNAL OUTCOMES:

- Prior cardiac failure, arrhythmia or transient ischemic attack ♦ Baseline NYHA class > 2 or associated cyanosis
- Left heart obstruction: Mitral valve area < 2 cm^2, aortic valve area < 1.5 cm^2, or peak ventricular outflow gradient > 30 mm Hg by echocardiography
- Left ventricular ejection fraction < 40%.

SPECIFIC HEART DISEASE DURING PREGNANCY AND THE MANAGEMENT

RHEUMATIC HEART DISEASE

MITRAL STENOSIS: Mitral stenosis is the commonest heart lesion met during pregnancy. Normal mitral valve area ranges between 4 and 6 cm^2. Symptoms usually appear when stenosis narrows this to less than 2.5 cm^2. Women with mitral valve area ≤1 cm^2, have the high rate of pulmonary edema (55%) and arrhythmia (33%). In asymptomatic cases, the mortality is < 1% but once it is significantly symptomatic, mortality ranges between 5% and 15%.

Diagnosis and management has been mentioned earlier. During labor continuous **epidural analgesia** is ideal and intravenous fluid overload is to be avoided.

PLACE OF VALVOTOMY: It is better to withheld elective cardiac surgery during pregnancy. Surgery should be considered in cases of unresponsive failure with pregnancy beyond 12 weeks. Best time of surgery is between 14 weeks and 18 weeks. Valve replacement, commissurotomy, balloon valvotomy can be carried out in early second trimester. Atrial fibrillation is a complication. Digoxin, β blockers and anticoagulation (heparin) should be used.

AORTIC STENOSIS: Most cases of aortic stenosis are congenital, some are rheumatic in origin. Normal aortic valve area is 3–4 cm^2. When it is reduced to less than or equal to 1 cm^2, stenosis is significant. Maternal mortality of significant aortic stenosis is about 15–20% with perinatal loss of about 30%. Epidural anesthesia is contraindicated. During labor, fluid therapy (125–150 mL/h) should **not be** restricted. Left ventricular after load is high and the pregnant patient is sensitive to hemorrhage.

Common symptoms are angina, syncope and left ventricular failure. Medical management is not helpful in a symptomatic patient. Valve replacement is the definitive treatment. Mechanical valves need anticoagulation. Open heart surgery is preferably avoided in pregnancy. Aortic balloon valvuloplasty may be done as a palliative procedure.

CONGENITAL HEART DISEASE: With increasing number of surgical correction of the congenital heart lesions from infancy to adulthood, more and more pregnancies with congenital lesions are met in day-to-day practice. These patients pose little problem in obstetrics. But when pregnancy occurs in uncorrected congenital lesions, problems are very much there especially in a cyanotic group. Risk to the offspring of congenital heart disease is high (3–13%). **Major maternal risks in pregnancy are**: (i) Cyanosis (ii) Left ventricular dysfunction and (iii) Pulmonary hypertension. T**he common maternal complications are**: (i) Congestive cardiac failure (ii) Pulmonary edema (iii) Arrhythmia and (iv) Hypertension. All women should have fetal echocardiography examination at mid pregnancy.

A. Acyanotic (L to R shunt)

- **Atrial Septal Defect (ASD)**: ASD (ostium secundum type) is the most common congenital heart lesion during pregnancy. Even uncorrected ASD tolerates pregnancy and labor well. Congestive cardiac failure unresponsive to medical therapy requires surgical correction. Shunt reversal is the major risk which may develop in hypovolemia. Such cases may occur in hemorrhagic conditions and following injudicious administration of epidural anesthesia. In the absence of arrhythmias, and pulmonary hypertension, ASD does not usually complicate pregnancy.

- **Patent Ductus Arteriosus (PDA)**: Presence of continuous murmur at the upper left sternal border is suggestive of diagnosis. Most patients with PDA tolerate pregnancy well. *Pulmonary hypertension* may cause maternal death. Surgical correction during pregnancy can be performed provided there is no pulmonary hypertension. *Epidural analgesia is better avoided to minimize shunt reversal due to systemic hypotension*. Fetal loss may be up to 7% and there is 4% chance that the child of this parent will suffer from the same abnormality. Endocarditis prophylaxis should be given.

- **Ventricular Septal Defect (VSD):** In general, if the defect is less than 1.25 cm^2, pulmonary hypertension and heart failure do not develop. Pregnancy is well tolerated with small to moderate left to right shunt or with moderate pulmonary hypertension. The major risk is shunt reversal leading to circulatory collapse and cyanosis. Hypotension is to be avoided. Fetal loss may be up to 20%.

- **Mitral Valve Prolapse (MVP):** Is the commonest congenital valvular lesion. Most of them are asymptomatic. Women tolerate pregnancy and labor well. Endocarditis prophylaxis is given.

B. Cyanotic (R to L shunt)

- **Fallot's tetralogy:** It is the most common form of cyanotic heart lesion. It is a combination of (a) ventricular septal defect, (b) pulmonary valve stenosis, (c) right ventricular hypertrophy and (d) an overriding aorta. After surgical correction, patients tolerate pregnancy well. Surgically uncorrected patients are at increased risk. Complications like bacterial endocarditis, brain abscess and cerebral embolism are more common. Maternal mortality is 5–10% and the perinatal mortality is 30–40%. IUGR is common. Systemic hypotension is dangerous which may lead even to death. Epidural or spinal anesthesia is avoided. Pregnancy is discouraged in women with uncorrected tetralogy.

- **Eisenmenger's syndrome:** Patients with Eisenmenger's syndrome have pulmonary hypertension with shunt (right to left) through an open ductus, an atrial or ventricular septal defect. Maternal mortality is about 50% and so also the perinatal loss (50%). Termination of pregnancy should be seriously considered. Heparin should be used throughout pregnancy as there is risk of systemic and pulmonary thromboembolism. Epidural anesthesia is contraindicated. Inhaled nitric oxide or I.V. prostacyclin is used as a pulmonary vasodilator. To maintain hemodynamic stability, pulmonary artery catheter and a peripheral artery catheter are used. Complications are: CCF, hemoptysis, arrhythmia, cerebrovascular accident and hypoxemia; hyperviscosity syndrome and sudden death.

C. Other congenital heart lesions

- **Coarctation of aorta:** The maternal risks are hypertension, aortic dissection, bacterial endocarditis and cerebral hemorrhage due to ruptured intracranial aneurysms. Maternal mortality is high 3–9%. Fetal loss is also increased to 25%. Surgical correction should be done prior to pregnancy. Termination of pregnancy should be seriously considered. Elective cesarean section is preferred to minimize dissection associated with labor.

- **Primary pulmonary hypertension** is characterized by increased thickening of muscular layer of pulmonary arterioles. The cause remains unknown. Maternal mortality is about 50%, majority die (75%) postpartum. The fetal outlook is also gloomy. Termination of pregnancy is indicated. Bed rest should be imposed from 20 weeks of pregnancy. Anticoagulant (heparin) is administered. Sildenafil is used as a potent vasodilator as it increases endogenous nitric oxide. Oral nifedipine or I.V. prostacyclin helps pulmonary vasodilatation. Epidural morphine gives effective analgesia without any hemodynamic change. Women with pulmonary hypertension and right ventricular dysfunction are strongly discouraged to become pregnant.
- **Marfan's syndrome:** Marfan's syndrome is an autosomal dominant condition. There is 50% chance of transmission to the offspring. Dilatation of aorta more than 40 mm as evidenced from echocardiography is a contraindication of pregnancy. Beta blocking drugs should be used to maintain resting heart rate around 70 bpm. Hypertension should be avoided to prevent aortic dissection. Vaginal delivery is desirable with shortening of second stage. When the aortic root diameter measures more than 4 cm, mortality increases to 25%. Women with aortic diameter more than 5.5 cm should have graft and valve replacement before pregnancy.
- **Prosthetic valves** are used for significant valvular disease. Mechanical valves are durable but require anticoagulation. The risk of thromboembolism is high with low molecular weight heparin rather than warfarin. Bioprosthetic valves (Porcine) are superior to mechanical valves. The ACOG guidelines for anticoagulation has been mentioned in p. 321.

D. Cardiomyopathies

- **Peripartum cardiomyopathy**: Important diagnostic criteria are: (i) Cardiac failure within last month of pregnancy or within 5 months postpartum. (ii) No determinable cause for failure. (iii) Absence of previous heart disease. (iv) Left ventricular dysfunction as evidenced on echocardiography—(a) Ejection fraction less than 45% and (b) Left ventricular end diastolic dimension more than 2.7 cm/m^2. Peripartum cardiomyopathy is a diagnosis of exclusion. The patients are usually multiparous and young (20–35 years). They complain of weakness, shortness of breath, cough, nocturnal dyspnea and palpitation. Examination reveals—tachycardia, arrhythmia, peripheral edema and pulmonary rales. Pregnancy is poorly tolerated in women with dilated cardiomyopathy.

The treatment is bed rest, digoxin, diuretics (preload reduction), hydralazine or ACE inhibitors (postpartum) (afterload reduction), β blocker and anticoagulant therapy. Vaginal delivery is preferred. Epidural anesthesia is ideal. There is no contraindication of breastfeeding. Mortality is high (20–50%)—due to CCF, arrhythmia or thromboembolism. It may recur in subsequent pregnancies.

Myocardial infarction is rare in pregnancy. Management is mostly as in nonpregnant state. Coronary angioplasty, stenting and thrombolytic therapy have been done in pregnancy when indicated. Supine position and hypotension should be avoided. **Labor**: managed as with standard cardiac care. Elective delivery within two weeks of infarction should be avoided. Regional analgesia for pain in labor and β blockers for tachycardia may be used. Maternal pushing is avoided and second stage is shortened by forceps or vacuum. **Syntocinon should be used in the third stage management as ergometrine may cause coronary artery spasm.** Diuretics to be used postpartum. Percutaneous transluminal coronary angioplasty can be done successfully around 36 weeks of pregnancy if needed.

DIABETES MELLITUS AND PREGNANCY

Diabetes mellitus is a chronic metabolic disorder due to either **insulin deficiency (relative or absolute)** or due to **peripheral tissue resistance** (decreased sensitivity) to the action of insulin (see p. 72). The pathophysiology involved are: (i) decreased sensitivity (see p. 72) of skeletal muscles and liver to insulin (insulin resistance) and (ii) inadequate secretion of insulin (β cell dysfunction). Pregnancy is a state of chronic low grade inflammation. This is associated with increased circulating levels of C-reactive protein (CRP) and interleukin-6 (IL-6). Both these factors enhance insulin resistance. The defect lies both in insulin secretion and action. The ultimate effect is the **hyperglycemia**. Two types are generally described.

A. Type–1 (IDDM) is characterized by young age onset (Juvenile) and **absolute insulinopenia**. They have genetic predisposition with presence of **autoantibodies**.

B. Type–2 (NIDDM) is characterized by late age onset, overweight woman and peripheral tissue (skeletal muscle, liver) **insulin resistance (hyperinsulinemia)**. Genetic predisposition is also observed. Others are:

C. Gestational Diabetes Mellitus (GDM)

D. Others: Genetic, Drugs, MODY

MODY (Maturity onset diabetes of the youth) is due to β cell dysfunction. It has an autosomal dominant mode of inheritance. It usually manifests in adulthood.

Alternation in lipid metabolism in diabetes: Decrease in HDL cholesterol is observed especially with type-1 diabetes. HDL acts as a plasma antioxidant. Fall in HDL may be a cause for congenital malformations as oxidative stress is a potential factor. Increased free fatty acids have been associated with fetal over-growth. Insulin inhibits lipolysis and decrease FFA levels.

About 1–14% of all pregnancies are complicated by diabetes mellitus and 90% of them are gestational diabetes mellitus (GDM). Nearly 50% of women with GDM will become overt diabetes (type-2) over a period of 5 to 20 years.

GLYCOSURIA IN PREGNANCY: Repeat and random urine samples taken on one or more occasions throughout pregnancy reveal glycosuria in about 5–50% cases. During pregnancy, **renal threshold is diminished** due to the combined effect of increased glomerular filtration and impaired tubular reabsorption of glucose. It is present most commonly in mid pregnancy. If glucose tolerance test is done, glucose leaks out in the urine even though the blood sugar level is well below 180 mg/100 mL (normal renal threshold). No treatment is required and the condition disappears after delivery.

SIGNIFICANCE: Glycosuria is specifically detected by testing a second fasting morning specimen of urine, collected a little later, after discarding the overnight urine. Fasting glycosuria if present, is ominous. Glycosuria on one occasion before 20th week and on two or more occasions, thereafter, is an indication for **glucose tolerance test**. Glycosuria occurring any time during pregnancy with a positive family history of diabetes or past history of having a baby weighing 4 kg or more should be similarly investigated.

INDICATIONS OF GLUCOSE TOLERANCE TEST

- Fasting glycosuria on one occasion before 20th week and on two or more occasions thereafter
- Following a positive "screening test"— (vide—Gestational diabetes)
- If fasting blood sugar exceeds 95 mg/100 mL or if that after 2 hours of ingestion of 100 gm (WHO-75 gm) glucose is over 120 mg/100 mL
- However, if the fasting plasma glucose value is more than or equal to 126 mg/dL and if confirmed on repeat test, there is no need to perform GTT as the woman is diabetic

GESTATIONAL DIABETES MELLITUS (GDM)

NOMENCLATURE: GDM is defined as carbohydrate intolerance of variable severity with onset or first recognition during the present pregnancy. The entity usually presents late in the second or during the third trimester. The definition is used irrespective of the fact that the condition persists after pregnancy or not. Majority of these women (>50%) with GDM ultimately develop overt diabetes by next 15 to 20 years. It appears that many of these women diagnosed as GDM, are already suffering from impaired β-cell function but remained undetected. These cases are basically pre-existing type 2 diabetes.

The potential candidates for GDM are: (a) Positive family history of diabetes (parents or sibling). Family history should include uncles, aunts and grandparents. (b) Having a previous birth of an overweight baby of 4 kg or more (c) Previous stillbirth with pancreatic islet hyperplasia revealed on autopsy (d) Unexplained perinatal loss (e) Presence of polyhydramnios or recurrent vaginal candidiasis in present pregnancy (f) Persistent glycosuria (g) Age over 30 years (h) Obesity (see p. 400) (i) Ethnic group (East Asian, Pacific island ancestry).

SCREENING: While some advocate screening routinely to all pregnant mothers, others reserve it only for the potential candidates. Screening strategy for detection of GDM are: (a) **Low risk**—Absence of any risk factors as mentioned above → blood glucose testing is not routinely required (b) **Average risk**—Some risk factors → perform screening test (see below) (c) **High risk**—Blood glucose test as soon as feasible. **The method employed is by using 50 gm oral glucose challenge test without regard to time of day or last meal, between 24 weeks and 28 weeks of pregnancy**. A plasma glucose value of 140 mg% or that of whole blood of 130 mg% at 1 hour is considered as cut off point **for consideration of a 100 gm (WHO– 75 gm) glucose tolerance test**.

DIPSI (Diabetes In Pregnancy Societies of India) recommends 1-step procedure with 75 gm oral glucose without regard to the time of the last meal. A venous plasma glucose value at 2-hour more than 140 mg/dL is diagnosed GDM.

HAZARDS: (1) Increased perinatal loss is associated with fasting hyperglycemia. Fetal anomalies are however not increased. This is due to the absence of metabolic disturbance during organogenesis (2) Increased incidence of macrosomia (3) Polyhydramnios (4) Birth trauma (5) Recurrence of GDM in subsequent pregnancies is about 50%.

Table 20.3: Criteria for Diagnosis of GDM with 100 gm Oral Glucose (O'Sullivan and Mahan Modified by Carpenter and Coustan) and National Diabetes Data Group

GTT: Venous Plasma (mg/dL)

Time	Carpenter & Coustan	NDDG
Fasting	95	105
1 hour	180	190
2 hours	155	165
3 hours	140	145

GDM is diagnosed when any two values are met or elevated

MANAGEMENT: Diet with 2,000–2,500 Kcal/day for normal weight woman and restriction to 1,200–1,800 Kcal/day for over weight woman is recommended. Carbohydrate should be 40–50% of total calories. **Complex carbohydrates are preferred** because simple carbohydrates produce significant post-prandial hyperglycemia. Women should perform self-blood glucose monitoring using reflectance meter. Women with well controlled GDM have reduced risk of complications like: IUFD, macrosomia, shoulder dystocia, preeclampsia, and cesarean delivery. The patient needs more frequent antenatal supervision with periodic checkup of **fasting plasma glucose level which should be less than 90 mg%**. Maintenance of mean plasma glucose level between 105 mg/dL and 110 mg/dL is desirable for good fetal outcome (DIPSI – 2009). The control of high blood glucose is done by restriction of **diet**, **exercise with or without insulin**. Human insulin should be started if fasting plasma glucose level exceeds 90 mg/dL and 2 hours postprandial value is greater than 120 mg/dL (repetitive) even on diet control. Nearly 25% women with GDM need insulin therapy. **Exercise** (aerobic, brisk walking) programs are safe in pregnancy and may obviate the need of insulin therapy.

Obstetric management: Women with good glycemic control and who do not require insulin may wait for spontaneous onset of labor. However, elective delivery (induction or cesarean section) is considered in patients requiring insulin or with complications (macrosomia) at around 38 weeks.

Follow-up: Nearly 50% of women with GDM would develop overt diabetes over a follow-up period of 5–20 years. Women with fasting hyperglycemia have got worse prognosis to develop type-2 diabetes and cardiovascular complications. Recurrence risk in subsequent pregnancy is more than 50%.

Risks of being overweight for the infants of mother with GDM is twofold and the risk for metabolic syndrome is about fourfold.

OVERT DIABETES

A patient with symptoms of diabetes mellitus (polyuria, polydipsia, weight loss) and random plasma glucose concentration of 200 mg/dL or more is considered overt diabetic. The condition may be pre-existing or detected for the first time during present pregnancy. According to American Diabetic Association diagnosis is positive if: (a) **the fasting plasma glucose exceeds 126 mg/dL**, (b) **the 2 hours post glucose (75 gm) value exceeds 200 mg/dL** and (c) **HbA1C ≥ 6.5%** (see Table 20.4).

Classification of pregnant diabetic women:

Fetal and maternal outcome of diabetic pregnancy depends on severity of the disease and its duration. **Priscilla White's classification** (Table 20.5) was originally used to assess the perinatal outcome and to formulate the obstetric management. But it is now mainly used for statistical correlation of different

Table 20.4: Criteria for Diagnosis of Impaired Glucose Tolerance and Diabetes with 75 gm Oral Glucose (American Diabetic Assocation)

Time	Normal Tolerance	Impaired Glucose Tolerance	Diabetes
Fasting	<100	≥100 and <126	≥126
2 hour post glucose	<140	≥140 and <200	≥200

- Venous whole blood values are 15% less than the plasma
- m mol/l = mg% × 0.0555

International Association of Diabetes and Pregnancy Study Groups (IADPSG) (75 gm Oral Glucose) (mg/ dL)

Time	Value
Fasting	92
1-hour	180
2-hour	153

Based on IADPSG, diagnosis of GDM is made if one or more thresholds are met or exceeded.

Table 20.5: White's Classification of Pregnant Diabetic Women

Class	Onset	Fasting Plasma Glucose	2-hour Postprandial	Treatment
A Gestational Diabetes	**Any Age**	**A1: Glucose < 105 mg/dl** **A2: > 105 mg/dl**	**< 120 mg/dl** **> 120 mg/dl**	**Diet** **Insulin**
Class	**Age of Onset**	**Duration (years)**	**Vascular Disease**	**Treatment**
B	>20 years	<10 years	None	Insulin
C	10–19 years	10–19 years	None	Insulin
D	<10 years	>20 years	Benign retinopathy	Insulin
F	Any	Any	Nephropathy	Insulin
H	Any	Any	Coronary artery disease	Insulin
R	Any	Any	Proliferative retinopathy	Insulin
T	Any	Any	Renal transplant	Insulin

types of pregnant diabetics. **Vasculopathy** is given more importance to predict the outcome. Patients with poor glycemic control and vasculopathy are at increased risk of complication like IUD, IUGR, Preeclampsia and Ketoacidosis.

EFFECTS OF PREGNANCY ON DIABETES

It is difficult to stabilize the blood glucose during pregnancy due to altered carbohydrate metabolism and an impaired insulin action. **The insulin antagonism is due to the** combined effect of human placental lactogen, estrogen, progesterone, free cortisol and degradation of the insulin by the placenta. The insulin requirement during pregnancy increases as pregnancy advances. **As more glucose leaks out in the urine due to renal glycosuria, control of insulin dose cannot be made by urine test and repeated blood glucose estimation becomes mandatory**. With the "accelerated starvation" concept, there is rapid activation of lipolysis with short period of fasting. Ketoacidosis can be precipitated during hyperemesis in early pregnancy, infection and fasting of labor. It can be iatrogenically induced by β sympathomimetics and corticosteroids used in the management of preterm labor. Insulin requirement falls significantly in puerperium. **Vascular changes**, especially retinopathy, nephropathy, coronary artery disease and neuropathy may be worsened during pregnancy (Table 20.5).

EFFECTS OF DIABETES ON PREGNANCY

Complications of diabetes (Hyperglycemia and adverse pregnancy outcome):

- **Maternal**
- **Fetal and Neonatal**

MATERNAL

During pregnancy:

- **Abortion**: Recurrent spontaneous abortion may be associated with uncontrolled diabetes.
- **Preterm labor (26%)** may be due to infection or polyhydramnios.
- **Infection:** Urinary tract infection and vulvovaginitis.
- **Increased incidence of preeclampsia (25%)**.
- **Polyhydramnios (25–50%)** is a common association. Large baby, large placenta, fetal hyperglycemia leading to polyuria, increased glucose concentration of liquor irritating the amniotic epithelium or increased osmosis, are some of the probabilities.
- **Maternal distress** may be due to the combined effects of an oversized fetus and polyhydramnios.
- **Diabetic retinopathy (Class R)** is characterized by the proliferative retinopathy having neovascularization and microaneurysms. These vessels may rupture and may cause vitreous hemorrhage, scarring, retinal detachment and loss of vision. Severity of retinal pathology depends on (a) age (time) of onset, (b) duration of the disease, (c) degree of rise in blood Hb AIC and (d) association of hypertension (Table 20.3). Laser photocoagulation is the preferred treatment.
- **Diabetic nephropathy (Class F)** is diagnosed when creatinine clearance is reduced or there is persistent proteinuria (≥300 mg/24 hours) during the first 20 weeks of gestation. Predictive factors for perinatal outcome (e.g., low birth weight, preterm delivery or preeclampsia) are: (a) Proteinuria > 3 g/24 hours, (b) serum creatinine > 1.5 mg/dL (see Table 20.3).

  Most women (90%) develop preeclampsia. Control of hypertension is important to prevent further deterioration of kidney function. Calcium channel blocker is commonly used.

  These women have significantly reduced life expectancy. The disease progression is characterized by hypertension, falling glomerular filtration rate and creatinine clearance. The **end stage disease** needs dialysis or renal transplantation. Renal transplantation improves survival of women with diabetic nephropathy.
- **Coronary artery disease (Class H):** These women run the high risk for ischemic heart disease especially when the disease is long standing.
- **Ketoacidosis**—see p. 332.

***During labor:* There is increased incidence of**: (1) Prolongation of labor due to **big baby**. (2) **Shoulder dystocia** (p. 406). Shoulder dystocia is due to disproportionate growth with increased shoulder/head ratio. (3) Perineal injuries. (4) Postpartum hemorrhage. (5) Operative interference.

Puerperium: (1) Puerperal sepsis. (2) Lactation failure.

FETAL AND NEONATAL HAZARDS:

- **Fetal macrosomia (40–50%)** with birth weight > 4 kg (Fig. 20.3) (>90th percentile) probably results from: (a) **Maternal hyperglycemia** → hypertrophy and hyperplasia of the fetal islets

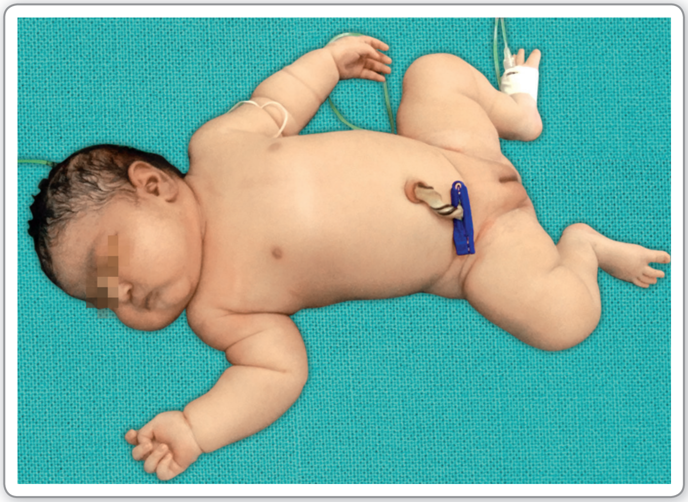


Fig. 20.3: Macrosomic baby, weighing 5.3 kg, of a diabetic mother, looked plethoric (due to polycythemia), with plumpy face, buried eyes and excessive buccal fat

of Langerhans → increased secretion of fetal insulin → stimulates carbohydrate utilization and accumulation of fat. Insulin like growth factors (IGF-I and II) are also involved in fetal growth and adiposity. With good diabetic control, incidence of macrosomia is markedly reduced. (b) **Elevation of maternal free fatty acid (FFA)** in diabetes leads to its increased transfer to the fetus → acceleration of triglyceride synthesis → adiposity.

- **Congenital malformation (6–10%)** is related to the severity of diabetes affecting organogenesis, in the first trimester (both in type 1 and type 2 diabetes). **The factors associated with teratogenesis are multifactorial**: (a) Genetic susceptibility (b) Hyperglycemia (c) Arachidonic acid deficiency (d) Ketone body excess (e) Somatomedin inhibition (f) Free oxygen radical excess (superoxide dismutase, an oxygen radical scavenging enzyme can protect excess malformation). Risks of fetal chromosomal abnormalities are not increased.

Early detection of fetal anomalies :

- **Estimation of glycosylated hemoglobin A (HbA1c)** before 14 weeks reflect the quality of diabetic control over the previous 3 months. Overall risk of fetal malformations are increased when the level of Hb A1c is high (normal < 6%). **Chance of major congenital malformation is about 8% and 23% when the values are 9.5 and 10 respectively.**
- **Maternal serum α-fetoprotein level** at 16 weeks and a detailed high resolution ultrasonography of the fetus including fetal echocardiography at 20–22 weeks are advocated (see p. 129).
- **A comprehensive ultrasound examination—**including fetal echocardiography is done at 20–22 weeks to detect any cardiac anomaly along with other structural malformation.
    - **Birth injuries** (brachial plexus) are associated with prolonged labor and shoulder dystocia due to macrosomic baby (see p. 558).
    - **Growth restriction** is less commonly observed and is associated with maternal vasculopathy. Placental amino acid transporters are involved in fetal macrosomia or IUGR in women with diabetes.
    - **Fetal death** has got multifactorial pathogenesis but the final event being **hypoxia and lactic acidemia**. It is observed more in patients with poor glycemic control, vasculopathy, preeclampsia, ketoacidosis and fetal macrosomia. Fetal hyperglycemia and hyperinsulinemia increase fetal oxygen demand. Glycosylated hemoglobin carries less oxygen molecule. It binds O_2 more avidly and releases O_2 less. Other factors involved are fetal polycythemia, and hyperviscosity.
    - **Neonatal complications include**—(a) **Hypoglycemia** (< 35 mg/dL) is due to hyperinsulinemia. It is common in macrosomic infants. (b) **Respiratory distress syndrome** is due to excess level of fetal insulin that blocks the action of cortisol. Cortisol activates type II pneumocytes for the synthesis of phospholipids (surfactant). Risk of RDS is reduced when diabetes is well controlled and delivery is done after 38 weeks of gestation (see p. 547). (c) **Hyperbilirubinemia** is high (25% to 50%) due to increased red cell production (polycythemia) and break down of red cells (p. 477). (d) **Polycythemia** (e) **Hypocalcemia** (≤7 mg/dL) (f) **Hypomagnesemia** (<7 mg/dL) (g) **Cardiomyopathy** is more common when diabetes is poorly controlled. Septal hypertrophy and cardiac hypertrophy are also observed.
    - **Long-term effects**—childhood obesity, neuropsychological effects and diabetes.

Table 20.6: Major Birth Defects in Infants of Diabetic Mothers (6–10%)

CNS and Skeletal	Cardiovascular	Renal	Gastrointestinal	Others
Neural tube defects	VSD, ASD	Renal agenesis	Duodenal atresia	Single umbilical artery
Anencephaly	Coarctation of aorta	Hydronephrosis	Anorectal atresia	
Microcephaly	Transposition of great vessels	Double ureter	Omphalocele	
Caudal regression syndrome	Situs inversus	Polycystic kidneys	Tracheoesophageal fistula	
Sacral agenesis	Fallot's tetralogy			

Good glycemic control with preconceptional counseling can reduce the incidence to 0.8–2%

PERINATAL MORTALITY: The overall perinatal mortality is **increased 2–3 times**. **The neonatal deaths are principally due to** hypoglycemia, respiratory distress syndrome, polycythemia and jaundice.

MANAGEMENT

Preconceptional counseling: Goal is to achieve tight control of diabetes before the onset of pregnancy. Ideally a diabetic woman should be seen jointly by the diabetologist, obstetrician and dietician. Fetal congenital malformations are significantly low (0.8–2%) in women who receive preconceptional counseling. Patients' glycemic control and vascular status are assessed. Folic acid supplementation (0.4 mg/day) should be started. HbA1C level should be measured to plan pregnancy. Women are taught for self-glucose monitoring. Appropriate advice about diet and insulin is given. Chance of having a diabetic child is about 3–4% when either parent is diabetic (type 1). Risk is high, about 40%, when both the parents are (type 2) diabetic.

Principles in the management are: (1) Careful antenatal supervision and glycemic control, so as to maintain the glucose level as near to physiological level as possible (2) To find out the optimum time and method of delivery (3) Arrangement for the care of the newborn.

ANTENATAL CARE: Antenatal supervision should be at monthly intervals up to 20 weeks and thereafter at 2 weeks intervals. At times patient needs admission for stabilization of blood glucose and for monitoring the fetus. **Diet**—30 Kcal/kg for normal weight women, 24 Kcal/kg for overweight women and 12 Kcal/kg for morbidly obese women. Ideal plasma glucose (mg/dL) levels in women with pregestational diabetes should be: fasting $\leq$ 95, premeal $\leq$ 100; 2 hour P.P. values $\leq$ 120 and morning (2 am–6 am) value $\geq$ 60. If values are exceeded even on diet, insulin therapy is suggested. Diet should contain carbohydrate 40%–50%, protein 20%, fat 30–40% and saturated fat <10%. Fat may be curtailed, if the patient is obese. Fiber containing diet (**complex carbohydrates**) is increased. Usually **three meal** regimen, with breakfast 25% of the total calorie intake, lunch 30%, dinner 30% and **several snacks** are quite suitable for most of the patients. **Frequent blood sugar estimation is required** as the urine examination for sugar is not informative. Monitoring of blood glucose by glucose meter can give an accurate idea about the control. In addition, glycosylated hemoglobin should be determined at the end of first trimester and trimonthly thereafter. **HbA1c level of ≤6% is desirable**. **Sonographic evaluation** (Level II) in pregnancy (at 3-4 weeks interval) is extremely helpful, not only to diagnose varieties of congenital malformation of fetus but also to detect fetal macrosomia or growth restriction (rare). **Assessment of fetal well-being is to be made from 28 weeks onwards (ch. 11)**. **Biophysical profile** and NST should be performed weekly **(see p. 109)**. Doppler **umbilical artery velocimetry** is useful in cases with vasculopathy.

***Insulin therapy*:** When diabetes is first detected during pregnancy and cannot be controlled by diet alone, it should be treated with insulin. **A postprandial (2 hours) plasma glucose level of more than 140 mg% even on diet control is an indication of insulin therapy**. There is frequent change in insulin need during pregnancy and changes in the dosage are made in small increments at a time. Glycemic goals should be around 90 mg/dL before meals and not to exceed 120 mg/dL, 2 hours after meals. During the stabilization process of the insulin dose, frequent blood sugar estimation especially at night (2 am–6 am) may be necessary using glucose meter (capillary whole blood, utilized for self-monitoring, is equivalent to venous plasma). However, as pregnancy advances, "a double mixed regime" may be employed. The patient should receive three to four daily injections of a regular (human act rapid) and an intermediate acting insulin (isophane), the latter is to be given before dinner. The aim is to maintain the blood sugar level as near to normal as possible **without causing troublesome hypoglycemia**. Use of subcutaneous insulin infusion by insulin pump is preferred as it is more physiological. Women are instructed on diet composition, insulin dose, recognition and treatment of hypoglycemia, hyperglycemia and ketosis, adjusting insulin dose in relation to exercise, food and sick days. Oral hypoglycemic drugs have been found to be effective and safe. Commonly used drugs are glibenclamide and metformin (biguanide). Both the drugs cross the placenta. However, no teratogenic effect has been observed as yet. Glibenclamide appears to be superior when compared to metformin.

ADMISSION: In uncomplicated cases, the patient is admitted at 34–36 weeks. **Early hospitalization facilities**: (1) Stabilization of diabetes (2) Minimizes the incidence of preeclampsia, polyhydramnios and preterm labor (3) To select out the appropriate time and method of delivery.

Induction of labor: **The indications are** — (i) Diabetic women controlled on insulin (GDM or class B diabetes) are considered for induction of labor after 38 completed weeks (ii) Women with vascular complications (preeclampsia, IUGR) often require induction after 37 weeks.

Methods: Prior to the day of induction of labor, the usual bed time dose of insulin is administered. No breakfast and no morning dose of insulin are given on the day of induction. Capillary blood glucose level is checked with a bed side glucose meter. Normal saline infusion is begun. Induction is done by low rupture of the membranes. Simultaneous oxytocin drip is started, if not contraindicated. An intravenous drip of one liter of 5% dextrose is set up with 10 units of soluble insulin. An infusion rate of 100–125 mL/hr (1–1.25 units/hr), will maintain a good glucose control to approximately 100 mg/dL (ACOG-2005). Insulin may also be infused from a syringe pump (0.25–2 units/hr). Blood glucose levels are estimated hourly with a glucose meter and the soluble insulin dose is adjusted accordingly. **Epidural analgesia is ideal** for pain relief. If the labor fails to start within 6–8 hours or if the labor progresses unsatisfactorily, cesarean section should be performed.

Cesarean section: **The indications are**—(1) Fetal macrosomia (>4 kg) (2) Diabetes with complications or difficult to control (3) Fetal compromise as observed in antepartum fetal monitoring (4) Elderly primigravidae (5) Multigravidae with a bad obstetric history (6) Obstetric complications like preeclampsia, polyhydramnios, malpresentation. **As such 50% of diabetic mothers are delivered by cesarean section**.

Procedure: Cesarean section is scheduled for early morning. On the day of operation, breakfast and the insulin dose are omitted. Capillary blood glucose level is checked with a glucose meter. A normal saline infusion is started. The administration of dextrose drip and the insulin dose are to be maintained as mentioned in induction until the patient is able to take fluids by mouth (ACOG-2005). Continuous subcutaneous insulin infusion with insulin pump is preferred as it is more physiological. The insulin requirement suddenly falls following delivery and after the omission of the drip, pre-pregnant dose of insulin is to be administered or adjusted from the blood glucose level. **Epidural or spinal anesthesia is better** than general anesthesia as oral feeding could be started soon following the operation.

To control blood glucose: (1) One liter of **5% dextrose** drip is started with 10 units of soluble insulin (2) A general guideline for insulin infusion rate is, 1 unit per hour for blood glucose of 100–140 mg/dL, 2 units per hour for blood glucose of 141–180 mg/dL and 3 units per hour for blood glucose of 181–220 mg/dL is followed. Use of motorized syringe pump for insulin infusion is convenient (3) Hourly estimation of blood glucose levels is done with glucose meter and the insulin dose is adjusted accordingly. The blood glucose level should be maintained between 80 and 100 mg per 100 mL.

PLACE OF AWAITING SPONTANEOUS ONSET OF LABOR AT TERM: The following are the conditions where the pregnancy may be continued awaiting spontaneous onset of labor and vaginal delivery. (1) Young primigravidae or multiparae with good obstetric history (2) Diabetes well controlled either by diet or insulin and without any obstetrical complication. However, in the absence of gadgets for assessment of fetal well-being, it is risky to continue the pregnancy in such cases up to the EDD. **In any case, the pregnancy should not be allowed to overrun the expected date**.

Fetal monitoring: Constant watch to note the fetal condition is mandatory, preferably with continuous electronic fetal monitoring. CTG using a scalp electrode is maintained (Fig. 38.1). Fetal scalp blood pH sampling is done whenever indicated (see p. 696). The combination of fetal hyperglycemia and anoxia contribute not only to fetal distress but responsible for RDS. However, labor should not be allowed for more than an arbitrary 12 hours and should be augmented by low rupture of the membranes and oxytocin or delivered by cesarean section. **Shoulder dystocia** may be a problem (see p. 469). **The cord should be clamped immediately** after delivery to avoid hypervolemia.

Examination of the placenta and cord: Placenta is large, the cord is thick and there is **increased incidence of a single umbilical artery**. Microscopically, villi show edema and excessive syncytial knots, numerous cytotrophoblasts and thickened basement membrane. The term placentosis is given to such features.

Diabetic ketoacidosis: Pathology is insulin resistance → lipolysis → enhanced ketogenesis → fall in plasma HCO_3^- and pH (< 7.30). It may be precipitated with the use of β-mimetic agents (Isoxsuprine) and corticosteroids.

Management is done in an acute care unit where both neonatal care is also available. **Parameters to assess are**: Degree of acidosis, alterations in the level of arterial blood gas, blood glucose, ketones and electrolytes.

- **IV insulin** → 0.1–0.2 units/kg (loading dose) → 0.1 U/kg/hr. (to adjust with frequent capillary glucose estimation) → to keep plasma glucose levels between 100 and 150 mg/dL
- **Fluids**—NaCl total: 4-6L in first 12 hours. 5% dextrose with 0.45% NaCl at 150 mL/hr.
- **IV Potassium:** if reduced—infusion 15-20 mEq/hr until serum $K^+ > 3.3$ but <5.3 mEq/L
- **Bicarbonate:** if PH < 7.0: $NaHCO_3$ (50 mmol) in 200 mL water over 1 hr → repeat serum $NaHCO_3$ levels.

PUERPERIUM: Antibiotics should be given prophylactically to minimize infection. **Insulin requirement falls dramatically following delivery**. She is to revert to the insulin regime as was prior to pregnancy. A fresh blood glucose level after 24 hours will help to adjust the dose of insulin. Breastfeeding is encouraged. Women who breast feed should have additional 500 Kcal daily in diet. In lactating women insulin dose is lower.

CARE OF THE BABY: A neonatologist should be present at the time of delivery. The baby should preferably be kept in an intensive neonatal care unit and to remain vigilant for at least 48 hours, to detect and to treat effectively any complication likely to arise.

- Asphyxia is anticipated and be treated effectively (see p. 544).
- To look for any congenital malformation.
- All babies should have blood glucose to be checked within 2 hours of birth to avoid problems of hypoglycemia (blood glucose < 35 mg/dL).
- All babies should receive 1 mg vitamin K intramuscularly.
- Early breastfeeding within half to 1 hour is advocated and to be repeated at three to four hourly intervals thereafter to minimize hypoglycemia and hyperbilirubinemia.

Improvement in the care of diabetes in pregnancy has reduced perinatal mortality significantly (< 5%).

CONTRACEPTION: Barrier method of contraceptives is ideal for spacing of births. **Low dose combined oral pills** containing third generation progestins, are effective and have got minimal effect on carbohydrate metabolism. Main worry is their effect on vascular disease (thromboembolism and myocardial infarction). **Progestin only pill** may be an alternative (see p. 627). Long acting progestins are not used as a first line method. The **IUCD** may be used once diabetes is well controlled. **Permanent sterilization** is considered when family is completed.

KEY POINTS

- **Gestational Diabetes Mellitus (GDM)** is defined as carbohydrate intolerance of variable severity with onset or fist recognition during the present pregnancy.
- **Potential risk factors for GDM are:** (see p. 326)
- **Complications of GDM are:** (see p. 327)
- **Pregnancy is a diabetogenic state** due to several contra-insulin factors causing decreased sensitivity of the peripheral tissues to insulin (insulin resistance).
- **Metabolic events** for a woman with diabetes in pregnancy is **maternal hyperglycemia** → Fetal hyperglycemia → Fetal pancreatic islet cells hyperplasia and hypertrophy → Increased fetal insulin secretion → Excessive fetal growth → Fetal macrosomia → Increased birth injury.
- **Fetal congenital malformations** are high in diabetic woman (pregestational diabetes); commonest is cardiovascular (see p. 330).
- **Complications of diabetes in pregnancy** are increased both for the mother, fetus and the neonates (see p. 329).
- **Screening for GDM** is done between 24 and 28 weeks of pregnancy (see p. 326).
- **Treatment of women with diabetes in pregnancy:** Dietary therapy. Insulin is given when glycemic control is not achieved with dietary therapy. Oral hypoglycemic agents (glibenclamide, metformin) are also being used to maintain glycemic control.
- **Close antenatal fetal assessment** for women with both GDM and pregestational diabetes is maintained. It depends on degree of glycemic control, presence of vascular disease (nephropathy, retinopathy) or hypertension.

contd...

contd...

- **Self monitoring of capillary blood glucose** is encouraged for optimum glycemic control. The goals of capillary blood glucose levels (mg/dL) are: fasting ≤ 95; 2 hr. postprandial ≤ 120; 2 am to 6 am ≥ 60; Mean 100 and HbA1C ≤ 6 (ACOG-2005).
- **Timing of delivery** should be with fetal maturation provided diabetes is well controlled and fetal surveillance is normal.
- **Mode of delivery** depends on level of glycemic control, fetal size, associated complication if any (preeclampsia) and pelvic adequacy. As such 50% of diabetic mothers are delivered by cesarean section. However selected women may be considered for induction of labor and/or vaginal delivery.
- **Pre-pregnancy counseling** ideally be done for all such women for optimum glycemic control and pregnancy outcome.

THYROID DYSFUNCTION AND PREGNANCY

HYPERTHYROIDISM: Physiological changes during pregnancy such as increase in cardiac output, oxygen consumption and heat production may mimic mild thyrotoxicosis (ch. 5). Hyperthyroidism occurs in about 2 per 1,000 pregnancies. Autoimmune hyperthyroidism (Graves' disease) due to **thyroid stimulating antibodies** is the commonest cause. Other causes are : Nodular thyroid disease, sub-acute thyroiditis, hyperemesis gravidarum and trophoblastic disease.

MATERNAL AND FETAL/NEONATAL COMPLICATIONS IN UNTREATED HYPERTHYROIDISM:

Maternal: Miscarriage, preterm delivery, preeclampsia, congestive cardiac failure, placental abruption, thyroid storm and infection.

Fetal/Neonatal: IUGR, prematurity, stillbirth, hyperthyroidism, hypothyroidism, increased perinatal morbidity and mortality.

Thyroid stimulating antibodies cross the placenta and produce neonatal thyrotoxicosis with increased neonatal death. The risk is increased if the antithyroid drug is stopped in late pregnancy or following surgery. **Clinical diagnosis** of hyperthyroidism should always be confirmed by measuring free T_4 (FT_4 – high), free T_4 index (FT_4 1– high), FT_3 (high) and TSH (suppressed) levels. Ultrasonography of the fetal thyroid gland is done when the mother is taking antithyroid drugs. Thyroid peroxidase antibodies (TPOAb), (antimicrosomal antibodies) and thyroid stimulating immunoglobulin should be measured. Radioactive iodine uptake and scans **should not be done** during pregnancy as it will cross the placenta and damage the fetal thyroid gland permanently.

The main stay of treatment is use of antithyroid drugs [propylthiouracil (PTU) or methimazole (MM)]. Both the drugs are effective. Methimazole is preferably avoided in the first trimester of pregnancy if PTU is available. Methimazole has the risk of **embryopathy**. Carbimazole is given orally with a daily dose of 10–40 mg and maintained at this dose until the patient becomes euthyroid. Then it is progressively reduced to a maintenance of between 5 mg and 15 mg daily. Propylthiouracil is given at a daily dose of 300–450 mg and continued till the patient becomes euthyroid—the maintenance dose being 50 and 150 mg daily. Both the drugs may cause fetal goiter and hypothyroidism. Methimazole may be associated with **aplasia cutis** of the neonate. Normalization of TSH and FT4 is an indicator to reduce the dose of drugs. Patients having marked tachycardia or arrhythmias should also have propranolol (β blocking agent).

Fetal surveillance is maintained with serial USG, NST and BPP (see p. 121). **The drugs are not contraindicated during breastfeeding provided** the dose is kept relatively low and close monitoring of the neonatal thyroid functions is carried out. Cord blood should be taken for TSH and free T_4 at the time of delivery to detect neonatal hyperthyroidism.

Thyroidectomy, when required to relieve the pressure symptoms can be done safely in the second trimester with prior biochemical control.

Preconceptional counseling: Considering the hazards during pregnancy, preconceptional counseling is important. Adequate treatment should be instituted to bring down the thyroid function profile to normal. **Radioactive iodine (^{131}I) therapy should not be given to patients wanting pregnancy within one year**. If pregnancy occurs inadvertently, termination should be done. **Oral pill is to be withheld** because of accelerated metabolism and disturbed liver function.

HYPOTHYROIDISM: May be subclinical (elevated TSH and normal FT_4) or overt (elevated TSH and low FT_4). The clinical association of hypothyroidism in pregnancy may be due to (i) first time diagnosis in pregnancy (ii) hypothyroid women who either discontinue thyroid therapy or who need larger doses in pregnancy (iii) hyperthyroid women on excessive amounts of antithyroid drugs (iv) women with lithium or amiodarone therapy. **Primary hypothyroidism** met in pregnancy is mostly related to thyroid autoimmunity (Hashimoto thyroiditis). Myxedema rarely presents in pregnancy because they tend to be infertile. Untreated hypothyroidism in early pregnancy has a high fetal wastage in the form of abortion, stillbirth and prematurity and deficient intellectual development of the child. However, pregnancy complications like preeclampsia and anemia are high. Serum thyroid peroxidase antibodies (TPO-Ab) or antimicrosomal antibodies are elevated in autoimmune thyroiditis. Serum TSH should be repeated at an interval of 6–8 weeks as there is increased demand of thyroid hormone in the second half of pregnancy. If the patient is having substitution therapy in pre-pregnant state, the dose of levothyroxine need to be increased in pregnancy. Generally, therapy is started 2 to 2.4 mcg/kg/day. The maintenance dose for most patients is between 75 and 150 mcg of L-thyroxin per day. The serum TSH should be repeated every 2 to 6 weeks. In order to keep serum TSH, serum FT_4 or $FT_4$1 values within the normal range, the dose need to be increased in the second trimester of pregnancy. After delivery, dose is reduced.

Postpartum thyroiditis: It is an autoimmunothyroid disease observed by 6–12 weeks postpartum due to anti-microsomal antibodies. Most women present with transient hyperthyroid state of which nearly two-thirds become euthyroid and the remaining one-third become hypothyroid. High risk factors are infection, traumatic delivery, dehydration and stress. Treatment is symptomatic (β blockers and/or levothyroxine). **Thyroid storm** is a life threatening condition. It is diagnosed by features of severe thyrotoxicosis like hyperpyrexia (> 103°F), tachycardia (> 140 beats/min), CCF, neuropsychiatric symptoms, nausea and vomiting. Mortality rate is about 25%. Laboratory tests show hyperthyroid changes: **Management includes**: aggressive approach. (i) Supportive therapy in an intensive care unit: fluids and electrolytes balance, O_2 therapy and acetaminophen for hyperpyrexia (ii) management of CCF (iii) β blockers (propranolol) therapy to control hyperdynamic symptoms (iv) Antithyroid drug (PTU or MM) to block thyroid hormone synthesis (v) Lugol's iodine solution, and (vi) Glucocorticoids (Hydrocortisone) to block peripheral conversion of serum T_4 to T_3.

Thyroid storm is a life threatening condition with a mortality rate of about 25%.

JAUNDICE IN PREGNANCY

When the serum bilirubin level exceeds 2 mg% (normal being 0.2–0.8 mg%), visible yellow staining of the tissue appear. Overall incidence in India is 1–4 per 1,000 deliveries. **The causes of jaundice during pregnancy may be grouped as follows**:

Jaundice Peculiar to the Pregnant State	Jaundice Unrelated to Pregnant State	Jaundice When Pregnancy is Superimposed on Chronic Liver Disease
♦ Intrahepatic cholestasis (obstetric hepatosis)—may be recurrent ♦ Severe preeclampsia, eclampsia, HELLP syndrome ♦ Acute fatty liver (acute yellow atrophy of the liver) ♦ Severe hyperemesis gravidarum ♦ Endotoxic shock—disseminated intravascular coagulation (DIC)	♦ Viral hepatitis: virus A – E, G ♦ Gall stone—obstructive jaundice ♦ Drug induced—isoniazid, phenothiazines ♦ Hemolytic jaundice—mismatched blood transfusion, malaria, *Clostridium welchii* infection, etc.	♦ Chronic hepatitis ♦ Cirrhosis, tumors

OBSTETRIC CHOLESTASIS (*Syn*: *Obstetric hepatosis, icterus gravidarum*)

Obstetric cholestasis (OC) is the second most common cause of jaundice in pregnancy, the first one being viral hepatitis. Overall incidence is 1.2–1.5% of pregnant Indian women. The stasis of bile in the bile canaliculi with rise in conjugated bilirubin is probably due to excess circulating estrogen. Similar manifestation is also observed in women taking contraceptive pills. Genetic, familial and abnormal progesterone metabolism have been observed.

The manifestations usually appear in the last trimester. The onset is insidious; generalized pruritus is the predominant symptom; there may be weakness, nausea or even vomiting. Jaundice is slight. There is rise in the levels of AST, ALT and serum alkaline phosphatase. Bilirubin level rarely exceeds 5 mg%. Liver biopsy shows no evidence of necrosis but shows the features of intrahepatic cholestasis.

There are increased risks of preterm labor, low birthweight babies, meconium stained liquor, IUD and postpartum hemorrhage. Prothrombin time should be monitored. The features subside within two weeks postpartum. Treatment of (OC) with medications, are not supported by any evidence. Cholestyramine is effective for itching. All women with OC should be given vit K to reduce postpartum hemorrhage and neonatal bleeding. The neonate should be given vit K as a routine. Combined oral contraceptives should be avoided in women with history of obstetric cholestasis. Prothrombin time should be monitored. Ursodeoxycholic acid (UDCA) is found helpful. It increases bile acid excretion. It improves pruritus. **Recurrence** rate is high (50–60%).

VIRAL HEPATITIS

Viral hepatitis is the commonest cause of jaundice in pregnancy in the tropics. Hepatitis is mostly restricted to the ill-nourished mothers, living in unhygienic environment. In the tropics, it often occurs as an epidemic form. There is also increased incidence of its affection in the pregnant state compared to the non-pregnant one. At present **six distinct types** of highly contagious hepatitis virus have been identified. Each type (mentioned below) has different clinical effect to the pregnant women and her fetus.

- **Hepatitis – A (RNA)**
- **Hepatitis – B (DNA)**
- **Hepatitis – C (RNA)**
- **Hepatitis – D (RNA)**
- **Hepatitis – E (RNA)**
- **Hepatitis – G (RNA)**

Hepatitis A (HAV): Infection is spread by fecal-oral route. Diagnosis is confirmed by detection of IgM antibody to hepatitis A. Disease is usually self limited and fulminant hepatitis is rare. Perinatal transmission is rare, chronic carrier state does not exist. The virus is not teratogenic. Pregnant woman **exposed to HAV infection** should receive immunoglobulin 0.02 mL/kg within 2 weeks of exposure. She should also have hepatitis A vaccine single dose 0.06 mL IM. It is safe in pregnancy.

Hepatitis B virus (HBV): It is a global public health problem. The virus is **transmitted by parenteral route, sexual contact, vertical transmission and rarely through breast milk**. The risk of transmission to fetus ranges from 10% in first trimester to as high as 90% in third trimester and it is **specially high (90%)** from those mothers who are **seropositive to hepatitis B surface antigen (HBsAg) and 'e'-antigen (HBeAg)**.

Neonatal transmission mainly occurs at or around the time of delivery through mixing of maternal blood and genital secretions. Approximately 25% of the carrier neonate will die from cirrhosis or hepatic carcinoma, between late childhood to early adulthood. HBV is not teratogenic.

Maternal infection: The acute infection is manifested by flu like illness as malaise, anorexia, nausea and vomiting. There may be arthralgia and skin rash. In majority, it remains asymptomatic. Jaundice is rare and fever is uncommon.

Clinical course (HBV): Nearly 90–95% of patients clear the infection and have full recovery. 1% develop fulminant hepatitis resulting massive hepatic necrosis. 10–15% become chronic and 10% of these chronic cases suffer from chronic active hepatitis, cirrhosis and hepatocellular carcinoma.

Diagnosis is confirmed by serological detection of HBsAg, HBeAg, (denote high infectivity) and antibody to hepatitis B core antigen (HBc) and HBV DNA titer. Chronic carriers are diagnosed by presence of HBsAg or HBeAg and anti-HBc antibody and HBV-DNA titer (10^7 to 10^{11}) 6 months after the initial infection. Liver enzymes are elevated during the initial phase.

Screening: All pregnant women should be screened for HBV infection at first antenatal visit and it should be repeated during the third trimester for "high risk" groups (intravenous drug abusers, sexual promiscuity, hemophilics, patients on hemodialysis or having multiple sex partners).

Hepatitis C (HCV): It is recognized as the major cause of non-A, non-B hepatitis. Transmission is mainly blood borne and to a lesser extent by fecal-oral route. It is responsible for chronic active hepatitis and hepatic failure. Perinatal transmission (10–40%) is high when viral load is high and presence of coinfection with HIV and HBV. Detection is by antibody to HCV by EIA, which develops usually late in the infection. Confirmation is done by recombinant immunoblot assay (RIBA-3). Chronic carrier state is present. No effective vaccine against HCV is available. Women should be immunized against hepatitis A and B if not immune. Breastfeeding is not contraindicated.

Hepatitis D (HDV): It is seen in patients infected with HBV either as a co-infection or super infection. Perinatal transmission is known. Chronic carrier state is seen. Neonatal immunoprophylaxis for HBV is almost effective against HDV. Acute infection with fulminant course results in high maternal mortality (2–20%) due to hepatic failure.

Hepatitis E (HEV): It behaves similar to hepatitis A virus infection. It is observed in Asia (Kashmir) and South America. It may lead to fulminant hepatitis. ELISA can detect HEV specific IgG and IgM antibodies or by PCR. Chronic carrier state is present. Perinatal transmission is uncommon. Maternal mortality following acute infection is high (15–20%).

Hepatitis G (HGV): It is related to hepatitis C virus. It is more prevalent but less virulent than HCV. Co-infection with hepatitis A, B, C and HIV is common. Chronic carrier state is known and perinatal transmission is documented.

Prognosis: Fulminant hepatitis is more common in hepatitis E, less common in hepatitis C and rare in hepatitis A. Mortality is very high in fulminant type.

Maternal: There is increased incidence of postpartum hemorrhage, hepatic coma, renal failure, coagulopathy, infection and hepatorenal syndrome. All these lead to increased maternal morbidity and mortality. Medical termination of pregnancy does not alter the prognosis of the patient.

Fetal: There is increased incidence of abortion, preterm birth and intrauterine death leading to increased fetal wastage. Congenital malformation of the fetus following viral hepatitis in early pregnancy is inconclusive. Perinatal mortality is about 20–70%.

MANAGEMENT

PROPHYLAXIS: Improvement in sanitation, supply of safe drinking water and adequate care of personal hygiene are the essential prerequisites. Use of disposable syringe or boiling of syringe prior to use are the positive steps in prevention. **Screening of blood donors for HBsAg** should be routinely done.

Management of HBV during pregnancy: HBV infection can be prevented by vaccination and the recombinant vaccine is safe in pregnancy.

Pregnant woman who is seronegative, should have HB immunoglobulin (HBIG), 0.06 mL/kg IM, soon following exposure and a second dose after 1 month. Then she should be given recombinant DNA vaccine intramuscularly 1 mL, 3 doses at 0, 1 and 6 months. All infants born to HBsAg positive mothers should have HBIG 0.5 mL IM within 12 hours of birth. Active immunization with HB vaccine (0.5 mL) is also given IM at a separate site at the same time schedule. This is very effective (85–95%) to protect the infant from HBV infection. Breastfeeding is not contraindicated. Similar to HIV, perinatal transmission of HBV depends on maternal viral load. Lamivudine and HBIG are effective to reduce the transplacental transmission of HBV to the fetus. Lamivudine is given 150 mg/day from 34 weeks.

Hepatitis A: Both passive immunization (HAIG) and active immunization with killed virus vaccine are available for the mother.

Health-care workers should receive hepatitis B vaccine and they should avoid needle stick injury and blood to blood contact.

TREATMENT: There is no specific treatment. It is generally supportive. Consultation with a hepatologist is ideal.

- **Rest:** The patient should be put to bed rest, if necessary by hospitalization.
- **Isolation:** The patient should be kept in isolation. Blood samples are to be collected with gloved hand. Disposable syringes should be used. The excreta is to be disposed carefully.
- **Nutrition:** Diet rich in carbohydrate and adequate protein is to be prescribed. Initially, glucose drink, fruit juice may be given. Dietary fat restriction is not necessary. If the patient cannot tolerate oral feeding, 10% glucose may be given intravenously.
- **Drugs:** To prevent formation of the toxic nitrogenous compound from the bacterial flora of the gut, oral neomycin (1 gm to be given 6 hourly) is helpful. Lactulose (15–30 mL three times daily), reduces colonic ammonia absorption and it acts as an osmotic laxative. Hepatotoxic drugs should not be used. **There is no place for termination of pregnancy**.
- **Prevention of complications:** Hypokalemia, hypoglycemia and hypocalcemia are corrected by regular blood checkup. Hemorrhagic complications are managed by giving blood or fresh frozen plasma.
- **During labor:** (a) Hepatotoxic drugs should be avoided. (b) To administer vitamin K, 5 mg intramuscularly to raise the prothrombin level (c) Prophylactic oxytocin is to be given.
- **Hepatologists** to be involved. Patient may need ICU management depending on liver function tests.

EPILEPSY IN PREGNANCY

The effect of pregnancy on epilepsy is uncertain. Frequency of convulsions is unchanged in majority (50%). The frequency of convulsions is unchanged in majority (50%), increased in 45% and decreased in about 5% of women. Serum concentration of anticonvulsant falls in pregnancy. All anticonvulsants interfere with folic acid metabolism. Folic acid deficiency has been associated with neural tube defects and other congenital malformations.

Effects of epilepsy on pregnancy—Incidence of fetal malformations, IUGR, oligohydramnios, preeclampsia and stillbirths are increased. Birth defects are increased by twofold. This could be related to the severity of the disease with its genetic predilection and also due to the anticonvulsants used. Pattern of abnormalities is related to the type of anticonvulsant drugs (valproate 5.9%, Carbamazepine 2.3% and Lamotrigine 2.1%).

The malformations include—Cleft lip and/or palate, mental retardation, cardiac abnormalities, limb defects and hypoplasia of the terminal phalanges. Sodium valproate is associated with neural tube defects. There is chance of neonatal hemorrhage and is related to anticonvulsant induced reduction of coagulation factors (vitamin K dependent). The risk of developing epilepsy to the offspring of an epileptic mother is 10%.

Preconception counseling includes—(1) To initiate monotherapy (if possible) replacing polytherapy. (2) To administer folic acid 4 mg daily. (3) Importance of prenatal diagnosis is to be discussed.

Management—The dose of the chosen drug should be kept as low as possible. Valproate and phenytoin are found to be most teratogenic. The **commonly used drugs are:** carbamazepine 0.8–1.2 mg daily in divided doses, phenytoin 150–300 mg daily in two divided doses. Lamotrigine 300–500 mg/day is given and it is not an enzyme inducer. Newer drugs used with safety are: topiramate (100–400 mg/day) and levetiracetam 1–3 gm/day (not enzyme inducer). Serum levels may be measured in patients with frequent seizures to assess therapeutic levels and compliance. Fits are controlled by IV phenytoin with a slow loading dose of 15–20 mg/kg. It is highly effective, has a long duration of action and side effects are less. Otherwise benzodiazepine 10–20 mg slow IV may be given. Folic acid 4 mg daily is to be started before pregnancy and to be continued throughout. Prenatal diagnosis with serum αFP at 16 weeks (p. 129) and detailed fetal anatomy scan at 18 weeks (p. 493) with real time ultrasonography (Level II) including fetal echocardiography is done. There is decrease in free level of most of the anticonvulsants in pregnancy. The reasons are: delayed gastric emptying, reduced absorption, increased protein binding, nausea, vomiting, increase in plasma volume, increased hepatic metabolism and renal clearance. Vitamin K 10 mg a day orally is to be given to mother in the last two weeks.

There is **no contraindication for breastfeeding**. Infant is given injection vitamin K 1 mg IM at birth to prevent neonatal hemorrhage due to decreased vit K dependent clotting factors. The infant may be drowsy. Readjustment of the anticonvulsant dosage is necessary and to bring down the dose to the pre-pregnant level by 4–6 weeks postpartum. Steroidal contraceptives are better to be avoided due to hepatic microsomal enzyme induction (see p. 624).

The risk of having epilepsy of an infant born to a mother with a seizure disorder is four times higher compared to a normal one.

ASTHMA IN PREGNANCY

During pregnancy some amount of breathlessness is common due to the effect of progesterone and fall in arterial CO_2 tension (p. 63). Such breathlessness is considered physiological where oxygen saturation is more than 95%. Asthma is a chronic airway inflammation due to its **hyper-responsiveness** to a number of irritants. The incidence of asthma is about 5–8% of all pregnant women.

EFFECTS OF PREGNANCY ON THE DISEASE: The course of the disease is very much unpredictable. In about 20%, the condition improves, in 30%, it deteriorates and in 50%, it remains unchanged. Bronchodilator influences are due to progesterone and cortisol and bronchoconstrictor influences are due to reduced residual volume and increased PGF_2 and thromboxane. Asthma increases maternal morbidity.

EFFECTS OF ASTHMA ON PREGNANCY: There is increased incidence of preterm labor, PROM, preeclampsia, FGR or LBW and neonatal hypoxia. Maternal risk increases with **status asthmaticus**. Life threatening complications include pneumothorax, cor pulmonale, cardiac arrhythmias and respiratory failure.

MANAGEMENT: Pre-conception counseling: The overall risk of any child having asthma is about 4%. If one parent has asthma, the risk that the child will have asthma increases to 8–16%. If both the parents have asthma and also atopic (allergic), the risk may be as high as 30%.

Pregnancy: *Step therapy of asthma* is currently recommended.

Step Therapy of Asthma	
Mild Intermittent	Inhaled β agonist (albuterol) as needed
Mild Persistent	Low dose inhaled corticosteroid (budesonide) or LTRA
Moderate Persistent	Low dose or medium dose inhaled corticosteroids and LABA (salmeterol)
Severe Persistent	High dose inhaled corticosteroid and LABA ± OCS

(**LTRA**: leukotriene receptor agonist; **LABA**: long acting β agonist; **OCS**: Oral corticosteroids)

MANAGEMENT OF ACUTE ATTACKS OF ASTHMA (ASTHMA EXACERBATION) IN PREGNANCY

— Avoidance of asthma triggers (allergens, irritant) to minimize airway inflammation and hyper-responsiveness.

— **Oxygen inhalation** with mask to maintain O_2 saturation > 95% (pulse oximeter).

- **High dose albuterol** by nebulization every 20 minutes and inhaled ipratropium bromide and systemic corticosteroid. Repeat assessment of symptom, physical examination and O_2 saturation to be done.
- **Corticosteroids:** Intravenous hydrocortisone 200 mg stat and to be repeated after 4 hours. Because of long onset of action, corticosteroids should be given along with β_2-agonists.
- **Forced expiratory volume (FEV_1)** in one second or peak expiratory flow rate (PEFR) < 50% and PCO_2 > 42 mm Hg necessitates intubation and mechanical ventilation with 100% O_2 in ICU.
- **Mechanical ventilation** is needed for status asthmaticus to avoid hypoxemia and carbon dioxide retention.

LABOR

- **Clinical evaluation** of the patient in labor may be inaccurate to predict the severity. PEFR or FEV_1 should be determined.
- Asthma medications to be continued. $FEV_1 \geq 70\%$, and reassuring fetal status on EFM indicates good response to therapy.
- **Opiate analgesics** should be avoided as they are bronchoconstrictor and respiratory depressant. Maternal oxygenation should be adequately maintained. Labetalol should be avoided as it may precipitate asthma.
- **Hydrocortisone** 100 mg IV 8 hourly during labor and 24 hours postpartum is to be given if the patient had steroids within the previous 4 weeks. Inhaled corticosteroid (fluticasone, budesonide) prevents bronchial hyper-responsiveness to allergens.
- **Syntocinon** is better than ergometrine because of bronchoconstrictor effect of the latter. **$PGF_2\alpha$** should not be used, as it precipitates bronchospasm. PGE_1 and PGE_2 compounds can be used locally for induction of labor or abortion.
- **Epidural anesthesia** is preferable to general anesthesia because of risk of atelectasis and subsequent chest infection following the latter. Halothane is better in general anesthesia. However, it produces uterine atony.
- **Ketamine** is used for induction of general anesthesia as it prevents bronchospasm.
- **O_2 saturation is assessed** with pulse oximeter or arterial blood gases.
- **Postnatal physiotherapy** is maintained and drugs are continued.

Breastfeeding should be encouraged, as it delays the onset of allergic problems in the child. Drugs used in asthma: Prednisolone, corticosteroids, LABA, LTRA do not contraindicate breast feeding.

Contraception: Barrier method is the best. For terminal contraception, husband is to be motivated for vasectomy.

SYSTEMIC LUPUS ERYTHEMATOSUS (SLE)

SLE is a serious autoimmune disease with autoantibodies causing specific multisystem (skin, joints, kidneys, lungs, liver, nervous system and other organs) affection. It is 10 times more common in adult women than the adult men. It is first diagnosed during pregnancy in 10–30% cases.

Clinical features: Common presenting features are: fatigue, fever, weight loss, arthralgias, arthritis and myalgias. Joint pains are often migratory in nature.

Effects of pregnancy on SLE: Long-term prognosis remains unaffected. There is chance of flare ups especially during first half and maximum in puerperium. Majority of maternal deaths occur in puerperium, the cause being pulmonary hemorrhage and lupus pneumonitis and exacerbation of lupus nephritis.

Effects of SLE on pregnancy: Risks of lupus rash, anemia, leukopenia, thrombocytopenia and renal failure are increased. There are increased risks of first trimester miscarriage, lupus nephritis, recurrent deep vein thrombosis, PIH, prematurity, IUGR and stillbirths. **Neonatal lupus syndrome** is due to crossing of maternal lupus antibodies (anti-Ro or anti-La) to the fetus causing hemolytic anemia, leukopenia and thrombocytopenia. Isolated congenital heart block is present in about one-third of cases. An apparently healthy woman delivering a baby with congenital heart block should be observed for the development of SLE.

Investigation: **Antinuclear antibodies** are the standard screening test for the disease. Presence of autoantibodies to double-stranded DNA **(dsDNA)** is highly specific to the diagnosis. Antibodies to Sm antigen (RNA protein) found in 30–40% of SLE patients are highly specific and are correlated with renal involvement. Other antibodies for diagnosis are: Lupus anticoagulant, antiphospholipid antibodies, anti-Ro and anti-La. Baseline laboratory tests are done to assess anemia, thrombocytopenia, renal function tests and serum antibodies (LA, ACL, anti-RO/SSA and anti-La/SSB).

Management: Pre-conception planning is extremely important since conception during a period of quiescence is most likely to result in a live birth. **Lupus** can flare any time in pregnancy and postpartum period (15%–60%). The **predictive factors for successful pregnancy outcome are:** a) Phase of sustained remission in the past 6 months; b) Continued use of hydroxychloroquine (HCQ) through pregnancy. Corticosteroids (prednisolone) are the commonly used drugs. As an analgesic, paracetamol is the best agent. Non-steroidal anti-inflammatory agents can be used during puerperium. Low dose aspirin 80 mg daily is prescribed with advantages (minimizes PIH and IUGR). It should be stopped at least two weeks before delivery. Prednisolone 1–2 mg/kg/day is started initially and the dose is gradually tapered. **Hydroxychloroquine is safe in pregnancy and found to be the best for maintenance**. It reduces the risks of prolonged corticosteroid therapy (PE, bone demineralization, GDM and FGR). Women, who are on chronic corticosteroid should receive hydrocortisone 100 mg every 8 hours (IV) for three doses during labor or at the time of cesarean delivery. **Immunosuppressive drugs (azathioprine) may be needed to control severe cases of SLE. Mode of delivery** is guided by the obstetric behavior. Fetus with congenital heart block should be delivered by cesarean section. Patients should receive corticosteroids during the peripartum period.

Oral contraceptives may affect SLE and IUCD may predispose to infection in an immunocompromised patient, as such, **barrier method of contraceptives** are recommended. Progestins do not flare lupus.

TUBERCULOSIS IN PREGNANCY

INCIDENCE: The incidence ranges between 1% and 2% amongst the hospital deliveries in the tropics, being confined predominantly to the underprivileged sectors of society. Incidence of tuberculosis is rising worldwide with the rising prevalence of HIV infected patients. In 2000, WHO showed the emergence of multidrug resistant tuberculosis (MDR-TB) all over the world. It is a "global health emergency".

RISK FACTORS: ♦ Positive family history or past history ♦ Low socioeconomic status ♦ Area with high prevalence of tuberculosis ♦ HIV infection ♦ Alcohol addiction ♦ Intravenous drug abuse ♦ Diabetes, jejunoileal bypass, underweight by ≥ 15% ♦ Chronic renal failure.

DIAGNOSIS: (i) **Tuberculin skin test** with Purified Protein Derivative (PPD) induration ≥ 5 mm is considered positive especially in presence of risk factors (HIV) (ii) **X-ray chest** (after 12 weeks) (iii) **Early morning sputum** (three samples) for acid-fast bacilli (iv) **Gastric washings** (v) **Diagnostic bronchoscopy** (vi) **Extrapulmonary sites**—lymph nodes, bones (rare in pregnancy) (vii) **Direct amplification tests for** 16 S ribosomal DNA and gene probe can detect *M. tuberculosis* with greater sensitivity and specificity.

Congenital tuberculosis is diagnosed by—(1) Lesion noted in the first week of life (2) Infection of the maternal genital tract or placenta (3) Cavitating hepatic granuloma diagnosed by percutaneous liver biopsy at birth (4) No evidence of postnatal transmission. **Congenital tuberculosis is rare.** Concomitant HIV infection or when TB is drug resistant, the risk is increased.

PROGNOSIS: Provided the patient remains under medical supervision with adequate treatment, pregnancy has got **no deleterious effect on the course of the disease; nor has the disease any adverse effect on the course of pregnancy**. In active disease, fetus can be affected by transplacental route or by aspiration of amniotic fluid. Neonatal affection is mainly by postpartum maternal contact. **In untreated patients, the incidence of preterm labor, IUGR and perinatal mortality is high**. In such a situation maternal morbidity and mortality are also high.

MANAGEMENT

MEDICAL: Prevention—Women (age < 35 years) with positive PPD and no evidence of active disease (asymptomatic)—**Isoniazid prophylaxis 300 mg/day** is started after the first trimester and continued for 6–9 months. Pyridoxine (Vit B_6) 50 mg/day is added to prevent peripheral neuropathy. No major adverse fetal or neonatal effects are seen with these antituberculous drugs.

<table>
<tr><th>First Line Drugs</th><th>Daily Dose — PO</th><th>Major Side Effects</th></tr>
<tr><td>▪ Isoniazid (Pyridoxine)</td><td>5 mg/kg up to 300 mg (50 mg daily)</td><td>Hepatitis, peripheral neuropathy, hepatic enzyme elevation, hypersensitivity</td></tr>
<tr><td>▪ Rifampicin</td><td>10 mg/kg up to 600 mg</td><td>Nausea, vomiting, hepatitis, orange discoloration of urine and secretions, febrile reaction</td></tr>
<tr><td>▪ Ethambutol</td><td>15 mg/kg up to 2.5 gm</td><td>Skin rash. Optic neuritis, decreased visual activity</td></tr>
<tr><td>▪ Pyrazinamide</td><td>15–30 mg/kg up to 2 gm</td><td>Hepatotoxicity, skin rash, arthralgias, hyperuricemia, GI upset</td></tr>
<tr><td colspan="3">RNTCP recommends: Category 1 (All new cases irrespective of smear positivity): 2 H3 R3 Z3 E3 + 4 H3 R3 and Category II: (smear positive cases with relapse, failure, default or others): 2 H3 R3 Z3 E3 S3 + 1 H3 R3 Z3 E3 + 5 H3 R3 E3.</td></tr>
<tr><th colspan="3">Second Line Drugs</th></tr>
<tr><td>▪ Para-aminosalicylic acid (PO)</td><td>150 mg/kg up to 1 gm</td><td>G1 toxicity, hepatotoxicity</td></tr>
<tr><td>▪ Cycloserine (PO)</td><td>15-20 mg/kg up to 1 gm</td><td>Rash, convulsions</td></tr>
<tr><td>▪ Kanamycin (1M)</td><td>15-30 mg/kg up to 1 gm</td><td>Auditory and renal toxicity, rarely vestibular</td></tr>
</table>

Treatment: Women with active tuberculosis should receive the following drugs orally daily for a minimum period of 9–18 months.

SURGICAL: Major thoracic surgery should be withheld, if possible, but if deemed necessary should be restricted to the first half of pregnancy beyond 12 weeks.

OBSTETRIC MANAGEMENT:

***Place of therapeutic termination*:** Tuberculosis *per se* is not an indication for termination of pregnancy.

Obstetric management is no different from other pregnant women, once tuberculosis is well managed.

Breastfeeding: Breastfeeding is not contraindicated when a woman is taking anti-tuberculous drugs. Breastfeeding should be avoided if the infant is also taking the drugs (to avoid excess drug level). **In active lesion, however, not only is breastfeeding contraindicated but the baby is to be isolated from the mother following delivery. Baby should be given prophylactic isoniazid** 10–20 mg/kg/day for 3 months when the mother is suffering from the active disease. However, if the mother is on effective chemotherapy for at least 2 weeks, there is no need to isolate the baby. BCG should be given to the baby as early as possible.

CONTRACEPTION: Pregnancy is to be avoided until quiescence is assured for about two years. Spacing can be achieved by any methods acceptable to the couple. **Oral contraceptives** should be avoided when rifampicin is used. Due to accelerated drug metabolism contraceptive failure is high (see p. 624). A barrier method may be used. **Puerperal sterilization** should be seriously considered, if the family is completed.

Morbidity and Mortality of TB in pregnancy is high when left untreated. Many drugs, once contraindicated (cycloserine, kanamycin), are found to have no adverse fetal or neonatal effects. Women with multidrug resistant TB in pregnancy need to be counseled about the benefits versus small risks of using the second line drugs.

SYPHILIS IN PREGNANCY

Syphilis is a sexually transmitted disease caused by ***Treponema pallidum***. Incidence is rising due to upsurge of HIV infection and the IV drug abuse. Overall frequency of **vertical transmission** (congenital syphilis) is high in primary (50%) and secondary (50%) syphilis. In tertiary syphilis it is about 10%. The symptoms may be suppressed during pregnancy.

EFFECTS ON PREGNANCY

Mother—Syphilis accelerates the course of HIV infection in pregnant woman.

Baby—Congenital infection results from transplacental migration of spirochete to the fetus. Congenital disease occurs with all stages of maternal infection and at any gestational age. The basic pathology is **obliterative endarteritis**. There is perivascular infiltration of lymphocytes and plasma cells within the developing fetus. **The placenta** becomes bulky from increased connective tissue. The villi become bulky due to increased cellularity, the vascularity being diminished. Spirochete can hardly be found in the placenta. **However, the baby may be affected without specific changes in the placenta**.

Depending upon the intensity and time of occurrence of the infiltration, **the fate of the fetus will be as follows**: (1) Abortion (2) Preterm birth (3) Intrauterine deaths leading to either a macerated or a fresh stillbirth (4) Non-immune fetal hydrops (ascites, hepatomegaly) (5) Delivery of a highly infected baby with early neonatal death (6) Survival with congenital syphilis.

DIAGNOSIS: Mother

- **Obstetric history in multigravidae**—With serial pregnancies, there have been gradually improved obstetric performances. A classic history shows—late abortion → macerated stillbirth → fresh stillbirth → congenital syphilitic baby → healthy baby.
- **Clinical findings** of various stages of syphilis—usually suppressed during pregnancy.
- **Investigations:** (a) Serological test—This should be done as a routine in the first antenatal visit. VDRL (positive within 4 weeks of infection) is commonly done (b) A positive VDRL test has to be confirmed by fluorescent treponemal antibody absorption test **(FTA-ABS)** and *Treponema pallidum* microhemagglutination (**MHA-TP**) test which are specific. Husband's blood should also be tested for VDRL (c) Detection of spirochetes from the cutaneous lesion if any, by dark field examination (d) Fetal infection could be diagnosed by **polymerase chain reaction (PCR)** of *T. pallidum* in amniotic fluid, fetal serum or spinal fluid. Spirochetes may be detected from fetal liver or spleen.

Clinical features of congenital syphilis:

- ***Early:*** Maculopapular rash, rhinitis, hepatosplenomegaly, jaundice, lymphadenopathy, chorioretinitis and pneumonia.
- ***Late:*** Hutchinson teeth, deafness, saddle nose, saber shins, hydrocephalus, mental retardation, clutton joint, interstitial keratitis and optic nerve atrophy.

If the baby is stillborn, spirochetes may be detected from the fetal liver or spleen or from the intimal scraping of umbilical vein.

TREATMENT: ***Mother:*** **Treatment should be started as soon as the diagnosis is established**. The baby may have the chance of protection even if the treatment is begun late in pregnancy. For **primary or secondary or latent syphilis** (< 1 year duration): benzathine penicillin 2.4 million units intramuscularly single dose. When the **duration is more than a year**—benzathine penicillin 2.4 million units IM weekly for 3 doses is given. If the patient is allergic to penicillin, oral azithromycin 2 gm as a single dose is given. **Tertiary disease**: Neurosyphilis—Aqueous crystalline penicillin G 18–24 million units IV daily for 10–14 days is given. If the treatment is given in early pregnancy, the treatment should be repeated in late pregnancy. **Irrespective of the serological report, treatment should be repeated in subsequent pregnancies**.

Baby:

- **Positive serological reaction without clinical evidences of the disease** —The baby is treated with a single intramuscular dose of penicillin G 50,000 units per kg body weight.
- **Infected baby with positive serological reaction:** (1) Isolation with the mother (2) Intramuscular administration of aqueous procaine penicillin G 50,000 units per kg body weight each day for 10 days.

- **An apparently healthy child of a known syphilitic mother:** Serological reaction should be tested weekly for the first month and then, **monthly for 6 months**.

LEPROSY (HANSEN DISEASE) IN PREGNANCY: Latent infection by *M. leprae* may become overt for the first time during pregnancy or within few months following delivery. With established leprosy, there is chance of exacerbation of the lesions during pregnancy. **Pregnancy may be regarded as a test of cure. Failure of signs of the disease to reappear during or after pregnancy indicates that the leprosy is definitely arrested.** Although *M. leprae* have been found in the placenta and cord, congenital infection of the baby is unlikely. **However, the baby should be separated from the infected mother, immediately after delivery.** When the disease becomes quiescent and non-infectious, the baby may be given to the mother. **Dapsone and clofazimine** appear safe in pregnancy. **Rifampicin** may also be used. **IUGR** is a common problem.

GONORRHEA IN PREGNANCY: The manifestations of the disease are the same as in the non-pregnant state. If the infection takes place during pregnancy, it tends to be more acute. **The diagnosis** is to be confirmed by bacteriological identification of intracellular Gram-negative diplococci from urethral or cervical smear. Serological test is to be done to exclude concurrent syphilitic infection. **Infection increases the risk of preterm labor, PROM, intrapartum and postpartum infection. Disseminated infection** includes : arthritis, meningitis, endocarditis and perihepatitis (Fitz-Hugh-Curtis syndrome). **The baby may be affected during labor while passing through the infected birth canal resulting in ophthalmia neonatorum.**

Treatment: Single dose injection of Ceftriaxone 125 mg IM or Cefixime 400 mg PO single dose or a single 2 gm IM dose of spectinomycin is effective. Both the parents are treated. Ophthalmia neonatorum is treated with either silver nitrate or tetracycline preparation. Infected neonate is treated with single dose of ceftriaxone 50 mg/kg IM.

CHLAMYDIAL INFECTIONS: Chlamydial infection is becoming the common sexually transmitted pathogen. The organisms are found in urethra, endocervix and rectum. *C. trachomatis* is an obligate intracellular bacteria. **The adverse effects in pregnancy are**: Preterm labor, PROM, Chorioamnionitis, stillbirth, perihepatitis (**Fitz-Hugh-Curtis syndrome**). Puerperal endometritis or acute salpingo-oophoritis may develop. Neonates may develop conjunctivitis or pneumonia.

Confirmation is only done by tissue culture methods which is expensive and time consuming. **The culture materials should be taken from both the cervix and urethra**. **Culture** is replaced by antigen detection method. Serological detection of chlamydial antigen (lipopolysaccharide) by ELISA is done using a kit. DNA amplification by PCR is more reliable.

Treatment is highly effective with erythromycin in doses 0.5 g 4 times a day for 7–10 days. Azithromycin 1 gm in a single dose could also be prescribed. Therapy should be instituted to the husband simultaneously. **As a prophylaxis to ophthalmia neonatorum**, tetracycline or erythromycin ointment 1% is to be applied to the infant's eyes soon following birth. Neonatal infection is treated with erythromycin 50 mg/kg/day 4 times a day for 14–21 days.

GROUP B STREPTOCOCCAL INFECTION (GBS): Maternal infection with GBS is an important cause of high perinatal mortality. Vaginal and anorectal colonization of GBS is the main source of infection. Main risk factors for neonatal infection are prolonged rupture of membranes, preterm labor, prolonged labor and low birth weight. Neontal infections include R.D.S., pneumonia, jaundice, hypotension, septicemia and meningitis. Neonatal mortality is about 30–40%.

Maternal complications include: Endometritis, UTI, PROM and preterm labor and wound infection. Diagnosis is made by culture of specimens obtained from vagina, perineum using a cotton swab. PCR can also be used.

Currently universal cultures for all patients is recommended (CDC, 2002) for prevention of GBS infection. Intrapartum therapy with ampicillin 2 gm initially, then 1 gm 6 hourly is effective.

PARASITIC AND PROTOZOAL INFESTATIONS IN PREGNANCY

MALARIA: Malaria is predominantly a tropical disease. In India and other south-east Asian countries there is resurgence of malaria. The diagnosis is confirmed by the detection of malarial parasites in peripheral thick blood smear.

Pathology: The infected erythrocytes become rigid, irregular and sticky. There is blockage of microcirculation due to the sequestrated red cells. The infected red cells are broken down (hemolysis). Maternal HIV or tuberculosis causes intense parasitization of the placenta.

*The fetal effects are due to **high fever** or due to **placental parasitization**. The intervillous spaces become blocked with macrophages and parasites and there is diminished placental blood flow. This is mostly seen with **P. falciparum infection** and in the second half of pregnancy. **Congenital malaria is rare (< 5%) unless the placenta is damaged.***

Effects of Malaria on the Mother	Effects on the Fetus
♦ Anemia (megaloblastic) due to hemolysis and folic acid deficiency ♦ Thrombocytopenia ♦ Hypoglycemia : due to increased glucose consumption both by the host and the parasites. There is ↓ hepatic gluconeogenesis and ↑ insulin secretion by the drugs (quinine) ♦ Metabolic acidosis (pH < 7.3) ♦ Jaundice due to hepatic dysfunction ♦ Renal failure — due to block of renal microcirculation ♦ Pulmonary edema and respiratory distress (RDS) ♦ Convulsions and coma — cerebral malaria ♦ Circulatory collapse ♦ Thrombocytopenia ♦ Abnormal bleeding (DIC)	Pregnancy complications are increased ♦ Miscarriage ♦ Preterm labor ♦ Pre-maturity ♦ IUGR ♦ Congenital malaria ♦ IUFD

Effects of pregnancy on malaria:

- Risk and severity of infection are high due to immunocompromised state
- Complications are high.

Management: Prevention from mosquito bites is done using permethrin and pyrethroids-spray kill mosquitoes. Electrically heated mats will kill mosquitoes in the room. **Chemoprophylaxis:** Chloroquine is given unless proved resistant. It is taken 300 mg base weekly, 2 weeks before travel and covering the period of exposure and 4 weeks after leaving the endemic zone. Mefloquine 5 mg/kg/week is the alternative drug in second and third trimesters when chloroquine is found resistant.

Treatment: Risks of malaria is life threatening in pregnancy. So benefits of treatment **outweigh the potential risk of antimalarial drugs**. **Chloroquine**—600 mg base P.O. followed by 300 mg 12 hours later. Then 300 mg daily for next 2 days. To prevent relapse during pregnancy, 300 mg is to be taken weekly until delivery. For radical cure, **primaquine should be postponed** until pregnancy is over. **Parasites resistant to chloroquine** should be given **quinine** (10 mg salt/kg P.O> every 8 hours for 7 days) under supervision. Patients with severe anemia may need blood transfusion. The antimalarial drugs when given in therapeutic doses, have got no effect on uterine contraction unless the uterus is irritable. **Folic acid 10 mg should be given daily to prevent megaloblastic anemia**.

Complicated malaria: Artesunate IV 2.4 mg/kg at 0, 12 and 24 hours, then daily thereafter. Oral therapy (2 mg/kg) is started when the patient is stable. Alternatively Quinine IV followed by oral therapy is given. Artesunate act very fast and resistance is rare. It is as effective as IV Quinine. Use is limited in the second or third trimesters of pregnancy only when other drugs are found resistant.

Patient with severe malaria needs intensive care unit management. Supportive care is essential in cases with hyperpyrexia, pulmonary edema, renal failure or DIC. Multidisciplinary team approach is needed.

TOXOPLASMOSIS: Toxoplasmosis is a protozoan infestation caused by *Toxoplasma gondii*. **Infection is transmitted through** encysted organism by eating infected raw or uncooked meat or through contact with infected cat feces. It can also be acquired across the placenta.

The fetal risk of infection increases with duration of pregnancy and is approximately 15%, 30% and 60% in the first, second and third trimesters respectively. The fetus is only at risk if the mother is seronegative. **Risk of fetal damage decreases** from 60% in first trimester to almost zero percent at the end of pregnancy. There is increased rate of abortion, IUGR and stillbirth. **During parasitemia, transplacental infection to the fetus occurs.** The affected baby may develop hydrocephalus, chorioretinitis, cerebral calcification, microcephaly, seizures and mental retardation. Presence of IgM antibody in the neonates indicates congenital infection. IgG transmitted from the mother persists for many years.

Maternal infection is mostly asymptomatic when she is immunocompetent. **Acute infection** is diagnosed by detecting IgM specific antibody, high level of IgG antibody titer and detection of seroconversion for IgG from negative to positive. Neonatal infection is common (40%) when mother suffers with acute toxoplasmosis. Routine screening is usually not done. Chronic maternal toxoplasmosis is not considered to be a significant cause of recurrent abortion as parasitemia will not be repeated in subsequent pregnancies.

If current infection is confirmed the following tests are carried out: (1) Amniocentesis and cordocentesis for detection of IgM antibody in the amniotic fluid and fetal blood. PCR for T. gondii gene is also done (2) Ultrasonography findings suggestive of fetal infection are: ventriculomegaly, intracranial calcification, microcephaly, hepatosplenomegaly, ascites and IUGR at 20–22 weeks. If the fetus is infected and hydrocephalus is present, counseling for termination is to be done.

Treatment: Toxoplasmosis is a self-limited illness in an immunocompetent adult and does not require any treatment. Pyrimethamine 25 mg orally daily and oral sulfadiazine 1 gm four times a day is effective. Leucovorin is added to minimize toxicity. Four to six weeks course is usually given. Pyrimethamine is not given in the first trimester. Spiramycin (3 gm orally daily) has also been used as an alternative. Acute toxoplasmosis during pregnancy is treated with spiramycin. Extended courses may be needed in an immunocompromised patient to cure infection. Treatment to the mother reduces the risk of congenital infection and the late sequelae.

Prevention: Uncooked meat, unpasteurized milk and contact with stray cat or cat litter are to be avoided.

LISTERIOSIS: Listeria monocytogenes (LM) is an intracellular gram-positive bacillus. It is readily found in soil and vegetation. It can grow and multiply in temperature as low of 0.5°C. Infection is caused by eating infected food or through contact with infected miscarried products of animals. Maternal symptoms are 'flu-like' or "food poisoning". There is no reliable serological test for it except the blood culture during septicemia. Obstetric complications are: Late miscarriage, preterm labor and delivery and stillbirth. LM infections can cause meningitis, encephalitis, bacteriemia pneumonia, fever and gastroenteritis. Neonatal death due to septicemia is also high (10%). Overall perinatal mortality is 50%.

Treatment: Combined therapy with ampicillin, amoxicillin and gentamicin is preferred. Trimethoprim and sulfamethoxazole can also be used.

Prevention of listeriosis during pregnancy according to FDA includes: Not to drink or take—unpasteurized milk, soft cheese, refrigerated smoked seafood (salmon, trout, cod).

INTESTINAL WORMS: Hookworms (*Ancylostoma*) and round worms (*Ascaris lumbricoides*) are the common intestinal infestations in the tropics and subtropics. The diagnosis is by stool examination. The treatment consists of eradicating the worms along with treatment of anemia by iron therapy. **Deworming is not contraindicated during pregnancy excluding the first trimester.**

PYELONEPHRITIS IN PREGNANCY

There is increased chance of urinary tract infection in females as compared to males due to: (i) Short urethra (4 cm) (ii) Close proximity of the external urethral meatus to the areas (vulva and lower third of vagina) contaminated heavily with bacteria (iii) Catheterization (iv) Sexual intercourse.

INCIDENCE: The overall incidence of pyelonephritis in pregnancy is between 1% and 3%.

ETIOLOGY: (1) It is more common in primigravidae than multiparae (2) Previous history of urinary tract infection increases the chance by 50% (3) Presence of asymptomatic bacteriuria increases the chance by 25% (4) Abnormality in the renal tract is found in about 25%.

Physiologic changes responsible for acute pyelonephritis in pregnancy are: (1) Low ureteral peristalsis due to high progesterone levels. (2) Dextrorotation of gravid uterus causing compression of right ureter. Pyelonephritis occurs more on the right side (70–80%) compared the left (10–15%).

PATHOGENESIS: ***Predisposing factors***—Dilatation of the ureters and renal pelvis and stasis of the urine in the bladder and ureters are the normal physiological changes during pregnancy. **The organisms responsible are**

E. coli (70%), *Klebsiella pneumoniae* (10%), *Enterobacter*, *Proteus*, *Pseudomonas* and *Staphylococcus aureus* group. About 10% of women develop bacteremia following acute pyelonephritis. 70–80% of pyelonephritis occur on the right side, 10–15% on the left side and only few are bilateral.

CLINICAL TYPES: Depending upon the mode of onset and the presenting features, the cases are grouped into:

- **Acute or severe type**
- **Chronic type**

ACUTE PYELONEPHRITIS: Clinical features—the onset is acute and usually appears beyond the 16th week. The involvement is bilateral but if unilateral, it is more frequent on the right side. Clinical features are mainly due to endotoxemia. The chemical mediators (cytokines) released are: IL-1, TNF and endogenous pyrogen. **Important features are:**

Acute aching pain over the loins, often radiating to the groin and costovertebral angle tenderness, urgency, frequency, dysuria, hematuria. **Fever** (spiky 40°C) with chills and rigor followed by hypothermia (34°C); anorexia, nausea, vomiting and myalgias; respiratory distress and pulmonary edema (ARDS) due to endotoxin induced alveolar injury.

Investigations: Apart from the routine ones, serum level of creatinine, electrolytes and culture studies of urine and blood should be done.

DIFFERENTIAL DIAGNOSIS: Includes (1) Acute appendicitis (2) Abruptio placentae (3) Red degeneration of fibroid (4) Acute cholecystitis (5) Labor (6) Chorioamnionitis.

COMPLICATIONS: FETAL: There may be increased fetal loss due to abortion, preterm labor, intrauterine fetal death caused by hyperpyrexia and low birth weight babies (prematurity and dysmaturity).

MATERNAL: Anemia, Septicemia, septic shock, renal dysfunction and pulmonary insufficiency. ARDS may develop due to endotoxin induced alveolar capillary membrane damage following septicemia.

MANAGEMENT—The outlines of management are:

- Intravenous fluid (crystalloid) for adequate hydration.
- Evaluate hemogram, serum electrolytes, creatinine.
- Acetaminophen is given for the fever.
- Monitor urine output (≥ 60 mL/hr), temperature and BP.
- IV antibiotics—Cephalosporins, aminoglycosides (gentamicin), Cefazolin or Ceftriaxone, for 48 hours till culture report is available and then changed to oral therapy for another 10-14 days.
- Repeat urine culture after 2 weeks of antimicrobial therapy and is repeated at each trimester of pregnancy.
- If the symptoms recur or the dip stick test for nitrate and leukocyte esterase is positive, urine culture is repeated. The woman needs retreatment if the culture is positive.
- Patient not responding with this therapy needs to be evaluated (sonography, CT scan, radiography) for urinary tract obstruction.
- **Antimicrobial suppression therapy is continued till the end of pregnancy to prevent recurrence (30–40%). Nitrofurantoin 100 mg daily at bed time is effective.**

ASYMPTOMATIC BACTERIURIA (ASB)

The term asymptomatic bacteriuria is used when a bacterial count of the same species over 10^5/mL in mid stream clean catch specimen of urine on two occasions is detected without symptoms of urinary infection. This indicates actively multiplying bacteria within the urinary tract. *E. coli* is the offending organism in over 90% cases. Other pathogens are *Klebsiella pneumoniae* and *Proteus*. To exclude pre-existent ASB, all pregnant woman should ideally have a urine culture at their first antenatal visit (ACOG). The overall incidence during pregnancy ranges between 2% and 10%.

Considering its high incidence, screening techniques have a definite place. Urine should be screened for nitrites and leukocyte esterase. If either of these tests is positive, urine culture is indicated.

Twenty-five percent of these women are likely to develop acute pyelonephritis, usually in third trimester, if left untreated. The incidence of hypertension and anemia is stated to be high. The increased association of **premature labor and growth retarded babies** is probably related with the underlying chronic renal lesion.

Asymptomatic bacteriuria if recurrent is associated with **high incidence of urinary tract abnormality** (20%), congenital or acquired. The woman runs a greater **risk of developing chronic renal lesion in later life**.

Treatment: The antimicrobial agents should be appropriate to the mother and the fetus. Any one of these drugs could be prescribed, amoxicillin (500 mg tid), nitrofurantoin (100 mg bid), cephalexin (500 mg tid) or amoxicillin–clavulanic acid (875 mg bid). A course of 10–14 days will cure 70–100% of cases. A single dose therapy of nitrofurantoin 0.2 gm or amoxicillin 3 gm has been found effective. Long-term prophylaxis with nitrofurantoin (50 mg) or amoxicillin (250 mg) at night may have to be continued until delivery when the infection is recurrent. Women with recurrent UTI should undergo imaging of the upper urinary tract, 3 months postpartum.

PROTEINURIA IN PREGNANCY: The term proteinuria is preferable to albuminuria as globulin fraction also leaks out along with albumin. Proteinuria is present in the urine in about 5% of all pregnant women. **The causes are**: (1) Preeclampsia and eclampsia (2) Urinary tract infection (3) Chronic renal disease : nephritis and nephrotic syndrome (4) Essential hypertension (5) Orthostatic —Due to increased lumbar lordosis there is increased pressure on the inferior vena cava by the uterus or the left renal vein is compressed by the aorta. These lead to congestion of one or both the kidneys leading to proteinuria. In late pregnancy, the enlarged gravid uterus may compress the left renal vein when the patient is placed on supine position. Lying down on lateral position relieves the pressure and congestion and makes the urine free of protein.

Investigation: If the history (present and past) and clinical examination fail to find out the cause, the investigation protocols are designed to rule out infection of the urinary tract and renal parenchymal lesion. Microscopic examination (pus cells, casts, RBCs), bacteriological studies (including tuberculosis) and renal biopsy are done. **Management** is based on the etiology. **Orthostatic proteinuria** in the absence of bacteriuria or hypertension is not significant.

HEMATURIA IN PREGNANCY: Presence of few red cells under the microscope in routine examination of urine during pregnancy is mostly due to contamination. Examination of the "clean catch" midstream urine rules out contamination. The causes of hematuria are manifold and are broadly divided into :

I. Those related to the pregnant state **II. Those unrelated to pregnancy**

- ***Those related to the pregnant state are***: (1) Severe cystopyelitis (2) Rupture of bladder varicosities (3) Following rapid evacuation of urine in acute retention with a retroverted gravid uterus (4) Lower segment scar rupture involving the bladder (5) Operative delivery following obstructed labor due to congestion. The possibility of traumatic injury should be ruled out.
- ***Those unrelated to pregnancy*** as urinary calculi, renal tuberculosis, renal neoplasm, papilloma bladder.

Treatment consists of—(a) to increase the urinary output by adequate fluid intake and (b) to correct the pathology by medical or surgical treatment.

RETENTION OF URINE: Retention of urine is not an uncommon complication during pregnancy state. **The causes are divided into—**(A) ***During early pregnancy***— (1) Incarcerated retroverted gravid uterus (2) Impacted pelvic tumors. (B) ***During labor***—(1) Associated with abnormal uterine activity commonly with incoordinate uterine action (2) Obstructed labor. (C) ***During puerperium***—(a) diminished bladder tone (b) reflex from vulval injuries (3) Bruising and edema of the bladder neck. If simple measures fail, catheterization is to be done using a disposable catheter.

VIRAL INFECTIONS IN PREGNANCY

RUBELLA: Rubella or German measles (RNA virus) is transmitted by respiratory droplet exposure. Maternal Rubella infection is manifested by rash, malaise, fever, lymphadenopathy and polyarthritis. Fetal infection is by transplacental route throughout pregnancy. Risk of major anomalies when this infection occurs in first, second and third month is approximately 60%, 25% and 10%, respectively. Multisystem abnormalities are seen following congenital rubella infection. Congenital rubella syndrome (CRS) predominantly include cochlear (sensorineural deafness), cardiac (septal defects, PDA), hematologic (anemia, thrombocytopenia), liver and spleen (enlargement, jaundice), ophthalmic (cataracts, retinopathy, cloudy cornea), bone (osteopathy) and chromosomal abnormalities. **The virus predominantly affects** the fetus and is extremely teratogenic if contracted within the first trimester. **There is increased chance of** abortion, stillbirth and congenitally malformed baby. Infants born with congenital rubella shed the virus for many months and is a source of infection to others. Test for rubella specific antibody (IgM) should be done within 10 days of the exposure to know whether the patient is immune or not. Rubella specific IgG antibodies are present for life after natural infection or vaccination. **If the patient is not immune, question of therapeutic**

termination should be seriously considered. Detection of viral RNA by PCR is possible. Prenatal diagnosis of rubella virus infection using PCR can be done from chorionic villi, fetal blood and amniotic fluid samples.

Active immunity can be conferred in non-immune subjects by giving live attenuated rubella virus vaccine (MMR) preferably during 11–13 years. **It is not recommended in pregnant women. When given during the child-bearing period, pregnancy should be prevented within three months by contraceptive measure.** However, if pregnancy occurs during the period, termination of pregnancy is not recommended.

MEASLES: The virus (RNA) is not teratogenic. However, high fever may lead to miscarriage, FGR, microcephaly and oligohydramnios, stillbirth or premature delivery. Non-immunized women coming in contact with measles may be protected by intramuscular injection of immune serum globulin (5 mL) within 6 days of exposure. Mortality is high when complications like pneumonia, encephalitis develop. Diagnosis is made by assay of IgM and detection of viral RNA (RT-PCR). **Management** is supportive care. Antibiotics are given to prevent secondary bacterial infections. Ribavirin may be given for viral pneumonia. **Active vaccination (live attenuated) should not be given in pregnancy.**

INFLUENZA: Influenza virus (RNA) are enveloped. Hemagglutinin (H) and neuraminidase are present on the surface. Influenza strains are named according to their genus, species and H and N subtypes. The course of pregnancy remains unaffected unless the infection is severe. **Effects on pregnancy due to H1-N1 infection:** miscarriage, preterm labor, PROM, pneumonia, ARDS, renal failure, DIC and death. Severity of illness are high in pregnancy. There is no evidence of its teratogenic effect even if it is contracted in the first trimester. However, outbreak of Asian influenza showed increased incidence of congenital malformation (anencephaly) when the infection occurred in the first trimester. **Influenza (inactivated) vaccine is safe in pregnancy and also with breastfeeding. Diagnosis:** Rapid influenza diagnostic tests (RIDTs) are immunoassay used for detection of viral RNA by RT-PCR. **Management:** Treatment is supportive care. Acetaminophen is used for fever. Oseltamivir (neuraminidase inhibitor), an antiviral drug, reduces the severity, secondary complications and death. It is given 75 mg PO, twice daily for 5 days. High dose may be given for longer duration in cases with severe infection. During influenza season, all pregnant women should be given the inactivated vaccine (IM).

CHICKENPOX (Varicella): Varicella zoster virus (DNA) does cross the placenta and may cause congenital or neonatal chickenpox. **Maternal mortality is high due to varicella pneumonia.** Other maternal complications are: encephalitis and bacterial superinfection. **Congenital varicella syndrome (CVS) is characterized by:** hypoplasia of limb, psychomotor retardation, IUGR, chorioretinal scarring, cataracts, microcephaly and cutaneous scarring. The risk of congenital malformation is nearly absent when maternal infection occurs after 20 weeks. **Varicella (live attenuated virus) vaccine is not recommended in pregnancy.** Varicella PCR can identify VZV specific DNA from vesicular fluid. ELISA can detect VZV specific IgG and IgM. Varicella zoster immunoglobulin (VZIG) should be given to exposed non-immune patients as it reduces the morbidity. VZIG should also be given to newborn exposed within 5 days of delivery. Oral acyclovir, valacyclovir is safe in pregnancy and reduce the duration of illness when given within 24 hours of the rash. However, it cannot prevent congenital infection.

CYTOMEGALOVIRUS (CMV) INFECTION: It is a DNA virus. Transmission may be sexual, respiratory droplet or transplacental. Virus is also excreted with urine, cervix and breast milk. **Fetus is affected by transplacental route** in about 30–40% cases. Unlike rubella, CMV may damage fetal organs throughout gestation. Amongst all infected fetuses only 10% will suffer serious damage. The **consequences of infection (5%–18%) include** miscarriage, non immune hydrops, stillbirth, IUGR, microcephaly, intracranial calcification, hepatosplenomegaly, thrombocytopenia, choroidoretinitis, mental retardation and sensorineural deafness. Congenitally-affected infants may excrete virus (through urine and nasopharynx) for up to 5–7 years. Infection is confirmed by viral culture DNA PCR of urine and nasopharyngeal secretions. CMV specific IgM is present with 80% infected infants. Prenatal diagnosis by amniocentesis is possible using PCR to detect CMV DNA. A recombinant protein vaccine against CMV glycoprotein β has been shown to prevent maternal CMV infection. Maternal CMV infection is best diagnosed by DNA PCR of blood, urine, saliva, amniotic fluid or cervical secretions. Routine screening in pregnancy is not recommended.

PARVOVIRUSES: Parvovirus B 19 (DNA) is associated with human infection (fifth disease) during pregnancy. Fetal affection is by transplacental route. Fetal infection occurs in 33% cases following maternal infection. Infection is characterized by facial rash (slapped cheek appearance). It mainly affects the erythroid precursor cells resulting in anemia, thrombocytopenia, aplastic crises, congenital heart failure and hydrops. Fetal loss is more when infection occurs early (< 20 weeks) in pregnancy. Diagnosis is made by detection of virus specific IgM. PCR amplification of viral DNA from fetal and maternal blood is more sensitive than IgM antibody. Maternal infection is usually self limited. Serial USG should be performed 10 weeks after maternal illness to detect any fetal hydrops. Fetal middle cerebral artery peak Doppler velocity can be studied to detect any significant fetal anemia before hydrops develop. Intrauterine transfusion may improve the fetal outcome (p. 395). Nearly 33% of fetal hydrops resolve spontaneously. Otherwise mortality rate is about 30% (p. 571).

MUMPS (RNA): Maternal mumps has got no ill effects on the course of pregnancy. The virus is not teratogenic. Incidence is expected to be low with the introduction of measles-mumps-rubella vaccine (MMR) to childhood vaccination program (see p. 526). MMR vaccine (live-attenuated viruses) is contraindicated in pregnancy.

HERPES SIMPLEX (DNA) VIRUS (HSV): Genital tract infection is due to HSV-2. Infection may be primary, nonprimary, first episode and recurrent. It is transmitted by sexual contact. Primary infection may occur during pregnancy. Reactivation or recurrent infection occurs resulting in virus shedding with or without symptomatic lesions. HSV-1 infection is usually herpes simplex labialis.

Effect on pregnancy—Increased risk of miscarriage is inconclusive. If the primary infection is acquired in the last trimester there is chance of premature labor or IUGR. Transplacental infection is not usual. The fetus becomes affected by virus shed from the cervix or lower genital tract during vaginal delivery. The baby may be affected *in utero* from the contaminated liquor following rupture of the membranes. **Risk of fetal infection is high in primary genital HSV at term due to high virus shedding compared to a recurrent infection. Cesarean delivery is indicated (ACOG) in an active primary genital HSV infection** where the membranes are intact or recently ruptured. Acyclovir 400 mg three times daily for five days is the drug of choice when virus culture is positive.

Neonatal infection may be disseminated (fatal) or localized or it may be asymptomatic. **Diagnosis** is made by detection of the viral DNA by PCR. It is manifested as chorioretinitis, microcephaly, mental retardation, seizures and deaths. Neonatal HSV infection is treated with intravenous acyclovir. Neonatal mortality is high. **Prophylactic acyclovir** (400 mg twice daily) or valacyclovir (1 gm twice daily) can reduce HSV shedding, neonatal transmission and cesarean delivery. **Breastfeeding** is allowed provided the mother avoids any contact between her lesions, her hands and the baby.

DENGUE: Dengue (RNA) virus is transmitted by Aedes aegypti mosquito. Infected pregnant women present with acute febrile illness, headache, myalgia, facial flushing, retro-orbital pain, skin rashes (maculopapular) and rarely with hemorrhage. Laboratory findings are: leukopenia, thrombocytopenia and elevated serum levels of AST or ALT > 1000 U/L. Diagnosis is made by elevated levels of IgM (ELISA) or by antigen detection by ELISA or RT-PCR during the acute phase. **Triad of symptoms** are: hemorrhagic manifestations, plasma leakage and platelet counts <100000/mm^3. Maternal mortality is high specially when **shock syndrome** develops (50%). Pregnancy complications are: miscarriage, preterm labor, IUFD and still births. Vertical transmission may occur but fetal affection is not severe.

Management: Supportive care. Maintenance of intravascular volume, blood pressure and fluid replacement is to be done. Ribavirin may be useful, paracetamol is administered for control of pyrexia.

HUMAN IMMUNODEFICIENCY VIRUS INFECTION (HIV) AND ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS)

Human immunodeficiency virus (HIV) causes an incurable infection that leads ultimately to a terminal disease called **acquired immunodeficiency syndrome (AIDS)**. Worldwide 25–30% of infected patients are women and 90% of them are 20–49 years of age.

Incidence: Incidence is difficult to work out but the fact remains that the disease is alarmingly increasing both in the developed and in developing countries. It is now a global problem. The prevalence even in low-risk population in America is close to 1 in 1000. The seropositivity rate among US pregnant women is 1–2 per 1000. **In most Asian countries the infection rate is less than 0.5%.**

The virus: HIV viruses (HIV 1 and HIV 2) are **RNA retroviruses having the enzyme reverse transcriptase, which permits genomic RNA to be transcribed into double stranded DNA**. HIV preferentially targets lymphocytes expressing CD4 molecules whose action in the immune system is to combact viruses, bacteria and certain malignancies. Once the virus is into the genome of the host, it produces multiple copies of itself, which will eventually cause host cell damage. There is gradual depletion of CD_4^+ cells. There is also failure of B lymphocytes to produce antibodies to HIV. These events lead to progressive loss of host immune defence and development of AIDS. Primary infection → 3–6 weeks → Acute syndrome (1 week–3 months) → Immune response to HIV (1–2 weeks) → Clinical latency—about 10 years → AIDS.

The main modes of transmission of HIV are—(i) Sexual contact (homosexual or heterosexual) (ii) transplacental (iii) exposure to infected blood or tissue fluids and (iv) through breast milk.

Perinatal transmission of HIV: Vertical transmission to the neonates is about 14–25%. Transmission of HIV 2 is less frequent (1–4%) than for HIV 1 (15-40%). Transplacental transmission occurs: 20% before 36 weeks, over 80% of transmissions occur around the time of labor and delivery. Vertical transmission is more in cases with preterm birth and with prolonged membrane rupture. Risks of vertical transmission is directly related to maternal viral load (measured by HIV RNA) and inversely to maternal immune status (CD_4^+ count). Maternal anti-retroviral therapy reduces the risk of vertical transmission by 70% (see below). Breastfeeding doubles the risk of MTCT transmission (14% to 28%).

Male to female transmission is about double compared to female to male transmission. Rectal intercourse is more dangerous than vaginal. Parenteral transmission is the most potent route.

Immunopathogenesis: HIV virus mutates readily and produces several genotypes which are capable of immune control. Profound cell **mediated immunodeficiency is the basic pathology** as the HIV leads to slow but progressive destruction of T cells. The incubation period is about 1–3 weeks. After a peak viral load, there is gradual fall until a steady state of virus concentration is reached. This is known as **set point** which is a state of balance between the virus's ability to replicate and the host's ability to protect itself by neutralization and removal of virus. When the set point viral load is high → more destruction of host CD_4^+ cells → progressive immunosuppression → opportunistic infections and cancers.

Effects: Pregnancy *per se* has got no effect on the disease progression in HIV positive women. Increased incidence of abortion, prematurity, preeclampsia, IUGR and perinatal mortality in HIV seropositive mothers still remains inconclusive. Maternal mortality and morbidity are not increased by pregnancy.

Clinical presentation: Initial presentation of an infected patient may be fever, malaise, headache, sore throat, lymphadenopathy and maculopapular rash. Primary illness may be followed by an asymptomatic period. Progression of the disease may lead to multiple opportunistic infections (OI) with candida, tuberculosis, pneumocystis and others. Patient may present with neoplasms such as cervical carcinoma, lymphomas (Hodgkin's and non-Hodgkin's) and Kaposi's sarcoma. There may be associated constitutional symptoms like weight loss, lymphadenopathy or protracted diarrhea. CD_4^+ count < 200 cells/mm^3 is diagnostic of AIDS. **The median time from infection to AIDS is about 10 years**.

Diagnosis: HIV diagnosis is made by detecting HIV viral RNA in blood by PCR testing (HIV RNA PCR) or by detecting antibodies to HIV. The enzyme immunoassay (EIA) is used as a screening test for HIV antibodies. It is extremely sensitive (99.5%), inexpensive but less specific. EIA kits are commercially available. An initial positive EIA test must be confirmed with a second, more specific test Western blot or HIV RNA PCR. This is then **confirmed by Western blot, immunofluorescence assay (IFA) or HIV RNA PCR**. The western blot detects specific viral antigens P24 (Capsid), GP41 (envelope) and GP 120/160 (envelope). False positive rate of Western blot is less than 1 in 10,000.

MANAGEMENT

PRENATAL CARE:

(i) Integrated counseling and testing (ICT) in the antenatal clinic (ANC) to all pregnant women with an 'Opt Out' approach is offered.

(ii) **In seropositive cases** the following additional tests should be done. (1) Test for other STDs—such as hepatitis B and C viruses, syphilis, chlamydia, herpes and rubella, (2) Serological testing for cytomegalovirus and toxoplasmosis, (3) Tuberculosis, (4) Fungal opportunistic infections, (5) Husband should be offered serological testing for HIV.

(iii) **Counselling with education** to the patient is done about the impact of HIV infection on pregnancy; perinatal transmissions, side effects of medications and mode of delivery. Pregnancy does not affect the progression of HIV disease.

(iv) **Progression of the disease is assessed by—**(i) $CD4^+$ T lymphocyte counts and (ii) HIV RNA (viral load). Assessment is done at every 3–4 months interval. A patient with low viral load (< 3000 copies/mL) and high $CD4^+$ count (> 750 cells/mm^3) has nearly a zero probability of progressing to AIDS within 3 years. **Women with $CD4^+$ count (≤350 cells/mm^3) or HIV RNA level ≥ 50,000 copies/mm^3 should be initiated with HAART**.

Stages of HIV infection (CDC) – Stage - I (HIV infection): CD4$^+$ T-lymphocyte ≥500/mm^3; Stage-2 (HIV Infection): CD4$^+$ T-lymphocyte count of 200-499/mm^3; Stage-3 (AIDS): CD4$^+$ T-lymphocyte count of ≤200/mm^3.

(v) The patient should have T lymphocyte count in each trimester. If the count falls to less than 200 cells/mm^3, the patient should receive prophylaxis against *Pneumocystis carinii* and other opportunistic infections.

(vi) **Highly active antiretroviral therapy (HAART)** to HIV 1 positive women is effective in reducing the viral (HIV RNA) load. **Triple chemotherapy is preferred as a first line defence and to be started any time between 14 weeks and 28 weeks and then continued throughout pregnancy, labor and postpartum period.** Women taking HAART should be screened for gestational diabetes.

(vii) **Principles of HAART are to**: (a) suppress viral multiplication maximally (b) reduce perinatal transmission and (c) reduce the risk of drug resistance. Efavirenz is avoided in the first trimester due to its teratogenic risk.

(viii) Prophylactic antibiotics should be started when there is opportunistic infection (*Pneumocystis carinii* pneumonia).

Anti-HIV 1 drugs are grouped into—(A) **Nucleoside reverse transcriptase inhibitors** (NRTIs : Zidovudine, Didanosine, Lamivudine, Abacavir). (B) **Non-nucleoside reverse transcriptase inhibitors** (NNRTIs : Nevirapine, Efavirenz). (C) **Protease inhibitors** (PI : Indinavir, Saquinavir, Ritonavir). (D) **Entry inhibitors**: Enfuvirtide. **Treatment regimens change frequently. However, recommended regimens (USDHHS–2011) are: Two from: Group A plus one from either Gr. B or Gr. C. In resource poor setting Zidovudine 100 mg given five times daily PO can reduce perinatal transmission from 25% to 7%.** WHO recommends first line ART regimen to include: Zidovudine (ZDV), + Lamivudine (3 TC), + Nevirapine (NVP) or ZDV + 3 TC + EFV (Efavirenz).

Laboratory tests: (a) CD4$^+$ cell count (b) Viral load (HIV RNA-PCR) (c) HIV genotype (d) Others: complete blood count (CBC), platelet count, LFT, serum urea, creatinine, electrolytes, amylase and G6PD.

ANTENATAL CARE:

- Women needs screening against opportunistic infections specially when CD4$^+$ cell count is <200 cells/mm^3.
- Women on HAART should be screened for GDM.
- Screening for aneuploidy anomaly scan.
- Monitoring of plasma viral load and drug toxicities, vaccination against HBV and pneumococcal infection should be done.

INTRAPARTUM CARE:

- **Women presenting in labor;** need to check her recent viral load to plan mode of delivery. Women with viral load < 400 copies/mL, neonatal infection was 1%, whereas infection rate was >30% when maternal viral load was >100,000 copies/mL.
- **Zidovudine (ZDV)** is given IV infusion starting at the onset of labor (vaginal delivery) or 4 hours before cesarean section. Loading dose 2 mg/kg/hr, maintenance dose 1 mg/kg/hr **until cord clamping is done.**
- Women taking HAART can have **planned vaginal delivery** when plasma viral load is < 50 copies/mL (RCOG 2010).
- **Elective cesarean delivery** is recommended (RCOG-2010) at 38 weeks for women taking HAART who have plasma viral load > 50 copies/mL.
- **Elective cesarean delivery** reduces the risk of vertical transmission by about 50%. Avoidance of breastfeeding, HAART therapy and appropriate mode of delivery has reduced MTCT rates from 25–30% to < 1%. Baby should be bathed immediately.
- **Perioprätive or peripartum broad spectrum antibiotics** should be given as per hospital protocol.
- **Invasive procedures** that might result in break in the skin or mucous membrane of the infants (procedures like attachment of scalp electrode and determination of scalp blood pH) are contraindicated. Instrumentation (ventouse) is avoided.
- **Amniotomy and oxytocin augmentation** for vaginal delivery should be avoided whenever possible.
- **Place of cesarean delivery to reduce MTCT:** • Elective CS should be done at 38 weeks gestation. • CS to be done prior to onset of labor and prior to rupture of membranes if viral load is >1000 copies/mL or when viral load is unknown. • ZDV IV to be started 4 hours prior to the procedure. • Pediatrician should be involved.
- Caps, masks, gowns and double gloves should be worn. Protective eye wear (goggles) should be used.
- Mechanical suctioning devices should be used to remove secretions from the neonates airways.
- Blunt tipped needles should be used to avoid needle stick injury and washing on any blood contamination from the skin immediately. Appropriate sterilization of instruments and linens should be done.

- **Risk factors** for increased perinatal transmission are: (a) Maternal high plasma viral load (b) Low CD4 cell count (c) Co-existing other STDs and opportunistic infections (d) Prolonged rupture of membranes (e) Chorioamnionitis (f) Invasive intrapartum procedures (g) Preterm delivery (h) Vaginal delivery and (i) Breast-feeding.
- Health-care workers should be protected from contact with potentially infected body fluids. Estimated risk of infection after parenteral or mucous membrane exposure is 0.36%.
- **Post exposure prophylaxis** with triple therapy for 4 weeks, reduces the risk of seroconversion by more than 80%. (ZDV 200 mg tid + Lamivudin 150 mg bid + Indinavir 800 mg tid).
- Disposable syringes and needles are used and they are deposited in the puncture proof containers.
- **Long-term safety of anti-retroviral drugs** is unknown. Neuropathy, myopathy, lactic acidosis, pancreatitis, hepatitis and mitochondrial toxicity have been observed.

POSTPARTUM CARE:

- Women should continue HAART with CD4 count. She should be followed up according to adult guide lines.
- Formula feeding or breast-feeding to be started with counseling and informed consent.
- **Neonatal care:** Antiretroviral therapy (ARV) should be given to all neonates within 4 hours of birth. Usually ZDV monotherapy is started. When all these tests are negative and the baby is not breastfed a confirmatory HIV antibody test is done at 18 months. Once this test is negative, the child is declared to be free of HIV.
- **Breastfeeding**—Doubles the risk of MTCT (from 14% to 28%) but where alternative forms of infant nutrition are not safe, the risks associated with breastfeeding may be accepted. WHO recommends exclusive breast feeding in the developing countries for the first 6 months. Exclusive replacement feeding is done only when AFASS criteria is fulfilled. [A (affordable), F (feasible), A (acceptable), S (sustainable), S (safe)]. Mother is helped to make an informed choice.
- **Zidovudine syrup** – 2 mg/kg, is given to the neonate 4 times daily for first 6 weeks of life. High risk neonate should be treated with HAART. The infant is tested at D_1, weeks 6, 12 and at 18 months of age.

CONTRACEPTIVE COUNSELING AND CARE: Barrier methods of contraception (condom or female condom) is effective in preventing transmission of the virus. COCs are avoided as drug interactions with ARV agents affect their efficacy and safety. IUCD (Copper IUCD and LNG-IUS) are found safe and effective. However, condom use should be continued regardless of the use of other method of contraception. **The disease could be prevented predominantly by health education and by practice of safer sex**.

COUNSELING: Pre-pregnancy and early pregnancy counseling for HIV infected patient is essential. The woman needs ongoing care with a multidisciplinary team including social workers and counselors. The counselor must provide up to date knowledge which enables the patient to make an informed choice.

SURGICAL ILLNESS DURING PREGNANCY

Principles of general surgery during pregnancy:

- Try to avoid major elective surgery, specially abdominal, till delivery.
- Second trimester is the safest time for surgery as the risks of teratogenesis, miscarriage and preterm delivery are lowest.
- Diagnosis of acute abdomen is difficult in pregnant state.
- Emergency surgery has to be done at any time during pregnancy.
- Laparoscopic surgery can be performed safely during pregnancy (p. 354).
- Use of non-ionizing imaging procedures, e.g. USG, MRI is preferred to minimize fetal irradiation.
- Imaging of abdominal organs is difficult in pregnancy due to the presence of gravid uterus.
- Management of pregnant woman with trauma should always be to stabilize the mother first, with evaluation of the fetus thereafter.
- Operation should be done preferably by a senior surgeon with an expert anesthetist. An obstetrician should remain as a standby.
- Minimal handling of the uterus should be done.
- Postoperatively, the patient is to be given pain relief for 48 hours. Use of tocolytics may be helpful.

- Close observation is mandatory for evidences of miscarriage or preterm labor.
- In majority of cases, taken all the precautions, the risk of adverse perinatal outcome is low. However, risk of surgery must be balanced against the complications of the underlying pathology that need surgery.

ACUTE APPENDICITIS: Incidence is about 1 in 1,000 pregnancies. It is the commonest nongynecological cause of acute abdomen requiring surgery.

Diagnosis is difficult in pregnancy due to (a) Nausea and vomiting common in normal pregnancy are also the common symptoms of appendicitis (b) Leukocytosis is common in normal pregnancy (c) Appendix moves upwards and outwards as the uterus enlarges. So pain and tenderness may not be located in the right iliac fossa (Mc Burney's point) (d) Diagnosis is often confused with disturbed ectopic pregnancy, pyelonephritis, twisted ovarian cyst, abruptio placenta and red degeneration of a fibroid, preterm labor.

Effect of appendicitis on pregnancy—may lead to miscarriage, preterm delivery, increased perinatal mortality and maternal mortality.

Effect of pregnancy on appendicitis is adverse because of (a) late diagnosis (b) failure of localization due to displacement of the position and as such (c) peritonitis is more common, specially, in last trimester.

The risks of maternal and fetal mortality from appendicitis in pregnancy is high specially when associated with perforations. Ultrasonography is commonly done. Appendiceal mural thickening, periappendiceal fluid and a noncompressible tubal structure (6 mm or more) are suggestive. MRI may be used when ultrasound is inconclusive.

Treatment consists of laparotomy at the earliest opportunity. Once the diagnosis is suspected, it is essential to operate rather than to wait until generalized peritonitis has developed. Muscle splitting incision should be made at the point of maximum tenderness. Uterine manipulation is avoided to minimize the risk of preterm labor. Laparoscopic appendicectomy can be done before 28 weeks of gestation. Intraoperative fetal monitoring should be considered.

TRAUMA IN PREGNANCY: Trauma in pregnancy may be due to blunt trauma, motor vehicle accident, fall or following domestic violence. **Placental abruption** is the common complication following minor as well as major abdominal trauma. Common types of **penetrating trauma in pregnant women** are due to road traffic accidents, gunshot or stab wounds. Maternal death rates in penetrating trauma is two-thirds lower than in the non-gravid women. It is due to protective effects of the uterus to other abdominal organs. Fetal death is high (70%).

ACUTE PANCREATITIS: It is difficult to diagnose during pregnancy because of the physiological increase of amylase value during the second and last trimester. Serum amylase is elevated to 1000 IU/L or more, serum calcium is usually low. Ultrasound is of diagnostic value. Preterm labor is more common. Once the diagnosis is made, the treatment should be conservative rather than surgical. Medical management includes IV fluids, gastric acid suppression, analgesia and nasogastric suction.

SYMPTOMATIC CHOLELITHIASIS: Incidence is about 1 in 2,000 pregnancies. It is the second most common nongynecological condition that needs surgery during pregnancy. Initial management is conservative. Elective endocystectomy is done in the second trimester or puerperium. Deterioration of clinical condition despite medical therapy or recurrent billiary colic needs cholecystectomy regardless of trimester. Laparoscopic cholecystectomy can be done in the second trimester of pregnancy safely.

PEPTIC ULCER: It is rare during pregnancy to appear for the first time. The course of the disease is unpredictable. Perforation and hemorrhage are uncommon during pregnancy. Infection with *Helicobacter pylori* plays an important part in the pathogenesis. **TREATMENT**: Directed to inhibit acid production (H_2 blocker), acid neutralization (antacids) and eradication of *H. pylori* infection (antibiotic).

LAPAROSCOPY IN PREGNANCY: Laparoscopic surgery can be performed safely during pregnancy. Second trimester is the best time. Fetal risks and preterm labor are less as the uterine manipulation and the use of narcotics are less.

Guidelines of laparoscopic surgery during pregnancy (SAGES, 2008)

1. Obstetric consultation is essential for preoperative and postoperative management.
2. Diagnostic laparoscopy is safe and effective when done in a well selected case. Laparoscopy can be done safely during any trimester of pregnancy.
3. Gastric emptying time is prolonged in pregnancy. Risk of aspiration during anesthesia could be reduced using antacid, and H_2 blocker beforehand.
4. Patient should be in the left lateral decubitus with minimum reverse trendelenburg
5. Open technique (Hasson) for entering the abdominal cavity to be used. Veress needle may be avoided.

6. Antithrombotic prophylaxis are: use of pneumatic compression devices (intraoperative as well as postoperative) and early postoperative ambulation.
7. CO_2 pneumoperitoneum is maintained at 12–15 mm Hg, keeping intraperitoneal pressure minimum.
8. Maternal end tidal CO_2 should be maintained at 25–30 mm Hg (capnography) to minimize maternal and fetal acidosis.
9. Fetal monitoring is to be continued and pneumoperitoneum is to be released if fetal distress arises.
10. Operative time should be minimum as possible.

ACUTE PAIN IN ABDOMEN DURING PREGNANCY

Some amount of abdominal pain is common during pregnancy. One should be very careful to distinguish the pathological variety from the physiological one.

A meticulous history coupled with systematic and thorough examinations (general, abdominal and vaginal) are mandatory to arrive at a diagnosis on the real state of affairs. Many a times a delay in the diagnosis of a nongestational cause terminates fatally. Consultation with a surgeon or a physician should be done whenever felt necessary. Laboratory tests, ultrasonography and X-ray are helpful diagnostic parameters. However, their limitations and restrictions in pregnancy should be borne in mind. **The physician should be conscious of the entity of disturbed tubal pregnancy in early months and rupture of the uterus, in the later months while dealing with acute abdomen in pregnancy**.

Table 20.7: Causes of Acute Abdomen

I. Obstetrical			II. Nonobstetrical	
Early	**Late**	**Medical**	**Surgical**	**Gynecological**
▪ Abortion ▪ Disturbed ectopic ▪ Hydatidiform mole ▪ Acute polyhydramnios	▪ Abruptio placentae ▪ Preterm labor ▪ Labor pains ▪ Rupture uterus ▪ Polyhydramnios ▪ Acute fulminating preeclampsia ▪ Eclampsia ▪ HELLP syndrome ▪ Torsion of the uterus	▪ Pyelitis ▪ Pyelonephritis ▪ Pneumonia ▪ Cystitis ▪ Hepatitis ▪ Acute fatty liver ▪ Peptic ulcer	▪ Acute appendicitis ▪ Intestinal or gastric perforation ▪ Intestinal obstruction ▪ Volvulus ▪ Rectus sheath hematoma ▪ Cholecystitis ▪ Choledocholithiasis ▪ Biliary colic ▪ Renal or ureteric calculi ▪ Malignant disease	▪ Torsion of ovarian cyst ▪ Red degeneration of fibroid ▪ Retention of urine due to retroverted gravid uterus, impacted fibroid or ovarian tumor

QUESTIONS

1. Define anemia in pregnancy? Classify the different causes of anemia in pregnancy? Mention the different complications that are faced when a pregnant woman suffers from severe anemia in pregnancy? (p. 301, 307)
2. Outline in brief the place of iron therapy in the management of anemia in pregnancy? (p. 309)

Write Short Notes on:

A. Management of labor for a woman with heart disease in pregnancy (p. 322)
B. Effects of diabetes on pregnancy (p. 329)
C. Fetal and neonatal hazards for a woman with diabetes in pregnancy (p. 329)
D. Discuss perinatal transmission of HIV (p. 351)
E. Measures to reduce perinatal transmission of HIV (p. 352)
F. Enumerate the important causes of abdomen pain in pregnancy (p. 355)

Chapter 21

Gynecological Disorders in Pregnancy

ABNORMAL VAGINAL DISCHARGE: There is an increased cervical secretions and vaginal transudate during pregnancy due to increased vascularity and hyperestrogenic state. The discharge is thick, mucoid in nature and non-irritating. Microscopic examination reveals preponderance of cornified squamous cells with few pus cells. **Except improvement in personal hygiene, no treatment is required**.

TRICHOMONAS VAGINALIS: The infection is not increased during pregnancy. The clinical features remain the same as in non-pregnant state. Treatment consists of prescribing metronidazole (Flagyl) 200 mg thrice daily for 7 days. Metronidazole should be avoided in the first trimester. **The husband should be treated simultaneously**.

MONILIA VAGINITIS: Vaginitis due to *Candida albicans* is relatively more common than *Trichomonas vaginalis*. Its growth is favored by the high acidic pH of vaginal secretions and frequent presence of sugar in the urine during pregnancy. It is more prevalent in diabetic pregnancy. **Treatment** is by use of miconazole vaginal cream, one applicator full, high up in the vagina at bedtime for 7 nights.

CERVICAL ECTOPY (EROSION)

Hormonal ectopy — During pregnancy as a result of hyperestrinism, there is marked hyperplasia of the endocervical mucosa. This results in the downgrowth of the columnar epithelium to a variable extent beyond the external os replacing the squamous epithelium producing "pregnancy ectopy". It appears for the first time during pregnancy, circumoral in position and does not bleed to touch. The only symptom, if any, may be copious, non-irritating, mucoid discharge. **Spontaneous regression occurs usually 6–8 weeks postpartum**. Similar lesion may be observed in women taking oral contraceptives.

CERVICAL POLYP: During pregnancy, there is increased vascularity and as a result any pre-existent polyp bleeds, confusing the diagnosis with threatened abortion in early months and constitutes extra placental cause of APH in later months. The diagnosis is confirmed by speculum examination. **The polyp should be removed as in the non-pregnant state and should be sent for histological examination**.

ACQUIRED ABNORMALITY IN THE CERVIX: The cervix may be scarred following amputation during Fothergill's type of operation for prolapse, deep cauterization or diathermy. The cervix may fail to dilate during labor. Cesarean section may be needed in such a situation.

PREVIOUS HISTORY OF VAGINAL PLASTIC OPERATION: The patients with previous history of vaginal reconstructive operation should be delivered in hospital. Difficult repair for stress incontinence or vesicovaginal fistula indicates an elective cesarean section.

CONGENITAL MALFORMATION OF THE UTERUS AND VAGINA: Severe degrees of congenital malformations of the uterus usually lead to infertility; the minor degree has got little effect on obstetric performance and usually escapes attention. **It is the moderate degree of malformations that has got an adverse effect on pregnancy and labor. The**

diagnosis is made during (a) abdominal inspection — fundal notching, (b) cesarean section, (c) manual removal or evacuation operation, (d) hysterosalpingography or hysteroscopy, and (e) laparoscopy for infertility investigation. The adverse obstetric effects are: (1) Recurrent mid-trimester abortion; (2) Rupture pregnant rudimentary horn (cornual pregnancy); (3) Malpresentation — transverse, breech (common cause of recurrent malpresentations); (4) Abnormal uterine action—uterine inertia or asymmetrical uterine contractions; (5) Preterm labor; (6) Fetal growth restriction (FGR); (7) Postpartum hemorrhage; (8) Retained placenta; (9) Increased incidence of operative interference; (10) Obstructed labor by the non-pregnant horn of a bicornuate uterus.

The common types of malformations are: (a) Arcuate; (b) Subseptate; (c) Bicornuate with equal horn or unequal horn (rudimentary).

Septate vagina hardly produces any difficulty during delivery, but a transverse septum or partial atresia may necessitate delivery by cesarean section.

TORSION OF GRAVID UTERUS: Minor degrees of torsion (rotation) along the longitudinal axis is quite common as evidenced during cesarean section. But major degrees of torsion (≥ 270°) of the gravid uterus producing symptoms is extremely rare. The cases are associated with the presence of a fibroid or a bicornuate uterus. The diagnosis is confused with disturbed ectopic pregnancy in early months or abruptio placentae in later months. **The presenting features** are of acute abdomen with shock. The uterus is tense and tender. Internal examination reveals spiralling of the vagina. Sonography or MRI is helpful for correct diagnosis. The diagnosis is confirmed usually on laparotomy. Uterus must be repositioned anatomically prior to making any incision on it.

CARCINOMA CERVIX WITH PREGNANCY

INCIDENCE: The incidence of invasive carcinoma of the cervix is about 1 in 2,500 pregnancies.

DIAGNOSIS:

- ***Asymptomatic cases*** — Cytologic screening of all pregnant mothers is a routine during antenatal checkup in the organized sector. Cases showing **dyskaryotic smear** are subjected to colposcopic directed biopsy.
- ***Symptomatic cases*** — In cases of bleeding during pregnancy either in the early months simulating threatened abortion or in the later months constituting APH, the cervix should be inspected through a speculum at the earliest opportunity. If suspicion arises, **a biopsy** from the site of lesion confirms the diagnosis.

Pitfalls in diagnosis during pregnancy: Because of increased vascularity, softening of cervix and gestational hyperplasia of the cervical mucosa, **the following problems may arise in diagnosis**: (1) Indurated feel of malignancy may not be evident; (2) Benign lesions such as ectopy or polyp may bleed to touch; (3) Extension to the parametrium may not be well defined and as such, cases are often understaged.

Disease spread is better assessed by MRI. Cystoscopy and sigmoidoscopy can be done. CT scanning is avoided in pregnancy.

EFFECTS OF PREGNANCY ON CARCINOMA CERVIX: The malignant process remains unaffected. There may be a rapid spread following vaginal delivery and induced abortion.

EFFECTS OF CARCINOMA ON PREGNANCY: There is increased incidence of (1) abortion, (2) premature labor, (3) secondary cervical dystocia, (4) injury to the cervix and lower segment leading to traumatic PPH, (5) lochiometra and pyometra, and (6) uterine sepsis.

ABNORMAL CYTOLOGY IN PREGNANCY: There is intense basal cell hyperactivity of the cervical epithelium during pregnancy. This, however, reverts back to normal within 6 weeks postpartum. Abnormal cytology detected on routine screening procedure during antenatal checkup is placed under the following evaluation protocol. See Flowchart on cervical smear (p. 358).

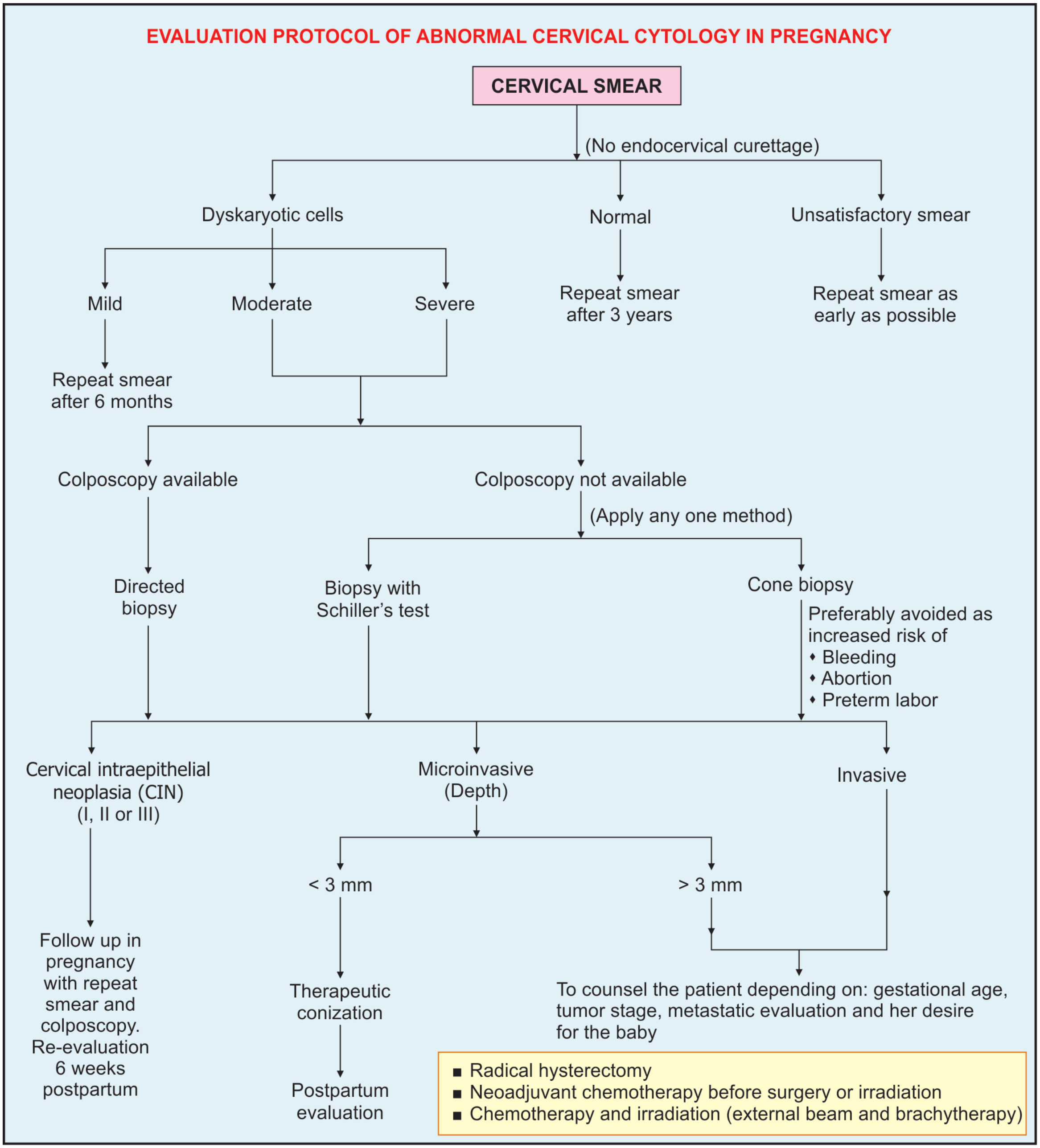
TREATMENT

CARCINOMA IN SITU: The pregnancy should be followed up as usual with periodic cytologic and colposcopic evaluation. There is no contraindication to vaginal delivery.

Microinvasive disease confirmed on cone biopsy → conservative management until delivery when the cone margins are negative → for definitive therapy → postpartum evaluation.

Early invasive (Stage IB, IIA): Treatment options depend on—gestational age, tumor stage, metastatic evaluation and maternal desire to continue pregnancy.

First trimester: Radical hysterectomy (with the fetus in uterus), pelvic lymphadenectomy and aortic node sampling are done. **Oophoropexy** at the time of hysterectomy may be done. Postoperative irradiation following evaluation of prognostic factors.


EVALUATION PROTOCOL OF ABNORMAL CERVICAL CYTOLOGY IN PREGNANCY
CERVICAL SMEAR
(No endocervical curettage)
Dyskaryotic cells
Normal
Unsatisfactory smear
Mild
Moderate
Severe
Repeat smear after 3 years
Repeat smear as early as possible
Repeat smear after 6 months
Colposcopy available
Colposcopy not available
(Apply any one method)
Directed biopsy
Biopsy with Schiller's test
Cone biopsy
Preferably avoided as increased risk of
♦ Bleeding
♦ Abortion
♦ Preterm labor
Cervical intraepithelial neoplasia (CIN) (I, II or III)
Microinvasive (Depth)
Invasive
< 3 mm
> 3 mm
Follow up in pregnancy with repeat smear and colposcopy. Re-evaluation 6 weeks postpartum
Therapeutic conization
Postpartum evaluation
To counsel the patient depending on: gestational age, tumor stage, metastatic evaluation and her desire for the baby
■ Radical hysterectomy
■ Neoadjuvant chemotherapy before surgery or irradiation
■ Chemotherapy and irradiation (external beam and brachytherapy)

Third trimester: Radical hysterectomy, pelvic lymphadenectomy after **classical cesarean delivery**. Dissection may be easy, but bleeding is often more in pregnancy.

Second trimester: Management decisions are more difficult.

Options are: (i) Neoadjuvant chemotherapy (platinum based) → continuation of pregnancy for 7–15 weeks (fetal maturation) → classical cesarean delivery and radical hysterectomy, pelvic lymphadenectomy (risk of prematurity far outweighs the risk of chemotherapy); (ii) Abdominal hysterectomy or classical cesarean section → radical surgery. Postoperative irradiation as on evaluation procedure.

ADVANCED INVASIVE DISEASE (Stage III, IV): ***First trimester***—Chemotherapy and external beam irradiation → spontaneous abortion (2–5 weeks) or uterine evacuation → brachytherapy.

Second or third trimester: Classical cesarean delivery → neoadjuvant chemotherapy and irradiation (external beam and brachytherapy).

Prognosis: Stage for the survival outcome of women with cervical cancer in pregnancy is no different when compared to the non-pregnant women.

LABOR AND DELIVERY: Vaginal delivery is avoided. This is due to the risk of disease spread following cervical injury. Moreover the risk of vaginal bleeding is significant as the tissues are friable. There may be cervical dystocia also. Delivery by classical cesarean section is commonly done. This is followed by definitive therapy.

LEIOMYOMAS WITH PREGNANCY

INCIDENCE: The incidence of fibroid in pregnancy is about 1 in 1,000 and it depends on population characteristics.

EFFECTS ON PREGNANCY: It depends on their location. (1) May be none; (2) Pressure symptoms due to impaction — (a) bladder—retention of urine and (b) rectum—constipation; (3) Abortion; (4) Malpresentation; (5) Non-engagement of the presenting part; (6) Preterm labor and prematurity; (7) Red degeneration; (8) Placental abruption.

EFFECTS ON LABOR: (1) May be unaffected; (2) Uterine inertia; (3) Dystocia due to: (a) cervical or broad ligament fibroid and (b) fibroid not pulled up above the presenting part during labor; (4) Obstructed labor; (5) Postpartum hemorrhage is due to atonicity or due to morbid adherent placenta; (6) Difficult cesarean section.

EFFECTS ON PUERPERIUM: (1) Subinvolution; (2) Sepsis is common when placenta is implanted over the myoma site which is a submucous or intramural type; (3) Secondary PPH; (4) Inversion of uterus; (5) Lochiometra and pyometra.

EFFECTS OF PREGNANCY ON FIBROID: (1) Increases in size due to increased vascularity, edema and hypertrophy and hyperplasia of the fibromuscular tissues. The tumor feels soft (2) changes in position, (3) changes in shape — becomes flattened, (4) degenerative changes, especially red degeneration, (5) torsion of pedunculated subserous fibroid, and (6) infection and polypoidal changes are more in puerperium.

RED DEGENERATION: It predominantly occurs in a large fibroid **during the second half of pregnancy or puerperium**. The cause is not known but is **actually a hemorrhagic infarction**. Infection does not play any part.

Naked eye appearance of the tumor shows dark red areas with cut section revealing raw-beef appearance often containing cystic spaces. The odor is often fishy. **The color is due to** the presence of hemolyzed red cells and hemoglobin. **Microscopically,** evidences of necrosis are present. Vessels are thrombosed, but extravasation of blood is unlikely.

Clinical features: (1) Acute onset of focal pain over the tumor; (2) Malaise or even rise of temperature; (3) Dry or furred tongue; (4) Rapid pulse; (5) Constipation; (6) Tenderness and rigidity over the tumor; (7) Blood count shows leukocytosis. **The diagnosis is confused with** acute appendicitis or twisted ovarian tumor. The diagnosis is often made only on laparotomy.

Treatment: Conservative treatment should be followed. Patient is put to bed. Ampicillin 500 mg capsule thrice daily for 7 days is given. Analgesic and sedative are frequently given. The symptoms usually clear off within 10 days. **When laparotomy is done with mistaken diagnosis, abdomen is to be closed without any intervention.** Pedunculated subserous fibroid may, however, be removed.

DIAGNOSIS: Clinically, it is difficult to diagnose a fibroid during pregnancy without fore knowledge of the existence. Marked softening and alteration in the shape (flattening) make it difficult to differentiate from the pregnant uterus. **In an uncomplicated tumor, it is confused with** ovarian tumor, retroverted gravid uterus, non-gravid half of a double uterus. **In early months, fibroid is diagnosed but pregnancy is missed whereas in later months, pregnancy is diagnosed but the fibroid is missed**. Sonography confirms the diagnosis with certainty. Color Doppler is helpful in some cases. MRI is more accurate for diagnosis and to know the dimensions, location and its relation to placental implantation.

TREATMENT

The basic principle in the management of pregnancy complicated by a fibroid is not to do anything to the fibroid whenever possible.

DURING PREGNANCY

- ***Uncomplicated*** — Usual antenatal care is followed. All cases are to be assessed at 38 weeks to formulate the method of delivery.
- ***Impaction in early months*** followed by retention of urine — The same management protocol as prescribed in retroverted gravid uterus is to be followed. **If manual correction fails**, laparotomy and myomectomy is rarely indicated leaving behind the undisturbed pregnancy.
- ***Acute pain following red degeneration*** — Medical management is usually done (mentioned earlier).

Place of elective cesarean section: (1) Cervical or broad ligament fibroid; (2) Associated complicating factors such as elderly primigravida or malpresentation.

DURING LABOR

- **Fibroid situated above the presenting part** usually results in uneventful vaginal delivery.
- **Fibroid situated below the presenting part**—spontaneous vaginal delivery may occur. If it fails, cesarean section is to be done.
- **Place of myomectomy:** Myomectomy is generally contraindicated in pregnancy. Pedunculated fibroid may be resected during cesarean delivery. Myomectomy for a case with intramural myoma during pregnancy or during cesarean delivery cause profuse hemorrhage. Few cases of successful surgery have been currently reported though.
- **One should be alert for postpartum hemorrhage and retained placenta**. The fibroid usually reverts to a smaller size during puerperium.

OVARIAN TUMOR IN PREGNANCY

INCIDENCE: The incidence of ovarian tumor with pregnancy is about 1 in 2,000. Although serous cystadenoma is common even during pregnancy **the incidence of germ cell tumor (dermoid) is increased two fold during pregnancy compared to the non-pregnant state**. This is because of its high prevalence during childbearing period and its detection due to increased complications during pregnancy. Malignant ovary is extremely rare during pregnancy.

EFFECTS OF TUMOR

- ***On pregnancy:*** **There is increased chance of** (1) impaction leading to retention of urine, (2) mechanical distress in presence of large tumor, (3) malpresentation, and (4) Non-engagement of the head at term.
- ***On labor:*** There is chance of obstructed labor if the tumor is impacted in the pelvis.

EFFECTS ON THE TUMOR: All the complications that occur in the non-pregnant state are likely to occur with increased frequency except malignancy. **(1) Ovarian masses relocate** (change their location) in the abdomen as pregnancy advances; **(2) Torsion of the pedicle** — usually occurs during 8–10 weeks of pregnancy as the tumor is out of the pelvis and in the early puerperium because of lax abdominal wall; **(3) Intracystic hemorrhage** is due to increased vascularity; **(4) Rupture** following intracystic hemorrhage or due to impaction in labor; **(5) Infection** is more common following abortion and delivery. Physiological event of thrombosis invites sepsis.

DIAGNOSIS: **Patient may remain asymptomatic or presents with the symptoms of (a) retention of urine due to impaction of the tumor, (b) mechanical distress due to the large cyst, and (c) acute abdomen due to complications of the tumor. Abdominal examination reveals the cystic swelling felt separated from the gravid uterus.** In later months of pregnancy, confusion may arise. The patient is examined vaginally in head down Trendelenburg position to elicit the groove between the two swellings, e.g. gravid uterus and the ovarian tumor **(Hingorani sign)**. **Ultrasonography** is useful to have the details of pregnancy and the ovarian tumor. MRI is very useful for some cases with more accurate information about the tumor, its nature and about the pregnancy profile.

Differential diagnosis includes pelvic kidney, uterine fibroids, colorectal or bladder tumors.

TREATMENT: **The principle** is to remove the tumor as soon as the diagnosis is made.

DURING PREGNANCY

- ***Uncomplicated*** — The best time of elective operation is between 14th week and 18th week, as the chance of abortion is less and access to the pedicle is easy. **Beyond 36 weeks** — The operation is better to be withheld till delivery and the tumor is removed as early in puerperium as possible.
- ***Complicated*** — **The tumor should be removed irrespective of the period of gestation**.

Adequate pain relief is ensured for 48 hours following surgery.

DURING LABOR: (1) **If the tumor is well above the presenting part**, a watchful expectancy hoping for vaginal delivery is followed; (2) **If the tumor is impacted in the pelvis** causing obstruction, cesarean section should be done followed by removal of the tumor in the same sitting.

DURING PUERPERIUM: On occasion, the diagnosis is made following delivery. **The tumor should be removed as early in puerperium as possible**. Following operation the specimen is sent for histological examination.

OVARIAN CANCER: Overall incidence of ovarian cancer in pregnancy is about 1 in 30,000. Most ovarian tumors in pregnancy are either germ cell tumors or epithelial cancer of early stage and low-grade variety. Treatment in majority of cases is continuation of pregnancy and preservation of fertility. When the tumor is found malignant on laparotomy, surgical intervention should be similar to that of non-pregnant patient. Preoperative tumor markers like serum CA-125, β-hCG and AFP levels increase in pregnancy. They have prognostic value to monitor the course of the disease. Pregnancy usually does not alter the prognosis of most ovarian cancers.

RETROVERTED GRAVID UTERUS

Retroverted uterus, either congenital or acquired, is considered as a normal variant of uterine position. Retroversion is either pre-existing or may be due to pregnancy. The incidence is about 10% during first trimester of pregnancy.

MORBID ANATOMIC CHANGES IF LEFT UNCARED FOR

FAVORABLE: **In the majority, spontaneous rectification occurs**. As the uterus grows, the fundus rises spontaneously from the pelvis beyond 12 weeks. Thereafter, the pregnancy continues uneventfully.

UNFAVORABLE: In the minority, spontaneous rectification fails to occur between 12 weeks and 16 weeks. The developing uterus gradually fills up the pelvic cavity and **becomes incarcerated**. **The probable causes of incarceration are**: (a) Projected sacral promontory; (b) Uterine adhesions; (c) Pelvic tumor; (d) Idiopathic (majority).

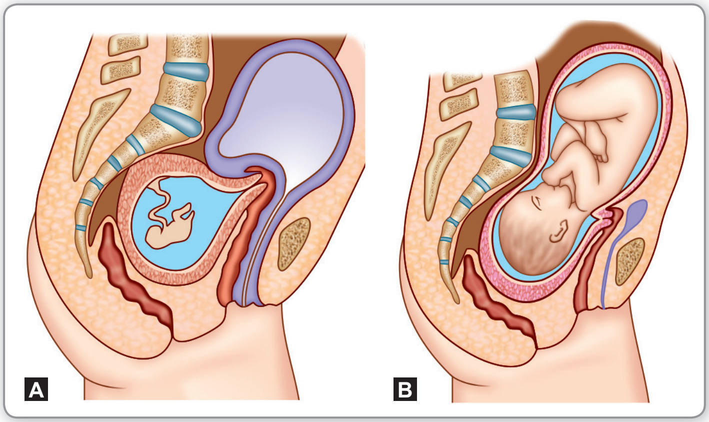
CHANGES FOLLOWING INCARCERATION

Changes in the uterus: (1) **The cervix is pointed upwards and forwards** and is placed even on the upper border of the symphysis pubis; (2) Rarely, the uterus continues to grow at the expense of the anterior wall called **anterior sacculation** while the thick posterior wall lies in the sacral hollow (Fig. 21.1).

Changes in the urethra and bladder: **Urethra:** Marked elongation along with the bladder base due to stretching of the anterior vaginal wall by the cervix. **There is retention of urine**. **The causes of retention are**: (1) Mechanical **compression of the urethra by** the cervix; (2) **Edema on the bladder neck**; (3) The woman passes small amount of urine with increased pressure (strain) even when the bladder is full (paradoxical incontinence).

Bladder changes: As a result of retention of urine, the bladder gets distended and becomes an abdominal organ reaching even upto the umbilicus. **If the retention is not relieved, the following may happen**: (1) The bladder walls become thickened due to edema; (2) Severe cystitis, pyelonephritis with uremia supervenes; (3) Intraperitoneal rupture may occur in grossly neglected cases resulting in infective peritonitis; (4) Obstructive nephropathy in a severe case may occur.

Effects on pregnancy: (1) Miscarriage; (2) If pregnancy continues with anterior sacculation, there is increased chance of (a) Malpresentation (Fig. 21.2), (b) Non-engagement of the head, (c) Preterm delivery and prematurity, and (d) Rupture of the uterus during labor.



Figs 21.1A and B: (A) Direction of the cervix following incarceration; (B) Anterior sacculation (sonography or MRI is needed to detect anatomical alterations)

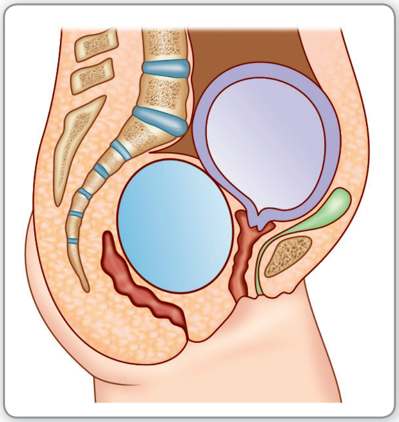


Fig. 21.2: Note the direction of the cervix—in pelvic tumor with pregnancy (compare with Fig. 21.1A)

TREATMENT

Diagnosis may be confirmed by ultrasonography or MRI.

BEFORE INCARCERATION: (1) Periodic checkup upto 12 weeks until the uterus becomes an abdominal organ; (2) She is advised to empty the bladder frequently and to lie in prone position as far as possible.

AFTER INCARCERATION: (1) To empty the bladder slowly by continuous drainage with a Foley's catheter; (2) To put the patient in bed and advise her to lie on her face or in Sims' position; (3) Urine is sent for culture and sensitivity test and urinary antiseptics—ampicillin 500 mg is given 8 hourly daily. With this simple regime, **the uterus is expected to be corrected spontaneously within 48 hours**.

If spontaneous correction fails:

- **Manual correction** by pushing the uterus digitally through the posterior fornix or through the rectum while drawing the cervix posteriorly at the same time by Allis or ring forceps is effective. The procedure should be done after putting the patient in Sims' or knee-chest position. Anesthesia may be required on occasion. **After correction, a Hodge-Smith pessary is to be inserted and kept upto 18th–20th week**.
- **In obstinate cases**, when the above method fails due to adhesions, laparotomy/laparoscopy may have to be done. Adhesiolysis is to be done for correction.
- **In diagnosed cases of anterior sacculation** of the uterus, delivery by cesarean section is the method of choice. One should be careful in delineating the anatomy to avoid the risk of inadvertent incision over the bladder, or anterior vaginal wall. Abdominal incision needs to be made above the umbilicus; uterus is to be delivered from the abdomen to restore the anatomy first and then to make the incision.

GENITAL PROLAPSE IN PREGNANCY

Pregnancy is not uncommon in first-degree uterine prolapse with cystocele and rectocele. Pregnancy is, however, unlikely when the cervix remains outside the introitus and continuation of pregnancy in third degree prolapse is an extremely rare event. The incidence of prolapse is about 1 in 250 pregnancies.

EFFECTS ON PROLAPSE: **There is aggravation of the morbid anatomical changes** in prolapse such as marked hypertrophy and edema of the cervix; first degree becomes second degree; cystocele and rectocele become pronounced and there is aggravation of stress incontinence. These are marked during early pregnancy and the effects are due to the weight of the uterus and increased vascularity (Fig. 21.3).

- **Vaginal discharge may be copious** and decubitus ulcer may develop when the cervix remains outside the introitus.
- **There is chance of incarceration**, if the uterus fails to rise above the pelvis by 16th week of pregnancy.

EFFECTS

- ***On pregnancy:*** **There is an increased chance of:** (1) Miscarriage; (2) Discomfort due to increased ailments; (3) Premature rupture of the membranes; (4) Chorioamnionitis.
- ***During labor:*** **There is an increased chance of:** (1) Early rupture of the membranes; (2) Cervical dystocia; (3) Prolonged labor due to non-dilatation of cervix and obstruction due to sagging cystocele and rectocele; (4) Operative interference.
- ***During puerperium:*** (1) Subinvolution; (2) Uterine sepsis.

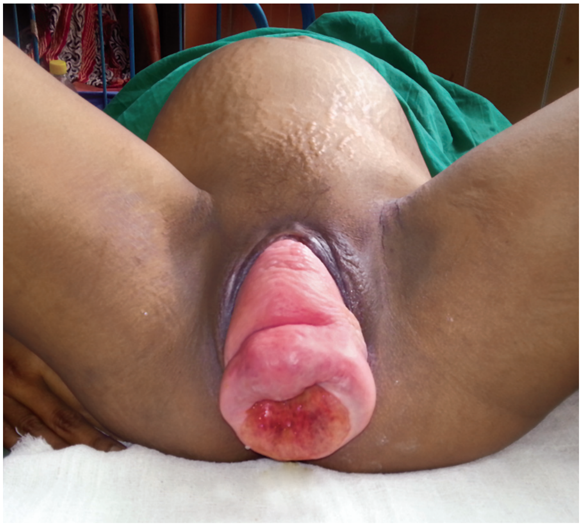


Fig. 21.3: Uterine prolapse in pregnancy with hugely edematous and hypertrophied cervix

TREATMENT

DURING PREGNANCY: The symptoms are mostly pronounced in **early pregnancy**.

- *If the cervix is outside the introitus* — The cervix is to be replaced inside the vagina and is kept in position by a ring pessary. **The pessary is to be kept until 18th–20th week of pregnancy** when the body of the uterus will be sufficiently enlarged to sit on the brim of the pelvis.

The pelvic floor is too much lax — The patient is to lie in bed with the foot end raised by about 20 cm. To relieve edema and congestion of the prolapsed mass, it should be covered by gauze soaked with glycerine and acriflavine. The treatment is continued until 18th–20th week of pregnancy till the prolapsed mass is reduced in size and replaced inside the vagina. Thereafter, the patient is allowed to walk about.

- ***If the cervix remains outside the introitus even in the later months***, it is preferable to admit the patient at 36th week.

DURING LABOR

- **The patient should be in bed**, not only to prevent early rupture of the membranes but also to facilitate replacement of the prolapsed cervix inside the vagina.
- **Intravaginal plugging soaked with glycerine and acriflavine** not only helps in reduction of cervical edema but also facilitates its dilatation.
- **Prophylactic antibiotic**, in cases of premature rupture of the membranes or when the cervix remains outside, should be administered.
- **Manual stretching of the cervix** or pushing up the cystocele or rectocele past the presenting part during uterine contractions facilitates progressive descent of the head.
- **If the head is deeply engaged with the cervix remaining thin but undilated**, delivery may be facilitated by Dührssen’s incision at 2 O’clock and 10 O’clock positions followed by ventouse extraction or forceps application.
- **If the head is high up and/or the cervix remains edematous, thick or undilated,** cesarean section is a safe procedure.

PUERPERIUM: (1) The patient should lie flat on the bed; (2) If the mass remains outside, it should be covered with gauze soaked in glycerine and acriflavine; (3) If subinvolution is evident, a ring pessary may be put in until involution is completed; (4) Prophylactic antibiotic is administered.

QUESTIONS

Write Short Notes on:

A. Mention the effects of pregnancy on fibroid? (p. 359)
B. Enumerate the complications of ovarian tumor in pregnancy? (p. 361)
C. Mention changes in the urethra and bladder due to a retroverted ground uterus? (p. 362)

Chapter 22

Preterm Labor, Preterm Rupture of the Membranes, Postmaturity, Intrauterine Fetal Death

PRETERM LABOR

(Syn: Premature Labor)

DEFINITION: Preterm labor (PTL) is defined as one where the labor starts before the 37th completed week (< 259 days), counting from the first day of the last menstrual period. The lower limit of gestation is not uniformly defined; whereas in developed countries it has been brought down to 20 weeks, in developing countries it is 28 weeks. **Preterm birth** is the significant cause of perinatal morbidity and mortality.

INCIDENCE: The prevalence widely varies and ranges between 5% and 10%.

ETIOLOGY

In about 50%, the cause of preterm labor is not known. Often it is **multifactorial**. **The following are, however, related with increased incidence of preterm labor**.

High risk factors:

(A) ***History:*** There is an increased incidence of preterm labor in cases such as: (1) Previous history of induced or spontaneous abortion or preterm delivery; (2) Pregnancy following assisted reproductive techniques (ART); (3) Asymptomatic bacteriuria or recurrent urinary tract infection; (4) Smoking habits (5) Low socioeconomic and nutritional status and (6) Maternal stress.

(B) ***Complications in present pregnancy:*** May be due to **maternal**, **fetal or placental**.

- *Maternal*: (a) **Pregnancy complications:** Preeclampsia, antepartum hemorrhage, premature rupture of the membranes, polyhydramnios; (b) **Uterine anomalies:** Cervical incompetence, malformation of uterus; (c) **Medical and surgical illness:** Acute fever, acute pyelonephritis, diarrhea, acute appendicitis, toxoplasmosis and abdominal operation. **Chronic diseases:** Hypertension, nephritis, diabetes, decompensated heart lesion, severe anemia, low body mass index (LBMI); (d) **Genital tract infection:** Bacterial vaginosis, beta-hemolytic *Streptococcus*, bacteroides, chlamydia and mycoplasma.
- *Fetal*: Multiple pregnancy, congenital malformations and intrauterine death.
- *Placental:* Infarction, thrombosis, placenta previa or abruption.

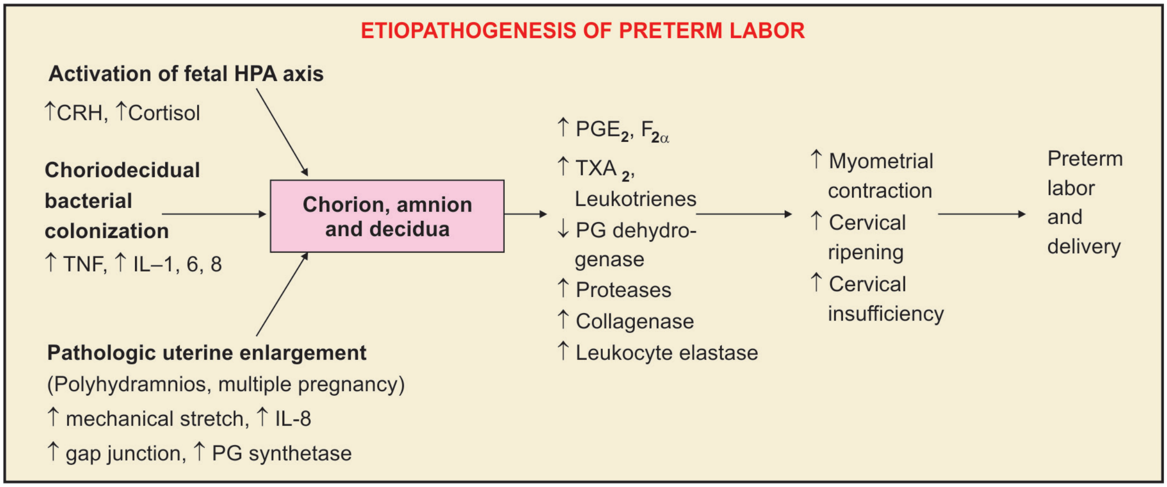
(C) ***Iatrogenic:*** Indicated preterm delivery due to medical or obstetric complications.

(D) ***Idiopathic:*** (Majority)—Premature effacement of the cervix with irritable uterus and early engagement of the head are often associated. In the absence of any complicating factors, it is presumed that there is premature activation of the same systems involved in initiating labor at term.

Etiopathogenesis of preterm labor: See below.

DIAGNOSIS: (1) **Regular uterine contractions with or without pain (at least one in every 10 minutes);** (2) **Dilatation** ($\geq$ 2 cm) and **effacement** (80%) of the cervix; (3) Length of the cervix (measured by TVS) $\leq$ 2.5 cm and funneling of the internal os (see p. 198) and (4) Pelvic pressure, backache and/or vaginal discharge or bleeding. It is better to overdiagnose preterm labor than to ignore the possibility of its presence.

Preterm labor is very unlikely when cervical length is > 30 mm, irrespective of uterine contractions.


ETIOPATHOGENESIS OF PRETERM LABOR
Activation of fetal HPA axis
↑CRH, ↑Cortisol
Choriodecidual bacterial colonization
↑ TNF, ↑ IL–1, 6, 8
Chorion, amnion and decidua
Pathologic uterine enlargement
(Polyhydramnios, multiple pregnancy)
↑ mechanical stretch, ↑ IL-8
↑ gap junction, ↑ PG synthetase
↑ PGE2, F2α
↑ TXA 2, Leukotrienes
↓ PG dehydrogenase
↑ Proteases
↑ Collagenase
↑ Leukocyte elastase
↑ Myometrial contraction
↑ Cervical ripening
↑ Cervical insufficiency
Preterm labor and delivery

MANAGEMENT OF PRETERM LABOR

The management includes: (1) To prevent preterm onset of labor, if possible; (2) To arrest preterm labor, if not contraindicated; (3) Appropriate management of labor; (4) Effective neonatal care.

Predictors of preterm labor: A. Clinical predictors: (i) History of prior preterm birth; (ii) Multiple pregnancy; (iii) Presence of genital tract infection; (iv) Symptoms of PTL (see p. 365). **B. Biophysical predictors:** (i) Uterine contractions (UC) $\geq$ 4/hr; (ii) Bishop score $\geq$ 4; (iii) Cervical length (TVS) $\leq$ 25 mm. **C. Biochemical predictors:** (i) Fetal fibronectin (fFN) in cervicovaginal discharge (see below) (ii) Others IL-6, IL-8, TNF-α.

Fibronectin is a glycoprotein that binds the fetal membranes to the decidua. Normally it is found in the cervicovaginal discharge before 22 weeks and again after 37 weeks of pregnancy. Presence of fibronectin in the cervicovaginal discharge **between 24 weeks and 34 weeks** is a predictor of preterm labor. When the test is negative it reassures that delivery will not occur within next 7 days.

Principles of Management of Women with Preterm Labor

- **Glucocorticoids** to the mother to reduce neonatal RDS, IVH, NEC, BPD and PDA (see p. 367).
- **Antenatal transfer** of the mother with fetus in utero to a tertiary center equipped with NICU (see p. 367).
- **Tocolytic drugs** (see p. 583) to the mother for a short period unless contraindicated (see p. 367).
- **Antibiotics** to prevent neonatal infection with Group B *Streptococcus* (GBS).
- **Magnesium sulfate** (neuroprotector) to the mother to reduce neonatal cerebral palsy when pregnancy is <34 weeks.
- **Careful intrapartum monitoring** (see p. 692), minimal trauma and presence of a neonatologist during delivery.
- **Vaginal delivery** is preferred, unless otherwise indicated for cesarean birth.

PREVENTION OF PRETERM LABOR

In about 50%, the cause remains unknown. Among the remaining complicated groups, decision has to be taken whether to allow the pregnancy to continue or not. The risk of delivery of a low birth weight baby has to be weighed against the risks involved to the fetus and/or to the mother in continued pregnancy. **However, the following guidelines are adopted**.

- **Primary care** is aimed to reduce the incidence of preterm labor by reducing the high-risk factors (e.g. infection, etc.).
- **Secondary care** includes screening tests for early detection and prophylactic treatment (e.g. tocolytics).
- **Tertiary care** is aimed to reduce the perinatal morbidity and mortality after the diagnosis (e.g. use of corticosteroids).

Investigations: (1) Full blood count; (2) Urine for routine analysis, culture and sensitivity; (3) Cervicovaginal swab for culture and fibronectin; (4) Ultrasonography for fetal well being, cervical length and placental localization and (5) Serum electrolytes and glucose levels when tocolytic agents are to be used (see Chapter 33).

MEASURES TO ARREST PRETERM LABOR

The scope to arrest preterm labor is limited, as majority is associated with maternal and/or fetal complicating factors where the early expulsion of the fetus may be beneficial. It is indeed unwise to attempt to arrest the onset of labor in such cases. **Thus, in only negligible proportion of cases** (about 10–20%) where the fetus is not compromised, the maternal condition remains good and membranes are intact, **the following regime may be instituted in an attempt to arrest premature labor.**

- **Bed rest**—The patient is to lie preferably in left lateral position though the benefits are doubtful.
- **Adequate hydration** is maintained. **Prophylactic antibiotic** is not routinely given. It is recommended when infection is evident or culture report suggests.
- **Prophylactic cervical cerclage** (see p. 199) for women with prior preterm birth and short cervix in the present pregnancy may be beneficial.
- **Tocolytic agents:** Various drugs nifedipine, atosiban, progesterone (micronized) have been used to inhibit uterine contractions. Drugs that can be used are described in Chapter 34. The tocolytic agents can be used as short-term (1–3 days) or long-term therapy. Tocolytics should preferably be avoided as there is no clear benefit (RCOG–2002).
- Dose schedule of $MgSO_4$ and monitoring are same as used for seizure prophylaxis of preeclampsia (4 g IV over 3–5 minutes followed by an infusion of 1 g/hr).

Short-term therapy: It is commonly employed with success. **The objectives are:** (1) **To delay** delivery for at least 48 hours for glucocorticoid therapy to the mother to enhance fetal lung maturation and (2) **In utero transfer** of the patient to a unit with an advanced neonatal intensive care unit (NICU).

Contraindications are — A. Maternal: Uncontrolled diabetes, thyrotoxicosis, severe hypertension, cardiac disease, hemorrhage in pregnancy, e.g. placenta previa or abruption. **B. Fetal**: Fetal distress, fetal death, congenital malformation and pregnancy beyond 34 weeks. **C. Others**: Rupture of membranes, chorioamnionitis and cervical dilatation more than 4 cm.

Glucocorticoid therapy: Maternal administration of glucocorticoids is advocated where the pregnancy is less than 34 weeks. This helps in fetal lung maturation so that the incidence of RDS, IVH and NEC are minimized. This is beneficial when the delivery is delayed beyond 48 hours of the first dose. Benefit persists as long as 18 days. Either betamethasone (Betnesol) 12 mg IM 24 hours apart for two doses or dexamethasone 6 mg IM every 12 hours for 4 doses is given. Betamethasone is the steroid of choice (RCOG – 2004).

Risks of antenatal corticosteroid use: (a) Premature rupture of the membranes especially with evidence of infection as the infection may flare-up; (b) Insulin-dependent diabetes mellitus where patients need insulin dose readjustment; (c) Transient reduction of fetal breathing and body movements.

MANAGEMENT IN LABOR

The principles in management of preterm labor are: (1) To prevent birth asphyxia and development of RDS; (2) To prevent birth trauma. *Duration of labor is usually short.*

First Stage	Second Stage
■ **The patient is put to bed** to prevent early rupture of the membranes	♦ **The birth should be gentle and slow** to avoid rapid compression and decompression of the head
■ **To ensure adequate fetal oxygenation** by giving oxygen to the mother by mask	♦ **Episiotomy** may be done to minimize head compression if there is perineal resistance
■ **Epidural analgesia** is of choice	♦ **Tendency to delay** is curtailed by low forceps. As such, routine forceps is not indicated
■ **Labor should be** carefully monitored preferably with continuous EFM (see p. 693)	♦ **The cord is to be clamped** immediately at birth to prevent hypervolemia and hyperbilirubinemia
■ **Cesarean delivery** is done for obstetric reasons only (hypertension, abruption or malpresentation)	♦ **To shift the baby to neonatal intensive care unit** under the care of a **neonatologist**
■ **NICU is a *sine qua non* for good outcome**	

Place of cesarean section: Routine cesarean delivery is not recommended. Preterm fetuses before 34 weeks presented by breech are generally delivered by cesarean section. Lower segment vertical or "J"-shaped incision (see p. 671) may have to be made to minimize trauma during delivery. This is due to poor formation of the lower uterine segment.

Immediate management of the preterm baby following birth—see p. 530.

PROGNOSIS: Preterm labor and delivery of a low birth weight baby results **in high perinatal mortality and morbidity**. However, with NICU, the survival rate of the baby weighing between 1,000 g and 1,500 g is more than 90%. **With the use of surfactant** (see p. 549), survival rate of infants born at 26 weeks is about 80%.

Late preterm labor—Birth of infants between 34 weeks and 36 weeks gestation. These infants do better than those infants born before 34 weeks.

KEY POINTS

- **A preterm labor** is one when labor starts at less than 259 days (< 37 completed weeks) of pregnancy (see p. 365).
- **High risk factors** for preterm labor are many (see p. 365). Etiopathology of preterm labor is complex (see p. 366). Infection, uterine enlargement and/or fetal stress can initiate the pathology.
- **Risk of preterm labor** increases as the length of the cervix decreases (< 2.5 cm, see p. 366).
- **Presence of fetal fibronectin** in the cervicovaginal discharge between 22 weeks and 34 weeks of pregnancy is a good predictor of preterm labor (see p. 366).
- **To arrest preterm labor,** tocolytics may be used for a short-term basis (see p. 367). There are several **contraindications** for the use of tocolytics (see p. 367).
- **Complications** of a preterm infant are many (see p. 529) and are inversely related to the gestational age and birth weight.
- **Management of PTL** is aimed to reduce perinatal morbidity and mortality. **The principles of management are:**
  - To transfer the mother with the fetus in utero to a hospital where special care baby unit is available.
  - To give glucocorticoid to the woman to reduce neonatal RDS and IVH (see p. 367).
  - To start antibiotic to the woman if infection is present (see p. 367).
  - To start tocolytic medications to delay delivery at least for 48 hours.

PRELABOR RUPTURE OF THE MEMBRANES (PROM)

(Syn: Premature Rupture of Membranes)

DEFINITION: Spontaneous rupture of the membranes any time beyond 28th week of pregnancy but before the onset of labor is called prelabor rupture of the membranes (PROM). When rupture of membranes occur beyond 37th week but before the onset of labor, it is called **term PROM** and when it occurs before 37 completed weeks, it is called **preterm PROM**. Rupture of membranes for > 24 hours before delivery is called **prolonged rupture of membranes**.

INCIDENCE: PROM occurs in approximately 10% of all pregnancies.

CAUSES: In majority, the causes are not known. **The possible causes are:** (1) Increased friability of the membranes; (2) Decreased tensile strength of the membranes; (3) Polyhydramnios; (4) Cervical incompetence; (5) Multiple pregnancy; (6) Infection—Chorioamnionitis, urinary tract infection and lower genital tract infection; (7) Cervical length < 2.5 cm; (8) Prior preterm labor and (9) Low BMI (< 19 kg/m^2).

DIAGNOSIS: The only subjective symptom is escape of watery discharge per vaginum either in the form of a gush or slow leak. This is often confused with **:** (a) **Hydrorrhea gravidarum**—a state where periodic watery discharge occurs probably due to excessive decidual glandular secretion; (b) **Incontinence of urine** especially in later months.

Confirmation of diagnosis: (1) **Speculum examination** is done taking aseptic precautions to inspect the liquor escaping out through the cervix; (2) **To examine the collected fluid from the posterior fornix (vaginal pool) for:** (a) **Detection of pH** by litmus or Nitrazine paper. The pH becomes 6–6.2 (Normal vaginal pH during pregnancy is 4.5–5.5 whereas that of liquor amnii is 7–7.5). Nitrazine paper turns from yellow to blue at pH > 6; (b) To note the **characteristic ferning pattern** when a smeared slide is examined under microscope; (c) Centrifuged cells stained with 0.1% **Nile blue sulfate** showing orange blue coloration of the cells (exfoliated fat containing cells from sebaceous glands of the fetus); (3) **AmniSure**—A rapid immunoassay is accurate; (4) **Ultrasonography** is to be done not only to support the diagnosis but also to assess the fetal well being. Digital vaginal examination should be avoided.

INVESTIGATIONS: (1) Full blood count; (2) C-reactive protein (CRP); (3) Urine for routine analysis and culture; (4) High vaginal swab for culture (specially for Gr. *B Streptococcus*); (5) Vaginal pool for estimation of **phosphatidyl glycerol** and L: S ratio; (6) Ultrasonography for **fetal biophysical profile** and (7) Cardiotocography for **nonstress test** (see p. 122).

DANGERS: The implications are less serious when the rupture occurs near term than earlier in pregnancy. (1) In term PROM labor starts in 80–90% of cases within 24 hours. PROM is one of the important causes of preterm labor and prematurity; (2) Chance of ascending infection is more if labor fails to start within 24 hours. Liquor gets infected **(chorioamnionitis)** and fetal infection supervenes; (3) Cord prolapse, especially when associated with malpresentation; (4) Continuous escape of liquor for long duration may lead to dry labor; (5) Placental abruption; (6) Fetal pulmonary hypoplasia, especially in preterm PROM is a real threat when associated with oligohydramnios; (7) Neonatal sepsis, RDS, IVH and NEC in preterm PROM; (8) Perinatal morbidities (cerebral palsy) are high.

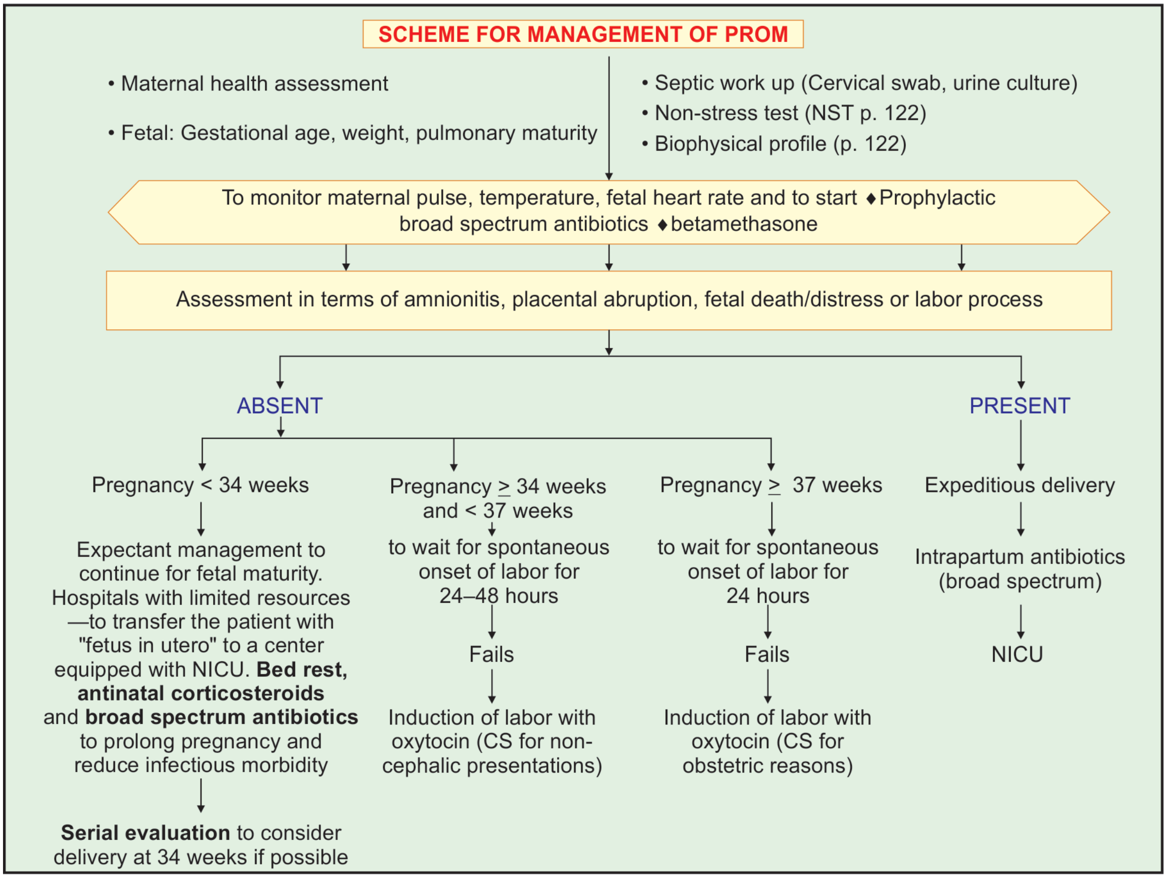
Maternal complications of PROM: Chorioamnionitis, placental abruption, retained placenta, endometritis, maternal sepsis and even death.

MANAGEMENT

PRELIMINARIES: (1) **Aseptic examination** with a sterile speculum is done not only to confirm the diagnosis but also to note the state of the cervix and to detect any cord prolapse; (2) Vaginal digital examination is generally avoided; (3) Patient is put to bed rest and sterile vulval pad is applied to observe any further leakage. Once the diagnosis is confirmed, management depends on—(i) Gestational age of the fetus; (ii) Whether the patient is in labor or not; (iii) Any evidence of sepsis and (iv) Prospect of fetal survival in that institution, if delivery occurs. Maternal pulse, temperature and fetal heart rate are monitored 4 hourly.

Term PROM: If the patient is not in labor and there is no evidence of infection or fetal distress, she is observed carefully in the hospital. Generally in 90% of cases spontaneous labor ensue within 24 hours. If labor does not start within the stipulated time or there are reasons not to wait, induction of labor with oxytocin is commenced forthwith. Cesarean section is performed with obstetric indications.

Preterm PROM: The main concern is to balance the risk of infection in expectant management (while pregnancy is continued) versus the risk of prematurity in active intervention. Ideally the patient should be transferred with the "fetus in utero" to an unit able to manage preterm neonates effectively.


SCHEME FOR MANAGEMENT OF PROM

- Maternal health assessment
- Fetal: Gestational age, weight, pulmonary maturity
- Septic work up (Cervical swab, urine culture)
- Non-stress test (NST p. 122)
- Biophysical profile (p. 122)

To monitor maternal pulse, temperature, fetal heart rate and to start ♦Prophylactic broad spectrum antibiotics ♦betamethasone

Assessment in terms of amnionitis, placental abruption, fetal death/distress or labor process

ABSENT

Pregnancy < 34 weeks → Expectant management to continue for fetal maturity. Hospitals with limited resources —to transfer the patient with "fetus in utero" to a center equipped with NICU. **Bed rest, antinatal corticosteroids** and **broad spectrum antibiotics** to prolong pregnancy and reduce infectious morbidity → **Serial evaluation** to consider delivery at 34 weeks if possible

Pregnancy ≥ 34 weeks and < 37 weeks → to wait for spontaneous onset of labor for 24–48 hours → Fails → Induction of labor with oxytocin (CS for non-cephalic presentations)

Pregnancy ≥ 37 weeks → to wait for spontaneous onset of labor for 24 hours → Fails → Induction of labor with oxytocin (CS for obstetric reasons)

PRESENT

Expeditious delivery → Intrapartum antibiotics (broad spectrum) → NICU

If the gestational age is 34 weeks or more, perinatal mortality from prematurity is less compared to infection (GBS). Labor generally starts spontaneously within 48 hours, otherwise induction with oxytocin is instituted. Presentation other than cephalic merits cesarean section. When gestational age is less than 34 weeks, conservative attitude generally followed in absence of any maternal or fetal indications. On rare occasion with bed rest, the leak seals spontaneously and pregnancy continues.

USE OF ANTIBIOTICS: Prophylactic antibiotics are given to minimize maternal and perinatal risks of infection. Intravenous ampicillin, amoxicillin or erythromycin for 48 hours followed by oral therapy for 5 days or until delivery is recommended. Pelvic rest and antibiotics help to seal the leak spontaneously and reduce infection.

Use of corticosteroids to stimulate surfactant synthesis against RDS in preterm neonates is advised. As such PROM alone may accelerate fetal lung maturation. However, combined use of antibiotics and corticosteroids (see p. 367) has reduced the risks of neonatal RDS, IVH and NEC, BPD and PDA (see p. 559).

Tocolysis, progesterone therapy, cervical cerclage in the management of PROM is not recommended.

KEY POINTS

- **PROM** is defined as the rupture of membranes any time beyond 28 weeks of pregnancy but before the onset of labor. PROM may be term or preterm (**PPROM**) when it occurs before 37 completed weeks of pregnancy.
- **Neonatal complications** after PROM is inversely related to the gestational age at the time of PROM and at delivery.
- **Fetal complications** after PROM include infection and fetal distress due to umbilical cord compression.
- **Neonatal complications** are RDS, NEC, IVH, BPD, PDH, sepsis and pulmonary hypoplasia.
- **Maternal complications** of PROM are: Chorioamnionitis, placental abruption, sepsis and maternal death.

contd...

contd...

- **Antenatal corticosteroid** therapy after PROM reduces the risk of RDS, IVH and NEC. It does not increase the risk of maternal and neonatal sepsis.
- **Antenatal antibiotic** treatment in PROM can prolong pregnancy and reduce chorioamnionitis and endometritis. It also prevents neonatal sepsis, pneumonia and IVH.
- **Major risk factors** for PTL are: (1) History of prior PTB; (2) Multiple pregnancy and (3) Vaginal bleeding after the first trimester of pregnancy.
- **A 7-day course of parenteral (first 48 hours) and oral antibiotic therapy** is given. The commonly used antibiotics are erythromycin, or amoxicillin–ampicillin.

PROLONGED AND POST-TERM PREGNANCY

(Syn: Postmaturity)

DEFINITION: Uniform criteria are lacking as to the precise definition of postmaturity. Literally, any pregnancy which has passed beyond the expected date of delivery, is called a prolonged or postdated pregnancy. **But for clinical purposes, a pregnancy continuing beyond 2 weeks of the expected date of delivery (> 294 days) is called postmaturity or post-term pregnancy**.

INCIDENCE: The incidence of pregnancies continuing beyond 42 completed weeks (> 294 days) ranges between 4% and 14%. The average is about 10%. Many suspected post-term pregnancies are actually wrongly dated. Incidence varies as different criteria are used for gestational age dating (clinical and sonography).

ETIOLOGY: So long as the complex mechanism in initiation of labor remains unknown, the cause of the prolongation of pregnancy will remain obscure. But certain factors are related with postmaturity.

(1) **Wrong dates**—due to inaccurate LMP (most common)
(2) **Biological variability (Hereditary) may be seen in the family**
(3) **Maternal factors:** Primiparity, previous prolonged pregnancy, sedentary habit, elderly multiparae
(4) **Fetal factors:** *Congenital anomalies*: Anencephaly → abnormal fetal HPA axis and adrenal hypoplasia → diminished fetal cortisol response
(5) **Placental factors:** Sulfatase deficiency → low estrogen.

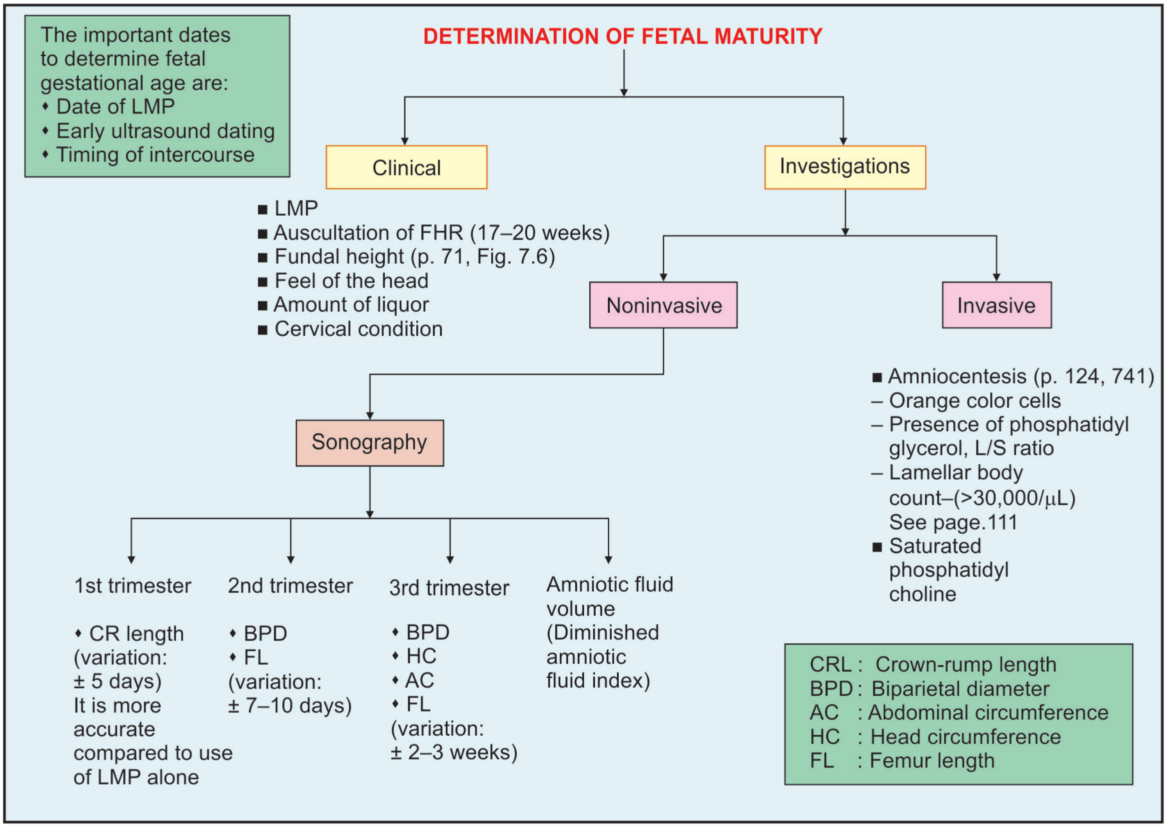
DIAGNOSIS

It is indeed difficult to diagnose postmaturity when the case is first seen beyond the expected date. The important dates to determine fetal gestational age are: (1) Date of LMP; (2) Early ultrasound dating and (3) Timing of intercourse. Every possible effort should be made with available resources to diagnose at least the maturity of the fetus, if not the postmaturity. **The following are the useful clinical guides:**

1. ***Menstrual history***—If the patient is sure about her date with previous history of regular cycles, it is a fairly reliable diagnostic aid in the calculation of the period of gestation. But in cases of mistaken maturity or pregnancy occurring during lactational amenorrhea or soon following withdrawal of the "pill", confusion arises. In such cases, the previous well-documented antenatal records of first visit in first trimester as mentioned on page 106, if available, are useful guides.

2. ***The suggested clinical findings*** when a pregnancy overruns the expected date by 2 weeks are:

- **Weight record:** Regular periodic weight checking reveals stationary or even falling weight.
- **Girth of the abdomen:** It diminishes gradually because of diminishing liquor (see p. 111).
- **History of false pain:** Appearance of false pain followed by its subsidence is suggestive.
- **Obstetric palpation:** The following findings, taken together are helpful. **These are** : height of the uterus, size of the fetus and hardness of the skull bones. As the liquor amnii diminishes, **the uterus feels "full of fetus"**— a feature usually associated with postmaturity.


The important dates to determine fetal gestational age are:
♦ Date of LMP
♦ Early ultrasound dating
♦ Timing of intercourse
DETERMINATION OF FETAL MATURITY
Clinical
Investigations
■ LMP
■ Auscultation of FHR (17–20 weeks)
■ Fundal height (p. 71, Fig. 7.6)
■ Feel of the head
■ Amount of liquor
■ Cervical condition
Noninvasive
Invasive
■ Amniocentesis (p. 124, 741)
– Orange color cells
– Presence of phosphatidyl glycerol, L/S ratio
– Lamellar body count–(>30,000/μL) See page.111
■ Saturated phosphatidyl choline
Sonography
1st trimester
♦ CR length (variation: ± 5 days) It is more accurate compared to use of LMP alone
2nd trimester
♦ BPD
♦ FL
(variation: ± 7–10 days)
3rd trimester
♦ BPD
♦ HC
♦ AC
♦ FL
(variation: ± 2–3 weeks)
Amniotic fluid volume (Diminished amniotic fluid index)
CRL : Crown-rump length
BPD : Biparietal diameter
AC : Abdominal circumference
HC : Head circumference
FL : Femur length

- **Internal examination:** While a ripe cervix is usually suggestive of fetal maturity, to find an unripe cervix does not exclude maturity. Feeling of hard skull bones either through the cervix or through the fornix usually suggests maturity.

INVESTIGATIONS: Aims are: ● **To confirm the fetal maturity** ● **To detect placental insufficiency**

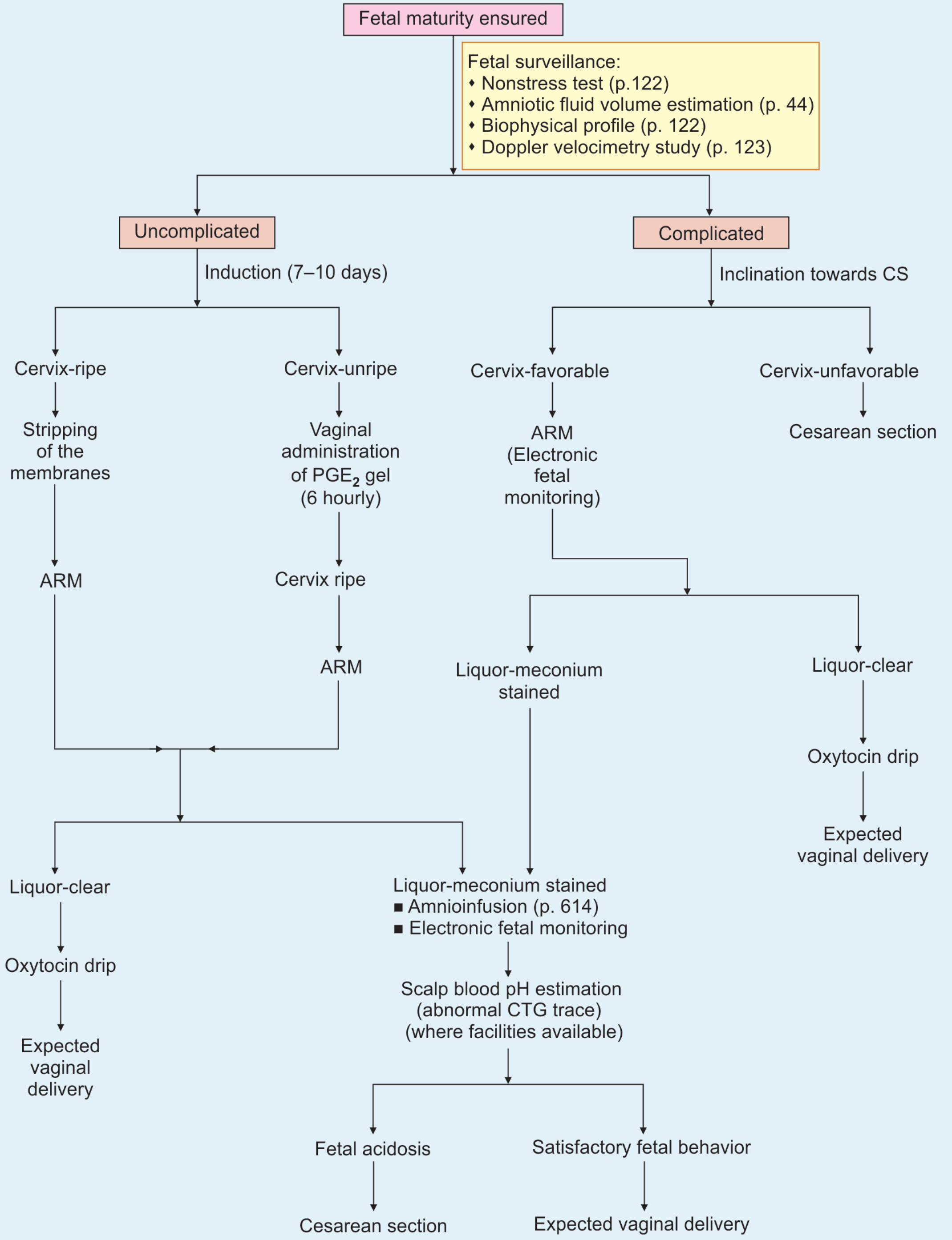
Assessment of fetal maturity:

- **Sonography:** Estimation of gestational age by early (first trimester) ultrasound is more accurate than by LMP. This is mainly due to poor recall of LMP by most patients and secondly LMP is not a good predictor of ovulation. Physiological variations in the duration of the follicular phase result in overestimation of true gestational age. Early ultrasound scan can reduce the incidence of true postmaturity (see fetal maturity flow chart above).
- **Amniocentesis:** The biochemical and cytological parameters as mentioned on page 124 are helpful. However, this invasive method has been mostly replaced by sonography.

Assesssment of fetal well being is done by twice weekly nonstress test (see p. 122), biophysical profile (see p. 122) and ultrasonographic estimation of amniotic fluid volume. Oligohydramnios has been associated with abnormal fetal CTG (see p. 123), umbilical cord compression and meconium stained liquor. **Modified biophysical profile** (NST and amniotic fluid volume) is commonly done (see p. 122). Amniotic fluid pocket < 2 cm and AFI ≤ 5 cm indicates induction of labor or delivery. Doppler velocimetry study of umbilical and middle cerebral arteries waveforms (see p. 123) are informative. Absence of umbilical artery end-diastolic velocity (see p. 124) indicates fetal jeopardy.

CLINICAL CONCEPT: The following criteria have been used to establish the diagnosis of postmaturity retrospectively, i.e. after the birth of the baby.

SCHEME FOR THE MANAGEMENT OF POSTMATURITY


Fetal maturity ensured
Fetal surveillance:
• Nonstress test (p.122)
• Amniotic fluid volume estimation (p. 44)
• Biophysical profile (p. 122)
• Doppler velocimetry study (p. 123)
Uncomplicated
Induction (7–10 days)
Cervix-ripe
Stripping of the membranes
ARM
Cervix-unripe
Vaginal administration of PGE2 gel (6 hourly)
Cervix ripe
ARM
Liquor-clear
Oxytocin drip
Expected vaginal delivery
Liquor-meconium stained
■ Amnioinfusion (p. 614)
■ Electronic fetal monitoring
Scalp blood pH estimation (abnormal CTG trace) (where facilities available)
Fetal acidosis
Cesarean section
Satisfactory fetal behavior
Expected vaginal delivery
Complicated
Inclination towards CS
Cervix-favorable
ARM (Electronic fetal monitoring)
Liquor-meconium stained
Liquor-clear
Oxytocin drip
Expected vaginal delivery
Cervix-unfavorable
Cesarean section

- ***Baby***—(1) **General appearance:** Baby looks thin and old. Skin is wrinkled. There is absence of vernix caseosa. Body and the cord are stained with greenish yellow color. Head is hard without much evidence of molding. Nails are protruding beyond the nail beds; (2) **Weight** often more than 3 kg and **length** is about 54 cm. Both are variable and even an IUGR baby may be born.
- ***Liquor amnii:*** Scanty and may be stained with meconium.
- ***Placenta:*** There is evidence of aging of the placenta manifested by excessive infarction and calcification.
- ***Cord:*** There is diminished quantity of Wharton's jelly which may precipitate cord compression.

COMPLICATIONS OF POST-TERM PREGNANCY: When pregnancy overruns the expected date, there is **risk of placental insufficiency due to placental aging**. This is manifested by placental calcification and infarction. Associated complications like hypertension and diabetes aggravates the pathology.

FETAL: *During pregnancy*—There is diminished placental function, oligohydramnios and meconium stained liquor. These lead to fetal hypoxia and fetal distress.

During labor—(1) Fetal hypoxia and acidosis; (2) Labor dysfunction; (3) Meconium aspiration; (4) Risks of cord compression due to oligohydramnios; (5) Shoulder dystocia; (6) Increased incidence of birth trauma due to big size baby and non-molding of head due to hardening of skull bones and (7) Increased incidence of operative delivery. **The main clinical significance** of post-term pregnancy is **dysmaturity or macrosomia**.

Following birth—(1) Chemical pneumonitis, atelectasis and pulmonary hypertension are due to **meconium aspiration;** (2) Hypoxia (low Apgar scores) and respiratory failure; (3) **Hypoglycemia and polycythemia** and (4) Increased NICU admissions.

Perinatal morbidity and mortality is calculated in terms of stillbirth. The risk of stillbirth is increased by about threefold from 37 weeks (0.4 per 1,000) to 43 weeks (11.5 per 1,000).

MATERNAL: There is increased morbidity, incidental to hazards of induction, instrumental and operative delivery. **Postmaturity** *per se* **does not put the mother at risk**.

MANAGEMENT

Before formulating the management, **one should be certain about the maturity of the fetus** as previously described. **Increased fetal surveillance is maintained**. Perinatal morbidity and mortality are increased when pregnancy continues beyond 41 weeks. Induction of labor may be considered at or beyond 41 weeks. Timely delivery reduces the risk of stillbirth. Increased fetal surveillance (twice weekly) is maintained (see p. 119) when conservative management is done. For the formulation of management, **the cases are grouped into**:

- **Uncomplicated**
- **Complicated**

UNCOMPLICATED:

- ***Selective induction:*** In this regime, the pregnancy may be allowed to continue till spontaneous onset of labor. Fetal surveillance is continued with **modified biophysical profile** twice a week (see p. 122).
- ***Routine induction:*** The expectant attitude is extended for 7–10 days past the expected date and thereafter labor is induced.

Induction: Induction of labor reduces the rate of cesarean delivery and perinatal mortality. *If the cervix is favorable (ripe),* induction is to be done by stripping of the membranes or by low rupture of the membranes. If the liquor is found clear, oxytocin infusion is added to be more effective. Careful fetal monitoring is mandatory. *If the cervix is unripe,* it is made favorable by vaginal administration of PGE2 gel. This is followed by low rupture of the membranes. Oxytocin infusion is added when required. Cervical length (TVS) < 25 mm is a predictor of successful induction of labor.

COMPLICATED GROUP: (Associated with complicating factors)

- ***Elective cesarean section*** is advisable when postmaturity is associated with high risk factors like: elderly primigravidae, preeclampsia, Rh-incompatibility, fetal compromise or oligohydramnios. ***Associated complications that are likely to produce placental insufficiency***—**Ideally, pregnancy should not be allowed to go past the expected date**.

CARE DURING LABOR:

Whether spontaneous or induced, **the labor is expected to be prolonged** because of a big baby and poor molding of the head. More analgesia is required for pain relief. **Possibility of shoulder dystocia is to be kept in mind**. Careful fetal monitoring with available gadgets is to be done. If fetal distress appears, prompt delivery either by cesarean section or by forceps/ventouse is to be done.

KEY POINTS

- Any pregnancy continuing beyond 2 weeks (>294 days) of the expected date of delivery is called **post-term pregnancy**.
- **Expected date of delivery (EDD)** is best assessed by LMP based ultrasonography in the first trimester rather than LMP alone.
- **Prolonged and postmature pregnancy** has got increased risk of perinatal morbidity and mortality, macrosomia and maternal morbidity.
- **Postmature pregnancy** needs careful antenatal fetal surveillance.
- **Fetal survillance** should include NST (see p. 122), amniotic fluid volume estimation (see p. 39), modified BPP (see p. 122) at least once a week.
- **Induction of labor** is done after 41 weeks if cervix is favorable.
- Either stripping of the membranes or PGE2 can be used for induction of labor in post-term pregnancy.

INTRAUTERINE FETAL DEATH (IUFD)

Literally, intrauterine fetal death (IUFD) embraces all fetal deaths weighing 500 g or more occurring both during pregnancy (antepartum death) or during labor (intrapartum). But death of a fetus weighing less than 500 g (before 22 weeks) has got a distinct etiology and is usually termed as abortion. Death during labor ends in delivery of a fresh stillborn and does not pose a problem for management. **Thus for practical purpose, antepartum death occurring beyond the period of viability is termed as intrauterine death**. It usually results in the delivery of a macerated fetus. There is a gradual decline in the incidence of IUD. Preconceptional care (see p. 116), care during pregnancy and labor, provision for prenatal diagnosis and selective termination in congenital anomalies are the possible reasons.

ETIOLOGY

The fetal deaths are related to maternal (5–10%), placental (20–35%) or fetal (25–40%) complications. Such a complication may be chronic (usual) or acute (rare) to produce placental insufficiency. However, in about 25–35% of cases the cause remains unknown (Table 22.1). Abnormal test results may not be the actual cause for IUFD.

MORBID PATHOLOGY: The dead fetus undergoes an aseptic degenerative process called maceration. The epidermis is the first structure to undergo the process, whereby **blistering and peeling off of the skin occur**. **It appears between 12 hours and 24 hours after death**. The fetus becomes swollen and looks dusky red. Gradually, aseptic autolysis of the ligamentous structure and liquefaction of the brain matter and other viscera take place. The changes vary in degree and are responsible for the characteristic radiological signs.

Table 22.1: Causes of Intrauterine Fetal Death

A. Maternal (5–10%) ■ Hypertensive disorders in pregnancy (p. 255) ■ Diabetes in pregnancy (p. 330) ■ Maternal infections (malaria, hepatitis, influenza, toxoplasma, syphilis) (p. 343) ■ Hyperpyrexia (temp > 39.4°C) ■ Antiphospholipid syndromes (APS) (p. 196) presence of lupus anticoagulant (LA), anticardiolipin antibodies (ACA) → decidual vasculopathy with fibrinoid necrosis, placental vascular atherosis and intervillous thrombosis → IUFD (p. 399) ■ Thrombophilias: Factor V Leiden, protein C, protein S-deficiency, hyperhomocysteinemia (p. 399) Mechanism of IUFD is similar to (APS) ■ Abnormal labor (p. 463) (prolonged or obstructed labor, ruptured uterus) ■ Post-term pregnancy (p. 371) ■ Systemic lupus erythematosus (p. 340)	■ Major structural anomalies (p. 567) ■ Infections (virus p. 349, bacteria p. 341, chorioamnionitis). ■ Rh-incompatibility (p. 388) ■ Non-immune hydrops (p. 571) ■ Growth restriction **C. Placental (20–35%)** ■ Antepartum hemorrhage p. 282 : Both placenta previa and abruptio placentae can cause fetal death by producing acute placental insufficiency (p. 289, 299). ■ Cord accident (prolapse, true knot, cord round the neck) p. 460 ■ Twin transfusion syndrome (TTTS) (p. 240). ■ Placental insufficiency (p. 533). **D. Iatrogenic** ■ External cephalic version (p. 663) ■ Drugs (quinine beyond therapeutic doses)
B. Fetal (25–40%) ■ Chromosomal abnormalities (p. 127)	**E. Idiopathic (25–35%)** ■ Cause remains unknown even with thorough clinical examination and investigations

DIAGNOSIS

Repeated examinations are often required to confirm the diagnosis.

SYMPTOMS—Absence of fetal movements which were previously noted by the patient.

SIGNS: Retrogression of the positive breast changes that occur during pregnancy is evident after variable period following death of the fetus.

Per abdomen

- **Gradual retrogression** of the fundal height and it becomes smaller than the period of gestation.
- **Uterine tone** is diminished and the uterus feels flaccid. Braxton-Hicks contraction is not easily felt.
- **Fetal movements** are not felt during palpation.
- **Fetal heart sound** is absent. Use of Doppler ultrasound is better than the stethoscope.
- **Cardiotocography (CTG):** Flat trace
- **Egg-shell crackling feel** of the fetal head is a late feature.

INVESTIGATIONS

- ***Sonography***—**Earliest diagnosis is possible with sonography**. The evidences are: (a) Lack of all fetal motions (including cardiac) during a 10-minute period of careful observation **with a real-time sonar is a strong presumptive evidence of fetal death** and (b) Oligohydramnios and collapsed cranial bones are evident (Fig. 22.1).

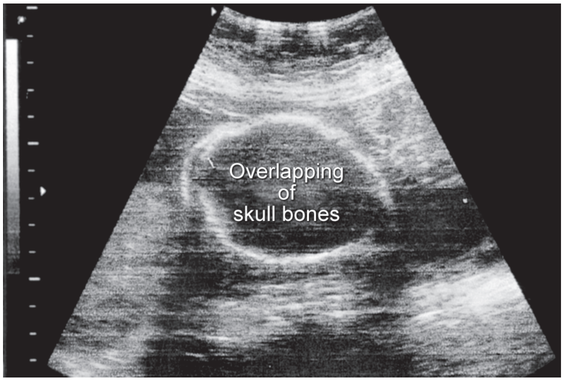


Fig. 22.1: Sonographic plate showing collapsed cranial bones—a late feature of IUD

- ***Straight X-ray abdomen***—Rarely done at present. The following features may be found, either singly or in combination.

  Spalding sign (Fig. 22.2)—The irregular overlapping of the cranial bones on one another is due to liquefaction of the brain matter and softening of the ligamentous structures supporting the vault. **It usually appears 7 days after death.**

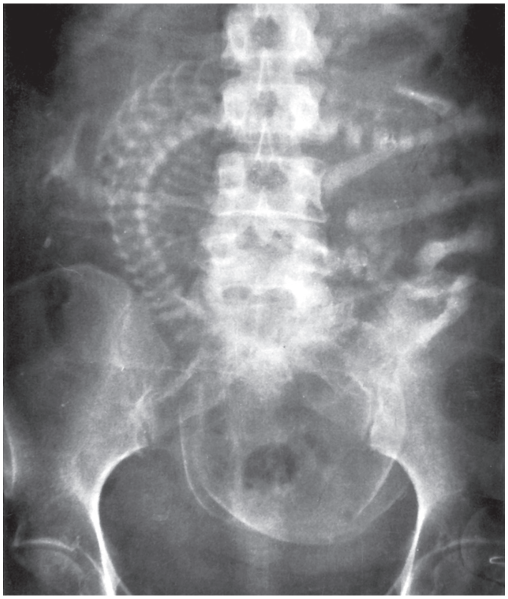


Fig. 22.2: Straight X-ray of abdomen showing single fetus with marked irregular overlapping of the skull bones (Spalding sign) and hyperflexion of the spine

- **Hyperflexion of the spine** is more common. In some cases hyperextension of the neck is seen. **Crowding of the ribs shadow** with loss of normal parallelism. **Appearance of gas shadow** (Robert's sign) in the chambers of the heart and great vessels may appear as early as 12 hours but difficult to interpret.

BLOOD—To estimate the blood fibrinogen level and partial thromboplastin time periodically, when the fetus is retained for more than 2 weeks.

RECOMMENDED EVALUATION FOR A STILLBIRTH

Hematological examination consists of ABO and Rh grouping, Kleihauer-Betke test, VDRL, postprandial blood sugar, HbA_1C, urea, creatinine estimations, thyroid profile, viral serology, lupus anticoagulant, anticardiolipin antibodies and thrombophilia studies. Urine examination for casts and pus cells. Thorough examination of the infant and placenta should be done: **Infant**—for malformations (skeletal X-ray) **umbilical cord** for entanglement, number of vessels (see p. 254), **placenta** for meconium staining, malformations (see p. 251) and the respective weights are to be recorded.

Autopsy and chromosome studies are done for fetuses with anomalies and dysmorphic features. It is also done if there is history of recurrent stillbirths or if either parent is a carrier for balanced translocation. Fetal skin, blood are usually taken for aneuploidy and single gene disorder study. For cytogenetic studies tissues must contain some viable cells.

COMPLICATIONS:

(1) **Psychological upset** often becomes a problem.

(2) **Infection**—As long as the membranes are intact, infection is unlikely but once the membranes rupture, infection, especially **by gas forming organisms like *Clostridium welchii* may occur**. The dead tissue favors their growth with disastrous consequences.

(3) **Blood coagulation disorders are rare**. If the fetus is retained for more than 4 weeks (10–20%), there is a possibility of defibrination from "silent" disseminated intravascular coagulopathy (DIC). **It is**

due to gradual absorption of thromboplastin, liberated from the dead placenta and decidua, into the maternal circulation.

(4) **During labor**—Uterine inertia, retained placenta and postpartum hemorrhage.

MANAGEMENT

PREVENTION: The overall risk of recurrence of stillbirth varies between 0% and 8%. The conditions that run the risks of recurrence are: hereditary disorders, diabetes, hypertension, thrombophilias, placental abruption and fetal congenital malformations. While IUD cannot be totally prevented, the following guidelines may help to reduce its recurrence:

- **Preconceptional counseling and care** (see p. 116) is essential to prevent its occurrence in the high-risk group.
- **Prenatal diagnosis** (see p. 127)—CVS or amniocentesis in selected cases (see p. 128).
- **To screen the "at-risk mothers"** during antenatal care. Careful **assessment of fetal well-being and to terminate pregnancy** with the earliest evidences of fetal compromise.

Breaking the bad news to the mother and the family members is a difficult task. This is mainly due to the fear of being blamed for the poor outcome and for the medical/legal problems. To listen to the patient and her family members actively and then to answer their concerns are important. It needs professional skill and abilities.

EXPECTANT ATTITUDE (NONINTERFERENCE): The patient and her relatives are likely to be upset psychologically but they should be assured of safety of noninterference. **In about 80% of cases, spontaneous expulsion occurs within 2 weeks of death**. The woman with intact membranes, no evidence of DIC or sepsis may remain at home with the advice to come to the hospital for delivery. Fibrinogen estimation should be done twice weekly.

REASONS FOR EARLY DELIVERY: (i) Reliable and early diagnosis could be made with real time ultrasonography; (ii) Prostaglandins are available for effective induction and (iii) Complications could be avoided.

INDICATIONS OF EARLY INTERFERENCE

- Psychological upset of the patient—common
- Manifestations of uterine infection
- Tendency of prolongation of pregnancy beyond 2 weeks
- Falling fibrinogen level (rare)

Methods of delivery—**The delivery should always be done by medical induction**:

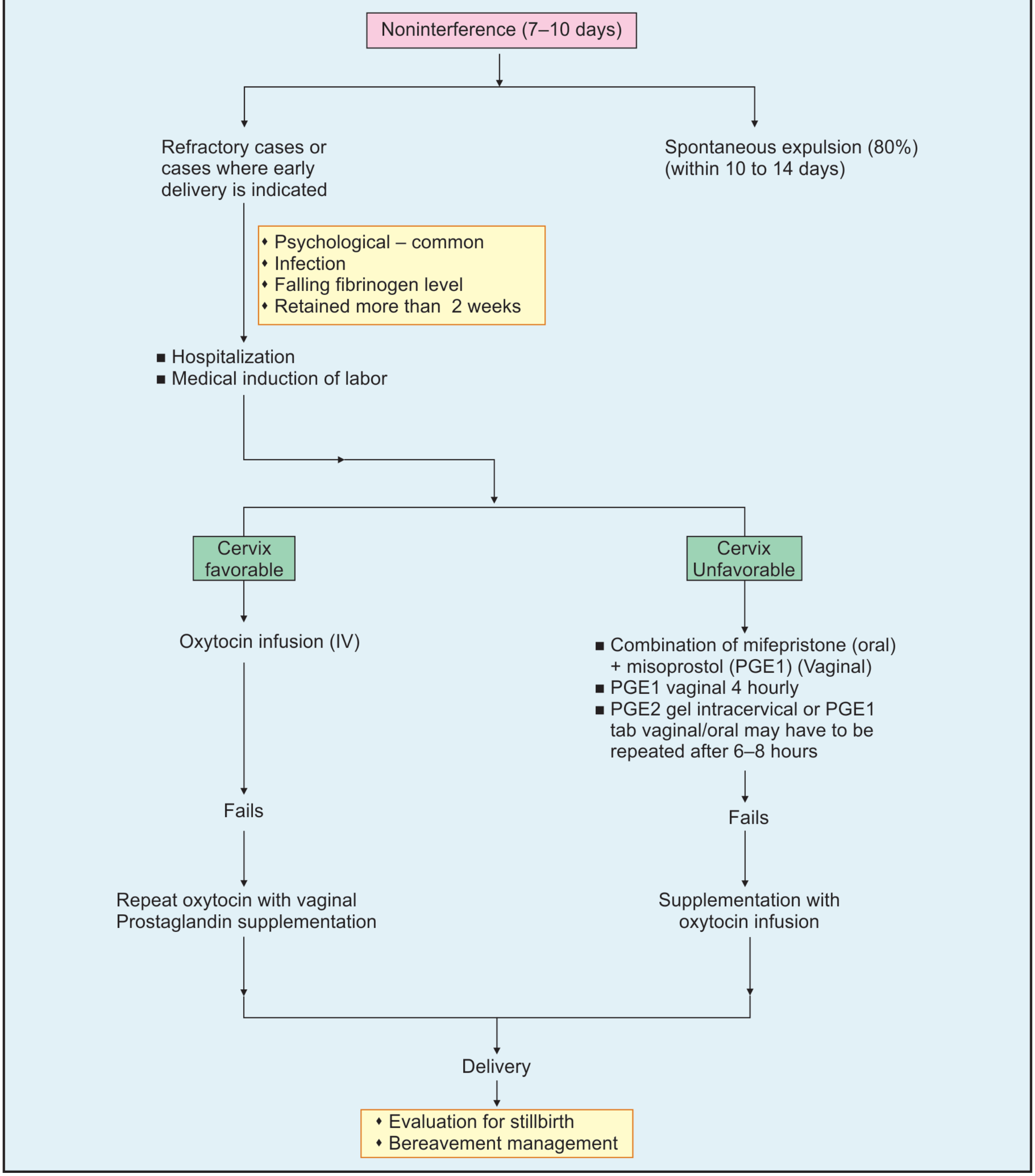
(a) A combination of mifepristone and a prostaglandin preparation is recommended **as the first-line choice for induction of labor**. A single dose (200 mg) of oral mifepristone and misoprostol (PGE1) intravaginal 25 µg 4 hourly are safe, effective and of low cost. Induction delivery interval was 8 hours. Mifepristone (600 mg daily for 2 days) alone can be used for induction also.

(b) Misoprostol (PGE1) 25–50 µg either vaginally or orally is also found effective (see p. 579). Vaginal route use is more effective compared to oral route. Misoprostol may be repeated at every 4 hours. Misoprostol is preferred to oxytocin or PGE2 as it is safe, effective and cheap.

(c) Prostaglandins (PGE2): Vaginal administration of prostaglandin (PGE2) gel or lipid pessary high in the posterior fornix is very effective for induction where the cervix is unfavorable. This may have to be repeated after 6–8 hours. The procedure may be supplemented with oxytocin infusion.

(d) Oxytocin infusion: This is widely practised and effective in case where the cervix is favorable. To begin with, 5–10 units of oxytocin in 500 mL of Ringer's solution is administered through intravenous infusion drip. In case of failure, an escalating dose of oxytocin is used on the next day. To start with, a drip is set up with 20 units of oxytocin in 500 mL of Ringer's solution and run 30 drops per minute (80 mU/minute). Oxytocin infusion may be used as a supplementary therapy when vaginal prostaglandins are used. One should **exclude the possibility of secondary abdominal pregnancy if repeated attempts fail** to start labor.

SCHEME OF MANAGEMENT OF IUFD


Noninterference (7–10 days)
Refractory cases or cases where early delivery is indicated
Spontaneous expulsion (80%) (within 10 to 14 days)
♦ Psychological – common
♦ Infection
♦ Falling fibrinogen level
♦ Retained more than 2 weeks
■ Hospitalization
■ Medical induction of labor
Cervix favorable
Cervix Unfavorable
Oxytocin infusion (IV)
■ Combination of mifepristone (oral) + misoprostol (PGE1) (Vaginal)
■ PGE1 vaginal 4 hourly
■ PGE2 gel intracervical or PGE1 tab vaginal/oral may have to be repeated after 6–8 hours
Fails
Fails
Repeat oxytocin with vaginal Prostaglandin supplementation
Supplementation with oxytocin infusion
Delivery
♦ Evaluation for stillbirth
♦ Bereavement management

Induction of labor in women with previous LSCS: PGE2 gel may be used safely in women with previous one LSCS, but for women with previous two LSCS, risk (rupture uterus) is slightly more.

Place of cesarean section in a case with IUD is limited. Previous cesarean section (two or more), major degree placenta previa and transverse lie are the rare conditions.

Postpartum suppression of lactation: Cabergoline (dopamine agonist), single dose (1 mg), is found effective. It should not be given to women with preeclampsia or hypertension.

Bereavement management and puerperium: The medical team and the nursing staff should provide all the support and sympathy to the bereaved couple. The couple should be explained in simple terms about the possible cause of fetal death. A psychologist or a counselor may see them to support. Recovery in postpartum ward is better avoided. The risk of postpartum depression is high (see p. 442). The couple is seen in the postpartum clinic after 6 weeks. The investigation reports are reviewed and counseling for future pregnancy is done (see p. 513).

KEY POINTS

- **Causes of IUFD** may be (A) Maternal (5-10%), (B) Fetal (24-40%), (C) Placental (20-35%), (D) Iatrogenic and (F) Unexplained (25–30%)
- **Earliest diagnosis** of IUFD is possible with USG. Absence of all fetal movements (including cardiac motion, using real time sonography) is a strong presumptive evidence of IUFD.
- **Maternal complications** of IUFD are: **During Pregnancy**: (a) Psychological upset, (b) Infection, (c) DIC (rare) and (d) **During labor**: Uterine inertia, PPH

Management of IUFD includes:

- **Expectant:** Waiting for spontaneous onset of labor (7–10 days) and delivery 80%, (b) **Medical method** of induction using Prostaglandin (PGE1, PGE2) with or without mifepristone or oxytocin IV (infusion).

QUESTIONS

1. Define preterm labor? How the diagnosis of preterm labor is made? Mention the important causes of preterm labor? (p. 365)
2. Outline the principles of management of a woman with preterm labor (p. 366). Mention the benefits of corticosteriod therapy in the management? (p. 367)

Related theory questions (Long & Short), Obstetric Case Discussions, Viva table discussions, Postoperative word round discussions, and MCQs are discussed in author's books:

1. **Bedside Clinic and Viva Voce:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

For further reading:

2. **Master pass in Obstetrics and Gynaecology:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

Chapter 23

Complicated Pregnancy

PREGNANCY WITH PRIOR CESAREAN DELIVERY

Pregnancy with prior cesarean delivery is quite prevalent in present day obstetric practice. This is due to liberalization of primary cesarean section with nonrecurrent indications. **These cases are loosely called "post-cesarean pregnancy"**.

EFFECTS ON PREGNANCY AND LABOR: Previous history of cesarean section does not appreciably alter the course of pregnancy and labor. However, **the following complications are likely to increase:** (1) abortion; (2) preterm labor; (3) normal pregnancy ailments; (4) operative interference and incidental morbidity; (5) placenta previa; (6) adherent placenta (placenta accreta); (7) postpartum hemorrhage; (8) peripartum hysterectomy.

EFFECTS ON THE SCAR: There is increased risk of scar rupture. Whereas the lower segment scar usually ruptures during labor that of classical or hysterotomy scar ruptures during late pregnancy and labor. **The incidence of scar rupture is about 0.2–1.5% in the former and about 4–9% in the latter**.

HEALING OF THE UTERINE WOUND: The uterine wound is healed by muscles and connective tissues, if the apposition of the margins is perfect. **The factors of prime importance in impaired healing of the uterine wound are:** (1) imperfect apposition of the cut margins; (2) presence of sepsis; (3) presence of hematoma in the wound; (4) poor general condition and (5) excessive stretching of the lower segment leading to diminished vascularity of the muscles.

SOUND SCAR: LOWER SEGMENT OR CLASSICAL?

The test of a sound scar is provided by its ability to withstand the strains of a future pregnancy and labor. **In identical conditions, the lower segment scar is more sound than the classical scar because of the following factors**.

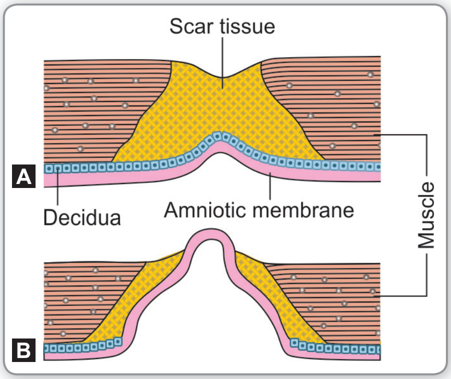
INTEGRITY OF THE SCAR

CLASSICAL OR HYSTEROTOMY SCAR: As stated above, the scar following classical section or hysterotomy is weak. The scar is more likely to give way during late pregnancy and labor with increased risks to the mother and the fetus. As such, these cases should be delivered by elective cesarean section.

Table 23.1: Differences Between Lower Segment Scar and Classical or Hysterotomy Scar

	Lower Segment Scar	**Classical or Hysterotomy Scar**
Apposition	Thin cut margins facilitate perfect apposition without leaving any pocket	Difficult to appose the thick muscle layer. Pockets are formed that contain blood, which is subsequently replaced by fibrous tissue. Formation of gutter on the inner surface is likely, as the decidua is more often than not excluded during suture (Fig. 23.1)
State of uterus during healing	The part of the uterus remains inert while healing process is going on	The part of the uterus contracts and retracts so that the sutures become loose leading to imperfect healing
Stretching effect	The scar is made to stretch during future pregnancy and normal labor more along the line of scar	The stretch is at right angles to the scar
Placental implantation in future pregnancy	Chance of weakening the scar by placental attachment is unlikely	The placenta is more likely to implant on the scar and weakens it by trophoblastic penetration or herniation of the amniotic sac through the gutter (Fig. 23.1)
Net effect	(a) Scar is sound; (b) rupture may occur only during labor (0.2–1.5%)	(a) Scar is weak; (b) rupture may occur both during pregnancy and labor (4–9%)
	Following rupture: Maternal death—less perinatal death—1 in 8	Following rupture: Maternal death—5% Perinatal death—6 in 8

LOWER SEGMENT TRANSVERSE SCAR: It usually heals better compared to the classical scar. During the course of labor the integrity of the scar needs to be assessed. It is indeed difficult to forecast precisely whether the particular scar is sound or not. High index of suspicion is essential. Factors that are to be considered while assessing scar are: evidences of **scar dehiscence during labor** (impending rupture). **Scar dehiscence** means asymptomatic separation or thinning of the scar without involving the peritoneal coat and without any hemorrhage.



Figs 23.1A and B: Herniation of the amniotic sac through the gutter of classical scar

- ***Previous operative notes:***
  - **Indication of cesarean section:** (a) **Placenta previa** makes a scar weak due to: (i) imperfect apposition due to quick surgery and (ii) thrombosis of the placental sinuses. (b) **Following prolonged labor**—increased chance of sepsis.
  - **Technical difficulty in the primary operation** leading to lateral extension or tears to involve the branches of uterine vessels or colporrhexis.
- ***Hysterography in interconceptional period:*** Hysterography, 6 months after the operation, may reveal defect on the scar (wedge depression of more than 5 mm).
- ***Pregnancy (present and past):*** (1) Pregnancy occurring soon after operation before the wound has got time for sound healing; (2) pregnancy complication such as twins or polyhydramnios puts stretching effect on the scar; (3) history of previous vaginal delivery following the operation may weaken the scar and (4) placenta previa in the present pregnancy may weaken the scar.
- ***Evaluation of uterine scar with ultrasonography:*** Assessment of thickness of the lower uterine segment is done. Risks of rupture is high (9%) when the full thickness was less than 2.3 mm.

EVIDENCES OF SCAR RUPTURE (OR SCAR DEHISCENCE) DURING LABOR

There is no single pathognomonic clinical feature that can indicate uterine scar dehiscence or rupture. Presence of any of the following features should be taken into consideration (RCOG-2007):

(1) Abnormal CTG—(abnormal FHR, bradycardia, variable and late decelerations)—most consistent finding (55–87%); (2) suprapubic pain if severe and especially persisting in between contractions; (3) shoulder tip pain or chest pain or sudden onset of shortness of breath; (4) acute onset of scar tenderness; (5) abnormal vaginal bleeding or hematuria; (6) cessation of uterine contractions which were previously adequate; (7) maternal tachycardia, hypotension or shock and (8) loss of station of the presenting part. Early diagnosis of scar dehiscence or rupture needs prompt laparotomy and resuscitation to reduce mortality and morbidity in mother and infant.

PROGNOSIS: Previous history of classical cesarean section or hysterotomy makes the woman vulnerable to unpredictable rupture of the uterus. This may occur either late during pregnancy or during labor and when it does, the maternal mortality is to the extent of 5% and the perinatal mortality to 75%.

The risk of lower segment scar-rupture is low (0.2–1.5%) and even if it does occur, maternal death is much less and the perinatal mortality is about one in eight. **The prognosis is dependent on scrupulous selection of cases as regards to the method of delivery**. Placed in ideal circumstances, the prognosis of the **vaginal birth after cesarean section (VBAC)** and that of repeat cesarean section group is almost identical so far as maternal and perinatal deaths are concerned. However, in circumstances where meticulous observation in labor is not possible, liberal repeat section is likely to offer better prognosis.

MANAGEMENT OF A PREGNANCY WITH PRIOR CESAREAN DELIVERY

The patient should be considered as "high risk" and as such regular antenatal check up is mandatory. **At each visit, enquiry is to be made about the pain or tenderness over the scar or any vaginal bleeding**.

HOSPITALIZATION: ***Elective***

- Cases with previous history of **lower segment operation should be admitted at 38th week because of**: (1) **to assess** the case and to formulate the line of treatment; (2) **to prevent** inconvenience of the patient, if the labor pain starts earlier especially where the communication to the hospital is a problem.
- Cases with previous history of **classical cesarean section or hysterotomy should be admitted at 36th week**. The chance of such type of scar rupture is more during the last few weeks of pregnancy.
- **All cases suggestive of weak lower segment scar (mentioned earlier) are also to be admitted at 36th week.**

Emergency: (1) Whenever labor starts; (2) any symptom suggestive of scar rupture such as acute pain in the abdomen, bleeding per vaginum and tenderness over the scar and (3) associated obstetrical complications.

FORMULATION OF THE METHOD OF DELIVERY—VAGINAL OR ABDOMINAL?

Previous classical cesarean section or hysterotomy: **The choice is elective, repeat section as soon as pregnancy reaches 38 weeks**.

Previous lower segment operation: The dictum which is widely accepted in the formulation of line of treatment is "mandatory hospital delivery and

Predictors for Successful VBAC-TOL

- Prior nonrecurring indication (breech, fetal distress)
- Woman having prior vaginal delivery
- Fetal birth weight (estimated)—higher the fetal weight, lower is the success
- Spontaneous onset of labor in the present pregnancy: higher success
- Cervical dilatation (on admission) > 4 cm—higher (86%) the success
- Women with prior LSCS due to breech presentation: higher success (89%)
- Women who are obese and elderly: lower success

individualization of the case". **Overall assessment of the case has to be made with due consideration to:**

- Indication of primary cesarean section: recurrent or nonrecurrent
- Integrity of the scar as evaluated clinically (p. 381) and with sonography (p. 382)
- Associated obstetric complications, if any
- Number of previous cesarean section
- Estimated weight of the baby

Risk Factors for Scar Rupture

- More the number (>2) of prior cesarean delivery
- Interpregnancy interval <24 months
- Induced labor
- Augmentation of labor [with high dose oxytocin (>20 mU/min)]
- Women having single layer uterine closure in prior cesarean delivery compared to double layer closure.

VAGINAL BIRTH AFTER PREVIOUS CESAREAN (VBAC) DELIVERY

VBAC and trial of labor after cesarean (TOLAC) is successful in 70–76% of cases (RCOG-2007). Benefits of successful VBAC are many (Table 23.3). Maternal and perinatal mortality rates following VBAC-TOL are the same when compared for elective repeat cesarean births.

In suspected maturity, it is better to wait for the pains to start or membranes to rupture, whichever occurs earlier and then to do cesarean section.

Vaginal delivery: If the previous section was done for some **nonrecurrent indication** and the uterine scar is sound (Table 23.1), a vaginal delivery is to be planned. Women need to be counseled as with the current recommendation (ACOG-2004) by the team of obstetrician, anesthetist and the neonatologist.

MANAGEMENT OF LABOR AND DELIVERY FOR VBAC-TOL

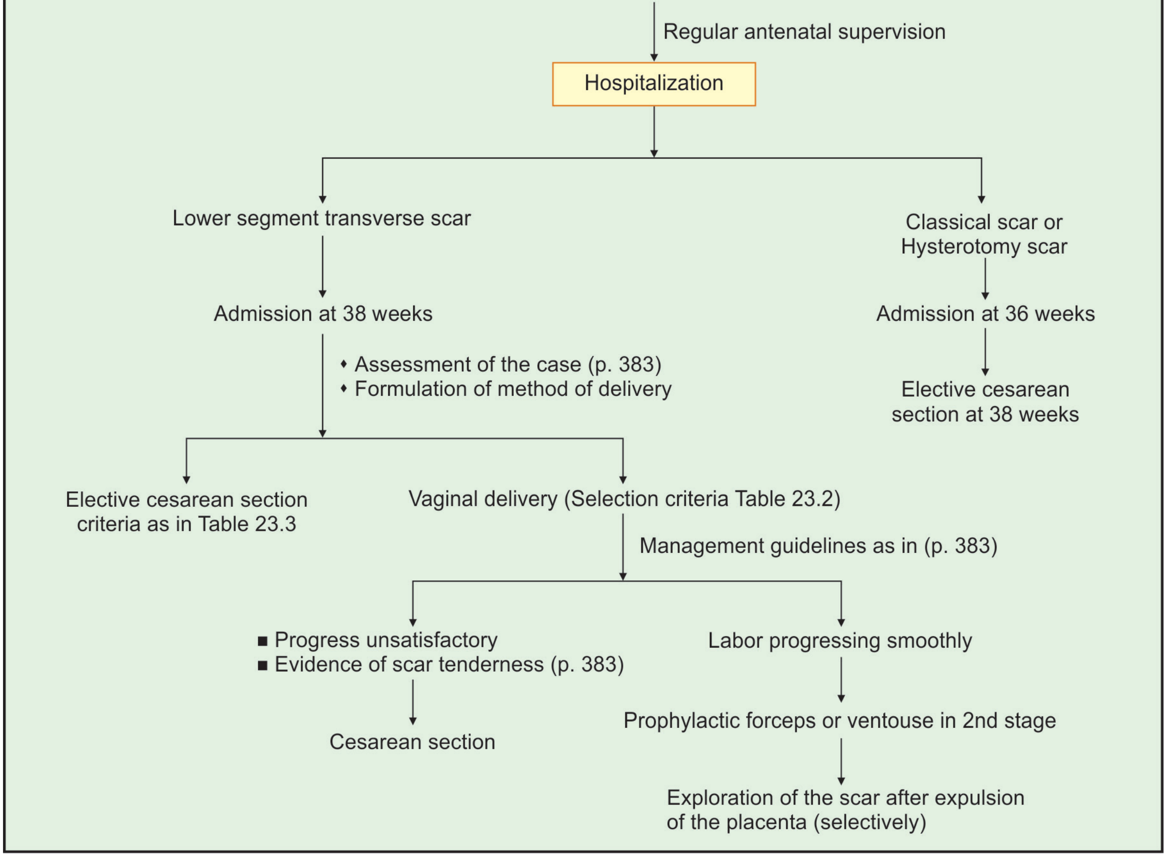
- Spontaneous onset of labor is desired. Induction of labor with prostaglandins increases the risk of uterine scar rupture.
- An intravenous line is commenced with Ringer's solution.
- Blood sample is sent for Hb%, group and cross-matching.
- Labor monitoring—clinically (for scar dehiscence) and electronically for fetal behavior. Careful serial clinical assessment is needed to ensure adequate cervicometric progress of labor.
- Analgesia—epidural is not contraindicated. It neither delays the course of labor nor delays the diagnosis of scar rupture.
- Oxytocin for augmentation of labor may be used selectively and judiciously. Augmentation of labor increases the risk of uterine scar rupture and the risk of cesarean section.
- Continuous EFM is desirable. Presence of nonreassuring pattern, severe variable decelerations, prolonged decelerations or bradycardia warns uterine rupture.
- Prophylactic forceps or ventouse to cut short the second stage is used.

Table 23.2: Selection Criteria of Cases for VBAC–TOL

- One or two previous lower segment transverse scar
- Nonrecurring indication for prior cesarean section
- Pelvis adequate for the fetus
- Continued labor monitoring possible
- Availability of resources (anesthesia, blood transfusion and theater) for emergency cesarean section within 30 minutes of decision
- Informed consent of the woman

Table 23.3: Contraindications for VBAC-TOL (Indications for Cesarean Delivery)

- Previous classical or inverted T-shaped uterine incision
- Previous two or more lower segment cesarean section
- Pelvis contracted or suspected CPD
- Presence of other complications in pregnancy: Obstetric (preeclampsia, malpresenation, placenta previa) or medical
- Resoruces limited for emergency cesarean delivery or patient refusal for VBAC-TOL
- History of prior uterine rupture



SCHEME OF MANAGEMENT OF PREGNANCY WITH PRIOR CESAREAN SECTION

Regular antenatal supervision
Hospitalization
Lower segment transverse scar
Classical scar or Hysterotomy scar
Admission at 38 weeks
Admission at 36 weeks
• Assessment of the case (p. 383)
• Formulation of method of delivery
Elective cesarean section at 38 weeks
Elective cesarean section criteria as in Table 23.3
Vaginal delivery (Selection criteria Table 23.2)
Management guidelines as in (p. 383)
■ Progress unsatisfactory
■ Evidence of scar tenderness (p. 383)
Labor progressing smoothly
Cesarean section
Prophylactic forceps or ventouse in 2nd stage
Exploration of the scar after expulsion of the placenta (selectively)

Table 23.4: VBAC-TOL: Benefits and the Risks

Benefits of VBAC-TOL	Risks of Elective Repeat Cesarean Delivery	Complications of Unsuccessful VBAC-TOL
■ Decreased maternal morbidity (infection and others) ■ Reduced length of hospital stay ■ Decreased need for blood transfusion ■ Decreased risk of abnormal placentation and need for successive cesarean delivery in next pregnancy	■ Increased maternal morbidity ■ Increased length of hospital stay ■ Increased risk of hemorrhage and need for blood transfusion ■ Increased risk of abnormal placentation (p. 36), hemorrhage and successive cesarean delivery in the next pregnancy	**Maternal** ■ Uterine wound dehiscence ■ Uterine rupture (0.5–1%) ■ Increased blood transfusion ■ Increased risks of hysterectomy due to uterine rupture ■ Infections, increased maternal morbidity **Perinatal:** Low apgar score, admission to NICU, hypoxic ischemic encephalopathy (HIE), neonatal death, rarely stillbirth

- *Routine exploration of the uterus*: Most prefer not to explore the uterine scar as a routine. It is done in selected cases only when there is continued and excessive vaginal bleeding or maternal hypotension inspite of well-contracted uterus. Others prefer to do it as a routine. Two fingers are introduced to palpate the scar internally for detection of any asymptomatic scar rupture. However, asymptomatic scar rupture or dehiscence generally heals well.

STERILIZATION: There is increasing risk after each cesarean operation. Before the third time cesarean delivery, the woman needs to be counseled and option may be obtained for sterilization operation.

KEY POINTS

PREGNANCY WITH HISTORY OF PRIOR CESAREAN SECTION

- **Complications** in pregnancy and labor are increased (p. 381). Healing of uterine wound is more perfect in lower segment cesarean section compared to the classical cesarean (Table 23.1). Incidence of lower segment scar rupture is (0.5–1%) less compared to classical scar (5–9%) (Table 23.1).

  Assessment of integrity of the uterine scar is done from history, investigation (USG) and examination (p. 381). **Diagnosis of scar rupture** (dehiscence) during labor is mainly clinical and is difficult (p. 383).
- **Woman with lower segment transverse scar** should preferably be admitted electively at 38 weeks and that with classical scar at 36 weeks of pregnancy (p. 383).
- **To formulate the method of delivery** (vaginal or abdominal) each case should be assessed on individual basis (Tables 23.1 and 23.2). Lower segment scar rupture may occur in **labor**, whereas classical scar rupture may occur **both in pregnancy and labor** (Table 23.1).
- **Management of labor** is important for successful VBAC-TOL (p. 385). Otherwise risks are high (p. 385).
- **Incidence of placenta previa** with accreta increases from 3% to 61% from previous one to four CS respectively.
- Labor should be monitored by continuous EFM. Presence of nonreassuring fetal heart pattern, significant variable deceleration or bradycardia indicates scar rupture.
- Cases that may be considered for VBAC-TOL are: (a) Nonrecurrent indication of previous CS, (b) lower segment transverse scar, (c) pelvis adequate, availability of resources in the hospital round the clock and (d) informed consent of the women (p. Table 23.2).

PREGNANCY IN A Rh-NEGATIVE WOMAN

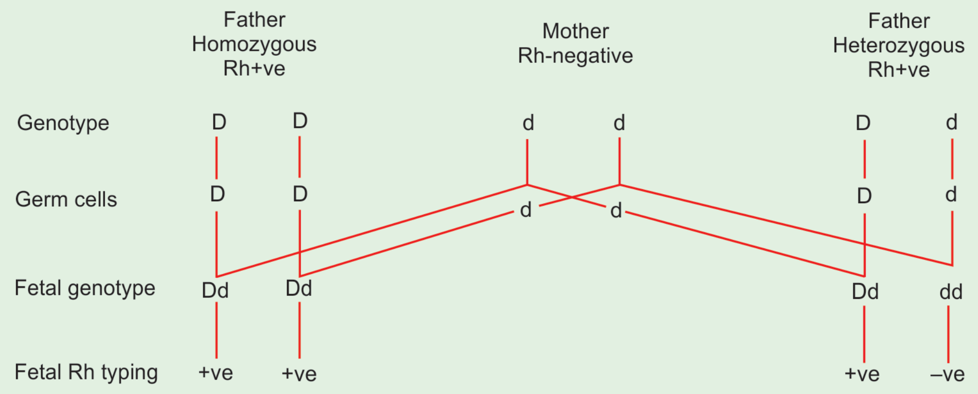
(Syn: Red Cell Alloimmunization)

NOMENCLATURE: Landsteiner and Weiner in the year 1940, discovered specific unknown antigen in the human red cells. As it was also present in the Rhesus monkeys, the antigen was named Rh. **The individual having the antigen is called Rh-positive and in whom it is not present, is called Rh-negative.**

INCIDENCE: The incidence of Rh-negative in the European and American whites, is about 15–17%; it is very much insignificant in China (1%) and almost nil in Japan. About 60% of Rh-positive men are heterozygous and 40% are homozygous at the D locus. Overall a Rh-negative woman having the chance of Rh-positive fetus is 60%, irrespective of the father's genotype. **In India, the incidence is about 5–10% (South India – 5%,** North India – 10%) in hospital statistics.

GENOTYPES: All pregnant patients should have ABO–Rh group and typing and also have serum antibody testing at the first antenatal visit. **The complete genetic make up of the Rh blood group of an individual is its genotype.** Because of lack of proper antisera against anti-e, anti-c and anti-k **the D antigen is the most potent and accounts for almost all damages (95%) due to Rh blood groups, its presence or absence denotes an individual to be Rh-positive or Rh-negative respectively.** An individual carrying D on both sets of antigens (DD) is called homozygous

Mating of Rh-positive Male with Rh-negative Female and the Resultant Possible Rh-group of the Baby



and when carrying D only (Dd) in one set, it is called heterozygous, the former constituting 65% and the latter 35%. **Heterozygous persons, are always classified as Rh-positive because D is dominant to d.** The common genotypes are CDe/cde, CDe/CDe and CDe/cDE. **In the discussion that follows, Rh-positive is taken to mean D-positive and Rh-negative to mean its absence.**

Genetic expression: The genetic locus for the Rh antigen complex is on the short arm of chromosome 1. Rh CcEe and RhD are the two distinct genes located within the Rh locus. Depending upon the presence of D antigen on one or both the chromosomes 1, it would be heterozygous or homozygous. When the normal RhD gene sequence on both the chromosomes 1 is absent, the subject is D negative.

The father's genotype may be tested, when Rh-negative wife becomes pregnant, to find out whether he is homozygous or heterozygous. This can predict whether all the subsequent babies are likely to be incompatible and liable to be affected or not. **The above scheme gives an idea** about the genetic make up of the offsprings due to mating of Rh-positive male with a Rh-negative female.

When the genotype of the father is heterozygous, half of his genes will be Rh-negative and compatible with a Rh-negative mother; the other half being Rh-positive will be incompatible, and the children will be Rh-positive (Dd) and liable to be affected. **When the father's genotype is homozygous,** all his genes will be incompatible with a Rh-negative mother and as such all the children will be Rh-positive (Dd) and may be affected by hemolytic disease.

Noninvasive fetal genotype (ffDNA) using maternal blood for D, C, c, E, e and K antigens could be done (see p. 131 Ch 12). This test is done in the first instance for the relevant antigen when maternal red cell antibodies are present.

RED CELL ALLOIMMUNIZATION

Alloimmunization (isoimmunization) is defined as a production of immune antibodies in an individual in response to foreign red cell antigen derived from another individual of the same species provided the first one lacks the antigen. It occurs in two stages: (1) sensitization and (2) immunization. This is in contrast to ABO groups, where there are naturally occurring isoimmune anti-A and anti-B antibodies.

METHODS

(1) ***Transfusion of mismatched blood:*** In ABO group incompatibility, there are naturally occurring anti-A and anti-B isoagglutinins, which result in immediate adverse reaction. In case of Rh group, there is no such naturally occurring antibody and as such there is no immediate reaction but the red cells carrying the Rh antigen sensitize the immunologically competent cells in the body, provided the amount is sufficiently large. This takes at least 1 week. Following a subsequent exposure to the antigen, the cells are stimulated to produce more specific anti-D antibody. The women may suffer a severe hemolytic reaction to the subsequent mismatched transfusion.

(2) As a result of pregnancy (**Rh-negative woman bearing a Rh-positive fetus**). Normally, the fetal red cells containing the Rh antigen rarely enter the maternal circulation. The following are the conditions where the risk chance of fetomaternal bleed is present: miscarriage (p. 188), MTP (p. 202), genetic amniocentesis (p. 130), embryoreduction, ectopic pregnancy (p. 218), hydatidiform mole (p. 227), CVS (p. 129), cordocentesis (p. 130), placenta previa with bleeding (p. 292), placental abruption (p. 294), IUFD (p. 378), external cephalic version (p. 663), abdominal trauma and the delivery of a Rh-D positive infant to a Rh negative mother. This is much more (15–50%) likely to occur during third stage of labor and following cesarean section or manual removal of placenta. However, recent studies show a continuous fetomaternal bleed occurring throughout normal pregnancies (1%).

Immunization is unlikely to occur unless at least 0.1 mL of fetal blood enters the maternal circulation. Not all "at risk" Rh-negative women become alloimmunized. About 17% of Rh-negative women will become alloimmunized by a single Rh-incompatible pregnancy. Rh-sensitization due to antepartum fetomaternal hemorrhage is about 1–2% before delivery. Fetal Rh antigens are present by 38th day after conception. Spontaneous first trimester abortion carries 3–4% risk and that of induced abortion about 5% risk of sensitization. Thus, affection of the baby due to Rh incompatibility is low considering the increased number of Rh-positive babies delivered to Rh-negative mothers. **The reasons are:**

- **Insufficient placental transfer** of fetal antigens or maternal antibodies.
- **Inborn inability to respond** to the Rh antigen stimulus.
- **Immunological nonresponder**—as found in 30% of Rh-negative women.
- **ABO incompatibility** has a protective effect against the development of Rh sensitization. This protective effect is significant when the mother is type O and the father is type A, B or AB. The reasons are—(i) ABO incompatible fetal cells are cleared from the maternal circulation rapidly before they are trapped by the spleen (ii) maternal anti-A or anti-B antibodies damage the Rh-antigen so that it is no longer immunogenic.

- **Variable antigenic stimulus** of the D antigen which depends on the Rh genotype of the fetal blood, e.g. CDe/cde genotype.
- **Volume of fetal blood entering** into the maternal circulation (0.1 mL is considered as critical sensitizing volume).

MECHANISM OF ANTIBODY FORMATION IN THE MOTHER: If the ABO compatible (mother and fetus have the same ABO group or when the fetus is group "O"), Rh-positive fetal red cells enter the mother's blood, they remain in the circulation for their remaining lifespan. Thereafter, they are removed from the circulation by the reticuloendothelial tissues and are broken down with liberation of the antigen. The antibody production is related not only to the responsiveness of the reticuloendothelial system but also to the amount of Rh antigen liberated, therefore to the number of red cells that have entered the maternal blood. Because this takes a long time, immunization in a first pregnancy is unlikely. **Detectable antibodies usually develop after 6 months following larger volume of fetomaternal bleed.** But, if the fetomaternal bleed is less than 0.1 mL, the antibody may not be detected until boosted by further Rh stimulus. **Antibodies once formed remain throughout life**.

TYPES OF ANTIBODIES—*Two types of antibodies are formed:*

(1) **IgM**—This type of antibody is the first to appear in the maternal circulation and agglutinates red cells containing D when suspended in saline. **IgM being larger molecules cannot pass through the placental barrier and is not harmful to the fetus**.

(2) **IgG—**It is also called incomplete or blocking antibody. It will agglutinate the red cells containing D only when suspended in 20% albumin. **Because of its small molecule, it can cross the placental barrier and cause damage to the fetus**. It appears at a later period than does the IgM antibody. It is important to recognize the preponderance of one or the other type of antibody than the actual level of the titer.

FETAL AFFECTION BY THE Rh ANTIBODY

The antibody formed in the maternal system (IgG) crosses the placental barrier and enters into the fetal circulation. **The antibody will not have any effect on Rh-negative fetus**. If the fetus is Rh-positive, the antibody becomes attached to the antigen sites on the surface of the fetal erythrocytes. The affected cells are rapidly removed from the circulation by the reticuloendothelial system. **Depending upon the degree of agglutination and destruction of the fetal red cells, various types of fetal hemolytic diseases appear**. These are loosely termed as erythroblastosis fetalis, since many babies may have a large number of nucleated cells in the peripheral blood as a result of compensatory erythropoiesis in response to anemia due to any cause other than Rh factor.

MANIFESTATIONS OF THE HEMOLYTIC DISEASE OF THE FETUS AND NEWBORN (HDFN)

Clinical manifestations of the hemolytic disease of the fetus and newborn are:

- **Hydrops fetalis**
- **Icterus gravis neonatorum**
- **Congenital anemia of the newborn**

HYDROPS FETALIS: This is the most serious form of Rh hemolytic disease (HDFN). Excessive destruction of the fetal red cells leads to **severe anemia**, **tissue anoxemia** and **metabolic acidosis**. These have got adverse effects on the fetal heart and brain and on the placenta. **Hyperplasia of the placental tissue** occurs in an effort to increase the transfer of oxygen but the available fetal red cells (oxygen carrying cells) are progressively diminished due to hemolysis. As a result of fetal anoxemia, there is damage to the liver leading to **hypoproteinemia** which is responsible for generalized edema **(hydrops fetalis)** (Fig. 23.2), ascites and hydrothorax. **Fetal death occurs** sooner or later due to cardiac failure. The baby is either stillborn or macerated and even if born alive, dies soon after. Other nonimmune causes of hydrops fetalis are discussed in p. 571.

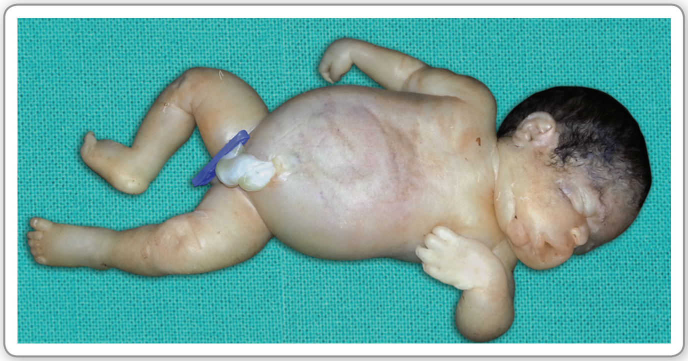


Fig. 23.2: Hydrops fetalis – stillborn edematous infant due to Rhesus alloimmunization (HDFN)

The following are the diagnostic features: (1) mother is Rh-negative; (2) serological examination reveals presence of Rh-antibody; (3) there may be presence of polyhydramnios; (4) previous history of affection of a baby due to hemolytic disease; (5) **sonography**—(real time combined with pulse Doppler) to detect edema in the skin, scalp and pleural or pericardial effusion and echogenic bowel; (6) straight X-ray abdomen showing—**"Buddha" position** of the fetus with a halo around the head due to edematous scalp; (7) the baby at birth looks pale and edematous with an enlarged abdomen due to ascites. There is enlargement of liver and spleen and (8) **placenta** is large, pale and edematous with fluid oozing from it. The placental weight may be increased to about half or even almost equal to the fetal weight. There is undue persistence of Langhans layer with marked swelling of the villi. If the fetus is not hydropic, the placenta usually looks normal.

ICTERUS GRAVIS NEONATORUM: This clinical entity is the effect of lesser form of HDFN. **The baby is born alive without evidences of jaundice but soon develops it within 24 hours of birth**.

While the fetus is in-utero, there is destruction of fetal red cells with liberation of unconjugated bilirubin which is mostly excreted through the placenta into the maternal system. A portion of the bilirubin enters the amniotic fluid perhaps from the fetal lung or through the skin or across the surface of the placenta or cord. This is the reason why baby is not born with jaundice. **But as soon as the umbilical cord is clamped**, with continuing hemolysis, the bilirubin concentration is increased. Sooner or later the baby becomes jaundiced. The liver particularly of a premature baby fails to conjugate the excessive amount of bilirubin to make it soluble and nontoxic.

If the bilirubin rises to the critical level of 20 mg per 100 mL (340 µmol/L—normal 30 µmol/L), the bilirubin crosses the blood-brain barrier to damage the basal nuclei of the brain permanently producing the clinical manifestation of kernicterus.

CONGENITAL ANEMIA OF THE NEWBORN: This is the mildest form of the disease where hemolysis is going on slowly. Although the anemia develops slowly within first few weeks of life, the jaundice is not usually evident. The destruction of the red cells continues up to 6 weeks after which the antibodies are not available for hemolysis. The liver and spleen are enlarged, the sites of extramedullary erythropoiesis.

Affection of the mother: The impact of Rh incompatibility mainly falls on the baby. The mother may also be affected somewhat. There is increased incidence of: (1) preeclampsia; (2) polyhydramnios; (3) big size baby with its hazards; (4) hypofibrinogenemia due to prolonged retention of dead fetus in uterus; (5) postpartum hemorrhage due to big placenta and blood coagulopathy; (6) "maternal syndrome"—the salient features are generalized edema, proteinuria and pruritus due to cholestasis. These features are ominous indicating imminent fetal death in utero.

PREVENTION OF Rh-IMMUNIZATION

- **To prevent active immunization**
- **To avoid mismatched transfusion**
- **To prevent or minimize fetomaternal bleed**

TO PREVENT ACTIVE IMMUNIZATION: To prevent active immunization of Rh-negative yet unimmunized, **Rh anti-D immunoglobulin (IgG) is administered intramuscularly to the mother following childbirth**. The other conditions that the Rh anti-D immunoglobulin should be given are mentioned before (p. 387).

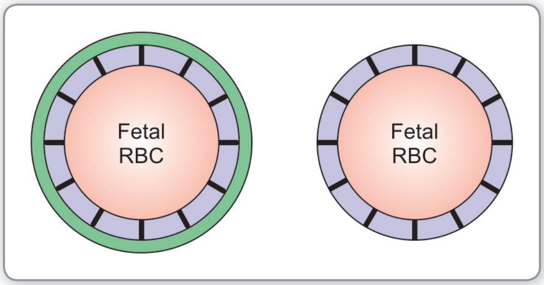


Fig. 23.3: Anti-D immunoglobulin injected into the mother surrounds the fetal RBC (thick green circle) preventing the antigen sites (spikes) to form antibody

Mode of action is antibody-mediated immune suppression (AMIS). The possible mechanisms are: (i) the anti-D antibody when injected, blocks the Rh-antigen of the fetal cells (Fig. 23.3); (ii) the intact antibody coated fetal red cells are removed from the maternal circulation by the spleen or lymph nodes; (iii) central inhibition—the fetal red cells, coated with anti D antibodies interfere the production of IgG from the B cells.

When to administer? **It should be administered within 72 hours or preferably earlier** following delivery or abortion. It should be given provided the baby born is Rh-positive and the direct Coombs' test is negative. In case, where the specified time limit is over (>72 hours), she may be given up to 14–28 days after delivery to avoid sensitization. Similarly, when the Rh factor of the fetus cannot be determined, it should be administered without any harm.

Dose: Anti D-gammaglobulin is administered intramuscularly **to the mother** 300 μg following delivery. All Rh-negative unsensitized women should receive 50 μg of Rh-immune globulin IM within 72 hours of induced or spontaneous abortion, ectopic or molar pregnancy or CVS in the first trimester. Women with pregnancy beyond 12 weeks should have full dose of 300 μg. Generally 300 μg dose will protect a woman from fetal hemorrhage of upto 30 mL of fetal whole blood.

Calculation of the dose: Approximate volume of fetal blood entering into the maternal circulation is to be estimated by "**Kleihauer-Betke test**" using acid elution technique to note the number of fetal red cells (dark, refractile bodies) per 50 low power fields (Fig. 23.6). **If there are 80 fetal erythrocytes in 50 low power fields in maternal peripheral blood films, it represents a transplacental hemorrhage to the extent of 4 mL of fetal blood**. More accurate tests are immunofluorescence and flow cytometry. If the volume of fetomaternal hemorrhage is greater than 30 mL whole blood, the dose of Rh-immune globulin calculated is **10 μg for every 1 mL of fetal whole blood**.

DURING PREGNANCY: In spite of postpartum Rh immune globulin prophylaxis, failure rate is about 1–2%. This is due to antepartum fetomaternal hemorrhage and sensitization (1–2%). If the woman is Rh-negative and has no antibody, she should have one dose of 300 μg Rh immune globulin as prophylaxis at around 28 weeks (ACOG–1999) and again after birth (within 72 hours).

TO PREVENT OR MINIMIZE FETOMATERNAL BLEED:

- **Precautions during cesarean section**: (i) to prevent blood spilling into the peritoneal cavity (ii) manual removal of placenta should not be done as a routine.
- **Prophylactic ergometrine** with the delivery of the anterior shoulder should preferably be withheld, as it may facilitate more fetoplacental bleed.
- **Amniocentesis** should be done after sonographic localization of the placenta to prevent its injury.
- **Forcible attempt** to perform external version under anesthesia should be avoided.
- **Manual removal of placenta** should be done gently.
- **To refrain from abdominal palpation** as far as possible in abruptio placentae.

TO AVOID **giving Rh-positive blood to one Rh-negative female from her birth to the menopause**.

All such women including women with multiple pregnancy may need more than usual 300 μg of anti-D immunoglobulin.

ANTENATAL INVESTIGATION PROTOCOL OF Rh-NEGATIVE MOTHERS

Investigation of blood for Rh and ABO grouping becomes almost a routine during the first antenatal visit in first trimester. **If the woman is found Rh-negative, Rh grouping of the husband is to be done** to find out whether the pregnancy is a result of incompatible or compatible mating. If the husband is also Rh-negative, i.e. compatible mating, there is no problem so far as Rh factor is concerned. But **if the husband is found to be Rh-positive, further investigations are to be carried out which aim at:**

(i) **To detect** whether the woman has already been immunized to Rh antigen; (ii) **To forecast** the likely affection of the baby; (iii) **To anticipate and formulate** the line of management of a likely affected baby (see p. 394-95).

OBSTETRIC HISTORY: (a) **If the woman is a primigravida** with no previous history of blood transfusion, it is quite unlikely that the baby will be affected; (b) **In a parous woman**, a detailed obstetric history has to be taken. **The classic history of fetal affection** in the form of stillbirth or neonatal death due to severe jaundice following one or two uneventful births is quite suggestive. **History of prophylactic administration of anti-D immunoglobulin** following abortion or delivery should be enquired (see also key points p. 401).

ANTIBODY DETECTION: In all cases of Rh-negative women irrespective of blood grouping and parity, **IgG antibody is detected by indirect Coombs' test.**

- *If the test is found negative at 12th week,* it is to be repeated at 28th and 36th week in primigravida. In multigravida, the test is to be repeated at monthly intervals upto 24 weeks and at every 2 weeks thereafter.
- *If the test is found positive*: The patient should be supervised in centers equipped to tackle with Rh problem (specialized fetal medicine unit).

— **Genotype of the husband** is to be determined. If he is found to be **homozygous**, the fetus is likely to be affected and in **heterozygous**, the fetus may be affected in 50% cases. In that case fetal blood group is determined (see below). **If the fetus is found to be Rh(D) negative, no further tests are required and routine care is continued.**

— **Fetal Rh status:** Amniotic fluid or chorionic villi (CVS) (p. 129) → uncultured amniocytes or trophoblasts → PCR for free-fetal DNA testing (ffDNA) to detect fetal blood group. **Free fetal DNA present in maternal plasma can be genotyped (p. 131). This method has replaced amniocentesis which is an invasive method.**

— **Quantitative estimation of IgG antibody** at weekly intervals. Sudden marked rise in the titer from 1 : 8 to 1 : 256 is very much suggestive of fetal affection. **Some centers consider the titer of 1: 16 or antibody level more than 10 IU/mL as a critical one.** Critical titer means anti-D antibody level that causes hydrops fetalis. One should make a cautious interpretation of the rise or fall in the titer.

Automated measurement of antibody (specific anti-D) is a more accurate test. The safe level of antibody in the maternal serum is < 4 IU/mL. Anti-D level > 4 IU/mL but <15 IU/mL has moderate risk for HDFN. Anti-D level >15 IU/mL can cause severe HDFN. Those with levels of < 4 IU/mL should have antibody measurement monthly. Those with levels > 10 IU/mL should have amniotic fluid optical density measurements at 450 nm wavelength (OD 450). Levels >10 IU/mL should also have weekly ultrasound assessment to detect fetal hydrops. Antibody levels do not always correlate with fetal affection.

Doppler ultrasound: Serial Doppler study of middle cerebral artery (MCA)-peak systolic velocity (PSV) is the mainstay to assess fetal anemia. **A value >1.5 multiples of the median (MOMs) for the corresponding gestational age, predicts moderate to severe fetal anemia.** This value (between 24 weeks and 35 weeks of gestation), is an indication for cordocentesis and fetal transfusion (see p. 395). Most centers have replaced serial amniocentesis with serial MCA Doppler studies.

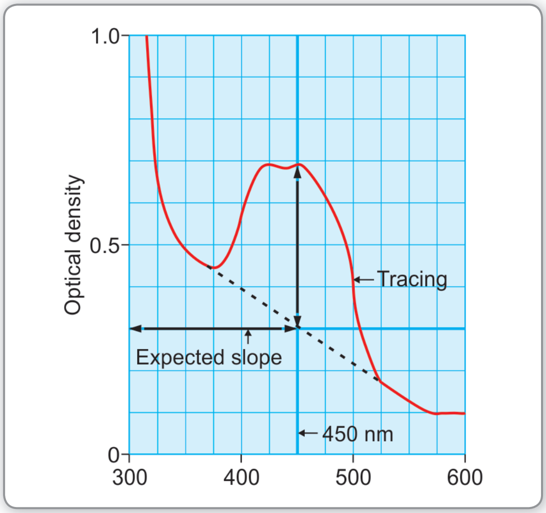


Fig. 23.4: Spectrophotometric analysis of amniotic fluid showing optical density difference at 450 nm wavelength with "deviation bulge" in Rh hemolytic disease

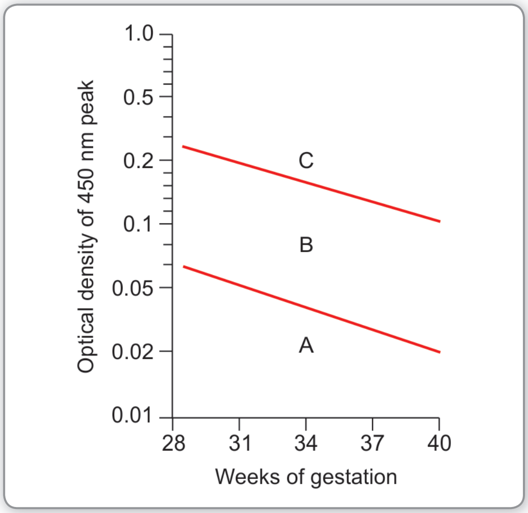


Fig. 23.5: Plotting of the "deviation bulge" in Liley's prediction chart at different periods of gestation

AMNIOCENTESIS: Amniocentesis and estimation of bilirubin in the amniotic fluid by spectrophotometry **are indicated in:** (1) **antibody titer rises** more than 1: 8 to determine whether the particular baby will be affected or not; (2) **previous history** of severely affected baby and (3) **father is heterozygous** to determine whether the particular baby will be affected or not. As such, if Rh antibodies are found in the current pregnancy, it is an essential procedure to guide the management.

Selection of time: (1) **No history of previously affected baby—**It is done at 30–32 weeks and a second test should be repeated after 3–4 weeks; (2) **positive history of previously affected baby—**It should be done at least 10 weeks prior to the date of previous stillbirth or other hemolytic manifestations on the baby. However, it is useless to perform prior to 20 weeks.

Inference: The optical density of the liquor containing the bilirubin pigment, is observed at 250–700 nm wavelength. **The optical density difference at 450 nm wavelength gives the prediction of the severity of fetal hemolysis**. In presence of bilirubin, there is a "deviation bulge" peaking at 450 nm wavelength (Fig. 23.4). The bigger the deviation bulge, the more severe is the affection of the baby. For any given period of gestation, the height of the spectrophotometric "deviation bulge" at ΔOD_{450} falls within one of the three zones when plotted in **Liley's chart** (Fig. 23.5).

Predictions:

- **Liley's zone I (low zone):** The fetus is unlikely to be affected and the pregnancy can be continued to term.
- **Liley's zone II (mid zone):** Repeat amniocentesis by 2 weeks → value upward → cordocentesis → hematocrit < 30% → intrauterine transfusion to raise hematocrit 40–45%. Preterm delivery may be needed after 34 weeks.
- **Liley's zone III (high zone):** The fetus is severely affected and death is imminent. Pregnancy > 34 weeks → delivery. Pregnancy <34 weeks → cordocentesis → hematocrit < 30% → intrauterine transfusion to raise hematocrit 40–45%. Preterm delivery may be needed after 34 weeks.

Advantages:

- **Spectrophotometric analysis** when plotted in relation to the Liley's zone can predict with fair degree of accuracy, the degree of hemolytic process in the fetus. This can give indications when to terminate the pregnancy and when to give intrauterine fetal transfusion.

- **Assessment of fetal anemia** is more accurate by doing fetal blood sampling (FBS). Ultrasound guided cordocentesis (p. 133) is done. FBS is done selectively in cases when MCA PSV is >1.5 MOM. Cordocentesis helps to detect fetal blood grouping, hematocrit, DCT, reticulocyte count and total bilirubin level. Fetal hematocrit value <15% is associated with hydrops.

Methods of antenatal assessment of fetal well-being: (1) **Serial ultrasonography** may detect fetal hydrops and anemia. The important features are: polyhydramnios, placental thickness >4 cm, pericardial or pleural effusion, echogenic bowel, dilatation of cardiac chambers and enlargement of spleen and liver; (2) **Cardiotocography:** Sinusoidal and decelerative pattern are observed (see ch. 39) in an affected fetus. (3) **Doppler flow velocity waveforms** in the umbilical artery, ductus venosus, **middle cerebral artery** have been used to detect fetal anemia and acidosis (see p. 127, 696) and (4) **cordocentesis** is done **when there is elevated ΔOD_{450} or elevated peak systolic MCA Doppler velocities (>1.5 MOM)**.

MCA PSV >1.5 multiples of the median (MoMs) for corresponding gestational age predicts moderate to severe fetal anemia.

Serial Dopper studies of MCA are the mainstay to assess fetal anemia in red cell alloimmunized pregnancy. This noninvasive test to detect fetal anemia has replaced serial amniocentesis for ΔOD_{450}.

PLAN OF DELIVERY

♦ **Unimmunized mothers** ♦ **Immunized mothers**

UNIMMUNIZED MOTHERS: In cases where there is no detectable antibody found during pregnancy, an expectant attitude is followed till term. **Tendency of pregnancy to overrun the expected date should not be allowed**.

IMMUNIZED MOTHERS: As mentioned previously, whenever there is evidence of hemolytic process in the fetus in utero, the patient should be shifted to an equipped center specialized to deal with Rh problems. **An intensive neonatal care unit, arrangements for exchange transfusion and an expert neonatologist are the basic requirements to tackle the affected babies**.

Delivery is to be done in all cases of immunized mothers with evidences of fetal hemolysis in utero. **The following factors are to be considered as to when to terminate the pregnancy:** (i) Previous history of stillbirth with father being homozygous; (ii) sudden rise in maternal antibody titer; (iii) the optical density difference at 450 nm wavelength as plotted on Liley's chart and (iv) Doppler and ultrasound features of fetal affection (see above).

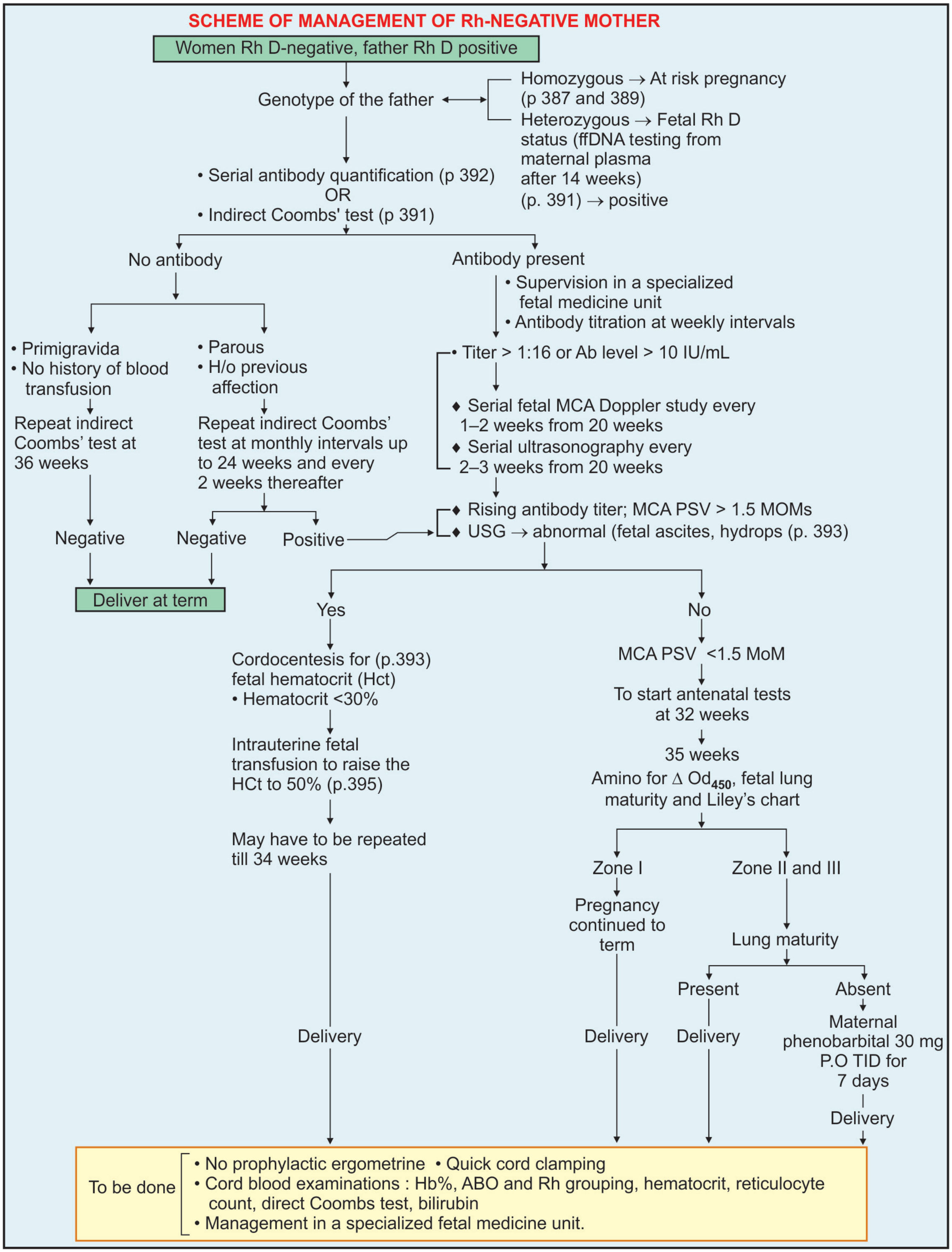
In mild affection, the pregnancy may be continued up to 38 weeks and then termination is to be done.

In severe affection: It is reasonable to terminate the pregnancy around 34 weeks after maternal steroid administration (p. 367). In every case of premature termination before 34 weeks, it is desirable to confirm the fetal lung maturation by measuring the L : S ratio in the amniotic fluid. In a specialized center where there is severe affection before 34 weeks, intrauterine fetal transfusion (intraperitoneal or intravascular) is done (p. 395) to continue pregnancy beyond 34 weeks (see below).

Methods of delivery: (1) **Amniotomy** (low rupture of the membranes) is quite effective, if termination is done near term. Vaginal prostaglandin gel (PGE_2) could be used to make the cervix ripe.

(2) **Cesarean section**: In cases when termination has to be done prematurely (say 34–37 weeks), the cervix will be unfavorable and considering the severity of affection and urgency of termination, cesarean section is a safe procedure.

CARE DURING DELIVERY: **Vaginal delivery:** (i) Careful fetal monitoring is to be done to detect at the earliest, evidences of distress; (ii) prophylactic ergometrine during second stage should be withheld; (iii) gentle handling of the uterus in the third stage and (iv) to take care of postpartum hemorrhage.


SCHEME OF MANAGEMENT OF Rh-NEGATIVE MOTHER
Women Rh D-negative, father Rh D positive
Genotype of the father
Homozygous → At risk pregnancy (p 387 and 389)
Heterozygous → Fetal Rh D status (ffDNA testing from maternal plasma after 14 weeks) (p. 391) → positive
• Serial antibody quantification (p 392)
OR
• Indirect Coombs' test (p 391)
No antibody
• Primigravida
• No history of blood transfusion
Repeat indirect Coombs' test at 36 weeks
Negative
• Parous
• H/o previous affection
Repeat indirect Coombs' test at monthly intervals up to 24 weeks and every 2 weeks thereafter
Negative
Positive
Deliver at term
Antibody present
• Supervision in a specialized fetal medicine unit
• Antibody titration at weekly intervals
• Titer > 1:16 or Ab level > 10 IU/mL
♦ Serial fetal MCA Doppler study every 1–2 weeks from 20 weeks
♦ Serial ultrasonography every 2–3 weeks from 20 weeks
♦ Rising antibody titer; MCA PSV > 1.5 MOMs
♦ USG → abnormal (fetal ascites, hydrops (p. 393)
Yes
Cordocentesis for (p.393) fetal hematocrit (Hct)
• Hematocrit <30%
Intrauterine fetal transfusion to raise the HCt to 50% (p.395)
May have to be repeated till 34 weeks
Delivery
No
MCA PSV <1.5 MoM
To start antenatal tests at 32 weeks
35 weeks
Amino for Δ Od450, fetal lung maturity and Liley's chart
Zone I
Pregnancy continued to term
Delivery
Zone II and III
Lung maturity
Present
Delivery
Absent
Maternal phenobarbital 30 mg P.O TID for 7 days
Delivery
To be done
• No prophylactic ergometrine • Quick cord clamping
• Cord blood examinations : Hb%, ABO and Rh grouping, hematocrit, reticulocyte count, direct Coombs test, bilirubin
• Management in a specialized fetal medicine unit.

Cesarean section: (i) To avoid spillage of blood into the peritoneal cavity and (ii) routine manual removal of placenta should be withheld.

Clamping the umbilical cord: In either methods, the cord is to be clamped as quickly as possible to minimize even minute amount of antibody to cross to the fetus from the mother. The cord should be kept long (15–20 cm) for exchange transfusion, if required.

Collection of cord blood for investigation: Cord blood sample is to be taken from the placental end of the cut cord. The cord should not be squeezed to prevent contamination with Wharton's jelly. About 5 mL of blood (2 mL oxalated and 3 mL clotted) should be collected for the following tests:

Clotted blood:	ABO and Rh grouping, reticulocyte count, direct Coombs' test and serum bilirubin.
Oxalated blood:	Hemoglobin estimation and blood smear for presence of immature RBC.

INTRAUTERINE FETAL TRANSFUSION (IFT): Significant advancement has been made in diagnosis (cordocentesis, real time ultrasonography) and therapy of rhesus isoimmunization. This can be carried out in a regional specialized center. It is indicated in **selected cases where there is severe affection of the fetus in utero prior to 34 weeks**. **The advantages are:** (i) Correction of fetal anemia and improvement of oxygenation and (ii) improved fetal hepatic function.

A. Intraperitoneal transfusion (IPT): *Principle:* Blood is transfused in the fetal peritoneal cavity under ultrasound guidance. Fetal anemia is corrected when the transfused erythrocytes are taken up by the sub-diaphragmatic lymphatics. It can be started at 18 weeks and repeated at intervals of 1–3 weeks up to 32–34 weeks. Pregnancy can thereafter be terminated. **Type and amount of blood—blood group "O", Rh-negative packed cells (hematocrit 80%)** cross matched with the mother, **are to be transfused. The quantity of blood** is to be calculated as number of weeks of gestation over 20 multiplied by 10 in mL, **e.g. at 32 weeks**, the amount of blood to be transfused is 12 × 10 = 120 mL. In case of repetition, at least a week gap is to be given. ***Procedure:*** The blood is to be infused slowly (5–10 mL/min) through a polythene tube that has been threaded, through an introducing needle inserted into the fetal abdomen under ultrasonic guidance. **Overall neonatal survival is approximately 90–100% for nonhydropic fetuses and about 50–70% for hydropic fetuses.**

B. Intravascular transfusion (IVT): There is poor correlation between amniotic fluid OD 450 values and ultrasound findings. Severity of fetal affection is best assessed by fetal hemoglobin and hematocrit levels as determined by cordocentesis. Generally a fetus whose hemoglobin deficit is 2 g/dL or more from the mean of a normal fetus of corresponding gestational age (hematocrit < 30%) should be transfused. Hydropic changes are observed when fetal hematocrit is less than 15%.

Procedure: Transfusion is generally made through umbilical cord vessel (vein) near its insertion into the placenta under real time ultrasound. It may be done into the intrahepatic portion of the fetal umbilical vein also. ***Type and amount of blood transfused:*** Blood group "O" Rh negative, CMV negative packed cells (hematocrit 90%) compatible with the mother's blood are transfused. Hematocrit level is checked at intervals during the procedure to determine the volume. Goal is to achieve a hematocrit of 50%. Repeat transfusion is given after 2 weeks. Fetal injury, volume overload, preterm labor and fetomaternal hemorrhage are the common complications. **Procedure-related perinatal loss** is 4.7%. **Fetal surveillance** with ultrasound and continuous electronic fetal monitoring is performed at the posttransfusion phase. Betamethasone (24 mg in three divided doses) should be administered to the mother 24 hours before transfusion from 26 weeks onwards to enhance pulmonary maturity, in case delivery becomes necessary during transfusion. Overall fetal survival rate is 90%.

OTHER THERAPIES: ♦ **Plasmapheresis** has been tried to remove several liters of maternal plasma with maternal **anti-D antibodies**. IVIG is then given. Due to its lack of definite benefit, it is not commonly done.

♦ **High-dose intravenous immunoglobulin (IVIG)** is thought to block placental transport of (FC mediated) antibody or to destroy anti D coated erythrocytes in fetal spleen and liver. A dose of 1,000 mg/kg IVIG weekly has been used.

♦ Delivery at around 34–36 weeks leads to neonatal survival to about 100% with low long-term morbidity.

♦ Artificial insemination with Rh-negative donor semen, surrogate pregnancy or PGD (when father is heterozygous) may be the option.

♦ To neutralize the established anti-D antibodies by developing antibody to the RhD antigen and monoclonal anti-D blocking antibodies are being investigated currently.

EXCHANGE TRANSFUSION IN THE NEWBORN

Exchange transfusion is a life-saving procedure in severely affected hemolytic disease of the newborn (HDFN). With the advent of wider use of prophylactic anti-D immunoglobulin, less and less problem babies are born and through exchange transfusion, the incidence of kernicterus has also been reduced.

INDICATIONS: Rh-positive with direct Coombs' test positive babies having:

- Cord blood bilirubin level more than 4 mg/dL and hemoglobin level is less than 11 g/dL
- Rising rate of bilirubin is over 1 mg/dL/hour despite phototherapy
- Total bilirubin level 20 mg/dL or more.

OBJECTIVES:

- **To stop hemolysis**, and bilirubin production
- **To correct anemia** and to improve congestive cardiac failure of the neonate
- **To remove** the circulatory antibodies
- **To remove** sensitized RBCs
- **To eliminate** the circulatory bilirubin
- To stop hemolysis and bilirubin production

While about 80–90% of the fetal blood is exchanged during the procedure, **transfusion of Rh-negative blood cannot alter the Rh-factor of the baby's blood**. The replacement temporarily helps to tide over the crisis from anemia and hyperbilirubinemia for about 2 weeks. Thereafter, the baby is quite capable to get rid of the maternal antibodies by producing sufficiently his own Rh-positive blood.

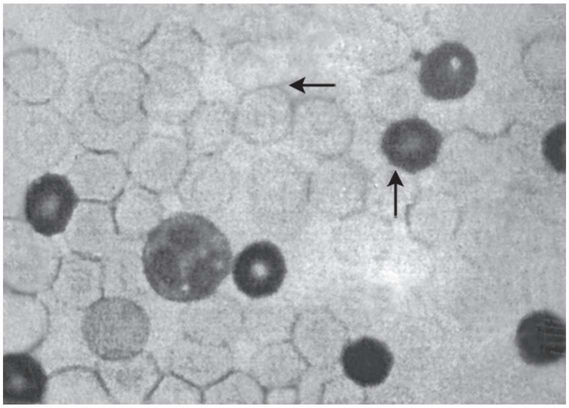


Fig. 23.6: Peripheral smear showing dark fetal red cells and light-colored maternal cells. (Modified Kleihauer and Betke acid elution test)

NATURE AND AMOUNT OF BLOOD TRANSFUSED

— **Blood for exchange should be Rh-negative whole blood with the same blood ABO grouping to that of the baby, otherwise group "O".** The blood should be cross matched with the mother's serum or with the infant's serum.

— **The blood should be collected relatively fresh** (< 7 days old).

— **The amount** is about 160 mL/kg body weight of the baby.

PROCEDURE (Fig. 23.7)

— **The procedure** is best to be carried out under a servo control radiant warmer.

— **The route of transfusion** should preferably be through the umbilical vein. A plastic catheter of 1 mm diameter is passed about 7 cm beyond the umbilicus so as to place it in the inferior vena cava. **In late transfusion,** femoral

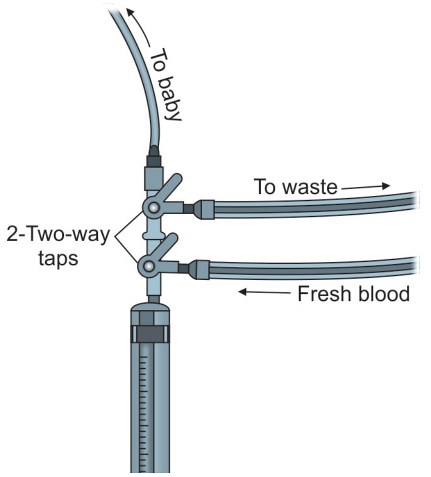


Fig. 23.7: Techniques of exchange transfusion

route through saphenous vein is the choice. **Entire set should be air tight** and to be periodically flushed with heparinized saline (1,000 units in 100 mL) to prevent clotting.

- **Blood** should be warmed to 37°C.
- **15 mL of fetal blood is withdrawn first** followed by 10 mL to be pushed in-return slowly (**push-pull method**), taking at least 1 minute time.
- **For every 100 mL of blood transfused,** one milliequivalent of sodium bicarbonate is given to combat metabolic acidosis and 1 mL of 10% calcium gluconate to prevent tetany due to transfusion of citrated blood.
- **To estimate the hemoglobin and bilirubin** concentration prior to and after the exchange transfusion.
- **The procedure should be supervised** by an expert team work.

POSTTRANSFUSION CARE: (1) **The baby is placed** under a radiant warmer; (2) **the umbilicus is to be inspected** frequently for any evidence of bleeding; (3) **serum bilirubin** is to be estimated 4 hours after transfusion and to be repeated as required. Occasionally, the level of conjugated bilirubin may remain higher and phototherapy should be continued and (4) **hypoglycemia** (due to increased insulin secretion) is to be checked by blood glucose estimation posttransfusion 4 hourly.

INDICATIONS OF REPEAT EXCHANGE TRANSFUSION: (1) Bilirubin level again rising to near the critical level of 20 mg% and (2) hemoglobin level again falls to less than 11 g%.

DANGERS: With considerable technical skill, the risks of exchange transfusion are reduced significantly.

Immediate complications: (1) Cardiac failure due to raised venous pressure and overloading of the heart; (2) air embolism; (3) clotting and massive embolism; (4) hyperkalemia; (5) tetany; (6) acidosis; (7) sepsis; (8) hypocalcemia; (9) hypoglycemia and (10) coagulopathies due to thrombocytopenia.

Delayed complications: (1) Necrotizing enterocolitis; (2) extrahepatic portal hypertension due to thrombosis of portal vein and (3) other complications are mostly attributed to prematurity, hyperbilirubinemia and hypoxia.

ADJUVANT THERAPY:

(1) ***Phototherapy:*** Phototherapy is to be continued for 24 hours. Phototherapy **(blue or blue green light of 420–470 nm wavelength)** degrades bilirubin by photooxidation and structural isomerization (lumibilirubin). Bilirubin is converted to less toxic polar isomer. These products are water soluble and therefore readily excreted in the bile and urine. **Ultraviolet light should be screened out and the baby's eyes should be protected by dark glasses**.

(2) ***Photochemical*** reactions convert bilirubin to less toxic and water soluble polar isomer, or to lumirubin.

(3) ***Phenobarbitone,*** 3–5 mg/kg body weight is to be administered thrice daily intramuscularly. Phenobarbitone increases the glucuronyl transferase enzyme activity in the fetal and neonatal liver to conjugate the bilirubin which hastens its clearance.

(4) ***Antibiotics*** should be administered for 3–5 days.

PROGNOSIS

So long as the RhD-negative mother remains unimmunized against RhD-antigen, the fetus is unaffected with any hemolytic process. **With alloimmunization of the mother, the prognosis of the baby depends on**: (1) Genotype of the father; (2) genotype of the fetus; (3) maternal antibody level; (4) history of previous affection of the baby due to hemolytic disease and (5) availability of sophisticated diagnostic and therapeutic facilities for the affected babies (specialist fetal medicine care unit).

However, with use of intensive management protocols such as repeated fetal MCA Doppler study, amniocentesis, cordocentesis, intrauterine fetal transfusions (when necessary) the neonatal survival is 100%. An immunized mother who has anti-D level more than 400 IU/L, should be advised for anti-D donation.

BREASTFEEDING: There is **no contraindication of breastfeeding** in immunized mothers, although trace amount of antibodies are excreted through the breast milk.

ELDERLY PRIMIGRAVIDA

Women having their first pregnancy at or above the age of 30 years (FIGO-35 years) are called elderly primigravidae. The age limit is arbitrary and is based on the fact that the outcome of the pregnancy is adversely affected beyond the specified age limit. **There are two groups of patients**: (1) one with high fecundity—a women married late but conceives soon after and (2) one with low fecundity—woman married early but conceives long after marriage. The latter one is prognostically more unfavorable so far as the obstetric outcome is concerned after conception occurs following treatment of infertility (ovulation induction or assisted reproductive technology).

COMPLICATIONS

During pregnancy: **There is increased incidence of**: (1) abortion; (2) preeclampsia because of increased association of hypertension; (3) abruptio placentae because of preeclampsia and folic acid deficiency; (4) uterine fibroid; (5) medical complications related with advancing age such as pregnancy-induced hypertension, gestational diabetes and organic heart lesion, placental abruption or previa; (6) tendency of postmaturity and (7) intrauterine growth restriction.

During labor: There is increased incidence of: (1) preterm labor; (2) prolonged labor due to (a) uterine inertia caused by anxiety or malposition (occipito-posterior); (b) impaired joint mobility and (c) inelasticity of the soft tissues of the birth canal; (3) maternal and fetal distress appears early; (4) increased cesarean delivery and (5) retained placenta due to uterine atony and increased association of fibroid.

Fetal risks: Preterm birth and prematurity either iatrogenic or spontaneous, IUGR, fetal congenital malformation (aneuploidy).

Puerperium: (1) Increased morbidity due to operative interference and (2) failing lactation.

PROGNOSIS: **The maternal morbidity is high** and the maternal mortality is slightly increased due to the increased complications and operative interference. **The perinatal mortality is increased** due to prematurity, increased congenital malformation (trisomy 21) and operative interference.

MANAGEMENT: Preconception counseling (p. 116) should be done. Considering the risks involved in pregnancy and labor, the patients are considered “high risk”. **They require meticulous antenatal supervision and should have a mandatory hospital delivery. The following principles are to be followed**: (1) result of induction is unsatisfactory and as such cesarean section is a preferred alternative; (2) prenatal diagnosis and sonography (targeted) are done to exclude fetal genetic or structural anomaly and (3) development of other complications should be viewed with concern.

GRAND MULTIPARA

A grand multipara relates to a pregnant mother who has got previous four or more viable births. The incidence has been gradually declining over the couple of decades due to acceptance of small family norm but it still constitutes to about one-tenth of the hospital population and accounts for one-third of the maternal deaths in the developing countries.

COMPLICATIONS: ***Pregnancy*****—There is increased incidence of**: (1) **abortion**—spontaneous and induced; (2) **inherent obstetric hazards such as:** (a) malpresentation due to pendulous abdomen and increased pelvic inclination resulting from associated lordosis; (b) multiple pregnancy and (c) placenta previa; (3) **medical disorders** such as anemia (both iron deficiency and megaloblastic), hypertension with or without superimposed preeclampsia, cardiac disability, exaggerated manifestations of hemorrhoids and varicose veins, hiatus hernia, etc. and (4) **prematurity.**

Labor: **There is increased incidence of:** (1) **cord prolapse** due to malpresentation and high floating head at the onset of labor (2) **Cephalopelvic disproportion due to—**(a) increasing size of the fetus; (b) secondary contracted pelvis which is mostly related to ill-nourished mothers and (c) forward projection of the sacrum due to subluxation of the sacroiliac joints, thereby diminishing the inlet conjugate; (3) **obstructed labor** due to malpresentation, malposition and CPD; (4) **rupture uterus**, if the obstruction remains undetected and left uncared for; (5) **postpartum hemorrhage** due to atonic uterus or increased association of adherent placenta; (6) **shock** due to severe anemia, hemorrhage or unrecognized uterine rupture and (7) **operative interference** because of the complications.

Puerperium: (1) Increased morbidity due to sepsis, intranatal hazards; (2) subinvolution and (3) failing lactation.

MANAGEMENT: The cases are considered as "high risk". As such they require adequate antenatal care and should have a mandatory hospital delivery.

During labor, the following guidelines are prescribed: (1) pelvic assessment should be done as a routine; (2) presentation and position are to be checked; (3) undue delay in progress should be viewed with concern and (4) to take prophylactic measures against PPH.

BAD OBSTETRIC HISTORY (BOH)

DEFINITION: The term bad obstetric history (BOH) is applied to a pregnant mother where her present obstetric outcome is likely to be affected adversely by the nature of previous obstetric disaster. The previous pregnancy loss should be obstetrically related and as such mishaps to the baby due to some other reasons should not come under the purview of BOH. (For further study author's book "Masterpass in Obstetrics and Gynaecology").

INVESTIGATIONS AND MANAGEMENT

The principles are: (1) To find out the cause; (2) to rectify the abnormality, if possible and (3) to remain vigilant till delivery.

To find out the etiological factor: **At the first antenatal visit, detailed in depth relevant history** should be taken in an attempt to find out the cause of mishaps. In the developing countries, too often the disaster is linked with inadequate or neglected antenatal, intranatal or neonatal care. **In significant cases, however, the cause remains undetermined. Common causes are:** endocrine disorders (diabetes mellitus, thyroid disorders), antiphospholipid syndromes, inherited thrombophilias (factor V Leiden mutation, Proteins C and S deficiency, hyperhomocysteinemia), structural abnormalities of the uterus and cervix (septate uterus, cervical incompetence) or maternal systemic disease (SLE). Previous history of congenital deformity of the baby, especially a neural tube defect should be excluded as there is likely chance of recurrence (see p. 471).

Antiphospholipid syndrome: Antiphospholipid antibodies (aPLs) including lupus anticoagulant (LA) and anticardiolipin antibodies (aCL) and antibodies to β_2-glycoprotein-1 (β_2GP-1) are to be estimated. They are the markers of adverse pregnancy outcome. These antibodies are either IgG or IgM or both and bind to negatively charged phospholipids. They prevent physiological changes in decidual vessels (see p. 36). There is inhibition in release of prostacyclin (vasodilator) from vascular endothelium with rise in the level of thromboxane (vasoconstrictor) from platelets.

Other pathological changes like placental vascular atherosis, intervillous and spiral artery thrombosis and decidual vasculopathy with fibrinoid necrosis lead to inadequate maternal blood supply to fetus. Common obstetric complications associated with antiphospholipid syndrome are: (a) recurrent fetal loss (≥ 10 weeks), preterm birth; (b) IUGR; (c) IUFD; (d) severe preeclampsia; (e) HELLP syndrome; (f) placental abruption; (g) recurrent thrombotic events and (h) thrombocytopenia.

Thrombophilias: Some regulatory proteins act as inhibitors in the coagulation cascade. Inherited or acquired deficiencies of these inhibitory proteins are collectively known as thrombophilias. Thrombophilias are associated with many pregnancy complications. Some inherited thrombophilias (factor V Leiden mutation, antithrombin deficiency, prothrombin gene mutation, proteins C and S deficiency, hyperhomocysteinemia) increases the risk like severe preeclampsia, eclampsia, HELLP syndrome, IUGR, placental abruption and stillbirths.

Hyperhomocysteinemia, an autosomal recessive inheritance, is commonly due to mutation of the enzyme 5, 10 methylenetetrahydrofolate reductase (MTHFR). In a normal pregnancy its plasma level is low (p. 313). Fasting plasma level more than 12 μmol/L is diagnostic. Hyperhomocysteinemia causes thromboembolism due to inactivation of protein C. It causes increased fetal loss due to premature placental vascular atherosis (p. 196). It also causes fetal neural tube defect. Routine folic acid supplementation should be given.

To treat the offending factor: **When the responsible factor is detected**, appropriate therapy can be directed so as to prevent repetition of the mishaps. A good example is to perform encerclage operation in recurrent midtrimester abortion. **Diabetes in pregnancy,** if discovered during such investigation, should be adequately supervised all throughout pregnancy and a suitable time is selected for its termination (see p. 331). **Proved cases of fetal wastage due to red cell alloimmunization** of the mother should be managed in specialized fetal medicine unit to deal with Rh problems. Approximate amount of fetomaternal bleed is estimated by **Kleihauer-Betke acid elution test** (Fig. 23.5). **If environmental factors are involved**, extension of adequate antenatal and intranatal care is enough to prevent repetition of the mishaps. Therapy for patients with positive antiphospholipid antibodies and with thrombophilias is low-dose aspirin (50 mg a day). **When there is history of previous thrombotic event, heparin (5,000 IU, subcutaneously twice daily) is the drug of choice. Low-molecular-weight heparin is also effective.** (Detail to read author's "Masterpass in Obstetrics and Gynaecology", see p. 400)

Prepregnancy folic acid therapy (4 mg daily) is given when there is any history of neural tube defect in previous birth. Therapy is started 1 month before conception and is continued in the first trimester.

To remain vigilant: **In obstetrics, any complicating factor, known or unknown, is likely to recur and if it recurs in two consecutive pregnancies, the chance of its recurrence in the third pregnancy is highly probable. When the cause remains unknown,** constant vigilance following hospitalization in early or later months of pregnancy as the case may be, is all that is required. History of unexplained intrauterine death (suspected chronic placental insufficiency) should preferably be terminated at a period, judiciously selected under the guidance of available gadgets for the assessment of fetal well being (see Chapters 12 and 32).

OBESITY IN PREGNANCY

There is no clear and universally accepted definition of obesity. However, a maternal weight of 90 kg is considered the upper limit of normal. **Body mass index (BMI)**, expressed as weight (Kg) divided by height^2 (m) is a better guide. Ideal BMI should be between 18.5 Kg/m^2 and 24.9 Kg/m^2. Arbitrary cut-off points of BMI more than or equal to 25 kg/m^2 and more than or equal to 30 kg/m^2 are considered as **overweight** and **obese** respectively. The obese patients have got adverse effects on obstetric performances. Obesity leads to development of type-2 diabetes, dyslipidemia and hypertension (metabolic syndrome).

Effects on pregnancy: (1) Patients feel uncomfortable and become dyspneic on exertion; (2) medical complications like **hypertension**, both essential and pregnancy induced, are increased and so also **gestational diabetes;** (3) there is difficulty in diagnosis of **presentation** and in hearing the FHS; (4) fetal macrosomia; (5) as such, there is more need for sonography; (6) increased risk of **miscarriage, fetal malformations**, especially neural tube defects; (7) postmaturity is also common and others are (8) IUFD and stillbirths.

Effects on labor: (1) There is increased incidence of abnormal uterine contraction and prolonged labor; (2) operative interference and cesarean delivery is increased and so also difficulty in cesarean section; (3) shoulder dystocia is likely and (4) anesthetic hazards (difficult intubation) are high. ***Puerperium:*** There is increased chance of puerperal urinary tract infection, PPH, deep vein thrombosis, poor wound healing and lactation failure.

Perinatal: Morbidity and mortality are high. There is increased risk of fetal neural tube defects, macrosomic infants, IUGR and stillbirth.

Management: The cases are considered as high-risk group. They require adequate antenatal supervision and mandatory hospital delivery. Fat and carbohydrate should be curtailed in the diet. There is increased insulin resistance. Obese women should limit weight gain to less than 8 kg (p. 51) and there is no need of weight loss during pregnancy. **During labor, shoulder dystocia should be kept in mind (macrosomia).** Early mobilization and heparin prophylaxis is given to prevent DVT.

Contraception: Oral contraceptives has got high failure rate. For further reading, author's book "Masterpass in Obstetrics and Gynaecology", see p. 464.

KEY POINTS

PREGNANCY IN A Rh-NEGATIVE WOMAN (Red Cell Alloimmunization)

- **A Rh negative woman** means she is D-negative (p. 386)
- **In alloimmunization,** it is the IgG antibodies that cross the placenta and damage the fetus (p. 388).
- **Degree of affection** of the fetus depends upon the degree of destruction of the fetal red cells (p. 388), by the antibodies.
- **Alloimmunization** is preventable by anti-D prophylaxis. It should be given to the mother within 72 hours or earlier following delivery (p. 389).
- **It is always better to give** anti-D immunoglobulin, where there is any doubt about whether to give or not.
- **Indications of anti-D immunoprophylaxis** for Rh (D) negative women are : (i) miscarriage; (ii) ectopic pregnancy; (iii) MTP; (iv) chorionic villous sampling; (v) amniocentesis; (vi) fetal blood sampling; (vii) APH; (viii) external cephalic version; (ix) routine at 28 weeks (p. 391); (x) after delivery; (xi) molar pregnancy and (xii) after abdominal trauma.
- **Antenatal investigations include :** Antibody detection (indirect Coombs' test), serial antibody measurement (p. 391). **Serial ultrasonography (p. 393)** including Dopper study is noninvasive and can reduce the need of amniocentesis (p. 395) and fetal blood sampling by cordocentesis (p. 395).
- **Ultrasonography** is helpful to identify an affected fetus and its severity. It is done at 1–3 weeks interval. Findings suggestive of fetal anemia are : skin edema, ascites, pleural or pericardial effusions, increased placental thickness and others (p. 393). Doppler ultrasound and cardiotocography are also informative and are noninvasive methods (p. 393).
- **Doppler blood flow** study in the middle cerebral artery (MCA) can predict fetal anemia. Peak systolic velocity in the fetal MCA greater than 1.5 multiples of the median (MOMs) for the corresponding gestational age predicts moderate to severe fetal anemia. Currently serial MCA Doppler studies have replaced serial amniocentesis (p. 391).
- **Management of a Rh-negative woman** (p. 394) should be in a tertiary center where facilities for intrauterine transfusion (p. 395) and exchange transfusion are available (p. 396). Specialized fetal medicine unit should deal the Rh problems.
- **HDFN depends on** the degree of destruction of the fetal red cells by the transplacental maternal antibodies (IgG). HDFN may be (a) congenital anemia of the newborn (b) icterus gravis neonatorum or (c) hydrops fetalis.
- **Fetal Rh status can be studied by ffDNA** present in maternal plasma. The method has replaced CVS and amniocentesis which are invasive methods (see p. 131).
- **Rh immune globulin prophylaxis** can be given to Rh-negative, nonimmune woman at 28 weeks of pregnancy. This is in addition to the dose given after delivery (300 μg within 72 hours of delivery).
- Anti D level of >4 IU/mL but <15 IU/mL indicates moderate risk of HDFN. Anti-D level of >15 IU/mL can cause severe HDFN. Woman should be referred to a fetal medicine unit when the anti-D levels are >4 IU/mL.

WOMEN WITH BAD OBSTETRIC HISTORY (BOH)

- Women with BOH need to be investigated for the underlying etiological factor(s) (see p. 399).
- **Common causes are:** Endocrine disorders (diabetes mellitus, thyroid disorder), antiphospholipid syndromes, inherited thrombophilias, anomalies of the uterus and the cervix (septate uterus, cervical insufficiency), red cell alloimmunization or chromosomal abnormalities.
- BOH cases need to be managed based on specific pathology.

QUESTIONS

Related theory questions (Long and Short), Obstetric Case Discussions, Viva table discussions, Postoperative word round discussions, and MCQs are discussed in author's books:

1. **Bedside Clinic and Viva Voce:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

For further reading:

2. **Master pass in Obstetrics and Gynaecology:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

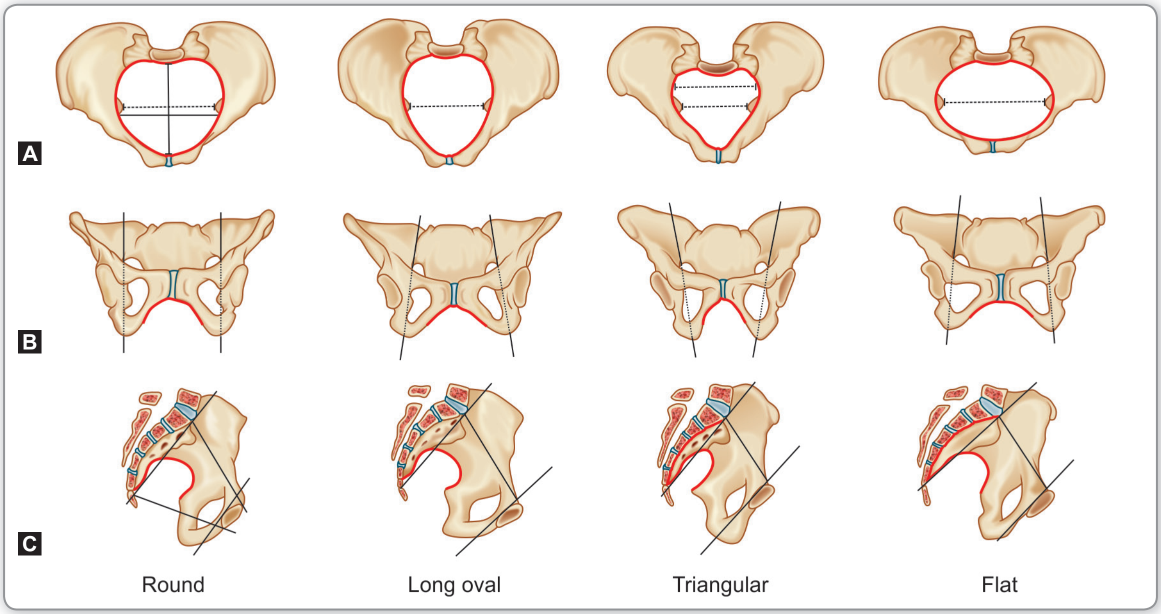
Chapter 24

Contracted Pelvis

DEFINITION: It is indeed difficult to define precisely what constitutes a contracted pelvis. Anatomically, contracted pelvis is defined as one where the essential diameters of one or more planes are shortened by 0.5 cm. But of more importance is the **obstetric definition which states alteration in the size and/or shape of the pelvis of sufficient degree as to alter the normal mechanism of labor in an average size baby**. Depending upon the degree of contraction, the head may pass through the pelvis by abnormal mechanism or fail to pass due to absolute obstruction.

VARIATIONS OF FEMALE PELVIS: The size and shape of the female pelvis differ so widely due to morphological factors such as developmental, sexual, racial and evolutionary that it is indeed difficult to define what the features of a normal pelvis are. **However, on the basis of the shape of the inlet, the female pelvis is divided into four parent types (Figs 24.1A to C and Table 24.1):**

- **Gynecoid (50%)**
- **Anthropoid (25%)**
- **Android (20%)**
- **Platypelloid (5%)**



Figs 24.1A to C: Anatomical features of parent pelvic types: (A) Inlet; (B) Cavity; (C) Outlet

But more commonly, intermediate forms with combination of features are found. They are termed as gyne-android or andro-gynecoid, etc. The first part of the nomenclature relates to features of the posterior segment and the second part relates to that of the anterior segment of the pelvis. All types of combinations are possible except anthropoid with platypelloid. **Thus, there may be 14 types of parent pelves either in pure form or in combination (Tables 24.1 and 24.2)**.

Table 24.1: Anatomical Features of Parent Pelvic Types (Figs 24.1A to C)

		Gynecoid	**Anthropoid**	**Android**	**Platypelloid**
Inlet	■ Shape	Round	Anteroposteriorly oval	Triangular	Transversely oval
	■ Anterior and posterior segment	Almost equal and spacious	Both increased with slight anterior narrowing	Posterior segment short and anterior segment narrow	Both reduced-flat
	■ Sacrum	Sacral angle (SA) more than 90°. Inclined backwards. Well curved from above down and side to side	SA more than 90°. Inclined posteriorly. Long and narrow. Usual curve	Sacral angle less than 90°. Inclined forwards and straight	SA more than 90°. Inclined posteriorly. Short and straight
Cavity	■ Sacrosciatic notch	Wide and shallow	More wide and shallow	Narrow and deep	Slightly narrow and small
	■ Sidewalls	Straight or slightly divergent	Straight or divergent	Convergent	Divergent
Outlet	■ Ischial spines	Not prominent	Not prominent	Prominent	Not prominent
	■ Pubic arch	Curved	Long and curved	Long and straight	Short and curved
	■ Subpubic angle	Wide (85°)	Slightly narrow	Narrow	Very wide (more than 90°)
	■ Bituberous diameter	Normal	Normal or short	Short	Wide

Table 24.2: Obstetric Outcome in Parent Pelvic Types

Inlet	■ Position	Occipitolateral or oblique occipito-anterior	Direct occipito-anterior or posterior	Occipitolateral or oblique occipito-posterior	Occipitolateral
	■ Diameter of engagement	Transverse or oblique	Anteroposterior	Transverse or oblique	Transverse
	■ Engagement	No difficulty Usual mechanism	No difficulty except flexion is delayed	Delayed and difficult	Difficult by exaggerated parietal presentation
Cavity	■ Internal rotation	Easy anterior rotation	Non-rotation common	Difficult anterior rotation. Not occurs early above the ischial spines, chance of arrest	Anterior rotation usually occurs late in the perineum
Outlet	■ Delivery	No difficulty	More incidence of face-to-pubis delivery	Difficult delivery with increased chance of perineal injuries	No difficulty

It should be clear that the pelves which are not typically female are not necessarily contracted, although there may be deviation of normal mechanism of labor. However, slight contraction if associated with any of the three nongynecoid pelves has a more serious consequence because of the unfavorable shape.

ETIOLOGY OF CONTRACTED PELVIS: Gross degree of contracted pelvis is nowadays a rarity. Severe malnutrition, rickets, osteomalacia and bone tuberculosis affecting grossly the pelvic architecture are now rarely met in clinical practice. Instead, **minor variation in size and/or shape of the pelvis is commonly found** which is often overlooked until complication arises.

Common causes of contracted pelvis are:

(1) **Nutritional and environmental defects** —

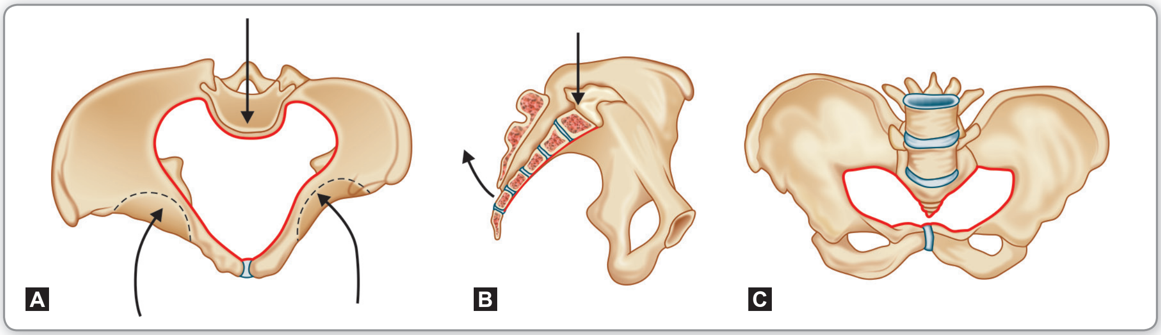
- *Minor variation:* Common
- *Major*: Rachitic and osteomalacic — rare

(2) **Diseases or injuries** affecting the bones of the **pelvis** — fracture, tumors, tubercular arthritis; **spine** — kyphosis, scoliosis, spondylolisthesis, coccygeal deformity; **lower limbs** — poliomyelitis, hip joint disease.

(3) **Development defects** — Naegele's pelvis, Robert's pelvis; high or low assimilation pelvis.

RACHITIC FLAT PELVIS: Rickets is predominantly a disease of early childhood when the bones remain soft and unossified. In childhood, changes occur in the bony pelvis due to weightbearing (Fig. 24.2). The classic changes in the pelvic bones are shown (Figs 24.2 A to C).

Inlet: Sacral promontory is pushed downwards and forwards producing a "reniform" shape of the inlet with marked shortening of the anteroposterior diameter without affecting the transverse diameter, which is often increased (Fig. 24.2C).



Figs 24.2A to C: Rachitic pelvis: (A) Effect of walking; (B) Effect on lying down position; (C) Reniform shape of the inlet

Cavity: Sacrum is flat and tilted backwards. There may be sharp angulation at the sacrococcygeal joint.

Outlet: Body weight transmitted through the ischium in sitting position results in widening of the transverse diameter of the outlet and the pubic arch.

OSTEOMALACIC PELVIS: The deformity is caused by softening of the pubic bones due to deficiency of calcium and vitamin D and lack of exposure to sunrays. It usually affects women after they have reached maturity. **The changes in the pelvic bones are (Fig. 24.3):**

- *The promontory* is pushed downwards and forwards and the **lateral pelvic walls** are pushed inwards causing the anterior wall

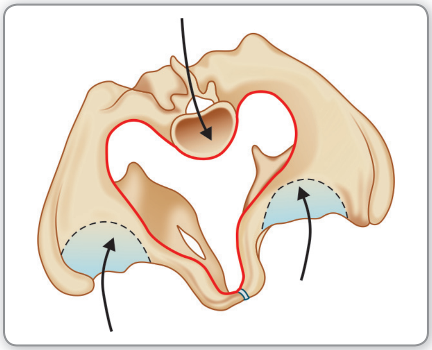


Fig. 24.3: Osteomalacic pelvis

to form a beak. **The shape of the inlet** thus becomes **triradiate.** *Approximation of the two ischial tuberosities* occurs. *Sacrum* is markedly shortened and *Coccyx* is pushed forward. **Vaginal delivery** is unlikely and **cesarean section is ideal.**

ASYMMETRICAL OR OBLIQUELY CONTRACTED PELVIS

It is seen in (1) Naegele's pelvis, (2) scoliotic pelvis, (3) due to disease affecting one hip or sacroiliac joint, and (4) tumors or fracture affecting one side of the pelvic bones during growing age.

- **Naegele's pelvis:** This type of pelvis is extremely rare. **It is produced due to arrested development of one ala of the sacrum (Fig. 24.4). It may be** (i) congenital or (ii) acquired (osteitis of sacroiliac joint). Congenital variety may be associated with urinary tract and may of the same side. The pelvis is obliquely contracted at all levels but more marked in the outlet. Iliopectineal line on the affected side is almost straight. **Method of delivery** is by cesarean section.

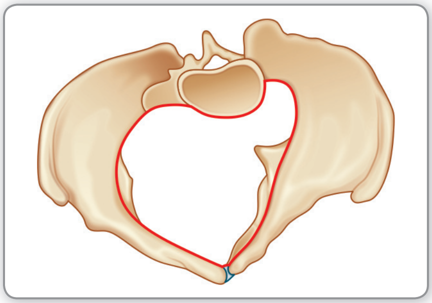


Fig. 24.4: Naegele's pelvis

Scoliosis involving only the lumbar region will cause deformity of the pelvis (Fig. 24.4A). The acetabulum is pushed inwards on the weightbearing side. This may be pronounced if the disease occurs during early life. Oblique asymmetry of the pelvis results in contraction of one of the oblique diameters. Cesarean section is the only safe method of delivery.

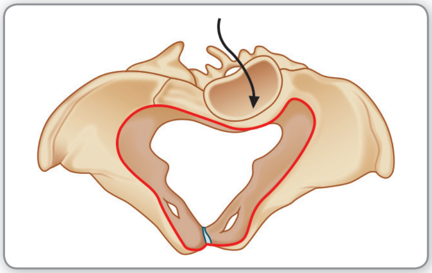


Fig. 24.4A: Scoliotic pelvis

Robert's pelvis (transversely contracted pelvis): This is an extremely rare abnormality. Ala of both the sides are absent and the sacrum is fused with the innominate bones. Delivery is done by cesarean section.

Kyphotic pelvis: This pelvic deformity is secondary to the kyphotic changes of the vertebral column either following tuberculosis or rickets. The deformities observed with lumbar kyphosis are:

The sacrum is tilted backwards in the upper part and forwards in the lower part. It is narrow and straight. The anteroposterior diameter of inlet is increased but is diminished at the outlet. Subpubic angle is narrow. Thus, **the feature is an extreme funneling of the pelvis.**

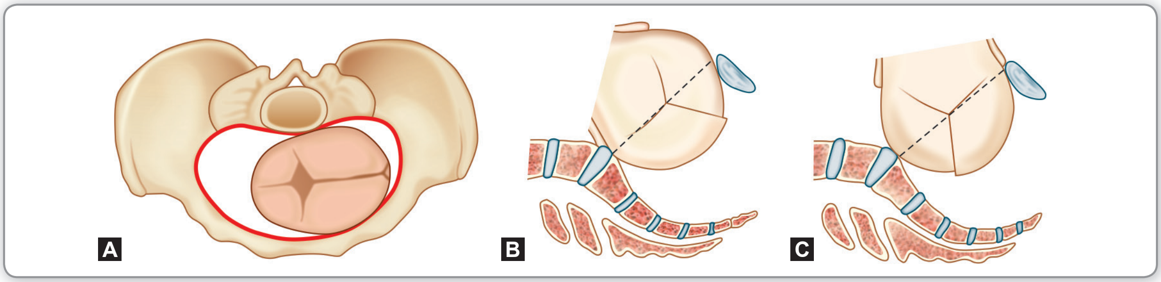
Abdomen becomes pendulous due to the shortened distance between the symphysis pubis and xiphisternum. Malpresentation is common. Mechanical distress is evident. **Cesarean section** is ideal and one may have to do the classical operation because of poor formation of the lower segment or for technical reasons.

MECHANISM OF LABOR IN CONTRACTED PELVIS WITH VERTEX PRESENTATION

FLAT PELVIS (Figs 24.5A to C)

In the flat pelvis, the head finds difficulty in negotiating the brim and once it passes through the brim, there is no difficulty in the cavity or outlet. **The head negotiates the brim by the following mechanism**:

- The head engages with the sagittal suture in the transverse diameter.
- Head remains deflexed and engagement is delayed.
- If the anteroposterior diameter is too short, the occiput is mobilized to the same side to occupy the sacral bay. The biparietal diameter is thus placed in the sacrocotyloid diameter (9.5 cm or 8.5 cm) and the narrow bitemporal diameter is placed in the narrow conjugate. If lateral mobilization is not possible, there is a chance of extension of the head leading to brow or face presentation.
- Engagement occurs by exaggerated parietal presentation so that the super-subparietal diameter (8.5 cm), instead of the biparietal diameter (9.5 cm), passes through the pelvic brim.



Figs 24.5A to C: Mechanism of labor in flat pelvis: (A) Lateralization of occiput to the sacral bay; (B and C) Engagement of the head by exaggerated parietal presentation

- Molding may be extreme and often there is an indentation or even a fracture of one parietal bone. However, the caput that forms is not big.
- Once the head negotiates the brim, there is no difficulty in the cavity and outlet and normal mechanism follows.

GENERALLY CONTRACTED PELVIS: In this type of pelvis the shape remains unaltered, but all the diameters in the different planes—inlet, cavity and outlet—are shortened. **There is difficulty from the beginning to the end.**

DIAGNOSIS OF CONTRACTED PELVIS

During the past couple of decades, there has been a gradual decline in the incidence of severe degree of contracted pelvis. This is due to an improved standard of living and of nutrition in particular. But of significance is the presence of **fetopelvic disproportion** due either to inadequate pelvis or big baby or more commonly a combination of the both.

Past History

Medical: Past history of fracture, rickets, osteomalacia, tuberculosis of the pelvic joints or spines and poliomyelitis is to be enquired.

Obstetrical: While an uncomplicated, previous safe vaginal delivery of an average size baby reasonably excludes pelvic contraction, a history of prolonged and a tedious labor followed by either spontaneous or difficult instrumental delivery is suggestive of pelvic contraction. Difficult vaginal delivery ending in stillborn or early neonatal death or late neurological stigmata following a difficult labor without any other etiological factor points towards contracted pelvis. Weight of the baby, evidences of maternal injuries such as complete perineal tear, vesicovaginal or rectovaginal fistula, if available, are of useful guide.

Physical Examination

Stature: A small woman of less than 5 ft is likely to have a small pelvis. She is likely to have a small baby also. However, this does not mean that tall women always have a good pelvis.

Stigma: Deformities (congenital or acquired) of pelvic bones, hip joint, spine.

Dystocia dystrophia syndrome: This syndrome is **characterized by the following features**: The patient is stockily built with bull neck, broad shoulders and short thighs. She is obese with a male distribution of hairs. They are usually subfertile, having dysmenorrhea, oligomenorrhea or irregular periods. There is increased incidence of pre-eclampsia and a tendency for postmaturity. Pelvis is of the android type. Occipito-posterior position is common. During labor, inertia is common and there is a tendency for deep transverse arrest or outlet dystocia leading to either increased incidence of difficult instrumental delivery or cesarean section. There is a chance of lactation failure.

Abdominal Examination

Inspection: Pendulous abdomen, especially in primigravidae is suspicious of inlet contraction.

Obstetrical: In primigravidae, usually there is engagement of the head before the onset of labor. **Presence of malpresentation in primigravidae** gives rise to a suspicion of pelvic contraction.

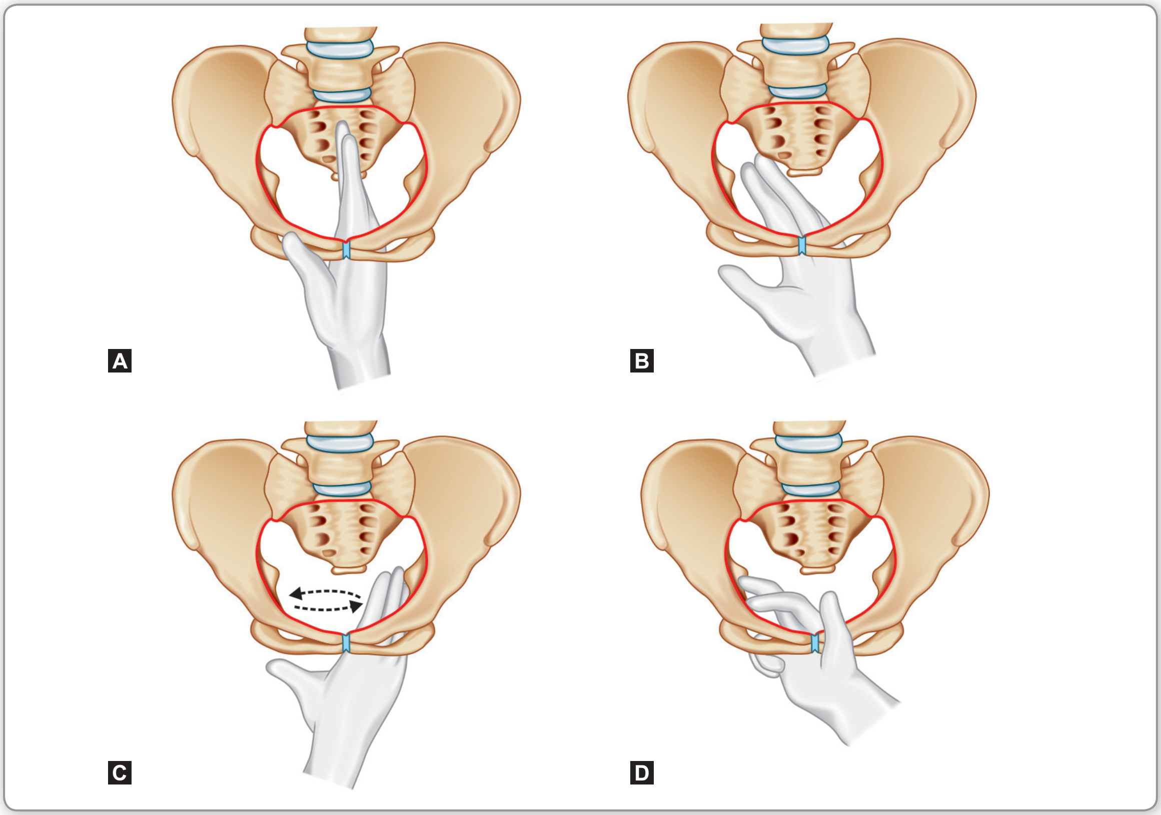
Assessment of the pelvis (pelvimetry): Assessment of the pelvis can be done by bimanual examination: **clinical pelvimetry or by imaging studies—radio-pelvimetry**, **computed tomography (CT)** and **magnetic resonance imaging (MRI)**.

Clinical pelvimetry: This is commonly done.

Time: In vertex presentation, the assessment is done at any time beyond 37th week but **better at the beginning of labor**. Because of softening of the tissues, assessment can be done effectively during this time. **Procedures**: The patient has to empty the bladder. The pelvic examination is done with the patient in dorsal position taking aseptic preparations. **The following features are to be noted simultaneously:** (1) State of the cervix; (2) To note the station of the presenting part in relation to ischial spines; (3) To test for cephalopelvic disproportion in nonengaged head (described later); (4) To note the resilience and elasticity of the perineal muscles.

Steps: The internal examination should be gentle, thorough, methodical and purposeful (Fig. 24.6). **It should be emphasized that the sterilized gloved fingers once taken out should not be reintroduced.**

Sacrum (Fig. 24.6A) — **The sacrum may be smooth, short and well curved, and the sacral promontory usually cannot be reached** or the sacrum may be long or straight.



Figs 24.6A to D: Clinical assessment of the pelvis

Sacrosciatic notch (Fig. 24.6B) — The notch is sufficiently wide so that two fingers can be easily placed over the sacrospinous ligament covering the notch. **The configuration of the notch denotes the capacity of the posterior segment of the pelvis and the sidewalls of the lower pelvis.**

Ischial spines (Fig. 24.6C) — Spines are usually smooth (everted) and difficult to palpate. They may be prominent and encroach to the cavity thereby diminishing the available space in the midpelvis.

Iliopectineal lines (Fig. 24.6D) — To note for any beaking suggestive of narrow fore pelvis (android feature).

Sidewalls — Normally they are parallel or divergent. They may be convergent.

Posterior surface of the symphysis pubis — It normally forms a smooth rounded curve. Presence of angulation or beaking suggests abnormality.

Sacrococcygeal joint — Its mobility and presence of hooked coccyx, if any, are noted.

Pubic arch — Normally, the pubic arch is rounded and should accommodate the palmar aspect of two fingers. Configuration of the arch is more important than pubic angle.

Diagonal conjugate — Procedure is described before (see p. 100). After the procedure, the fingers are now taken out.

Subpubic angle: The inferior pubic rami are defined and in female, the angle roughly corresponds to the fully abducted thumb and index fingers. In narrow angle, it roughly corresponds to the fully abducted middle and index fingers (Fig. 24.7B).

Transverse diameter of the outlet (TDO) — It is measured by placing the knuckles of the first interphalangeal joints or knuckles of the clinched fist between the two ischial tuberosities (Fig. 24.7). Normally, it accomodates four knuckles.

Anteroposterior diameter of the outlet—The distance between the inferior margin of the symphysis pubis and the skin over the sacrococcygeal joint can be measured either with the method employed for diagonal conjugate or by external calipers.

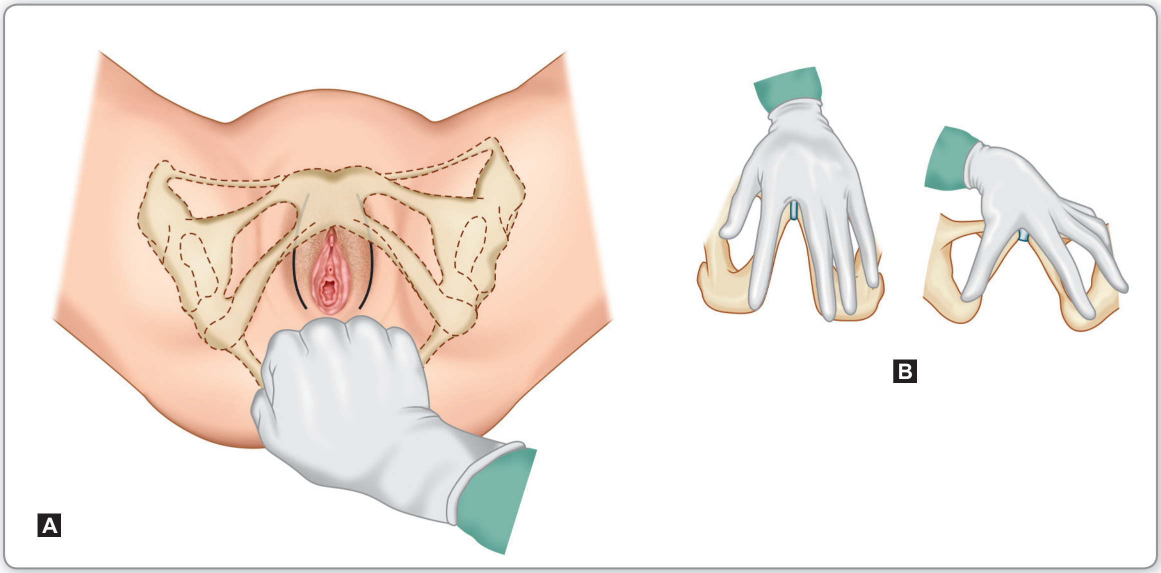


Fig. 24.7A and B: Measurement of transverse diameter of the outlet and subpubic angle

X-ray pelvimetry is of limited value in the diagnosis of pelvic contraction or cephalopelvic disproportion. Apart from pelvic capacity there are several other factors involved in successful vaginal delivery. These are the fetal size, presentation, position and the force of uterine contractions. X-ray pelvimetry cannot assess the other factors. It cannot reliably predict the likelihood of vaginal delivery neither in breech presentation nor in cases with previous cesarean section. **X-ray pelvimetry** is a poor predictor of pelvic adequacy and success of vaginal delivery. **However, X-ray pelvimetry is useful** in cases with fractured pelvis and for the important diameters which are inaccessible to clinical examination (Table 24.3).

Table 24.3: The Salient Features to be Noted to Detect Contraction at

Brim	Midpelvis	Outlet
▪ Diagonal conjugate ▪ Posterior surface of the symphysis pubis ▪ Iliopectineal line ▪ Sacrosciatic notch	▪ Sacrum ▪ Ischial spines ▪ Interspinous diameter ▪ Sacrosciatic notch ▪ Sidewalls	▪ Sidewalls ▪ Sacrococcygeal joint ▪ Subpubic arch ▪ Subpubic angle ▪ TDO

Techniques: For complete evaluation of the pelvis, three views are taken — anteroposterior, lateral and outlet. **But commonly, X-ray pelvimetry is restricted only to the erect lateral view** (the femoral head and acetabular margins must be superimposed) which gives **most of the useful information (Fig. 24.1C)**. **Anteroposterior view can give the accurate measurement** of the transverse diameter of the inlet and bispinous diameter.

Limitations of X-ray pelvimetry: The following are the prognostic significances of the successful outcome of labor: (1) Size and shape of the pelvis; (2) Presentation and position of the head; (3) Size of the head; (4) Molding of the head; (5) Give way of the pelvis; (6) Force of uterine contractions. **Out of these many unknown factors, X-ray pelvimetry can only identify one factor, i.e. size and shape of the pelvis. Thus, it can only supplement the clinical assessment of pelvis but cannot replace the clinical examination. Otherwise it is true that satisfactory progress of labor is the best indicator of pelvic adequacy**.

Hazards of X-ray pelvimetry includes radiation exposure to the mother and the fetus (see p. 740). With conventional X-ray pelvimetry, radiation exposure to the fetus is about 0.1–0.3 cGy. **So it is restricted to selected cases only**.

Computed tomography (CT) involves less radiation exposure (44–425 millirad) and is easier to perform. Accuracy is greater than that of conventional X-ray pelvimetry (Fig. 24.8). Three images (lateral, AP and axial slice) are taken.

Magnetic resonance imaging (MRI) is more accurate to assess the bony pelvis. It is also helpful to assess the fetal size and maternal soft tissues which are involved in dystocia. It has got no radiation risk, hence biologically safe. It is expensive, requires more time and availability is limited.

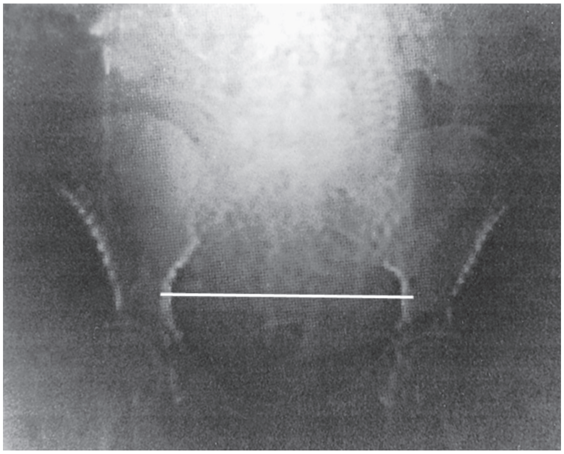


Fig. 24.8: Computed tomography (CT) of the maternal bony pelvis showing the widest transverse diameter at the brim

Ultrasonography is useful to measure the fetal head dimensions in the intrapartum phase.

DISPROPORTION

DEFINITION: Disproportion, in relation to the pelvis, is a state where the normal proportion between the size of fetus to the size of the pelvis is disturbed. **The disparity in the relation between the head and the pelvis is called cephalopelvic disproportion**. Disproportion may be either due to an average size baby with a small pelvis or due to a big baby (hydrocephalus) with normal size pelvis or due to a combination of both the factors. **Pelvic inlet contraction** is considered when the obstetric conjugate is < 10 cm or the greatest transverse diameter is < 12 cm or diagonal conjugate is < 11 cm. **Contracted Midpelvis:** Midpelvis is considered contracted when the sum of the interischial spinous and posterior sagittal diameters of the midpelvis (normal: 10.0 + 5 = 15.0 cm) is 13.0 cm or below.

Contracted outlet is suspected when the interischial tuberous diameter is 8 cm or less. A contracted outlet is often associated with midpelvic contraction. Isolated outlet contraction is a rarity. Disproportion at the outlet may not give rise to severe dystocia, but may cause perineal tears. The head is pushed backwards as it cannot be accommodated beneath the symphysis pubis.

As the head is the largest part of the fetus, it is more important to know whether the greatest diameter of the head passes through the different planes of the pelvis. Thus, from the clinical point of view, identification of the cephalopelvic disproportion is more logical than to concentrate entirely on the measurements of a given pelvis, as the **fetal head is the best pelvimeter**. Thus, disproportion may be limited to one or more planes. Absence of cephalopelvic disproportion at the brim usually, but not always, negates its presence at the midpelvic plane. On the other hand, **isolated outlet contraction without midpelvic contraction is a rarity**. Thus, a thorough assessment of the pelvis and identification of the presence and degree of cephalopelvic disproportion are to be noted while evaluating a case of contracted pelvis.

DIAGNOSIS OF CEPHALOPELVIC DISPROPORTION (CPD) AT THE BRIM

The presence and degree of cephalopelvic disproportion at the brim can be ascertained by the following:

- **Clinical** — (a) Abdominal method; (b) Abdominovaginal (Muller-Munro Kerr)
- **Imaging pelvimetry** (see above)
- **Cephalometry** — (a) Ultrasound; (b) Magnetic Resonance Imaging; (c) X-ray

Clinical: In multigravida, a previous history of spontaneous delivery of an average size baby reasonably rules out contracted pelvis. But in a primigravida with nonengagement of the head even at labor, one should rule out disproportion.

Abdominal method: The patient is placed in dorsal position with the thighs slightly flexed and separated. The head is grasped by the left hand. Two fingers (index and middle) of the right hand are placed above the symphysis pubis keeping the inner surface of the fingers in line with the anterior surface of the symphysis pubis to note the degree of overlapping, if any, when the head is pushed downwards and backwards (Fig. 24.8A).

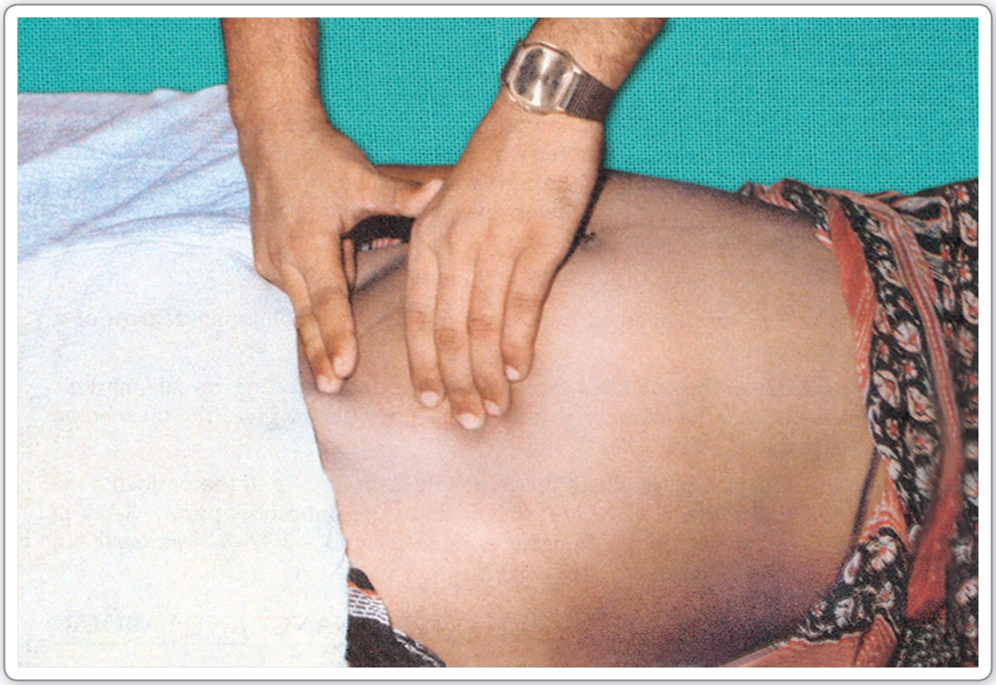


Fig. 24.8A: Abdominal method of testing cephalopelvic disproportion

Inferences:

- The head can be pushed down in the pelvis without overlapping of the parietal bone on the symphysis pubis — *no disproportion.*
- Head can be pushed down a little but there is slight overlapping of the parietal bone evidenced by touch on the under surface of the fingers (overlapping by 0.5 cm or 1/4" which is the thickness of the symphysis pubis) — *moderate disproportion.*
- Head cannot be pushed down and instead the parietal bone overhangs the symphysis pubis displacing the fingers — *severe disproportion.*

The abdominal method can be used as a screening procedure. At times, it is **difficult to elicit due** to deflexed head, thick abdominal wall, irritable uterus and high-floating head.

***Abdominovaginal method (Muller-Munro Kerr)*: This bimanual method is superior to the abdominal** method as the pelvic assessment can be done simultaneously. **Muller introduced the method by placing the vaginal finger tips at the level of ischial spines to note the descent of the head. Munro Kerr** added placement of the thumb over the symphysis pubis to note the degree of overlapping (Fig. 24.9).

Lower bowel is emptied, preferably by enema. The patient is asked to empty the bladder. The patient is placed in lithotomy position and the internal examination is done taking all aseptic precautions. Two fingers of the right hand are introduced into the vagina with the **finger tips placed at the level of ischial spines and thumb is placed over the symphysis pubis. The head is grasped by the left hand and is pushed** in a downward and backward direction into the pelvis (Fig. 24.9).

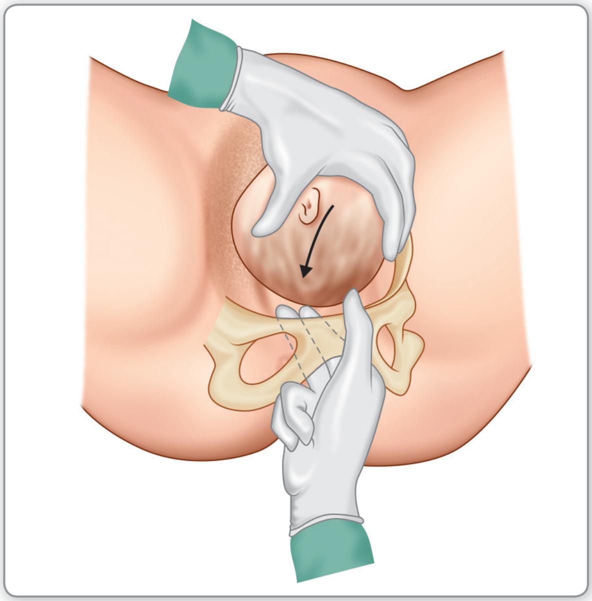


Fig. 24.9: Bimanual method of testing CPD. The finger tips are at the level of spines

Inferences: (1) The head can be pushed down up to the level of ischial spines and there is no overlapping of the parietal bone over the symphysis pubis — *no disproportion;* (2) The head can be pushed down a little but not up to the level of ischial spines and there is slight overlapping of the parietal bone — *slight or moderate disproportion;* (3) The head cannot be pushed down and instead the parietal bone overhangs the symphysis pubis displacing the thumb — *severe disproportion.*

Limitations of clinical assessment: (1) The method is only applicable to note the presence or absence of disproportion at the brim and not at all applicable to elicit midpelvic or outlet contraction; (2) The fetal head can be used as a pelvimeter to elicit only the contraction in the anteroposterior plane of the inlet but when the contraction affects the transverse diameter of the inlet, it is of less use.

X-ray pelvimetry: Lateral X-ray view with the patient in standing position is helpful in assessing cephalopelvic proportion in all planes of the pelvis — inlet, midpelvic and outlet.

Cephalometry: While a rough estimation of the size of the head can be assessed clinically, accurate measurement of the biparietal diameter would have been ideal to elicit its relation with the diameters of the planes of a given pelvis through which it has to pass. In this respect, **ultrasonographic measurement** of the biparietal diameter or Magnetic Resonance Imaging (MRI) gives superior information. The average biparietal diameter measures 9.4–9.8 cm at term.

Magnetic Resonance Imaging (MRI): MRI is useful to assess the pelvic capacity at different planes. It is equally informative to assess the fetal size, fetal head volume and pelvic soft tissues which are also important for successful vaginal delivery (p. 739).

Degree of disproportion and contracted pelvis: Based on the clinical and supplemented by imaging pelvimetry, the following degrees of disproportion at the brim are evaluated:

(1) ***Severe disproportion:*** Where obstetric conjugate is < 7.5 cm (3"). Such type is rare to see.

(2) ***Borderline:*** Where obstetric conjugate is between 9.5 cm and 10 cm. When both the anteroposterior diameter (< 10 cm) and the transverse diameter (< 12 cm) of the inlet are reduced, the risk of dystocia is high than when only one diameter is contracted.

EFFECTS OF CONTRACTED PELVIS ON PREGNANCY AND LABOR

Pregnancy: The general course of pregnancy is not much affected. However, **the following may occur:** (1) There is more chance of incarceration of the retroverted gravid uterus in flat pelvis; (2) Abdomen becomes pendulous especially in multigravida with lax abdominal wall; (3) Malpresentations are increased three to four times and so also increased frequency of unstable lie.

Labor: The course of events in labor is greatly modified depending upon the degree of pelvic contraction and presentation of the fetus: (1) There is increased incidence of early rupture of the membranes; (2) Incidence of cord prolapse is increased; (3) Cervical dilatation is slowed; (4) There is increased tendency of prolonged labor and in neglected cases, obstructed labor with features of exhaustion, dehydration, ketoacidosis and sepsis (see p. 467); (5) There is increased incidence of operative interference, shock, postpartum; and hemorrhage and sepsis.

Maternal injuries: The injuries of the genital tract may occur spontaneously or following operative delivery (see p. 489). There is increased maternal morbidity and mortality (see p. 498).

Fetal hazards: Fetal risks are due to trauma and asphyxia (see p. 465). The net effect leads to increased perinatal mortality and morbidity.

MANAGEMENT OF CONTRACTED PELVIS (INLET CONTRACTION)

The prerequisite in the formulation of the line of management of contracted inlet is to ascertain the degree of disproportion by clinical examination and supplemented by imaging pelvimetry. Due consideration is given to the associated complicating factor, if any.

Minor degrees of inlet contraction does not give rise to much problem and the cases are left to have a spontaneous vaginal delivery at term. **The moderate and the severe degrees are to be dealt by any one of the following: ♦ Induction of labor ♦ Elective cesarean section at term ♦ Trial labor**

Induction of labor prior EDC: Induction 2–3 weeks prior to the EDC may be considered only in cases with minor to moderate degrees of pelvic contraction. It is not favored nowadays. However, in a selected multigravida with previous history of difficult vaginal delivery, this method may be considered 2–3 weeks before the date. **In any case, one should be certain about the fetal gestational age**.

Elective cesarean section at term: This is commonly done. **Elective cesarean section at term is indicated in**—(1) major degree of inlet contraction and also in (2) moderate degree of inlet contraction associated with outlet contraction or complicating factors like elderly primigravida, malpresentation, post-cesarean pregnancy, etc. **If there is no doubt about the maturity of the fetus**, the operation is done in planned way any time during last week of pregnancy. **In doubtful maturity**, investigations are done to ascertain maturity (see p. 372); otherwise the operation is withheld till the pains start or the membranes rupture, whichever occurs early.

TRIAL LABOR

Definition: It is the conduction of spontaneous labor in a moderate degree of cephalopelvic disproportion, in an institution under supervision with watchful expectancy, hoping for a vaginal

delivery. Every arrangement should be made available for operative delivery, either vaginal or abdominal, if the condition so arises.

Aims: A trial labor aims at avoiding an unnecessary cesarean section and at delivering a healthy baby.

The phrase "trial" was used originally to test for pelvic adequacy but subsequently its use has been extended to test numerous factors other than the pelvic capacity. For example, the trial is conducted to test the integrity of the scar in a woman with prior cesarean delivery when she goes into labor.

Contraindications: (1) Associated midpelvic and outlet contraction; (2) Presence of complicating factors like elderly primigravida, malpresentation, postmaturity, post-cesarean pregnancy, pre-eclampsia, medical disorders like heart disease, diabetes, tuberculosis, etc.; (3) Where facilities for cesarean section is not available round the clock.

Conduction of trial labor: The management of a trial labor requires careful supervision and consideration. The following guidelines are prescribed.

- **The labor should ideally be** spontaneous in onset. But in cases where the labor fails to start even on due date, induction of labor may be done.
- **Oral feeding** remains suspended and hydration is maintained by intravenous drip. Adequate analgesic is administered (p. 157).
- **The progress of the labor is mapped with a partograph** (p. 606)—(a) progressive descent of the head (see p. 156) and (b) progressive dilatation of the cervix (partograph p. 606).
- **To monitor the maternal health** (p. 158). Fetal monitoring is done clinically and/or using EFM (see p. 692).
- **If there is failure to progress** due to inadequate uterine contraction, augmentation of labor may be done by amniotomy along with oxytocin infusion. **On no account should the procedure be employed before the cervix is at least 3 cm (2 fingers) dilated**.
- **After the membranes rupture**, pelvic examination is to be done: (a) To exclude cord prolapse; (b) To note the color of liquor; (c) To assess the pelvis once more and (d) To note the condition of the cervix including pressure of the presenting part on the cervix.

Successful outcome depends on: (1) Degree of pelvic contraction; (2) Shape of the pelvis—flat pelvis is better than android or generally contracted pelvis; (3) Favorable vertex presentation—anterior parietal presentation with less parietal obliquity is favorable; (4) Intact membranes till full dilatation of cervix; (5) Effective uterine contractions and (6) Emotional stability of the woman.

Unfavorable features: (1) Appearance of abnormal uterine contraction; (2) Cervical dilatation less than 1 cm per hour in the active phase (protracted active phase); (3) Descent of fetal head less than 1 cm per hour (protracted active phase) inspite of regular uterine contractions; (4) Arrest of cervical dilatation and nondescent of fetal head in spite of oxytocin therapy; (5) Early rupture of the membranes; (6) Formation of caput and evidence of excessive molding; (7) Fetal distress.

How long the trial to be continued? It is indeed difficult to set an arbitrary time limit which is applicable to all cases. **One should individualize the case**. So long as the progress is satisfactory (evidenced by descent of the head and progressive cervical dilatation) and the maternal and fetal condition remain good, trial may be continued safely. However, if any ominous feature appears, trial is to be terminated forthwith. Nowadays, there is a tendency to shorten the duration of trial. In spite of adequate uterine contractions, if there is arrest of descent or dilatation of the cervix for a reasonable period (3-4 hours) in the active phase, labor is terminated by cesarean section.

Termination of trial labor: The methods of termination are any one of the following:

- **Spontaneous delivery** with or without episiotomy (30%).
- **Forceps or ventouse (30%)**—Difficult forceps delivery is to be avoided.
- **Cesarean section (40%)**—Judicious and timely decision for cesarean delivery is to be taken. However, in significant cases, the section is done even before full dilatation of the cervix, the indication being uterine inertia or fetal distress.

Successful trial: A trial is called successful, if a healthy baby is born vaginally, spontaneously or by forceps or ventouse with the mother in good condition. Delivery by cesarean section or delivery of a dead baby, spontaneously or by craniotomy, is called failure of trial labor.

Advantages of trial labor: (1) It eliminates unnecessary cesarean section electively decided upon; (2) It eliminates injudicious use of premature induction of labor with its antecedent hazards; (3) A successful trial ensures the woman a good future obstetrics.

Disadvantages of trial labor: (1) **Test of disproportion remains unproven** when cesarean delivery is done due to fetal distress or uterine dysfunction; (2) **Increased perinatal morbidity or mortality** due to asphyxia or intracranial hemorrhage when the trial is prolonged and/or ends in difficult delivery; (3) **Increased maternal morbidity** due to the effects of prolonged labor and/or operative delivery; (4) Increased psychological morbidity when trial ends with a traumatic vaginal delivery or in cesarean delivery.

MIDPELVIC AND OUTLET DISPROPORTION

In clinical assessment, it is difficult to determine where the midpelvis ends and outlet begins. **Moreover, isolated outlet contraction without midpelvic contraction is a rarity**. As such, in practice the two problems are jointly considered as outlet contraction. **Cephalopelvic disproportion at the outlet is defined as one where the biparietal-suboccipitobregmatic plane fails to pass through the bispinous and anteroposterior planes of the outlet**.

Management: Unlike inlet disproportion, clinical diagnosis of midpelvic and outlet disproportion can only be made after the head sufficiently comes down into the pelvis.

(1) **Elective cesarean section**: Contraction of both the transverse and anteroposterior diameters of the midpelvic plane or minor contraction associated with other complicating factors is dealt by elective cesarean section.

(2) **To allow vaginal delivery**: In otherwise uncomplicated cases with minor contraction, vaginal delivery is allowed under supervision with watchful expectancy. Molding and adaptation of the head and "give" of the pelvis may allow the head to pass through the contracted zone. **Delivery** is accomplished by **forceps** or **ventouse** with deep episiotomy to prevent perineal injuries, especially with narrow pubic arch. Labor progress should be mapped with a **partograph** to make an early diagnosis of dysfunctional labor due to disproportion. Oxytocin may be used to augment labor for adequate uterine contractions.

If there is no dilatation of cervix or descent of the fetal head after a period of 2 hours in the active phase of labor, arrest of labor is considered. Once arrest disorder is diagnosed, cesarean delivery is the option.

CASES SEEN LATE IN LABOR is not an uncommon problem in the developing countries. The **principles of management** rest on: (i) **Cesarean section** to avoid difficult forceps; (ii) **Forceps** with deep episiotomy; (iii) **Symphysiotomy** (see p. 679) followed by ventouse or (iv) **Craniotomy** if the fetus is dead.

QUESTIONS

1. Define cephalopelvic disproportion (CPD)? (p. 409)
2. What are the methods of diagnosis of CPD? (p. 410)

Related theory questions (Long & Short), Obstetric Case Discussions, Viva table discussions, Post operative word round discussions, and MCQs are discussed in author's books:

1. **Bedside Clinic and Viva Voce:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

For further reading:

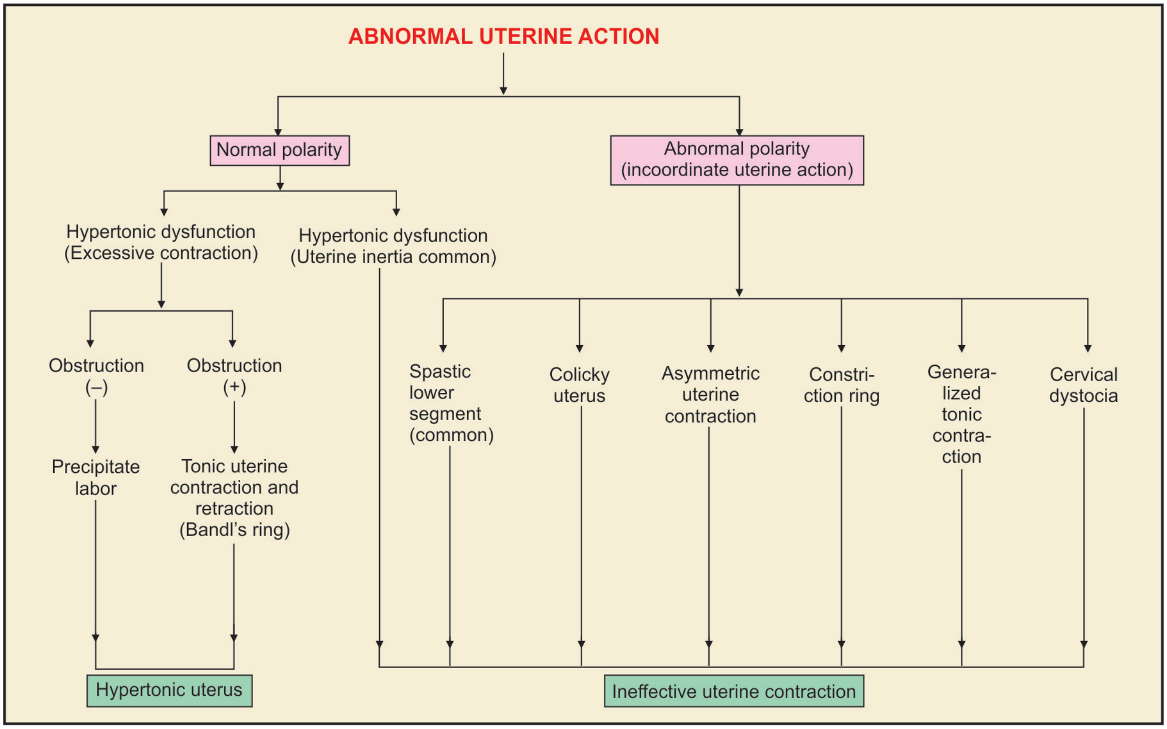
2. **Master pass in Obstetrics and Gynaecology:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

Chapter 25

Abnormal Uterine Action

Normal labor is characterized by **coordinated uterine contractions (Fig. 25.2A)** associated with progressive dilatation of the cervix and descent of the fetal head. Normal labor is associated with cervical dilatation more than or equal to 1 cm/hr in a nulliparous woman and 1.5 cm/hr for a multiparous woman. This results in successful vaginal delivery. Overall labor abnormalities occur in about 25% of the nulliparous women and 10% of multiparous women. Abnormal active phase of labor may be protraction or arrest disorder (see p. 463). The most common cause of protraction disorder is inadequate or abnormal uterine contractions. **Any deviation of the normal pattern of uterine contractions (as mentioned in page 138) affecting the course of labor is designated as disordered or abnormal uterine action**.

TYPES: The following are the different types (see below Flowchart):


ABNORMAL UTERINE ACTION
Normal polarity
Abnormal polarity (incoordinate uterine action)
Hypertonic dysfunction (Excessive contraction)
Hypertonic dysfunction (Uterine inertia common)
Obstruction (–)
Obstruction (+)
Precipitate labor
Tonic uterine contraction and retraction (Bandl's ring)
Spastic lower segment (common)
Colicky uterus
Asymmetric uterine contraction
Constriction ring
Generalized tonic contraction
Cervical dystocia
Hypertonic uterus
Ineffective uterine contraction

ETIOLOGY: As the physiology of normal uterine contraction is not fully understood, the cause of its disordered action remains obscure. **However, the following clinical conditions are often associated**: (1) Prevalent in first birth, especially with elderly women; (2) Prolonged pregnancy; (3) Overdistension of the uterus (twins and fibroids); (4) Emotional factor (anxiety, stress); (5) Constitutional factor (obesity); (6) Contracted pelvis and malpresentation; (7) Injudicious administration of sedatives, analgesics and oxytocics; (8) Premature attempt at vaginal delivery (induction of labor or ARM) or attempted instrumental vaginal delivery under light anesthesia.

Normal uterine contractions: Polarity of the uterus means when the upper segment contracts, the lower segment relaxes. Normally, there are two **pacemakers**, one is situated at each cornua of the uterus. The uterine pacemakers generate uterine contractions in a coordinated fashion. The properties of a normal uterine contraction wave are: (i) The intensity of contraction diminishes from top to bottom of the uterus; (ii) The contraction wave starts from the pacemaker and propagates towards the lower uterine segment; (iii) The duration of contraction diminishes progressively as the wave moves away from the pacemaker. In dysfunctional labor, new pacemakers may come up anywhere in the uterus.

The uterine pacemaker is situated at the cornua of the uterus and this generates uterine contractions. Effective uterine contraction, **starts at the cornua and gradually sweeps downwards over the uterus (see Fig. 25.1)**. In a **primary dysfunctional labor**, uterine activity instead of being governed by a single dominant pacemaker, is shifted to less efficient contractions due to emergence of other pacemaker foci. Oxytocin therapy may be effective in restoring **the global and effective uterine contractions**.

Primary dysfunctional labor is defined when the cervix dilates less than 1 cm/hr following a normal latent phase of labor (see Fig. 27.1). It is the most common abnormality and mostly corrected by amniotomy and/or oxytocin augmentation. **Secondary arrest** is defined when the cervical dilatation stops after the active phase of labor has started normally. **Secondary arrest of dilatation may be due to** (a) Poor uterine contractions (myometrial fatigue), (b) Cessation of cervical dilatation despite strong uterine contractions (mechanical factors like disproportion and malpresentation).

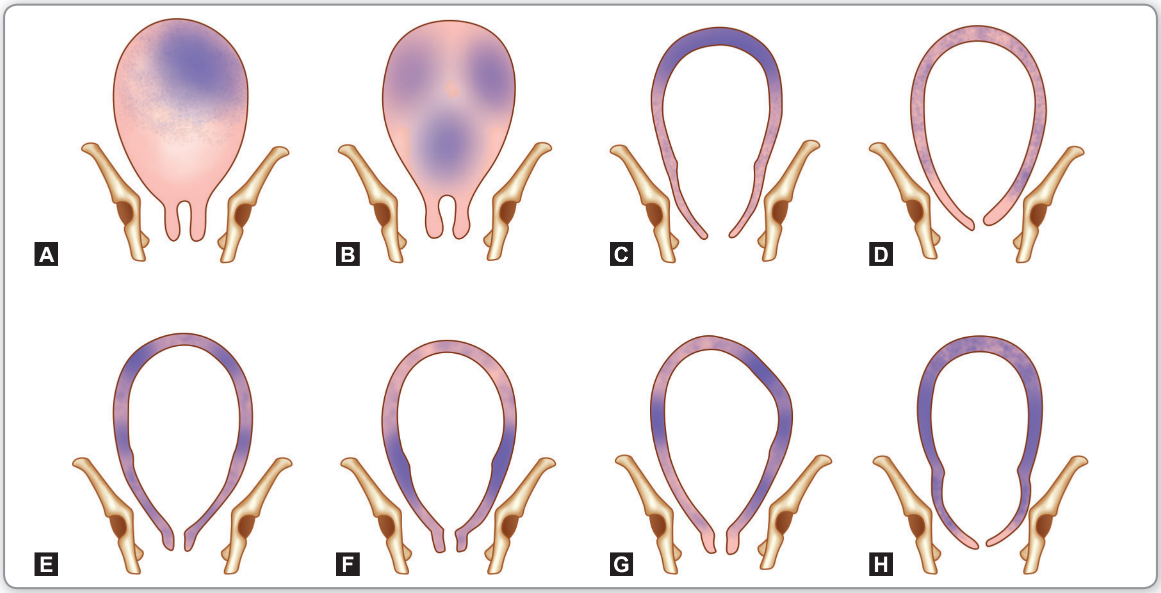
Uterine activity (contraction) is measured by noting (i) basal tone (ii) active (peak) pressure and (iii) frequency. **Assessment is usually done by**—(i) Clinical palpation—(inaccurate), (ii) Tocodynamometer with external transducer, (iii) Intrauterine pressure catheter (IUPC) is used to measure intrauterine pressure during uterine contractions. Normal baseline tonus is between 5 mm Hg and 20 mm Hg. Minimum uterine pressure required to dilate the cervix is 15 mm Hg over the baseline. Normal uterine contractions in labor create an intrauterine pressure up to 60 mm Hg. Oxytocin is to be used when uterine contractions are inadequate. Oxytocin dose is to be escalated till the optimum uterine contractions (3–4 per 10 minutes) with a peak intrauterine pressure of 50–60 mm Hg and a resting tone of 10–15 mm Hg is obtained.

UTERINE INERTIA (HYPOTONIC UTERINE DYSFUNCTION)

Uterine inertia is the **common type of abnormal uterine** contraction but is comparatively less serious. It may complicate any stage of labor. It may be present from the beginning of labor or may develop subsequently after a variable period of effective contractions.

UTERINE CONTRACTION: The intensity is diminished; **duration** is shortened; **good relaxation** in between contractions and **the intervals** are increased. General pattern of uterine contractions of labor is maintained (Fig. 25.1) but intrauterine pressure during contraction is less than 25 mm Hg.

DIAGNOSIS: (1) **Patient feels** less pain during uterine contraction; (2) **Hand placed over the uterus** during uterine contraction reveals less hardening of the uterus; (3) **Uterine wall is easily indentable** at the acme of a pain; (4) **Uterus becomes relaxed** after the contraction, fetal parts are well palpable and fetal heart rate remains normal; (5) **Internal examination reveals**—(a) Poor dilatation of the cervix



Figs 25.1A to H: Normal and abnormal patterns of uterine contractions (dark shade indicating strong contraction)—(A) Normal uterine contractions with single dominant pacemaker focus; (B) Uterus with three separate pacemakers firing sequentially; (C) Normal uterine contraction; (D) Uterine inertia; (E) Colicky uterus; (F) Spastic lower segment; (G) Asymmetrical contraction; (H) Cervical dystocia

(normal rate of dilatation in primigravida should be at the rate of 1 cm/hr beyond 4 cm dilatation); (b) Presence of cephalopelvic disproportion, malposition, deflexed head or malpresentation may be evident; (c) Membranes usually remain intact.

EFFECTS ON THE MOTHER AND FETUS: Maternal exhaustion and/or fetal distress are unusual and appear late.

MANAGEMENT: Case is **reassessed to exclude cephalopelvic disproportion or malpresentation**.

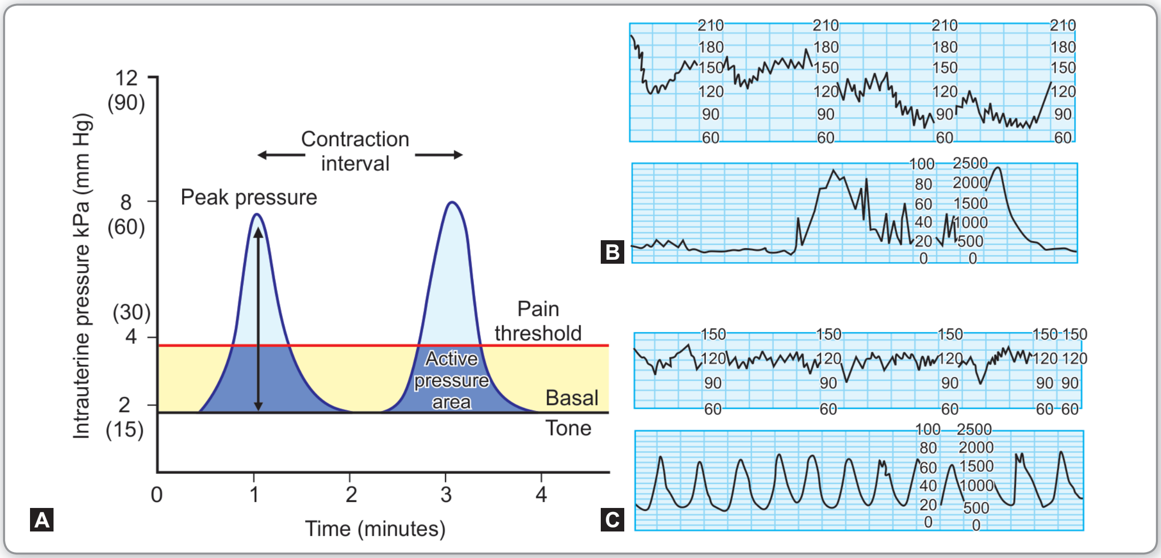
Place of cesarean section: (1) Presence of contracted pelvis (2) Malpresentation (3) Evidences of fetal or maternal distress.

Vaginal delivery — (A) ***General measures:*** (1) To keep up the morale of the patient. Maternal stress, pain and anxiety appear to inhibit uterine contractions through release of endogenous catecholamines. (2) Posture of the woman is changed. **Supine position is avoided** (see p. 159). (3) To empty the bladder, catheterization is made. (4) To maintain hydration by infusion of Ringer's solution. (5) Adequate pain relief (see p. 157).

(B) ***Active measures***: Acceleration of uterine contraction can be brought about by low rupture of the membranes followed by oxytocin drip. The drip rate is gradually increased until effective contractions are set up (see Chapter 35). **The drip is to be continued till 1 hour after delivery**.

INCOORDINATE UTERINE ACTION

It usually appears in active stage of labor. The **hypertonic state of the uterus** arises from any of the conditions such as spastic lower uterine segment, colicky uterus, asymmetrical uterine contraction, constriction ring or generalized tonic contraction of the uterus and all these states are collectively called **incoordinate uterine action**. **Increased frequency and/or duration of uterine contractions**



Figs 25.2A to C: (A) Normal uterine contractions showing peak pressure, contraction interval, pain threshold and rise of basal tone; (B) Hypertonic contractions with prolonged duration; and (C) Hypertonic contractions with increased frequency—both (B and C) showing changes in the fetal heart rate (decelerations) on CTG

cause rise in baseline tone and thereby diminish circulation in the placental intervillous space. These contractions fail to make progressive cervical effacement and dilatation. Frequent contraction of low amplitude causes elevation of basal intrauterine pressure. There is often maternal discomfort. Aminotomy with or without oxytocin augmentation is usually done when the women in the active phase of labor. Conservative management is done if it occurs in the latent phase. Uterine tonus is elevated. Pain is present before, during and after contractions. **This results in** fetal hypoxia in labor. **Placental abruption** is often associated with high baseline tone (> 25 mm Hg). **On CTG the FHR shows reduced variability and late decelerations (Figs 25.2B and C)**. **Uterine hyperstimulation due to oxytocics** (oxytocin, prostaglandins) are often associated with fetal tachycardia (fetal adrenergic activity) due to fetal stress. Constriction ring, generalized tonic uterine contraction and cervical dystocia have got their own separate clinical entity and as such will be discussed separately.

SPASTIC LOWER SEGMENT— UTERINE CONTRACTION: (1) **Fundal dominance** is lacking and often there is reversed polarity (see Fig. 25.1); (2) The pacemakers do not work in rhythm; (3) The lower segment contractions are stronger; (4) **Inadequate relaxation** in between contractions; (5) **Basal tone is raised** above the critical level of 20 mm Hg (Fig. 25.2A).

Diagnosis: (1) **The patient is in agony** with unbearable pain referred to the back. There are evidences of dehydration and ketoacidosis; (2) **Bladder is frequently distended** and often there is retention of urine; distension of the stomach and bowels are visible; (3) There are **premature attempts to bear down**; (4) **Abdominal palpation reveals**: (a) Uterus is tender and gentle manipulation excites hardening of the uterus with pain, (b) palpation of the fetal parts is difficult, (5) Fetal distress appears early; (6) **Internal examination may reveal:** (a) Cervix which is thick, edematous hangs loosely like a curtain; not well applied to the presenting part, (b) Inappropriate dilatation of the cervix, (c) Absence of the membranes, (d) Meconium stained liquor amnii may be there.

Effect on the fetus: **Fetal distress** appears early due to placental insufficiency caused by inadequate relaxation of the uterus.

Management: **There is no place of oxytocin augmentation with this abnormality**. Cesarean section is done in majority of cases. Prior correction of dehydration and ketoacidosis must be achieved by rapid infusion of Ringer's solution.

CONSTRICTION RING (***Syn*: *Contraction ring, Schroeder's ring)***: It is one form of incoordinate uterine action where there is localized myometrial contraction forming a ring of circular muscle fibers of the uterus. **It is usually situated at the junction of the upper and lower segment around** a constricted part of the fetus usually around the neck in cephalic presentation (Fig. 25.3). It may appear in all the stages of labor. It is usually reversible and complete.

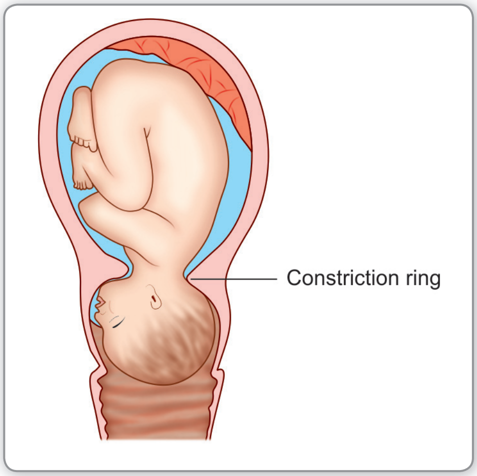


Fig. 25.3: Constriction ring

The common causes are: (1) injudicious administration of oxytocics, (2) premature rupture of the membranes, and (3) premature attempt at instrumental delivery.

Diagnosis: Diagnosis is difficult. **It is revealed during** cesarean section in the first stage, during forceps application in the second stage and during manual removal in the third stage (hour-glass contraction). **The ring is not felt per abdomen**. Maternal condition is not much affected but the fetus is in jeopardy because of the hypertonic state. Uterus never ruptures.

Treatment: Delivery is usually done by cesarean section. The ring usually passes off by deepening the plane of anesthesia otherwise the ring may have to be cut vertically to deliver the baby. The difficulties faced during forceps delivery (second stage) or during normal removal of placenta (third stage) can be overcome by using deep anesthesia that relaxes the constriction ring.

CERVICAL DYSTOCIA: Progressive cervical dilatation needs an effective stretching force by the presenting part. Failure of cervical dilatation may be due to—(a) Inefficient uterine contractions (see p. 140), (b) Malpresentation, malposition (abnormal relationship between the cervix and the presenting part), (c) Spasm (contractions) of the cervix. **Cervical dystocia may be primary or secondary**.

Primary: Commonly observed during the (i) First birth where the external os fails to dilate, (ii) Rigid cervix, (iii) Inefficient uterine contractions and the others (as mentioned earlier).

Treatment: In presence of associated complications (malpresentation, malposition), cesarean section is preferred. **If the head is sufficiently low down** with only thin rim of cervix left behind, the rim may be pushed up manually during contraction or traction is given by ventouse. In others, where the cervix is very much thinned out but only half dilated. Dührssen's incision at 2 and 10'O clock positions followed by forceps or ventouse extraction is quite safe and effective.

SECONDARY CERVICAL DYSTOCIA: This type of cervical dystocia results usually due to excess scarring or rigidity of the cervix from the effect of previous operation or disease (see Chapter 21). Others are: (i) Post-delivery (ii) Postoperative scarring (iii) Cervical cancer.

GENERALIZED TONIC CONTRACTION ***(Syn: Uterine tetany)***: In this condition, pronounced retraction occurs involving whole of the uterus up to the level of internal os. **Thus, there is no physiological differentiation of the active upper segment and the passive lower segment of the uterus**. The whole uterus undergoes a sort of tonic muscular spasm holding the fetus inside (active retention of the fetus) (Fig. 25.4). Usually there is no risk of rupture uterus. New pacemakers appear all over the uterus.

Causes: (i) Cephalopelvic disproportion (ii) Obstruction (iii) Injudicious use of oxytocics.

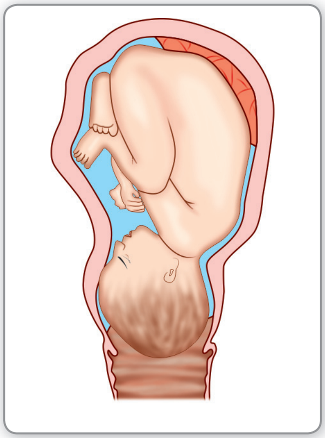


Fig. 25.4: Generalized tonic contraction of the uterus

Clinical features: The patient is in prolonged labor having severe and continuous pain. Abdominal examination reveals the uterus to be somewhat smaller in size, tense and tender. Fetal parts are neither well defined, nor is the fetal heart sound audible. Vaginal examination reveals jammed head with big caput, dry and edematous vagina.

Treatment:

- **Correction of dehydration and ketoacidosis**—by rapid infusion of Ringer's solution
- **Antibiotic**—to control infection
- **Adequate pain relief** (see p. 157).

Hypercontractility (tachysystole) may be induced by oxytocics (>5 contractions in 10 min). It may occur in spontaneous or with stimulated labor. Persistent tachysystole with FHR abnormality can cause fetal hypoxia. It can be managed by tocolytics (Terbutaline 0.25 mg SC). Oxytocin infusion should be stopped.

Cesarean delivery is done in majority of the cases, especially when obstruction is suspected.

PRECIPITATE LABOR

A labor is called precipitate when the combined duration of the first and second stage is less than 3 hours. Prevalence is about 2%. Short labors may be associated with: placental abruption and uterine tachysystole. It is common in multiparae and may be repetitive. Rapid expulsion is due to the combined effect of hyperactive uterine contractions associated with diminished soft tissue resistance. **Labor is short** as the rate of cervical dilatation is 5 cm/hr or more for the nulliparous women.

Maternal risks include: (1) Extensive laceration of the cervix, vagina and perineum (to the extent of complete perineal tear), (2) PPH due to uterine hypotonia that develops subsequent to unusual vigorous contractions, (3) Inversion, (4) Uterine rupture, (5) Infection, (6) Amniotic fluid embolism. **The fetal risks include**—intracranial stress and hemorrhage because of rapid expulsion without time for molding of the head. The baby may sustain serious injuries if delivery occurs in standing position; bleeding from the torn cord and direct hit on the skull, brachial plexus injury are real hazards.

Treatment: The patient having previous history of precipitate labor should be hospitalized prior to labor. During labor, the uterine contraction may be suppressed by administering ether or magnesium sulfate during contractions. **Delivery of the head should be controlled**. Episiotomy should be done liberally. Elective induction of labor by low rupture of membranes and conduction of controlled delivery is helpful. Oxytocin augmentation should be avoided.

TONIC UTERINE CONTRACTION AND RETRACTION

(Syn: Bandl's ring, Pathological retraction ring)

This type of uterine contraction is predominantly due to obstructed labor.

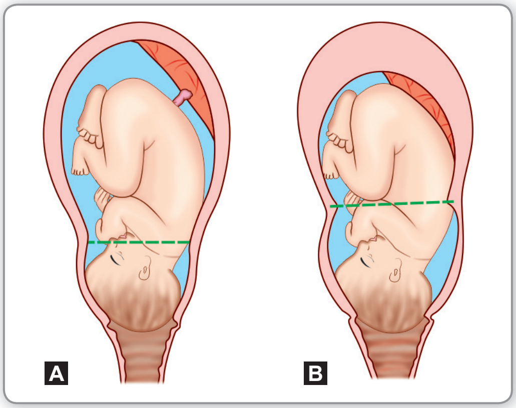
Pathological anatomy of the uterus: There is gradual increase in intensity, duration and frequency of uterine contraction. The relaxation phase becomes less and less; ultimately a state of tonic contraction develops. Retraction, however, continues. The lower segment elongates and becomes progressively thinner to accommodate the fetus driven from the upper segment (Fig. 25.5). **A circular groove encircling the uterus is formed between the active upper segment and the distended lower segment, called pathological retraction ring (Bandl's ring)**. Due to pronounced retraction, there is fetal jeopardy or even death.

In primigravidae, further retraction ceases in response to obstruction and labor comes to a stand still—a state of uterine exhaustion. Contractions may recommence after a brief period of rest with renewed vigor. **But in multiparae**, retraction continues with progressive circumferential dilatation and thinning of the lower segment. There is progressive rise of the Bandl's ring, moving nearer and nearer to the umbilicus and ultimately, the lower segment ruptures (Fig. 26.33).

Clinical features: (1) **Patient is in agony** from continuous pain and discomfort and becomes restless (2) **Features of exhaustion** and ketoacidosis are evident (3) **Abdominal palpation reveals**—(a) Upper segment is hard and tender (b) Lower segment is distended and tender.

For other features—see Table 25.1.

Management: Prevention—Partographic management of labor, early diagnosis of malpresentation, disproportion and delivery by cesarean section can prevent this condition completely.



Figs 25.5A and B: Pathogenesis of retraction ring (Bandl's ring). (A) Normal labor; (B) Late obstruction. Note the circumferential dilatation and progressive stretching of the lower segment with corresponding thickening of the upper segment and rise in the level of retraction ring following obstruction

Treatment:

- **Rupture of uterus** is to be excluded
- **Internal version is contraindicated**
- **Correction of dehydration and ketoacidosis** by infusion of Ringer's solution
- **Adequate pain relief** (see p. 157)
- **Parenteral antibiotic** is given (Ceftriaxone 1 g IV)
- **Cesarean delivery** is done in majority of the cases. Rupture of uterus must be excluded before attempting destructive operation.

Table 25.1 : Difference Between Constriction Ring and Retraction Ring

	Constriction Ring	**Retraction Ring**
Nature	It is a manifestation of localized incoordinate uterine contraction	It is an end result of tonic uterine contraction and retraction
Cause	Undue irritability of the uterus	Following obstructed labor
Situation	Usually at the junction of upper and lower segment but may occur in other places. Once formed the position does not alter	Always situated at the junction of upper and lower segment The position progressively moves upwards
Uterus	Upper segment contracts and retracts with relaxation in between; lower segment remains thick and loose. (Fig. 25.3) ▪ Polarity is abnormal (p. 416)	Upper segment is tonically contracted with no relaxation The wall becomes thicker; lower segment becomes distended and thinned out (Fig. 25.5) ▪ Polarity is normal
Maternal condition	Almost unaffected unless the labor is prolonged	Features of maternal exhaustion, sepsis appear early
Abdominal examination	(a) Uterus feels normal and not tender (b) Fetal parts are easily felt (c) Ring is not felt (d) Round ligament is not felt (e) FHS is usually present	(a) Uterus is tense and tender (b) Not easily felt (c) Ring is felt as a groove placed obliquely (d) Round ligaments are taut and tender (e) Usually absent
Vaginal examination	(a) Lower segment is not pressed by the presenting part (b) Ring is felt usually above the head (c) Features of obstructed labor are absent	(a) Lower segment is very much pressed by the forcibly driven presenting part (b) Ring cannot be felt vaginally (c) Features are present

contd...

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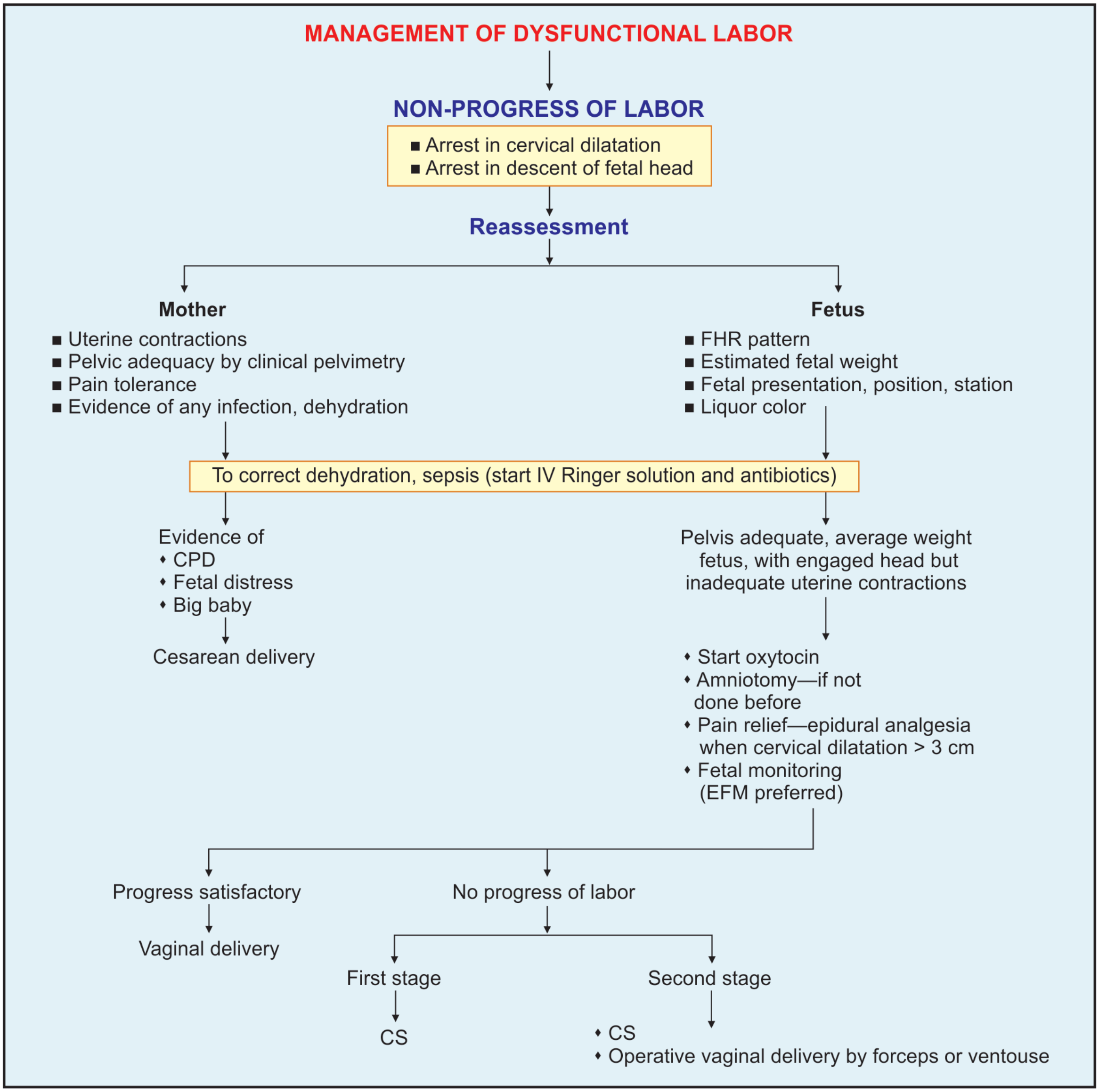
Table 25.1 : Difference Between Constriction Ring and Retraction Ring

	Constriction Ring	Retraction Ring
End result	(a) Maternal exhaustion is a late feature (b) Fetal anoxia usually appears late (c) Chance of uterine rupture is absent	(a) Maternal exhaustion and sepsis appear early (b) Fetal anoxia and even death are usually early (c) Rupture uterus in multigravidae is common
Principle of treatment	To relax the ring followed by delivery of the baby or to cut the ring during cesarean section. Cesarean delivery and to cut the ring, if needed	Cesarean delivery after excluding rupture uterus


MANAGEMENT OF DYSFUNCTIONAL LABOR
NON-PROGRESS OF LABOR
■ Arrest in cervical dilatation
■ Arrest in descent of fetal head
Reassessment
Mother
■ Uterine contractions
■ Pelvic adequacy by clinical pelvimetry
■ Pain tolerance
■ Evidence of any infection, dehydration
Fetus
■ FHR pattern
■ Estimated fetal weight
■ Fetal presentation, position, station
■ Liquor color
To correct dehydration, sepsis (start IV Ringer solution and antibiotics)
Evidence of
♦ CPD
♦ Fetal distress
♦ Big baby
Cesarean delivery
Pelvis adequate, average weight fetus, with engaged head but inadequate uterine contractions
♦ Start oxytocin
♦ Amniotomy—if not done before
♦ Pain relief—epidural analgesia when cervical dilatation > 3 cm
♦ Fetal monitoring (EFM preferred)
Progress satisfactory
Vaginal delivery
No progress of labor
First stage
CS
Second stage
♦ CS
♦ Operative vaginal delivery by forceps or ventouse

SUMMARY

Abnormal Uterine Action is due to development of abnormal polarity on the uterus. It may manifest as uterine inertia (common) or hypertonic dysfunction due to any mechanical factor (obstruction). Hypertonic dysfunction may end in either formation of Bandl's ring or precipitate labor. Incoordinate uterine action (asymmetric uterine contractions, constriction ring and cervical dystocia) can affect the health of both the mother and the fetus adversely. It is important to detect AUA early and to institute management appropriately to reduce maternal and neonatal morbidity and mortality.

Preventive Measures of Dystocia due to Abnormal Uterine Action:

(1) Quality antenatal care, emotional support to be parturient and close monitoring of labor can reduce abnormal uterine action.

(2) Induction of labor should be judicious, especially when the cervix is unfavorable.

(3) Amniotomy in the latent phase or as a routine procedure is to be avoided.

(4) During the course of labor the woman should be given adequate moral support, rest and analgesic. Her hydration should be maintained.

(5) Management of labor should be plotted partographically so that any deviation from the normal is detected and managed early.

KEY POINTS

- **Abnormal uterine action** is an important cause of abnormal labor. Abnormal uterine actions are due to many factors and are of different types (see p. 415). Slow progress of labor is commonly due to uterine inertia (see p. 416).
- **Supine position during labor should be avoided**. Left lateral tilt is preferable.
- **In a primary dysfunctional labor**, uterine contractions are less efficient as there is emergence of other pacemakers instead of a dominant single one.
- **Oxytocin is often effective** to correct the underlying pathology and to restore global uterine contractions.
- **Uterine contractions** are best assessed by measuring the intrauterine pressure. Manual assessment of uterine activity is inaccurate. It is the peak uterine contraction pressure that causes progress of labor (Fig. 25.2A).
- **Constriction ring dystocia** affects mainly the fetus whereas **retraction ring dystocia** affects both the fetus and the mother adversely (see p. 419). Rupture of uterus must be excluded in Bandl's (retraction) ring dystocia (see p. 420).
- **Cesarean deliveries** for dystocia should not be done unless adequate uterine contractions have been achieved (see p. 419).

QUESTIONS

Related theory questions (Long and Short), Obstetric Case Discussions, Viva table discussions, Post operative word round discussions, and MCQs are discussed in author's books:

1. **Bedside Clinic and Viva Voce:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

For further reading:

2. **Master pass in Obstetrics and Gynaecology:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

Chapter 26

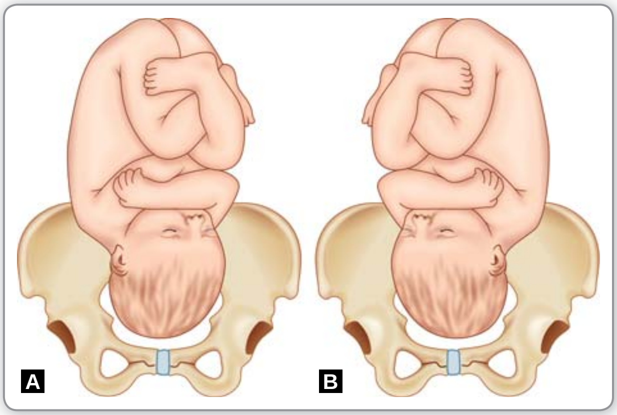
COMPLICATED LABOR-Malposition, Malpresentation and Cord Prolapse

OCCIPUT-POSTERIOR POSITION (OP)

Malposition refers to any position of the vertex other than flexed occipitoanterior one.

In a vertex presentation where the occiput is placed posteriorly over the sacroiliac joint or directly over the sacrum, it is called an **occiput-posterior position**. When the occiput is placed over the right sacroiliac joint, the position is called right occipitoposterior (ROP), traditionally called third position of the vertex and when placed over the left sacroiliac joint, is called left occipitoposterior (LOP), traditionally called fourth position of the vertex (Fig. 26.1) and when it points toward the sacrum, is called direct occipitoposterior. All the three positions may be primary (present before the onset of labor) or secondary (developing after labor starts).

Occiput-posterior is an abnormal position of the vertex rather than an abnormal presentation. In majority of cases (90%), anterior rotation of the occiput occurs and follows the course like that of an occipito-anterior and moreover, in certain type of pelvis (anthropoid), it is a favorable position. **But as the posterior position occasionally gives rise to dystocia, it is described along with malpresentation**.



Figs 26.1A and B: (A) Right occipitoposterior position (B) Left occipitoposterior position

Incidence: At the onset of labor, the incidence is about 10% of all the vertex presentations. The incidence is expected to be more during late pregnancy and is much less in late second stage of labor. Right occipito-posterior is five times more common than the left occipitoposterior. Dextrorotation of the uterus and the presence of the sigmoid colon on the left disfavor LOP position.

Cause: In majority, the cause of the abnormal position is not clear. **The following are the responsible factors:**

- **Shape of the pelvic inlet:** The shape of the inlet significantly determines the position of the head at the onset of labor. In more than 50%, the occipitoposterior position is associated with **either an anthropoid or android pelvis**. The wide occiput can comfortably be placed in the wider posterior segment of the pelvis.
- **Fetal factors:** Marked deflection of the fetal head, too often favors posterior position of the vertex. **The causes of deflexion are:** (1) High pelvic inclination. (2) Attachment of the placenta on the anterior wall of the uterus—this favors the well-flexed fetus ovoid looking toward the anterior wall of the uterus, i.e. remains in dorsoposterior position. Thus, the convexities of the fetal and maternal spines are apposed, leading to tendency of extension of the fetal spines with persistent deflexed attitude of the head, (3) primary brachycephaly—this shortens the length of the lever from the frontal to atlantooccipital joint, and thereby diminishes the effective movement of flexion.
- **Uterine factor:** Abnormal uterine contraction, which may be the cause or effect, leads to persistent deflexion and occipitoposterior position.

DIAGNOSIS

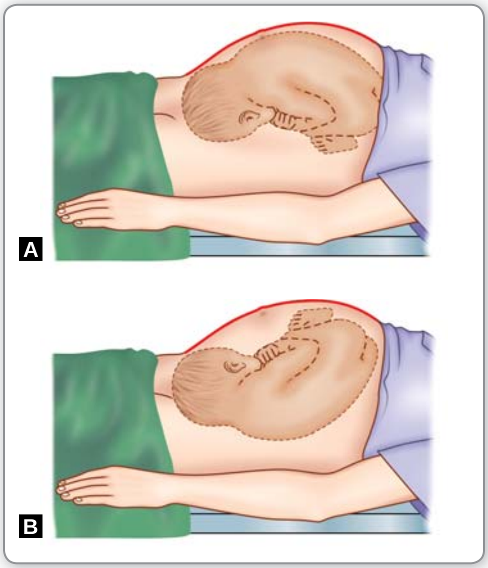
ABDOMINAL EXAMINATION

Inspection: The abdomen looks flat, below the umbilicus (Fig. 26.2).

Umbilical grip: The findings are: (1) The fetal limbs are more easily felt near the midline on either side. (2) The fetal back is felt far away from the midline on the flank and often difficult to outline clearly. (3) The anterior shoulder lies far away from the midline.

Pelvic grips: The findings are: (1) The head is not engaged. (2) The cephalic prominence (sinciput) is not felt so prominent as found in well-flexed occipitoanterior. **In direct occipitoposterior, the small sinciput is confused with breech**.

Auscultation: The maximum intensity of the fetal heart sounds is heard on the flank and often difficult to locate especially in LOP. However, in direct occipitoposterior, the FHS is distinctly felt in the midline.



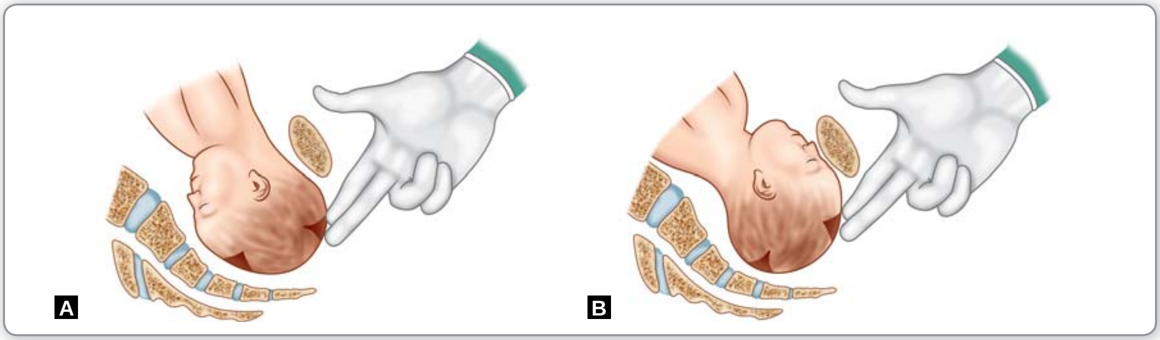
Figs 26.2A and B: Abdominal contour in: (A) Anterior; (B) Posterior positions of vertex presentation

VAGINAL EXAMINATION

The findings in early labor are: (1) Elongated bag of membranes which is likely to rupture during examination. (2) The sagittal suture occupies any of the oblique diameters of the pelvis. (3) Posterior fontanel is felt near the sacroiliac joint. (4) The anterior fontanel is felt more easily because of deflexion of the head and at times, is felt at a lower level than the posterior one (Fig. 26.3).

In late labor, the diagnosis is often difficult because of caput formation which obliterates the sutures and fontanels. In such cases, the ear is to be located and unfolded pinna points toward the occiput. Simultaneous assessment of the pelvis should be done.

Imaging: Ultrasonography is rarely done. It is helpful to know the descent, attitude of the head and its relation to the pelvic walls (position).



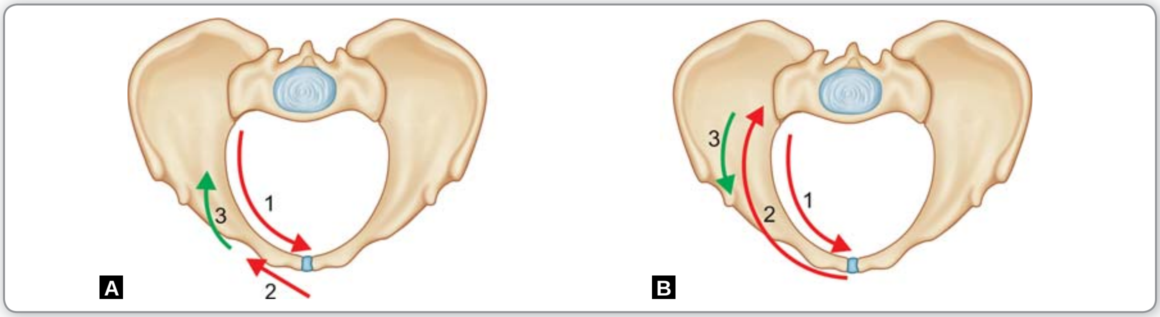
Figs 26.3A and B: Diagnosis of the attitude of the head. (A) Occipitoanterior—well flexed head—posterior fontanel easily felt; (B) Occipitoposterior—deflexed head—anterior fontanel easily felt

MECHANISM OF LABOR

The head engages through the right oblique diameter in ROP and left oblique diameter in LOP. The engaging transverse diameter of the head is biparietal (9.5 cm) and that of anteroposterior diameter is either suboccipitofrontal (10 cm) or occipitofrontal (11.5 cm). **Because of deflexion, engagement is delayed**.

In Favorable Circumstances (90%)

- **Flexion:** Good uterine contractions result in good flexion of the head. Descent occurs until the head reaches the pelvic floor.
- **Internal rotation of the head:** As the occiput is the leading part, it rotates 3/8th of a circle (135°) anteriorly to lie behind the symphysis pubis. **As the neck cannot sustain such amount of torsion, the shoulders rotate about 2/8th of a circle** to occupy the right oblique diameter in ROP and the left oblique in LOP with 1/8th of a circle torsion of the neck still left behind. Thus, the rest of the mechanism is like that of right occipitoanterior in ROP and that of left occipitoanterior in LOP.
- **Further descent** and delivery of the head occurs like that of occipitoanterior position.
- **Restitution:** There is movement of restitution to the extent of 1/8th of a circle in the opposite direction of internal rotation of the head.
- **External rotation:** The external rotation of the head occurs through 1/8th of a circle in the same direction of restitution as the shoulders rotate from the oblique to anteroposterior diameter of the pelvis (Fig. 26.4A).
- **Birth of the shoulders and trunk:** The process of expulsion is the same as that of occipitoanterior.



Figs 26.4A and B: Diagram to illustrate the rotation of the occiput in ROP (3rd) position. (A) Common; (B) Uncommon. 1 = Internal rotation; 2 = Restitution; 3 = External rotation

Alternative mechanism (uncommon) [Fig. 26.4B]: If the shoulders fail to follow the anterior rotation of the occiput, the neck sustains a torsion equal to 3/8th of a circle (135°) and the shoulders remain static in the left oblique diameter in ROP and in the right oblique diameter in LOP. In such cases, restitution occurs 3/8th of a circle and external rotation occurs through 1/8th of a circle in the opposite direction of restitution. However, the mechanism is quite unlikely.

In unfavorable circumstances: (***Nonrotation or malrotation***)—10%.

In certain circumstances, the occiput fails to rotate as described previously (Fig. 26.5). The causes are deflexion of the head, weak uterine contraction, faulty shape of the pelvis such as flat sacrum, prominent ischial spines or convergent side walls and weak pelvic floor muscles. Big baby and immobility of the fetal trunk consequent to the drainage of liquor amnii also contribute to faulty rotation.

- **Incomplete forward rotation:** In this condition, the occiput rotates through 1/8th of a circle anteriorly and the sagittal suture comes to lie in the bispinous diameter. Thereafter, further anterior rotation is unlikely and arrest in this position is called **deep transverse arrest**.
- **Nonrotation:** Both the sinciput and the occiput touch the pelvic floor simultaneously due to moderate deflexion of the head resulting in nonrotation of the occiput. The sagittal suture lies in the oblique diameter. Further mechanism is unlikely and the condition is called **oblique posterior arrest**.
- **Malrotation:** In extreme deflexion, the sinciput touches the pelvic floor first resulting in anterior rotation of the sinciput to 1/8th of a circle and putting the occiput to the sacral hollow. This position is termed as occipitosacral position. **This is, in the true sense, "Persistent Occipitoposterior Position" (POP) of the vertex**. In favorable circumstances, i.e. with an average size baby, good uterine contractions and an adequate pelvis such as an anthropoid or spacious gynecoid—spontaneous delivery may occur as "face to pubis". In unfavorable circumstances, when arrest occurs, it is called **occipitosacral arrest**.

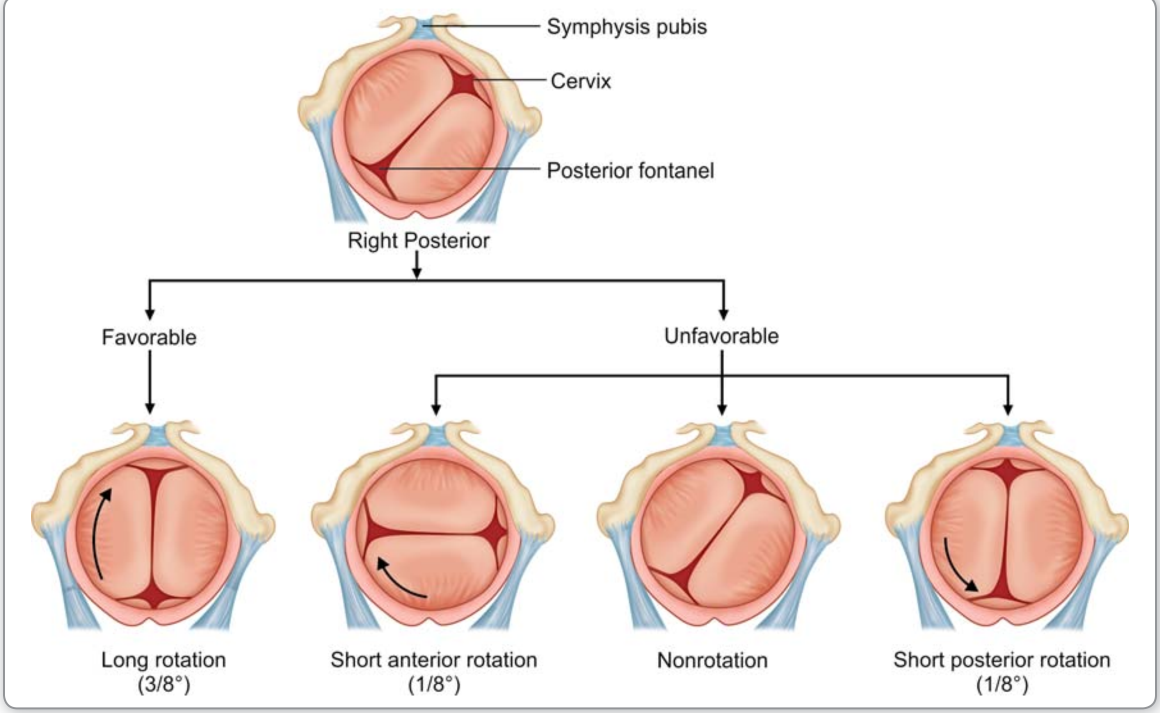
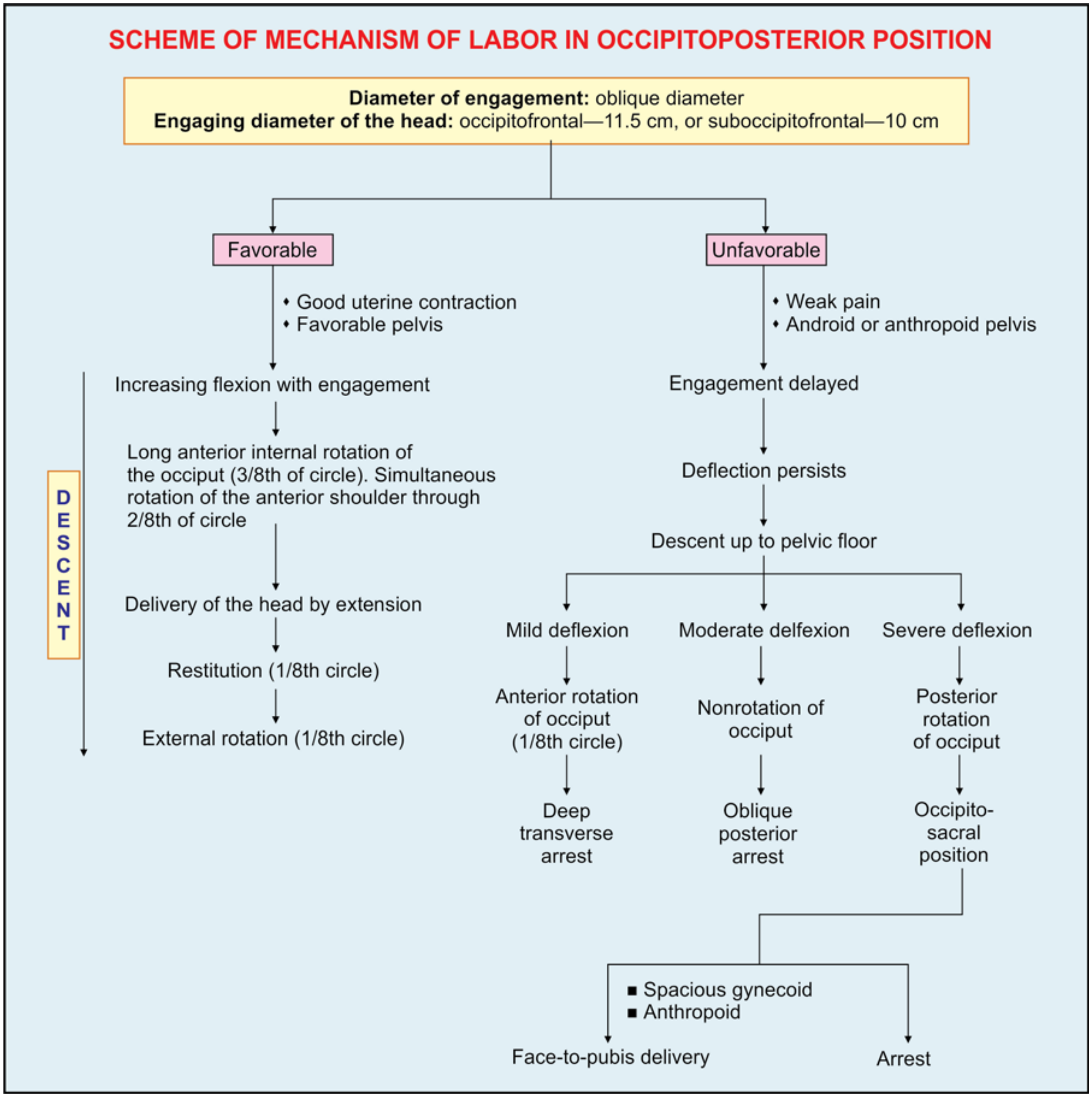


Fig. 26.5: Diagrammatic representation showing favorable and unfavorable rotation of occipitoposterior position

Mechanism of "face to pubis" delivery (Fig. 26.6)

- **Further descent occurs until the root of the nose** hinges under the symphysis pubis.
- **Flexion occurs**—It releasing successively the brow, vertex and occiput out of the stretched perineum and then **the face is born by extension**.
- **Restitution:** The head moves 1/8th of a circle in the opposite direction of internal rotation thus turning the face to look toward the mother's left thigh in ROP and right thigh in LOP.
- **External rotation:** The occiput further rotates to the same direction of restitution to 1/8th of a circle placing finally the face looking directly toward the left thigh in ROP and the right thigh in LOP.

Persistent occipitoposterior: In the true sense, it is an abnormal mechanism of the occipitoposterior position where there is malrotation of the occiput posteriorly toward the sacral hollow (occipitosacral


SCHEME OF MECHANISM OF LABOR IN OCCIPITOPOSTERIOR POSITION
Diameter of engagement: oblique diameter
Engaging diameter of the head: occipitofrontal—11.5 cm, or suboccipitofrontal—10 cm
Favorable
• Good uterine contraction
• Favorable pelvis
Increasing flexion with engagement
Long anterior internal rotation of the occiput (3/8th of circle). Simultaneous rotation of the anterior shoulder through 2/8th of circle
Delivery of the head by extension
Restitution (1/8th circle)
External rotation (1/8th circle)
DESCENT
Unfavorable
• Weak pain
• Android or anthropoid pelvis
Engagement delayed
Deflection persists
Descent up to pelvic floor
Mild deflexion
Moderate delfexion
Severe deflexion
Anterior rotation of occiput (1/8th circle)
Nonrotation of occiput
Posterior rotation of occiput
Deep transverse arrest
Oblique posterior arrest
Occipito-sacral position
■ Spacious gynecoid
■ Anthropoid
Face-to-pubis delivery
Arrest

position). As previously mentioned, delivery may occur spontaneously as face-to-pubis but arrest may occur in this position and is called occipitosacral arrest.

In the wider sense, it also includes two other arrested positions of the occipitoposterior, namely deep transverse arrest and oblique posterior arrest.

COURSE OF LABOR

Unlike the occipitoanterior, the course of events in labor is likely to be modified in occipitoposterior position. **The average duration of both the first and second stage of labor is increased**.

First stage: There is tendency to delay.

(1) ***Engagement*: Engagement is delayed due to**: (i) Persistence of deflexion of the head thereby increasing the diameter of engagement [occipitofrontal—11.5 cm (4 1/2")]. (ii) The driving force transmitted through the fetal axis is not in alignment with the axis of the inlet.

(2) ***Membrane status*:** Deflexed head becomes ovoid and this cannot fit well to the spherical lower segment → loss of ball valve action during uterine contraction → early rupture of the membranes and drainage of liquor.

(3) ***Uterine contraction*:** Because of ill-fitting of the deflexed head to the lower uterine segment, there is lack of stimulus for uterine contraction. This results in abnormal uterine contraction with slow dilatation of the cervix. Pressure on the rectum by the wide occiput results in premature desire of bearing-down effort even in the first stage. The patient, as a result, becomes exhausted. **There is prolongation of the first stage**.

Second stage: The second stage is often delayed due to long internal rotation or malrotation, with at times, arrest of the head. This may happen in android pelvis or in midpelvic contraction. If felt uncared for, arrest of the head may lead to obstructed labor.

Third stage: There is increased incidence of postpartum hemorrhage and trauma of the genital tract.

MODE OF DELIVERY:

(1) **Long anterior rotation of the occiput**: Spontaneous or assisted vaginal delivery usually occurs (90%).

(2) **Short posterior rotation**: Spontaneous or as assisted vaginal delivery may occur as face-to-pubis. **There is more chance of perineal injuries than with anterior vertex delivery. This is because** (a) wide biparietal diameter 9.5 cm (3 3/4") stretches the perineum and (b) occipitofrontal diameter 11.5 cm (4 1/2") emerges out of the introitus (Fig. 26.6).

(3) **Nonrotation or short anterior rotation:** Spontaneous vaginal delivery is unlikely except in favorable circumstances. If left uncared for, the case presents features of prolonged and obstructed labor. Vaginal operative delivery in such cases may, at times, become risky producing trauma to the genital tract (complete perineal tear) or injury to the fetal head.

Molding: The characteristic molding of the head observed in "face-to-pubis" delivery has been shown in the Figure 9.5. There is compression of the occipitofrontal diameter with elongation of the vault at right angle to it. The frontal bones are displaced beneath the parietal bones. **This type of molding favors tentorial tear because of extreme elevation of falx cerebri**.

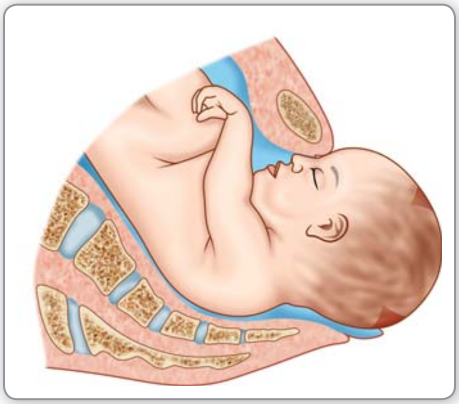


Fig. 26.6: Face-to-pubis delivery with occipitofrontal diameter emerges out of the introitus

Prognosis: There is increased maternal morbidity, incidental to prolonged labor and increased incidence of operative delivery (1 in 5). There is also increased perinatal morbidity and mortality (10%) due to asphyxia or trauma during vaginal operative delivery. **However, it is to be morbidity and remembered that in 4 out of 5 cases, there is usually no trouble and the fetus is delivered spontaneously**.

MANAGEMENT OF LABOR

Principles: The underlying principles in the management of the occipitoposterior position are—(1) early diagnosis, (2) strict vigilance with watchful expectancy hoping for descent and anterior rotation of the occiput, and (3) judicious and timely interference, if necessary.

Diagnosis and evaluation: ***Fetal back on the flank with the FHS not being easily located, early rupture of the membranes should arouse suspicion***. Internal examination is confirmatory (mentioned earlier).

Apart from the overall assessment of the case, the pelvic assessment is mandatory. **Pelvic adequacy is assessed clinically**. Inclination of the pelvis, configuration of the inlet, sacrum, ischial spines and the side walls are to be noted.

Early cesarean section: Occipitoposterior *per se* is not an indication of cesarean section. Pelvic inadequacy or its unfavorable configuration, along with obstetric complications such as, preeclampsia, postcesarean pregnancy, big baby usually need cesarean section.

First stage: In otherwise uncomplicated cases, the labor is allowed to proceed in a manner similar to normal labor. Forward leaning position (kneeling, hands and knees position) may help forward rotation and reduce back pain. **The following are the special instructions:**

- **Anticipating prolonged labor**, intravenous infusion line is sited and Ringer's solution drip is started.
- **Progress of labor is judged by**—(a) progressive descent of the head (b) rotation of the back and the anterior shoulder toward the midline, (c) increasing flexion of the head, (d) position of the sagittal suture on vaginal examination and (e) cervical dilatation.
- **Weak pain**, **persistence of deflexion and nonrotation of the occiput are the triad** too often coexistent. In such a situation, oxytocin infusion is started for augmentation of labor.
- **Indication of cesarean section:** (a) Arrest of labor (failure of rotation), (b) incoordinate uterine action and (c) fetal distress.

Second stage: In majority, anterior rotation of the occiput is completed and the delivery is either spontaneous or can be accomplished by low forceps or ventouse.

In minority (unrotated and malrotated): Provided the fetal and maternal conditions permit, one should take a watchful expectancy for the anterior rotation of the occiput and descent of the head. In occipitosacral position, spontaneous delivery as face-to-pubis may occur. In such cases, proper conduction of delivery and **liberal episiotomy should be done to prevent complete perineal tear**.

Third stage: Because of prolongation of labor, tendency of postpartum hemorrhage can be prevented by prophylactic intravenous ergometrine 0.25 mg with the delivery of anterior shoulder. Following vaginal operative delivery, meticulous inspection of the cervix and lower genital tract should be made to detect any injury.

ARRESTED OCCIPITOPOSTERIOR POSITION

If there is failure to progress (arrest) in spite of good uterine contractions for about 1/2–1 hour after full dilatation of the cervix, interference is indicated. The case is once more to be assessed abdominally and vaginally before formulating the suitable method of interference.

Per abdomen: The following conditions are assessed: (1) Size of the baby, (2) Engagement of the head, (3) Amount of liquor, (4) FHS.

Vaginal examination: The following conditions are to be noted—(1) Station of the head, (2) Position of the sagittal suture and the occiput, (3) Degree of deflexion of the head, (4) Degree of molding and caput formation, (5) Assessment of the pelvis at and below the level of obstruction, i.e. ischial spines, side walls of the pelvis, sacrococcygeal plateau, pubic arch and transverse diameter of the outlet.

I. ARREST IN OCCIPITOTRANSVERSE OR OBLIQUE OCCIPITOPOSTERIOR POSITION

(1) **Ventouse (Vacuum extraction)**: It is suitable in cases where the pelvis is adequate and the non-rotation of the occiput is either due to weak contractions or lack of tone of pelvic floor muscles. The cup is placed more toward the occiput to promote flexion and the rotation is expected during its descent on traction (see p. 660).

(2) **Cesarean section:** If the case is unsuitable for manual rotation especially in the presence of **mid-pelvic contraction**, cesarean section is much safer even at this stage.

(3) **Alternative methods**

(a) **Manual rotation followed by forceps extraction:** The objectives are **first** to rotate the head manually until the occiput is placed behind the symphysis pubis and **secondly** in that position forceps blades are applied. **The pelvis should be adequate; the baby is of average size and there is good amount of liquor**.

(b) **Forceps rotation and extraction: In the hands of experts**, forceps rotation followed by extraction can be achieved by using Kielland forceps (see p. 658). Its advantages over manual rotation are—(1) no chance of displacement of the head, (2) accidental cord prolapse is absent and (3) rotation can be done at, above or below the level of obstruction—depending upon the type of pelvis.

(c) **Craniotomy:** The dead baby should be delivered by **craniotomy**.

II. OCCIPITOSACRAL ARREST

If the head is engaged and the occiput descends below the ischial spines, forceps application in unrotated head followed by extraction as **face-to-pubis** is an effective procedure. Liberal mediolateral episiotomy should be done. **If the occiput remains at or above the level of ischial spines**, cesarean section should be considered.

DEEP TRANSVERSE ARREST (DTA)

The head is deep into the cavity; the sagittal suture is placed in the transverse bispinous diameter and there is no progress in descent of the head even after ½–1 hour following full dilatation of the cervix. The arrest in occipitotransverse position may be the end result of incomplete anterior rotation (1/8th of circle) of oblique occipitoposterior position, or it may be due to nonrotation of the commonly primary occipitotransverse position of normal mechanism of labor.

Causes: (a) Faulty pelvic architecture such as prominent ischial spines, flat sacrum and convergent side walls, (b) Deflexion of the head, (c) Weak uterine contraction, (d) Laxity of the pelvic floor muscles.

Diagnosis: (a) The head is engaged, (b) The sagittal suture lies in the transverse bispinous diameter, (c) Anterior fontanel is palpable, (d) Faulty pelvic architecture may be detected.

Management: The fetal condition and pelvic assessment give the guide as to the line of management (mentioned earlier).

(1) *Vaginal delivery is found not safe* (big baby and or inadequate pelvis): Cesarean section.

(2) *Vaginal delivery is found safe* (any of the methods may be employed): (1) **Ventouse**—Excessive traction force should not be used (see p. 662). (2) **Manual rotation and application of forceps**. (3) **Forceps rotation and delivery with Kielland** in the hands of an expert. **Operative vaginal delivery for DTA** should only be performed by a skilled obstetrician. Otherwise cesarean delivery is always preferred.

MANUAL ROTATION FOR OCCIPITOPOSTERIOR POSITION

The manual rotation can be accomplished with whole hand method or with half hand method.

Steps: The patient is put under general anesthesia and in lithotomy position. Full surgical asepsis is maintained. Bladder is catheterized. Vaginal examination is done to identify the direction of the occiput. **If a big caput has been formed, the direction of the unfolded pinna of the ear which points toward the occiput, can be taken for help.**

WHOLE HAND METHOD: Whole of the hand is introduced inside the vagina for rotation.

Step—I: *Gripping of the head:* In ROP or ROT the left hand and in LOP or LOT, the right hand is usually used. The corresponding hand is introduced into the vagina in a cone-shaped manner after separating the labia by two fingers of the other hand. In **occipitotransverse position,** the four fingers are pushed in the sacral hollow to be placed over the posterior parietal bone and the thumb is placed over the anterior parietal bone. In **oblique posterior position**, the four fingers of partially supinated hand are placed over the occiput and the thumb is placed over the sinciput (Fig. 26.7).

Step—II: *Rotation of the head:* Slight disimpaction may be needed for good grip. By a movement of pronation of the hand, the head is rotated to bring the occiput anterior along the shortest route. **Simultaneously, the back of the fetus is rotated by the external hand from the flank to the midline (Fig. 26.7).** This is an essential prerequisite for anterior rotation of the head. A little over rotation is desirable anticipating slight recurrence of malposition before the application of forceps.

In the alternative method, the four fingers of the pronated right hand are placed over the sinciput and the thumb over the occiput in ROP (Fig. 26.7). The head is rotated by supination movement of the hand.

Step—III: *Application of the forceps*: Following rotation, when the right hand is placed on the left side of the pelvis, left blade of the forceps is introduced. When the left hand is used, it is placed on the right side of the pelvis after rotation, as such the right blade is to be introduced first and **the left blade is then to be introduced underneath the right blade.** While introducing the blades, it is preferable that an assistant fixes the head by suprapubic pressure in a manner of first pelvic grip. As it is a mid-forceps application, axis traction device should be used.

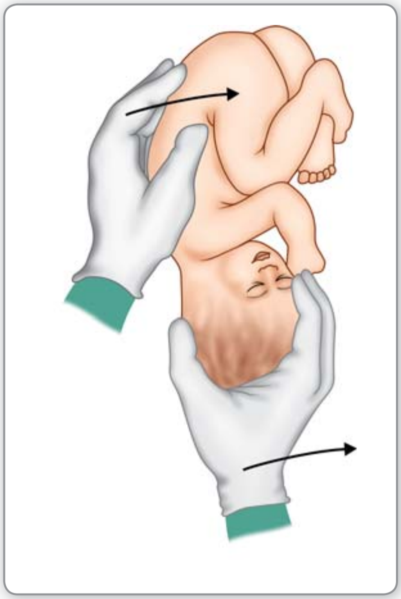


Fig. 26.7: Alternative method of gripping the head in ROP with the right hand

Difficulties and dangers: *The difficulties are due to*—(1) Failure to grip the head adequately due to lack of space, (2) Failure to dislodge the head from the impacted position, (3) Inadequate anesthesia, (4) Wrong case selection.

Dangers—**The chief dangers are accidental slipping of the head above the pelvic brim and prolapse of the cord.** It is better to perform cesarean section in such a situation.

HALF HAND METHOD: In this method, the four fingers and not the thumb are introduced into the vagina. **Its advantages over the whole hand method are:** (i) less space is required and (ii) less chance to displacement of the head.

Steps: The rotation is done only by using the right hand. The four fingers are introduced into the vagina and **tangential pressure is applied on the head at the level of diameter of engagement.** Thus, the pressure is applied on the side and the parietal eminence of the head. In ROP or ROT positions, the fingers are placed anterior to the head and the pressure is applied by the ulnar border of the hand (Fig. 26.8). In LOP or LOT positions, the fingers are placed posteriorly and the pressure is applied by the radial border of the hand. The force is applied intermittently till the occiput is placed behind the symphysis pubis.

Such maneuvers are only practiced by a skilled and experienced obstetrician. Simulated learning using mannequins and model pelvis with an experienced trainer is needed to acquire the skill.

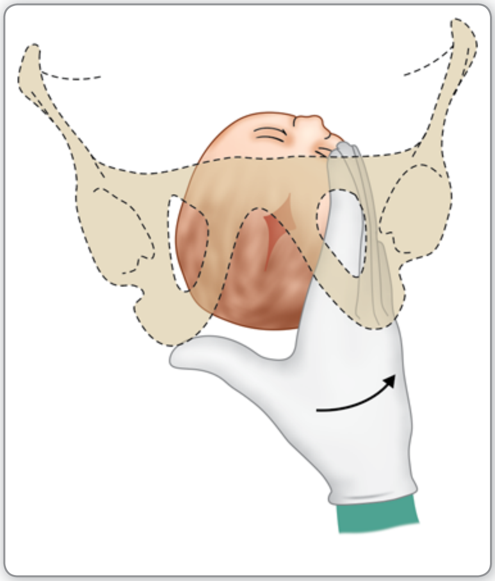
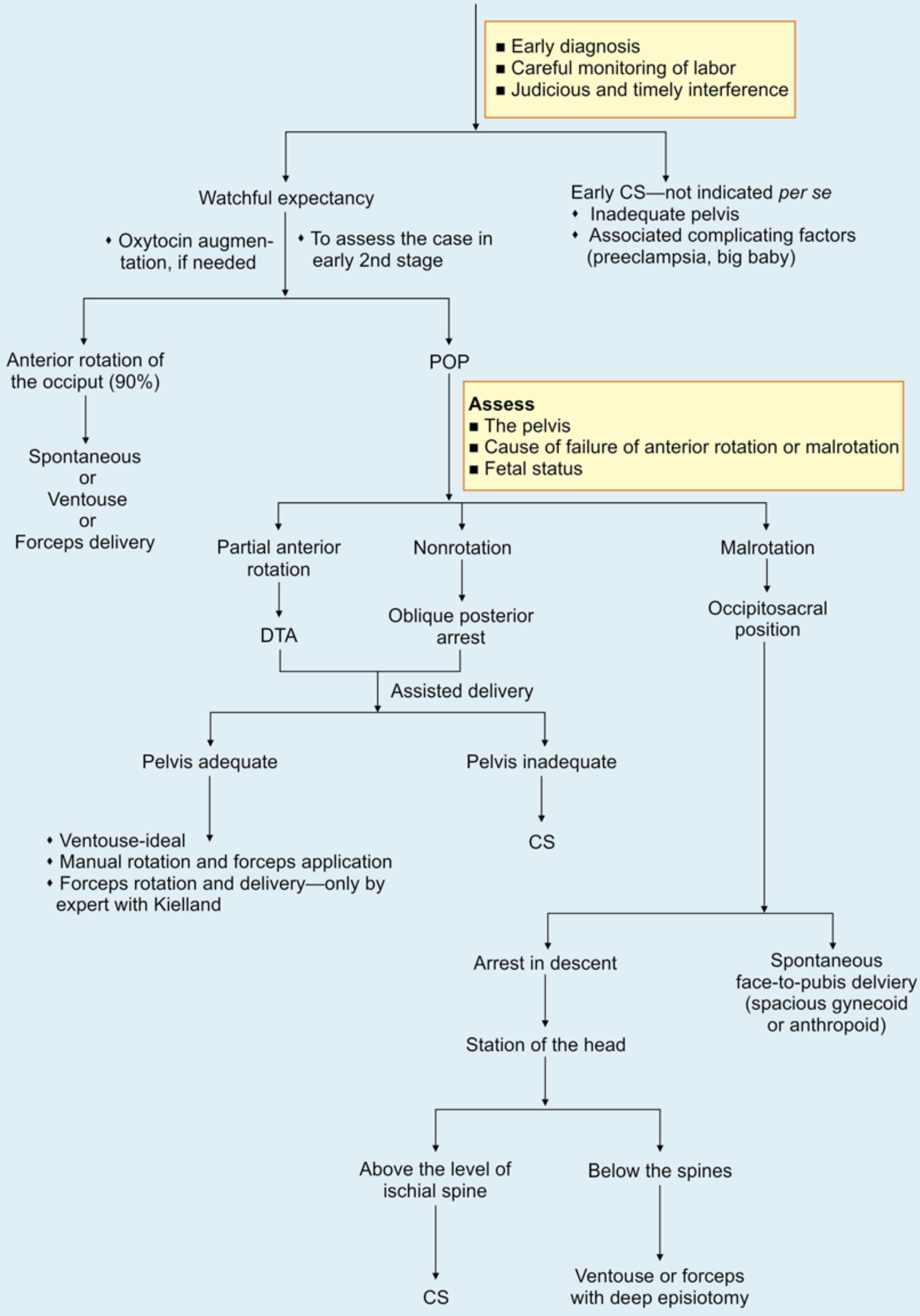


Fig. 26.8: Half hand method of manual rotation

SCHEME OF MANAGEMENT OF OCCIPITOPOSTERIOR POSITION


■ Early diagnosis
■ Careful monitoring of labor
■ Judicious and timely interference
Watchful expectancy
• Oxytocin augmentation, if needed
• To assess the case in early 2nd stage
Early CS—not indicated *per se*
• Inadequate pelvis
• Associated complicating factors (preeclampsia, big baby)
Anterior rotation of the occiput (90%)
Spontaneous or Ventouse or Forceps delivery
POP
Assess
■ The pelvis
■ Cause of failure of anterior rotation or malrotation
■ Fetal status
Partial anterior rotation
DTA
Nonrotation
Oblique posterior arrest
Malrotation
Occipitosacral position
Assisted delivery
Pelvis adequate
• Ventouse-ideal
• Manual rotation and forceps application
• Forceps rotation and delivery—only by expert with Kielland
Pelvis inadequate
CS
Arrest in descent
Station of the head
Above the level of ischial spine
CS
Below the spines
Ventouse or forceps with deep episiotomy
Spontaneous face-to-pubis delviery (spacious gynecoid or anthropoid)

KEY POINTS

- **Occiput posterior (OP)** is an abnormal position of the vertex rather than an abnormal presentation.
- **Common causes of OP** are: Anthropoid or android pelvis, deflection of fetal head and abnormal uterine contraction.
- Engagement of head is delayed due to deflection. In favorable circumstances (90%) occiput rotates anteriorly 3/8th of a circle anteriorly and delivery occurs as in occiput anterior.
- **Cases may develop other abnormal positions.**

  Depending upon the extent of rotation of the occiput, three positions may develop

  (a) Incomplete anterior rotation – deep transverse position
  (b) Nonrotation – oblique posterior position
  (c) Malrotation – occiput sacral position

  Depending upon the pelvic adequacy, degree of flexion and uterine contraction delivery may occur or there may be arrest of labor.
- **Labor in OP position**, expectant management is the policy, as most OP rotate anteriorly and baby is delivered spontaneously or by instruments (ventouse or forceps).
- **Indications of interventions are:** (a) lack of progress (CPD, android pelvis, deflection of the head, weak pelvic floor failing to guide occiput anteriorly); (b) Fetal distress (FHR < 100 or >160 bpm, irregular or meconium stained liquor; (c) Maternal distress (emotional instability, dehydration, high-colored urine, pulse > 100 bpm).

BREECH PRESENTATION

In breech presentation, **the lie** is longitudinal and the **podalic pole** presents at the pelvic brim. **It is the most common malpresentation.**

Incidence: The incidence is about 20% at 28th week and drops to 5% at 34th week and to 3–4% at term. Thus in 3 out of 4, spontaneous correction into vertex presentation occurs by 34th week. The incidence is expected to be low in hospitals where high parity births are minimal and routine external cephalic version is done in antenatal period.

VARIETIES

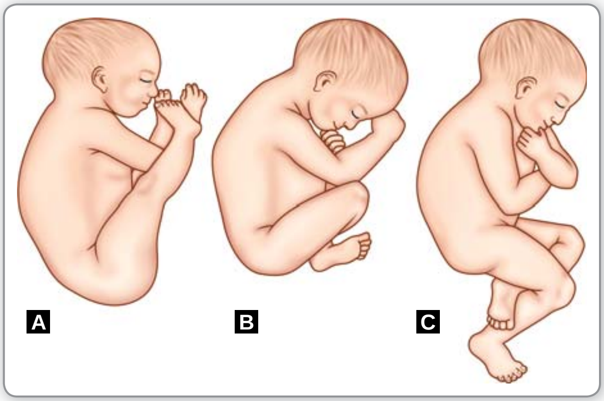
There are two varieties of breech presentation (Fig. 26.9):

- **Complete**
- **Incomplete**

Complete (Flexed breech): The normal attitude of full flexion is maintained. Thighs are flexed at hips and legs at knees. **The presenting part** consists of two buttocks, external genitalia and two feet. **It is commonly present in multiparae (10%)**.

Incomplete: This is due to varying degrees of extension of thighs or legs at the podalic pole. Three varieties are possible:

- **Breech with extended legs (Frank breech):** In this condition, thighs are flexed on the trunk and legs are extended at the knee joints (Fig. 26.10). The presenting part consists of the two buttocks and external genitalia only. **It is commonly present in primigravidae**, about 70%. The increased prevalence in primigravida is due to a tight abdominal wall, good uterine tone and early engagement of breech.



Figs 26.9A to C: Varieties of breech presentation—(A) Breech with extended legs (frank breech); (B) Flexed breech (complete breech); (C) Footling presentation

- **Footling presentation (25%):** Both thighs and legs are partially extended bringing the legs to present at brim.
- **Knee presentation:** Thighs are extended but the knees are flexed, bringing the knees down to present at the brim. The latter two varieties are not common.

Clinical varieties: In an attempt to find out the dangers inherent to breech, breech presentation is clinically classified as:

(1) *Uncomplicated*—It is defined as one where there is no other associated obstetric complications apart from the breech, **prematurity being excluded**.

(2) *Complicated*—When the presentation is associated with conditions which adversely influence the prognosis such as prematurity, twins, contracted pelvis, placenta previa, etc. It is called complicated breech. Extended legs, extended arms, cord prolapse or difficulty encountered during breech delivery should not be called complicated breech but are called complicated or abnormal breech delivery.

ETIOLOGY OF BREECH PRESENTATION

There is higher incidence of breech in earlier weeks of pregnancy. Smaller size of the fetus and comparatively larger volume of amniotic fluid allow the fetus to undergo spontaneous version by kicking movements until by 36th week when the position becomes stabilized. The following are the **known factors responsible for breech presentation**. In a significant number of cases, the cause remains obscure.

- **Prematurity:** It is the most common cause of breech presentation.
- **Factors preventing spontaneous version:** (a) Breech with extended legs, (b) Twins, (c) Oligohydramnios, (d) Congenital malformation of the uterus such as septate or bicornuate uterus, (e) Short cord, relative or absolute, (f) Intrauterine death of the fetus.
- **Favorable adaptation:** (a) Hydrocephalus—big head can be well accommodated in the wide fundus, (b) Placenta previa, (c) Contracted pelvis, (d) Cornu-fundal attachment of the placenta—minimizes the space of the fundus where the smaller head can be placed comfortably.
- **Undue mobility of the fetus:** (a) Hydramnios, (b) Multiparae with lax abdominal wall.
- **Fetal abnormality: Trisomies** 13, 18, 21, anencephaly and myotonic dystrophy due to alteration of fetal muscular tone and mobility.

Recurrent breech: On occasion, the breech presentation recurs in successive pregnancies. When it recurs in three or more consecutive pregnancies, it is called habitual or recurrent breech. The probable causes are congenital malformation of the uterus, septate or bicornuate, and repeated cornu-fundal attachment of the placenta.

DIAGNOSIS OF BREECH PRESENTATION

- **Clinical**
- **Sonography**

Clinical: The diagnostic features of a complete breech and a frank breech are given in a tabulated form (Box 1).

ULTRASONOGRAPHY is most informative. (1) **It confirms the clinical diagnosis**—especially in primigravidae with engaged frank breech or with tense abdominal wall and irritable uterus. (2) **It can detect fetal congenital abnormality** and also congenital anomalies of the uterus. (3) **Type of breech** (complete or incomplete). (4) **It measures** biparietal diameter, gestational age and estimated weight of the fetus. (5) **It also localizes** the placenta. (6) **Assessment of** liquor volume (important for ECV).

BOX – 1	**Complete Breech**	**Frank Breech**
Per abdomen		
Fundal grip	- Head—suggested by hard and globular mass - Head is ballottable	- Head - Irregular small parts of the feet may be felt by the side of the head - Head is nonballottable due to splinting action of the legs on the trunk
Lateral grip	- Fetal back is to one side and the irregular limbs to the other	- Irregular parts are less felt on the side
Pelvic grip	- Breech—suggested by soft, broad and irregular mass - Breech is usually not engaged during pregnancy	- Small, hard and a conical mass is felt - The breech is usually engaged
FHS	- Usually located at a higher level round about the umbilicus	- Located at a lower level in the midline due to early engagement of the breech
Per vaginam		
During pregnancy	- Soft and irregular parts are felt through the fornix	- Hard feel of the sacrum is felt, often mistaken for the head
During labor	- Palpation of ischial tuberosities, sacrum and the feet by the sides of the buttocks The foot felt is identified by the prominence of the heel and lesser mobility of the great toe	- Palpation of ischial tuberosities, anal opening and sacrum only

(7) **Attitude of the head (Fig. 26.12)**—flexion or hyperextension (important for decision making at the time of delivery). CT and MRI can be used to assess the pelvic capacity in addition to all the above-mentioned information (see p. 739, 740).

POSITIONS: Sacrum is the denominator of breech and there are four positions. In anterior positions, sacrum is directed toward iliopubic eminences and in posterior positions, sacrum is directed to sacroiliac joints. **The positions are:** (1) First position—left sacroanterior (LSA)—being the most common (2) Second position—right sacroanterior (RSA) (3) Third position—right sacroposterior (RSP) and (4) Fourth position—left sacroposterior (LSP).

MECHANISM OF LABOR IN BREECH PRESENTATION

SACROANTERIOR POSITION: In the mechanism of breech delivery, the **principal movements occur at three places**—buttocks, shoulders and the head. The first two successive parts to be born are bigger but more compressible while the head because of nonmolding due to rapid descent, presents difficulties. Each of the three components undergo cardinal movements as those of normal mechanism.

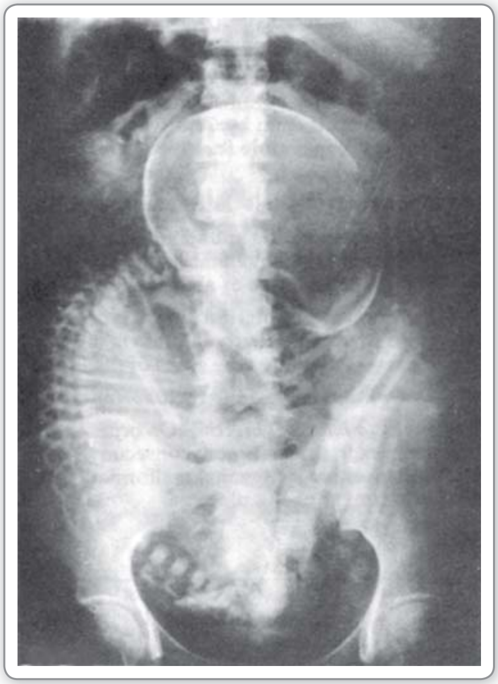
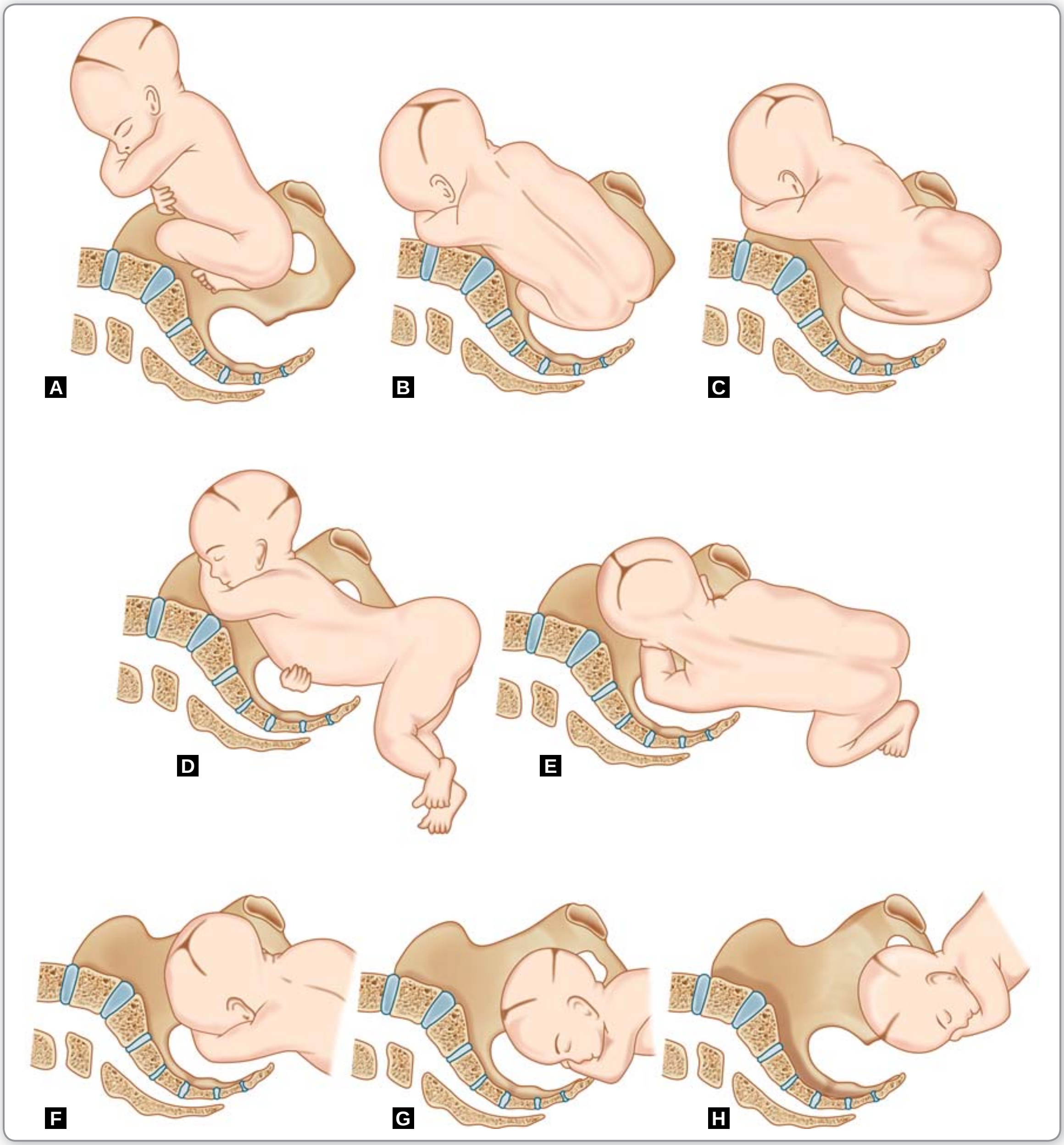


Fig. 26.10: Straight X-ray abdomen (*Not done these days*) showing a single fetus with breech presentation. The legs are extended

Buttocks (Fig. 26.11):

- **The diameter of engagement of the buttock is one of the oblique** diameters of the inlet. **The engaging diameter** is bitrochanteric (10 cm or 4") with the sacrum directed toward the iliopubic eminence. When the diameter passes through the pelvic brim, the breech is engaged.
- **Descent of the buttocks** occurs until the anterior buttock touches the pelvic floor.
- **Internal rotation of the anterior buttock** occurs through 1/8th of a circle placing it behind the symphysis pubis.



Figs 26.11A to H: Mechanism of labor in breech (RSA)

- **Further descent with lateral flexion** of the trunk occurs until the anterior hip hinges under the symphysis pubis which is released first followed by the posterior hip.
- **Delivery of the trunk** and the lower limbs follow.
- **Restitution occurs** so that the buttocks occupy the original position as during engagement in oblique diameter.

Shoulders (Fig. 26.11):

- **Bisacromial diameter (12 cm or 4 3/4") engages** in the same oblique diameter as that occupied by the buttocks at the brim soon after the delivery of the breech.
- **Descent occurs with internal rotation** of the shoulders bringing the shoulders to lie in the anteroposterior diameter of the pelvic outlet. The trunk simultaneously rotates externally through 1/8th of a circle.
- **Delivery of the posterior shoulder** followed by the anterior one is completed by anterior flexion of the delivered trunk.
- **Restitution and external rotation:** Untwisting of the trunk occurs putting the anterior shoulder toward the right thigh in LSA and left thigh in RSA. External rotation of the shoulders occurs to the same direction because of internal rotation of the occiput through 1/8th of a circle anteriorly. The fetal trunk is now positioned as dorsoanterior.

Head (Fig. 26.11):

- **Engagement** occurs either through the opposite oblique diameter as that occupied by the buttocks or through the transverse diameter. The engaging diameter of the head is suboccipitofrontal (10 cm).
- Descent with increasing flexion occurs.
- **Internal rotation of the occiput** occurs anteriorly, through 1/8th or 2/8th of a circle placing the occiput behind the symphysis pubis.
- **Further descent occurs** until the subocciput hinges under the symphysis pubis.
- **Head is born by flexion**—chin, mouth, nose, forehead, vertex and occiput appearing successively. The expulsion of the head from the pelvic cavity depends entirely upon the bearing-down efforts and not at all on uterine contractions.

Sacroposterior position: In sacroposterior position, the mechanism is not substantially modified. The head has to rotate through 3/8th of a circle to bring the occiput behind the symphysis pubis.

PROGNOSIS OF VAGINAL BREECH DELIVERY

MATERNAL: Labor is usually not prolonged. But because of increased frequency of **operative delivery including cesarean section**, the morbidity is increased. The **risks include** trauma to the genital tract, operative vaginal delivery (episiotomy, forceps), cesarean section, sepsis and anesthetic complications. As a consequence, **maternal morbidity** is slightly raised. Frank breech acts as an effective cervical dilator. Flexed breech, although, theoretically might cause delay in first stage, but rarely so because of its prevalence among multiparae.

FETAL: The fetal risk in terms of **perinatal mortality** is considerable in vaginal breech delivery. It is difficult to assess the magnitude of the real risk because the complicating factors, such as prematurity, birth trauma, congenital malformation of the fetus that contribute significantly to the fetal hazards. **The corrected** (excluding fetal abnormality) **perinatal mortality** ranges from 5 to 35 per 1,000 births. The overall perinatal mortality in breech still remains 9–25% compared with 1–2% for nonbreech deliveries. **Perinatal death (excluding congenital abnormalities) is 3 to 5 times higher than the nonbreech presentations. The factors which significantly influence the fetal risk are**—(a) skill of the obstetrician, (b) weight of the baby, (c) position of the legs and (d) type of pelvis. **The fetal mortality is least in frank breech and maximum in footling presentation**, where the chance of cord prolapse is also more. **Gynecoid and anthropoid pelvis are favorable for the aftercoming head**. The fetal risk in multipara is no less than that of primigravida. This is because of increased chance of cord prolapse associated with flexed breech.

THE DANGERS TO THE BABY

(1) ***Intrapartum fetal death specially with preterm babies***

(2) ***Injury to brain and skull*** — (a) **Intracranial hemorrhage: Compression followed by decompression during delivery of the unmolded after-coming head** results in tear of the tentorium cerebelli and **hemorrhage** in the subarachnoid space. The risk is more with preterm babies, (b) **Minute hemorrhages**, (c) **Fracture of the skull**.

(3) ***Birth asphyxia*: It is due to**—(1) Cord compression soon after the buttocks are delivered and also when the head enters into the pelvis. A period of more than 10 minutes will produce asphyxia of varying degrees. (2) Retraction of the placental site, (3) Premature attempt at respiration (amniotic fluid, vaginal fluid) while the head is still inside, (4) Delayed delivery of the head, (5) Cord prolapse and (6) Prolonged labor.

(4) ***Birth Injuries (7%)*:** The following injuries are inflicted during manipulative deliveries. It is 13 times more than the vertex presentation.

- **Hematoma—**over the sternomastoid or over the thighs.
- **Fractures—**The common sites are femur, humerus, clavicle and odontoid process. There may be dislocation of the hip joint, mandible or 5th and 6th cervical vertebrae and epiphyseal separation.
- **Visceral injuries** include rupture of the liver, kidneys, suprarenal glands, lungs and hemorrhage in the testicles.
- **Nerve**—Medullary coning, spinal cord injury, stretching of the cervical and brachial plexus to cause either Erb's or Klumpke's palsy (see p. 562).
- **Long-term neurological damage**.

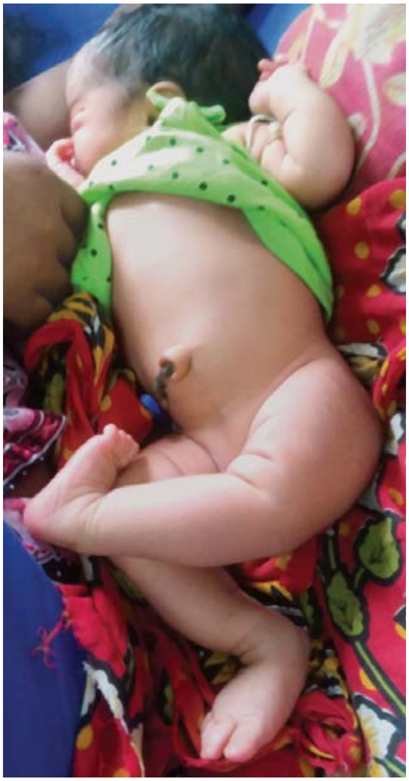


Fig. 26.12: Birth injury following assisted breech (frank) delivery showing dislocation of the left knee joint.

Some of the injuries may prove fatal and contribute to perinatal mortality. **Long-term** (neurological) **morbidity** of the surviving infants should not be underestimated.

(5) ***Congenital Malformations*** is double compared to babies with cephalic presentation (congenital dislocation of the hip, hydrocephalous, anencephaly are common).

PREVENTION OF THE FETAL HAZARDS

- The incidence of breech can be minimized by external cephalic version where possible.
- If the version fails or is contraindicated, delivery is done by elective cesarean section.
- Vaginal breech delivery should be conducted by a skilled obstetrician along with an organized team consisting of a skilled anesthetist and neonatologist (see p. 442).
- Vaginal manipulative delivery should be done by a skilled person with utmost gentleness, especially during delivery of the head.

ANTENATAL MANAGEMENT

Antenatal management in breech presentation consists of:

- **Identification of the complicating factors** related with breech presentation.
- **External cephalic version**, if not contraindicated.
- **Formulation of the line of management**, if the version fails or is contraindicated.

Identification of complicating factor: It can be detected by clinical examination, supplemented by sonography. **Sonography** is particularly useful to detect congenital malformations of the fetus, the precise location of the placental site and congenital anomalies of the uterus.

External Cephalic Version (ECV): There are protagonists and antagonists to external version. As such, in an institution or to an individual where the perinatal mortality in vaginal breech delivery is appreciably high, there is enough justification for its use. **The success rate of version is about 65%** (for technique—see p. 664). Successful version reduces the risk of cesarean section significantly. Prior sonography should be a routine. Cardiotocography (CTG) should ideally be done before and after the procedure (see p. 693).

Time of version: ECV has been considered from 36 weeks onward. While version in the early weeks is easy but chance of reversion is more. Late version may be difficult because of increasing size of the fetus and diminishing volume of liquor amnii. However, the use of uterine relaxant (tocolysis) has made the version at later weeks less difficult. It minimizes chance of reversion and should fetal complications develop, it can be effectively tackled by cesarean section. Hypertonus or irritable uterus can be overcome with the use of tocolytic drugs. (Tocolytic drugs with doses—see p. 583).

Contraindications of ECV

- **Antepartum hemorrhage** (placenta previa or abruption)—big risk of placental separation
- **Fetal causes**—hyperextension of the head, large fetus (> 3.5 kg), congenital abnormalities (major), dead fetus, fetal compromise (IUGR)
- **Multiple pregnancy**
- **Ruptured membranes**—with drainage of liquor
- **Known congenital malformation of the uterus**
- **Abnormal cardiotocography**
- **Contracted pelvis**
- **Previous cesarean delivery**—risk of scar rupture
- **Obstetric complications:** Severe pre-eclampsia, obesity, elderly primigravida, bad obstetric history (BOH), **oligohydramnios**
- **Rhesus isoimmunization**

Breech with extended legs is not a contraindication for version

Benefits of ECV are—(i) Reduction in the incidence of breech presentation at term, (ii) Reduction in the incidence of breech delivery (Vaginal or cesarean) and the associated complications (vide above) (iii) Reduction in the incidence of cesarean delivery by 5%.

Successful version is likely in cases of: (i) Complete breech, (ii) Nonengaged breech, (iii) Sacroanterior position (fetal back anteriorly), (iv) Adequate liquor, (v) Nonobese patient.

Causes of failure of version: (1) **Breech with extended legs**—early engagement of presenting part and difficult to flex the trunk because of splinting action of the limbs. (2) **Scanty liquor or** big size baby. (3) **Mechanical**—obesity, increased tone of the abdominal muscles and irritable uterus. (4) **Short cord**—either relative (common) or absolute. (5) **Uterine malformations**—septate or bicornuate.

Dangers of version: The dangers of version are—(1) premature onset of labor, (2) premature rupture of the membranes, (3) placental abruption and bleeding, (4) entanglement of the cord round the fetal part or formation of a true knot leading to impairment of fetal circulation and fetal death and (5) increased chance of fetomaternal bleed. (6) Amniotic fluid embolism. **Immunoprophylaxis with anti-D gammaglobulin** is to be administered in nonimmunized Rh-negative mother (Chapter 22). The perinatal mortality should not exceed beyond 1%. A reactive cardiotocographic trace should be obtained after the procedure (see p. 693).

Management, if version fails or is contraindicated: The pregnancy is to be continued with usual checkup and unexpectedly, one may find that spontaneous version has occurred. But if the breech persists, the assessment of the case is to be done with respect to—(1) age of the mother especially in primigravidae, (2) associated complicating factors, (3) size of the baby and (4) pelvic capacity. **Clinical assessment of the pelvis should be done** in all primigravidae and in selected multigravidae with previous history suggestive of pelvic inadequacy. X-ray pelvimetry is not favored. CT or MRI is a better alternative. Ultrasonographic examination is the gold standard for decision making (see p. 732). Two methods of delivery can be planned.

- To perform an **elective cesarean section**.
- To allow spontaneous labor to start and **vaginal breech delivery** to occur.

Elective Cesarean Section: Because of the complications involved in vaginal breech delivery, there is a tendency to liberalize the use of cesarean section in breech. **The indications of CS in breech are:** Big baby (estimated fetal weight >3.5 kg), small baby (<1.5 kg), estimated fetal weight <1.5 or >3.5 kg, hyperextension of the head (stargazing fetus), footling presentation (risk of cord prolapse), suspected pelvic contraction or severe IUGR. Any associated complications (obstetric or medical) is often considered for CS in breech. **The overall incidence of cesarean section in breech ranges from 15% to 50%, out of which about 80% is elective. Delivery of preterm breech** (weight <1,500 g) by cesarean section is commonly done but it should be reserved in selected centers, equipped with intensive neonatal care unit.

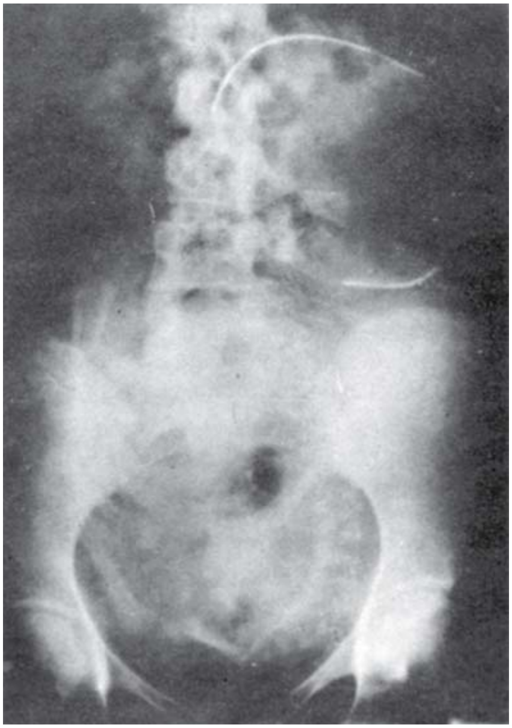


Fig. 26.13: Straight X-ray abdomen (*not done now*) showing breech with hyperextended head

Vaginal breech delivery: Criteria to be fulfilled are—(i) average fetal weight (between 1.5 kg and 3.5 kg), (ii) flexed fetal head, (iii) adequate pelvis, (iv) without any other (medical or obstetric) complications, (v) availability of facilities for emergency cesarean section (anesthetists, neonatologist), (vi) facilities for continuous labor monitoring (preferably electronic) and (vii) presence of obstetrician experienced with vaginal breech delivery, (viii) informed consent. Frank breech is preferred.

MANAGEMENT OF VAGINAL BREECH DELIVERY

FIRST STAGE: The management protocol is similar to that mentioned in normal labor. The following are the important considerations. Spontaneous onset of labor increases the chance of successful vaginal delivery.

- **Vaginal examination is indicated—**(a) at the onset of labor for pelvic assessment, (b) soon after rupture of the membranes to exclude cord prolapse.
- **An intravenous line is sited** with Ringer's solution, oral intake is avoided, blood is sent for group and cross matching (considering the chance of CS).
- **Adequate analgesia** is given, epidural is preferred.
- **Fetal status and progress of labor** are monitored.
- **Oxytocin infusion** may be used for augmentation of labor.

Indications of Cesarean Section (CS): (a) Cases seen for the first time in labor with presence of complications; (b) Arrest in the progress of labor; (c) Nonreassuring FHR pattern (Fetal distress); (d) Cord presentation or prolapse.

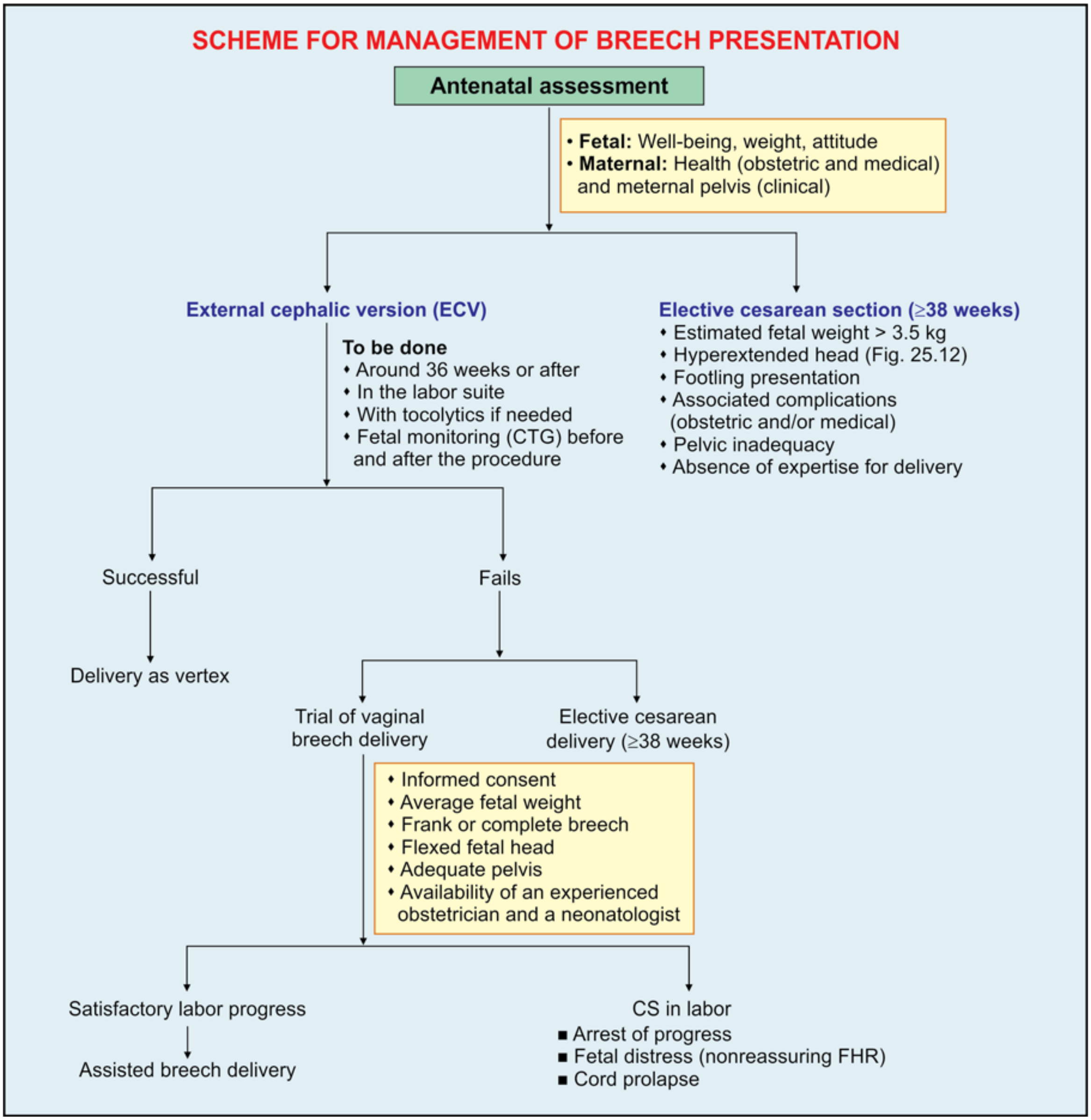
SECOND STAGE: There are three methods of vaginal breech delivery:

- **Spontaneous (10%):** Expulsion of the fetus occurs with very little assistance. This is not preferred.

- **Assisted breech:** The delivery of the fetus is by assistance from the beginning to the end. This method should be employed in all cases (see below).
- **Breech extraction (partial or total):** When part or the entire body of the fetus is extracted by the obstetrician. It is rarely done these days as it produces trauma to the fetus and the mother. **Indications are:** (a) Delivery of the **second twin after IPV** (see p. 242, 665), (b) Cord prolapse, (c) Extended legs arrested at the cavity or at the outlet.

ASSISTED BREECH DELIVERY

Breech delivery should be conducted by a skilled obstetrician. **The following are to be kept ready beforehand,** in addition to those required for conduction of normal labor: (1) **Anesthetist—**to administer



SCHEME FOR MANAGEMENT OF BREECH PRESENTATION

Antenatal assessment

- **Fetal:** Well-being, weight, attitude
- **Maternal:** Health (obstetric and medical) and meternal pelvis (clinical)

External cephalic version (ECV)

To be done
- Around 36 weeks or after
- In the labor suite
- With tocolytics if needed
- Fetal monitoring (CTG) before and after the procedure

Elective cesarean section (≥38 weeks)
- Estimated fetal weight > 3.5 kg
- Hyperextended head (Fig. 25.12)
- Footling presentation
- Associated complications (obstetric and/or medical)
- Pelvic inadequacy
- Absence of expertise for delivery

Successful

Delivery as vertex

Fails

Trial of vaginal breech delivery

Elective cesarean delivery (≥38 weeks)

- Informed consent
- Average fetal weight
- Frank or complete breech
- Flexed fetal head
- Adequate pelvis
- Availability of an experienced obstetrician and a neonatologist

Satisfactory labor progress

Assisted breech delivery

CS in labor
- Arrest of progress
- Fetal distress (nonreassuring FHR)
- Cord prolapse

anesthesia as and when required. (2) **An assistant**—to push down the fundus during contraction. (3) **Instruments and suture materials for episiotomy**. (4) **A pair of obstetric forceps** for the aftercoming head, if required. (5) **Appliances for resuscitation of the baby**, if asphyxiated. (6) Neonatologist.

Principles in conduction: (1) Never to rush, (2) Never pull from below but push from above (Fig. 26.14), (3) Always keep the fetus with the back anteriorly.

It is expected that good uterine contractions and maternal expulsive forces will maintain the flexion of the fetal head and result in descent and safe delivery.

Never to rush and never to pull—early aggressive and hasty pull affects breech delivery adversely by—(a) Entrapment of the aftercoming fetal head through the incompletely dilated cervix. (b) Traction from below results in deflection of the head posing longer occipitofrontal diameter (11.5 cm) at the pelvic inlet (Fig. 26.15). (c) Increased risk of nuchal displacement of arms.

Steps:

- The patient is brought to the table when the anterior buttock and fetal anus are visible. She is placed in lithotomy position when the posterior buttock distends the perineum.
- **To avoid aortocaval compression**, the woman is tilted laterally (15°) using a wedge under the back.
- **Antiseptic cleaning** is done, bladder is emptied with an "in and out" catheter.
- **Pudendal block** is done along with perineal infiltration if not epidural has been used earlier.
- **Episiotomy:** It should be made in all cases of primigravidae and selected multiparae. **Its advantages are**—(a) **to straighten** the birth canal which especially facilitates the delivery of breech with extended legs where lateral flexion is inadequate; (b) **to facilitate** intravaginal manipulation and for forceps delivery, (c) **to minimize** compression of the aftercoming head. **The best time for episiotomy** is when the perineum is distended and thinned by the breech as it is "climbing" the perineum.
- **The patient is encouraged to bear down** as the expulsive forces from above ensure flexion of the fetal head and safe descent. The "**no touch to the fetus**" policy is adopted until the buttocks are delivered along with the legs in flexed breech and the trunk slips up to the umbilicus.
- **Soon after the trunk up to the umbilicus is born. The following are to be done:**

  (a) **The extended legs (in frank breech)** are to be decomposed by pressure on the knees (popliteal fossa) in a manner of abduction and flexion of the thighs (Fig. 26.14).

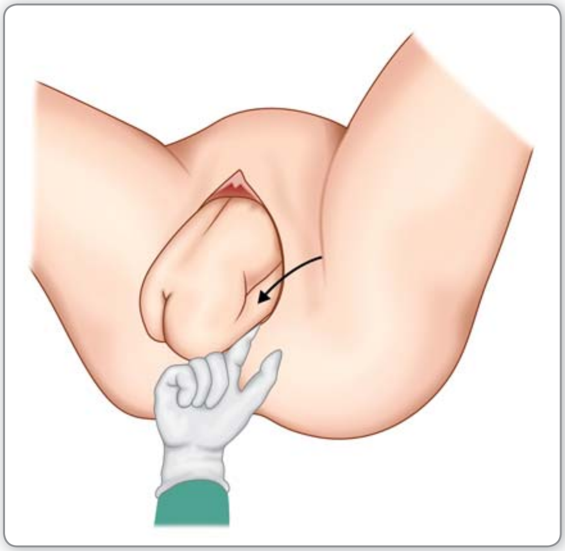


Fig. 26.14: Delivery of the extended leg by abduction at the knee

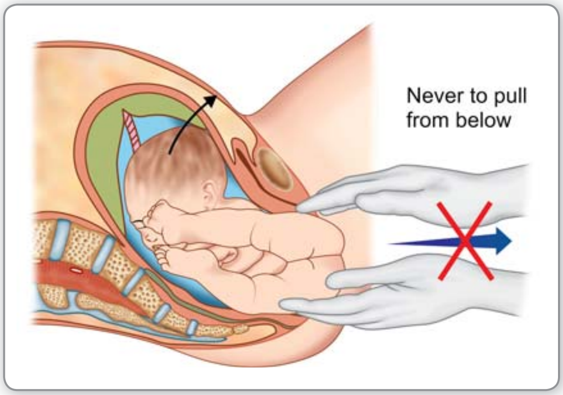


Fig. 26.15: Aggressive pull causes deflection and entrapment of the aftercoming head

(b) **The umbilical cord** is to be pulled down and to be mobilized to one side of the sacral bay to minimize compression. There may be transient abnormality in cord pulsation at this stage which has got no prognostic significance. An attempt of hasty delivery for this reason alone should be avoided.

(c) **If the back remains posteriorly**, rotate the trunk to bring the back anteriorly (sacroanterior).

(d) **The baby is wrapped** with a sterile towel to prevent slipping when held by the hands and to facilitate manipulation, if required.

- **Delivery of the arms:** The assistant is to place a hand over the fundus and keep a steady pressure during uterine contractions to prevent extension of the arms. Soon, the anterior scapula is visible. The position of the arm should be noted. **When the arms are flexed**, the vertebral border of the scapula remains parallel to the vertebral column and **when extended** there is winging of the scapula (parallelism is lost). **The arms are delivered one after the other only when one axilla is visible,** by simply hooking down each elbow with a finger. It is immaterial as to which arm is to be delivered first. The baby should be held by the feet over the sterile towel while the arms are delivered (Figs 26.16 and 26.18A).

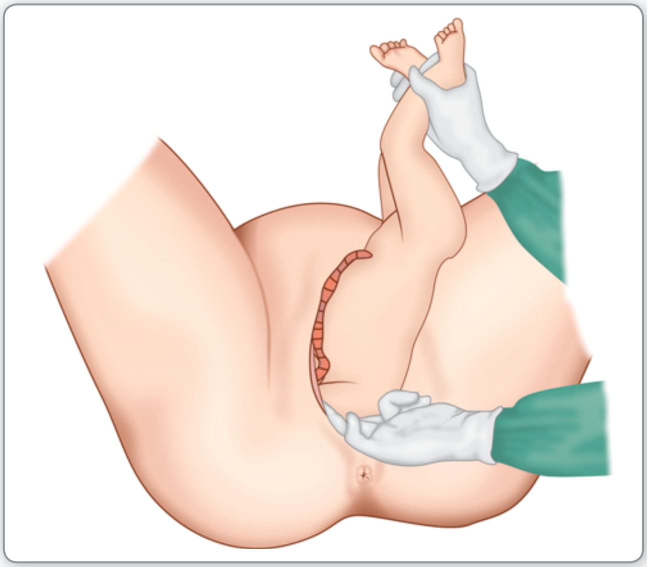


Fig. 26.16: Delivery of the posterior arm

- **Delivery of the aftercoming head:** This is the most crucial stage of the delivery. The time between the delivery of umbilicus to delivery of mouth should preferably be 5–10 minutes. There are various methods of delivery for the aftercoming head. Each one is quite safe and effective in the hands of an expert, conversant with that particular technique. **The following are the common methods employed**:

(a) ***Burns-Marshall method (Fig. 26.17):*** The baby is allowed to hang by its own weight. The assistant is asked to give suprapubic pressure with the flat of hand **in a downward and backward direction**, the pressure is to be exerted more toward the sinciput. The aim is to promote flexion of the head so that favorable diameter is presented to the pelvic cavity. Not more than 1–2 minutes are required to achieve the objective. **When the nape of the neck is visible under the pubic arch**, the baby is grasped by the ankles with a finger in between the two. Maintaining a steady traction and forming a wide arc of a circle, the trunk is swung in upward and forward direction (Fig. 26.18). Meanwhile, with the left hand to guard the perineum, slipping the perineum off successively the face and brow. **When the mouth is cleared off the vulva, there should be no hurry**. **Mucus of the mouth and pharynx is cleared by mucus sucker**. The trunk is depressed to deliver rest of the head.

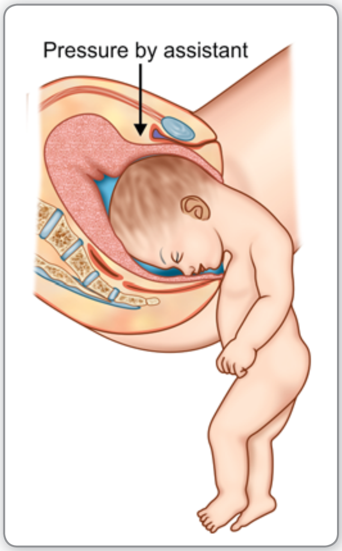


Fig. 26.17: Delivery of after-coming head by Burns-Marshall method

(b) ***Forceps delivery***: Forceps can be used as a routine. The head must be in the cavity. **The advantages are**—(a) delivery can be controlled by

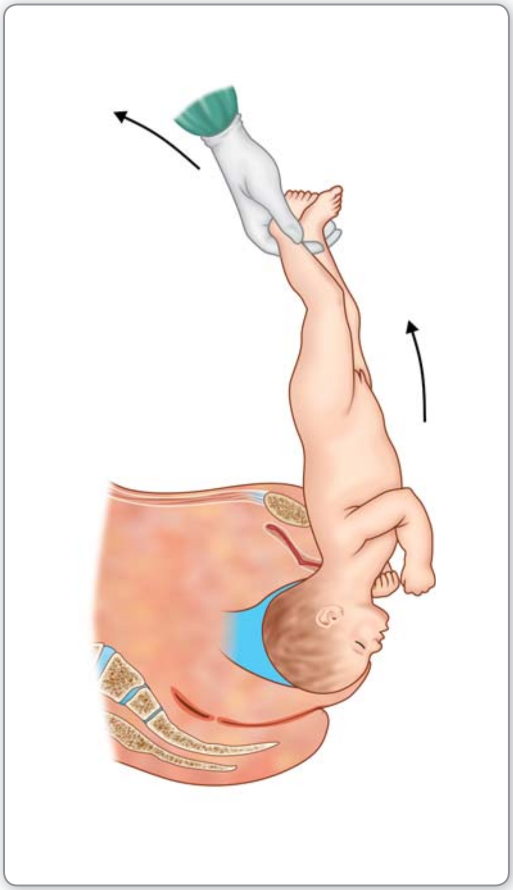


Fig. 26.18: Continuation of the Burns–Marshall method

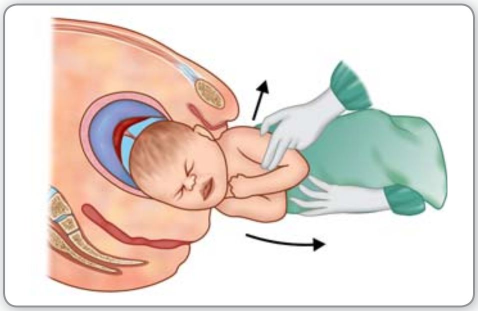


Fig. 26.18A: Delivery of the anterior arm

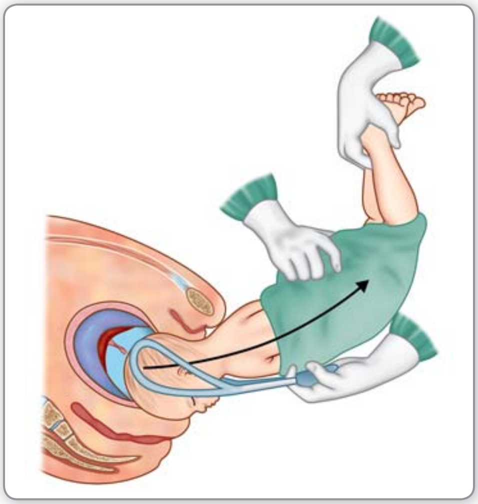
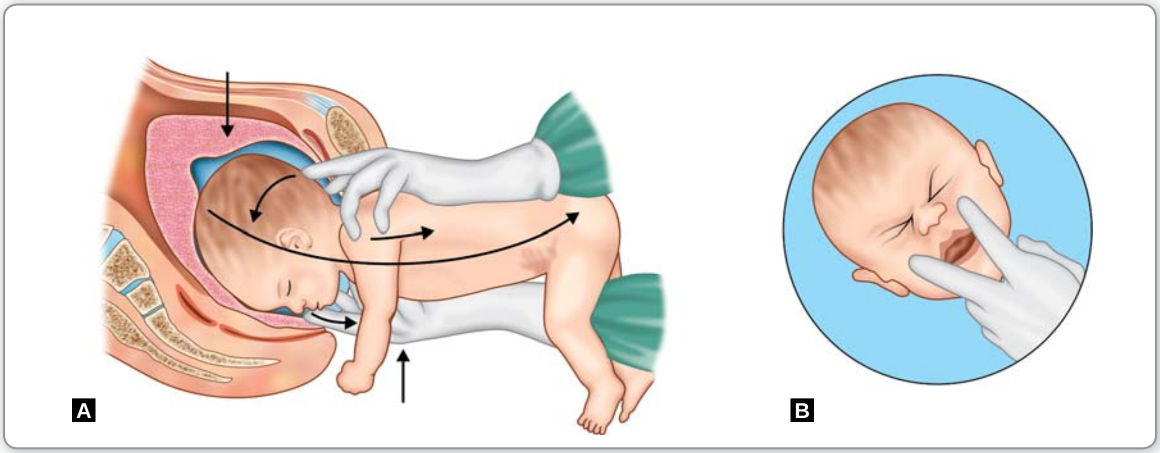


Fig. 26.19: Delivery of the aftercoming head by forceps

giving pull directly on the head and the force is not transmitted through the neck, (b) flexion is better maintained and (c) mucus can be sucked out from the mouth more effectively. The head should be brought as low down as possible by allowing the baby to hang by its own weight aided by suprapubic pressure. **When the occiput lies against the back of the symphysis pubis**, an assistant raises the legs of the child as much to facilitate introduction of the blades from below. Too much elevation of the trunk may cause extension of the head. The forceps pull maintains an arc, which follows the axis of the birth canal (Fig. 26.19). Ordinary forceps with usual length of shank, as in Das's variety, is quite effective. **Piper forceps** is especially designed (absent pelvic curve) for use in this condition. **The head should be delivered slowly (over 1 minute) to reduce compression-decompression forces as that may cause intracranial bleeding**.

(c) ***Malar flexion and shoulder traction*** **(modified Mauriceau-Smellie-Veit technique):** The technique is named after the three great obstetricians who described the use of the grip independently. The baby is placed on the supinated left forearm (preferred) with the limbs hanging on either sides. The middle and the index fingers of the left hand are placed over the malar bones on either sides (modification of the original method, where the index finger was introduced inside the mouth). This maintains flexion of the head. The ring and little fingers of the pronated right hand are placed on the child's right shoulder, the index finger is placed on the left shoulder and the middle finger is placed on the suboccipital region (Fig. 26.20). Traction is now given in downward and backward direction till the nape of the neck is visible



Figs 26.20A and B: Delivery of the aftercoming head by malar flexion and shoulder traction—(A) original Mauriceau-Smellie-Veit; (B) Modification (preferred)

under the pubic arch. **The assistant gives suprapubic pressure during the period to maintain flexion**. Thereafter, the fetus is carried in upward and forward direction toward the mother's abdomen releasing the face, brow and lastly, the trunk is depressed to release the occiput and vertex.

- **Resuscitation of the baby:** The baby may be asphyxiated and need to be resuscitated.

THIRD STAGE: The third stage is usually uneventful. The placenta is usually expelled out soon after delivery of the head. **If prophylactic ergometrine** is to be given, it should be administered intravenously with the crowning of the head.

Preterm breech: ECV with preterm breech presentation is not recommended. CS is commonly done when fetal weight is less than 1,500 g. However, it is not yet known whether the better perinatal outcome is due to the CS or the greater use of antenatal steroids and better neonatal care.

MANAGEMENT OF COMPLICATED BREECH DELIVERY

When a women presents in advanced labor, it may be difficult to decide what would be ideal mode of delivery. However, if breech is not visible at the perineum it may be possible to deliver the baby by CS. **Simulated teaching using mannequins and model pelvis with an experienced trainer can imporve the skill and performance of such maneuvers**.

DELAY IN DESCENT OF THE BREECH: The breech may be arrested**:**

- **At the outlet**
- **In the cavity**
- **At the brim**

- **Arrested at the outlet:** The causes are—(a) big size baby with extended legs (the most common), (b) weak uterine contractions, (c) rigid perineum and (d) outlet contraction.

Management: If the outlet is contracted and/or the baby is big, cesarean section even at this stage, is the method of choice.

- **In the absence of outlet contraction and feto-pelvic disproportion:** Liberal episiotomy and fundal pressure with or without groin traction (either single groin or both the groins) usually become effective (Fig. 26.21). The index finger(s) is placed in the groin fold and traction (along with uterine contraction) is exerted more toward the trunk than toward the femur (risk of fracture femur). **In present day obstetrics, such maneuvers are not advocated.**
- **Arrest of the breech at or above the level of ischial spines: The causes may be**: (i) Pelvic contraction, (ii) Big baby, (iii) Weak uterine contraction. **The best treatment** in such cases is delivery by **cesarean section**. By the time cervix is fully dilated, the breech should descend down to the perineum. This is called **trial of breech.** If this fails to occur, feto-pelvic disproportion is likely.

- **Frank breech extraction (Pinard's maneuver)**—is done by intrauterine manipulation (for breech decomposition) to convert a frank breech to a footling breech. This is possible when the membranes have ruptured recently. **In Pinard's maneuver,** the middle and the index fingers (Fig. 26.22) are carried up to the popliteal fossa. It is then pressed and abducted so that the fetal leg is flexed. The fetal foot is then grasped at the ankle and breech extraction is accomplished.

EXTENDED ARMS: One or both the arms may be fully stretched along the side of the head or lie behind the neck (nuchal displacement). The cause is usually due to faulty technique in delivery—using unnecessary traction, forgetting the principle of 'never pull but push from above'. Arrest occurs with the delivery of the trunk up to the costal margins. **The diagnosis is made** by noting the winging of the scapula and absence of the flexed limbs in front of the chest.

Management: The management calls for the urgent delivery of the arms, first the posterior and then the anterior one. The delivery of the arm may be accomplished by adopting any one of the following methods: (a) Classical, (b) Lovset.

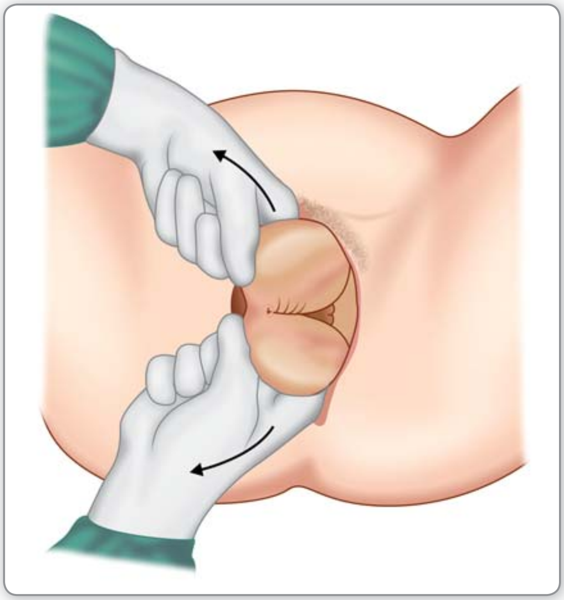


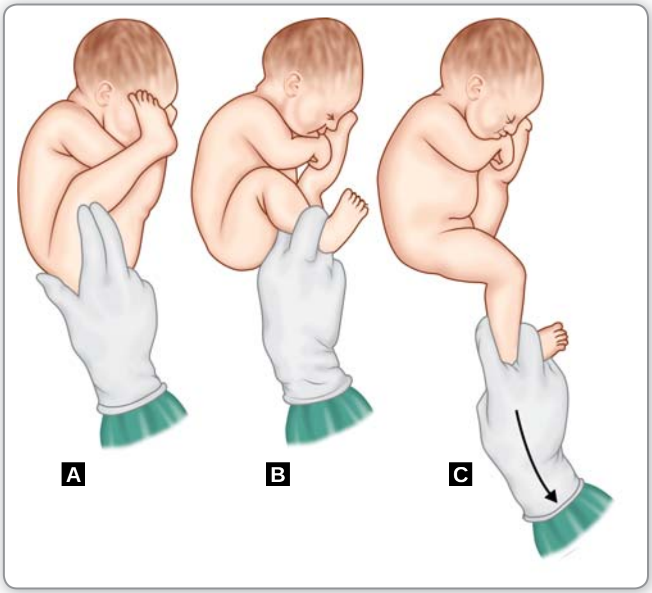
Fig. 26.21: Both groin traction

Classical: It works with the same principle as with **Lovset's maneuver**. In addition, it needs intrauterine manipulation while the patient is under general anesthesia. First, the posterior arm is delivered followed by the anterior arm. Left hand is introduced along the curve of the sacrum while the baby is pulled slightly upward. With firm pressure over the humerus, the posterior arm is pushed over the baby's face. The extended anterior arm is delivered from the anterior aspect by introducing the right hand in the same manner, while the baby's trunk is depressed toward the perineum. Details of steps have been discussed in author's book "Emergencies in Manipulative and Operative Obstetrics".

LOVSET'S MANEUVER: It is widely practiced in preference to the classical method of bringing down an arm. **The following are the advantages**: (1) Wider applicability—It can be applied even when the classical method becomes difficult. (2) Intrauterine manipulation is nil. (3) A single manipulation is effective to all types of displacement of the arms. (4) General anesthesia is usually not needed.

Principles: Because of the curved birth canal, when the anterior shoulder remains above the symphysis pubis, the posterior shoulder will be below the sacral promontory. If the fetal trunk is rotated keeping the back anterior and maintaining a downward traction, the posterior shoulder will appear below the symphysis pubis.

Procedure (Fig. 26.23): The baby (wrapped in a warm dry towel) is grasped, using both hands by femoropelvic grip



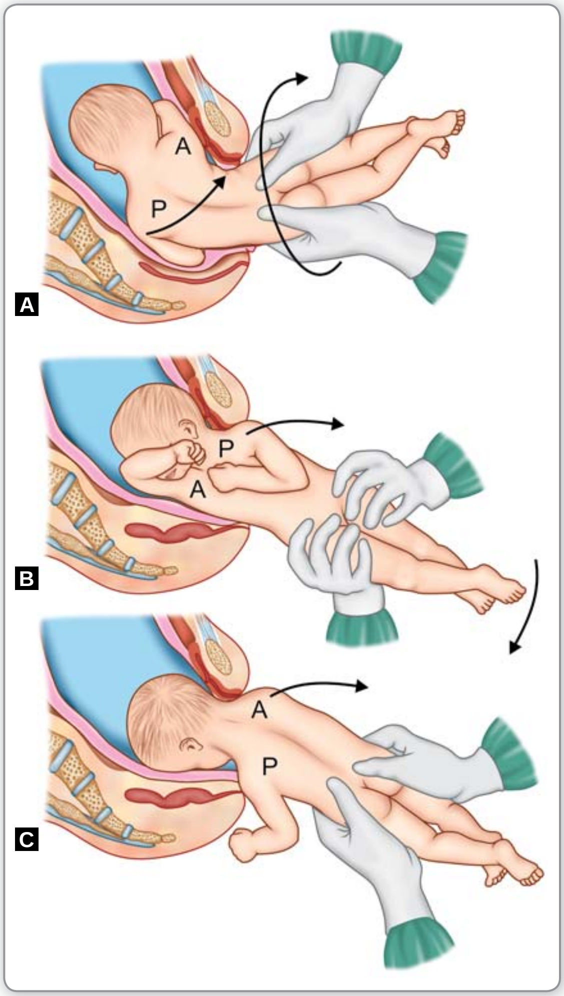
Figs 26.22A to C: Pinard's manuever—(A) Flexion and abduction of popliteal fossa; (B) To catch hold the ankle; (C) To pull down by movement of abduction

keeping the thumbs parallel to the vertebral column. The maneuver should start only when the inferior angle of the anterior scapula is visible underneath the pubic arch.

Step—1: The baby is lifted slightly to cause lateral flexion. The trunk is rotated through 180° keeping the back anterior and maintaining a downward traction. This will bring the posterior arm to emerge under the pubic arch which is then hooked out.

Step—2: The trunk is then rotated in the reverse direction keeping the back anterior to deliver the erstwhile anterior shoulder under the symphysis pubis.

Nuchal displacement of arm is where the arm is flexed at the elbow and extended at the shoulder and lies behind the fetal head. After grasping the baby at the pelvic girdle with thumbs along the sacrum, the trunk is rotated 180° toward the fingertips of the trapped arm. This may draw the elbow forward and render it amenable to **Lovsett's maneuver**. If this fails, the arm is forcibly extracted by hooking. In that case fracture almost always follows.



Figs 26.23A to C: Lovset's maneuver

ARREST OF THE AFTERCOMING HEAD

- **At the brim: The causes of arrest are**—(1) deflexed head (2) contracted pelvis and (3) hydrocephalus.

Management: (1) If the arrest is due to a deflexed head, the delivery is to be completed by malar flexion and shoulder traction along with suprapubic pressure by the assistant. The head is to be negotiated through the brim in the transverse diameter and rotated in the cavity. Forceps should not be applied in high head.

(2) If the arrest of the head is due to contracted pelvis or hydrocephalus, perforation of head is to be done (see p. 406 and 586).

- **In the cavity: The causes of arrest of the head in the cavity are**—(1) deflexed head and (2) contracted pelvis.

The best management is delivery of the head by forceps which is effective in both the circumstances. Malar flexion and shoulder traction may be effective only in deflexed head.

- **At the outlet:** The causes of arrest are—(1) rigid perineum and (2) deflexed head.

Episiotomy followed by forceps application or malar flexion and shoulder traction is quite effective.

- **Delivery of the head through an incompletely dilated cervix:** The common causes are—(1) premature baby, (2) macerated baby, (3) footling presentation and (4) hasty delivery of breech before the cervix is fully dilated.

Management: **If the baby is living,** the cervix is to be pushed up while traction of the fetal trunk is made by malar flexion and shoulder traction (shoe-horn method). If necessary, **Duhrssen's incision** can be made at 2 and 10 O' clock position on the cervix. If the baby is dead, perforation of the head is better than watchful expectancy, hoping for full dilatation of the cervix.

- **Occipitoposterior position of the head:** It usually occurs in spontaneous breech delivery. The fetal trunk and the head are rotated to bring them anteriorly. For rotation, **the fetal trunk and the head are to be grasped; the hand and the fingers are positioned like that in malar flexion and shoulder traction.** In premature baby, the delivery of the head may be completed as face-to-pubis by reversed malar flexion and shoulder traction (Prague) method or by forceps.

KEY POINTS

BREECH PRESENTATION

- In **breech presentation**, the podalic pole presents at the pelvic brim, and the lie is longitudinal. It is the most common malpresentation.
- **Types** of breech presentation are: complete, frank, footling and knee presentation.
- **Prematurity** is the most common cause of breech presentation.
- **Ultrasonography** is most informative besides confirmation of diagnosis (see p. 435).
- **Causes of damage and high mortality** to the breech baby are: prematurity, congenital malformation, injury to the skull, intracranial hemorrhage, birth asphyxia due to prolonged labor, cord compression or cord prolapse, birth injury (rupture of liver, fracture of femur, cervical and brachial plexus injury).
- **Management** of breech presentation: External cephalic version (ECV) is done after 36 weeks of pregnancy if not contraindicated. ECV has many benefits (see p. 440) in selected cases (see p. 440). However, it has got complications also (see p. 440).
- **Management** of breech delivery is by a) elective cesarean section after 38 weeks or b) vaginal delivery (assisted breech delivery) or c) cesarean delivery in labor.
- **Trial of labor** (vaginal breech delivery) is considered for cases with (a) fetal weight between 2.0 Kg and 3.5 Kg (b) adequate pelvis (c) frank or complete breech (d) flexed or neutral head (e) in presence of a skilled obstetrician, and with (f) informed consent.

FACE PRESENTATION

Face is a rare variety of cephalic presentation where the **presenting part** is the face. **The attitude** of the fetus shows complete flexion of the limbs with extension of the spine. **There is complete extension of the head** so that the occiput is in contact with the back. **The denominator** is mentum.

Position: There are four positions of the face according to the relation of the chin to the left and right sacroiliac joints or to the right and left iliopubic eminences. Face presentation results most likely from complete extension of deflexed head of a vertex presentation. **The numbering of the face positions is obtained as follows:**

1st vertex (LOA)	becomes	1st Face →	Right mentoposterior	(RMP)
2nd vertex (ROA)	becomes	2nd Face→	Left mentoposterior	(LMP)
3rd vertex (ROP)	becomes	3rd Face →	Left mentoanterior	(LMA)
4th vertex (LOP)	becomes	4th Face →	Right mentoanterior	(RMA)

The most common position is left mentoanterior (LMA)—As the ROP position is 5 times more common than LOP and as the conversion of face occurs from deflexed OP, LMA is the commonest. Overall anterior positions are more frequent than the posterior one.

Incidence: Its frequency is about 1 in 500 births. Face presentation present during pregnancy (primary) is rare, while that developing after the onset of labor (secondary) is common. It occurs more frequently in multiparae (70%).

Etiology: The cause of extreme extension of the head is not clear in all the cases. The following are the factors which are often associated.

Maternal: (1) Multiparity with pendulous abdomen, (2) Lateral obliquity of the uterus especially, if it is directed to the side toward which the occiput lies, (3) Contracted pelvis is associated in about 40% cases. Flat pelvis favors face presentation, (4) Pelvic tumors.

Fetal: (1) Congenital malformations (15%)—(a) The most common one is anencephaly. The almost nonexistent neck with absence of the cranium makes it easy to feel the facial structure even with semi-extended head, (b) Congenital goiter—prevalent in endemic areas, (c) Dolichocephalic head with long anteroposterior diameter, (d) Congenital bronchocele. (2) Twist of the cord several turns round the neck. (3) Increased tone of the extensor group of neck muscles.

MECHANISM OF LABOR

MENTOANTERIOR 60–80% (LMA OR RMA)

The principal movements are like those of corresponding occipitoanterior position. The exceptions are increasing extension instead of flexion and delivery by flexion instead of extension of the head.

Engagement: The diameter of engagement is the oblique diameter—right in LMA, left in RMA, with the mentum related to one iliopubic eminence and the glabella to the opposite sacroiliac joint. **The engaging diameter of the head** is submentobregmatic 9.5 cm (3 3/4") in fully extended head or submentovertical 11.5 cm (4 1/2") in partially extended head. **Engagement is delayed** because of long distance between the mentum and biparietal plane (7 cm). Descent with increasing extension occurs till the chin touches the pelvic floor.

Internal rotation—Internal rotation of the chin occurs through 1/8th of a circle anteriorly, placing the mentum behind the symphysis pubis. Further descent occurs till the submentum hinges under the pubic arch.

Delivery of the head—The head is born by flexion delivering the chin, face, brow, vertex and lastly the occiput. **The diameter distending the vulval outlet** is submentovertical—11.5 cm (4 1/2"). Restitution occurs through 1/8th of a circle opposite to the direction of internal rotation. External rotation occurs further 1/8th of circle to the same side of restitution so that ultimately the face looks directly to the left thigh in LMA and right thigh in RMA. This follows delivery of the anterior shoulder followed by the posterior shoulder and the rest of the trunk by lateral flexion.

MENTOPOSTERIOR (20–25%) (RMP OR LMP):

The cardinal movements in the mechanism of mentoposterior positions are like those of occipitoposterior position. **The salient differentiating features are—**(1) In the mentoposterior position, **anterior rotation of the mentum occurs in only 20–30% cases**.

(2) **In the rest (70–80%),** incomplete anterior rotation, nonrotation or short posterior rotation of the mentum occurs. Arrest occurs in all these positions with average size pelvis and fetalhead. Unlike persistent occipitoposterior, where occasional face-to-pubis delivery occurs, **there is no possibility of spontaneous delivery in persistent mentoposterior. This is because** the relatively short neck cannot clear off the total length of the sacrum (12 cm). As such the thorax is thrust in, resulting bregmaticosternal diameter (18 cm or 7") to occupy the pelvis (Fig. 26.24). As a result, the labor becomes inevitably obstructed.

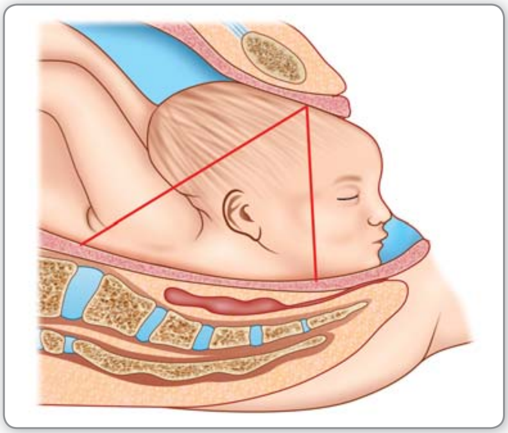


Fig. 26.24: Head cannot extend further, nor flexion is possible. Bregmaticsternal diameter (18 cm or 7") cannot be accommodated in the pelvis resulting in obstructed labor

DIAGNOSIS

Antenatal diagnosis is rarely made. Diagnosis is made only during labor but in about half, the detection is made at the time of delivery.

ABDOMINAL FINDINGS

Inspection: Because of “S”-shaped spine, there is no visible bulging of the flanks.

Palpation (Fig. 26.25): The diagnostic features in mentoanterior and mentoposterior are tabulated below.

	Mentoanterior	Mentoposterior
Lateral grip	(1) Fetal limbs are felt anteriorly. (2) Back is on the flank and is difficult to palpate. (3) The chest is thrown anteriorly against the uterine wall and is often mistaken for back	(1) Back is felt to the front and better palpated only towards the podalic pole because of extension of spine.
Pelvic grip	(1) Head seems big and is not engaged. (2) Cephalic prominence is to the side toward which back lies (3) Groove between the head and back is not so prominent.	(1) Same (2) Same (3) The groove is prominent.
Auscultation	FHS is distinctly audible anteriorly through the chest wall of the fetus toward the side of limbs	FHS is not so distinct and is audible on the flank toward the side of limbs.

VAGINAL EXAMINATION

The diagnostic features are palpating the mouth with hard alveolar margins, nose, malar eminences, supraorbital ridges and the mentum (Fig. 26.26). In early labor, because of high head and sausage shaped bag of membranes, the parts are not clearly defined. In late labor, the parts are often obscured due to edema. It is often confused with breech presentation. The distinguishing features are—(1) the mouth and the malar eminences are not in a line; but in breech, the anus and the ischial tuberosities are in one line, (2) sucking effect of mouth, (3) hard alveolar margins and (4) absence of meconium staining on the examination fingers. The mentum and the mouth should be clearly identified to exclude brow presentation and to identify the position. The examination should be conducted gently, as there is chance of injury to the eyes. Assessment of the pelvis should be done as a routine.

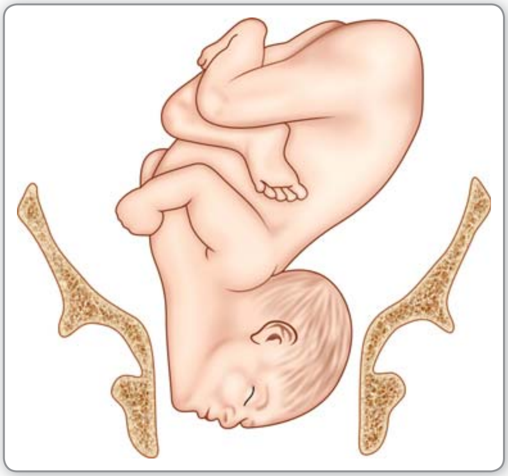


Fig. 26.25: Chief features to be recognized on abdominal examination in face presentation

SONOGRAPHY/RADIOGRAPHY: This should be done to confirm the diagnosis, to exclude bony congenital malformation of the fetus and to note the size of the baby.

CLINICAL COURSE: In spite of the fact that the engaging diameter of the head in flexed vertex and the extended face presentation is the same—9.5 cm (3 3/4"), **the clinical course of the latter is adversely affected because of the following**:

- **Irregular face ill fits** with the lower uterine segment. The poor ball valve action results in formation of elongated bag of membranes which is likely to rupture early.
- **Chance of cord prolapse is more**.
- **Delay of labor**, in all the stages, is common. **The causes are**—(a) weak uterine contractions, (b) absence of molding of the facial bones, (c) delayed engagement—the distance between the biparietal plane to chin is 7 cm and to occiput is only 3 cm (Fig. 26.27), (d) late internal rotation and (e) arrest and at times, insuperable obstruction if mentoposterior fails to rotate anteriorly.

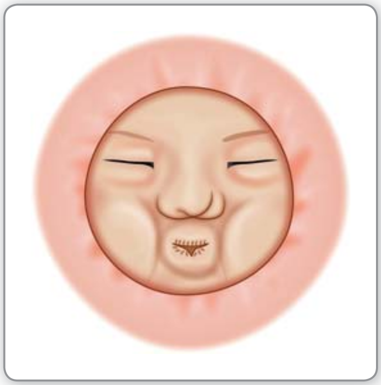
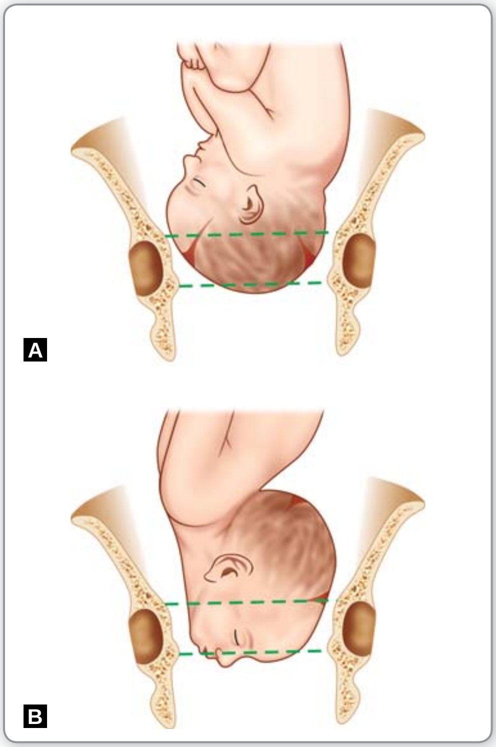


Fig. 26.26: Vaginal touch picture showing landmarks in mentoanterior position



Figs 26.27A and B: (A) Vertex presentation—head engaged; (B) Face presentation—head not yet engaged

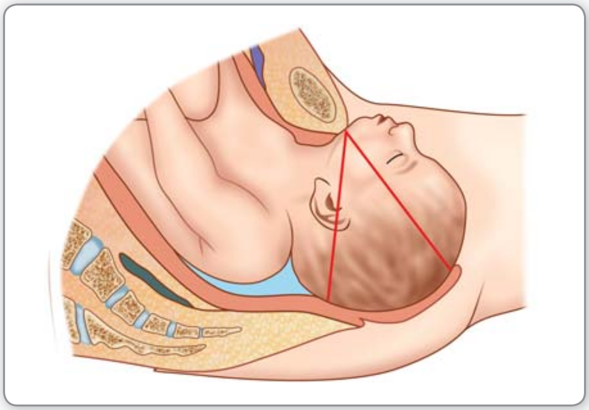


Fig. 26.28: Submentovertical diameter (11.5 cm or 4 1/2") emerges out of the introitus

- **Chance of perineal damage** is more because of a wide biparietal diameter—9.5 cm (3 3/4") stretches the perineum and submentovertical diameter 11.5 cm (4 1/2") emerges out of the introitus (Fig. 26.28).
- **Postpartum hemorrhage** is more likely due to atonic uterus and trauma following operative delivery.

PROGNOSIS: Maternal—In mentoanterior, the maternal risk is not much increased. However, there is increased morbidity due to operative delivery and vaginal manipulation. In neglected cases, the risks of impacted mentoposterior leading to obstructed labor and ruptured uterus are not uncommon.

Fetal—Fetal prognosis is, however, adversely affected due to—(a) cord prolapse, (b) increased operative delivery, (c) cerebral congestion due to poor venous return from the head and neck and (d) neonatal infection due to bacterial contamination within the vagina.

CAPUT AND MOLDING: Due to poor venous return from the head and neck, marked caput forms, distorting the entire face. The lips and the eyelids are markedly swollen with considerable appearance of bruising. There is no compression of the facial bones but there is elongation of occipitofrontal diameter (see Fig. 9.5). The extended attitude of the head, swelling of the face and the elongation of the head subside within a few days.

MANAGEMENT

Overall assessment of the case is to be done to note—(1) pelvic adequacy (clinical), (2) size of the baby, (3) associated complicating factors, if any, like elderly primigravidae, severe preeclampsia, post-cesarean pregnancy and postmaturity, (4) congenital fetal malformation and (5) position of the mentum.

Indications of elective or early cesarean section: (1) Contracted pelvis, (2) Big baby, (3) Associated complicating factors.

VAGINAL DELIVERY

MENTOANTERIOR

First stage: In uncomplicated cases, a wait and watch policy is adopted. Labor is conducted in the usual procedure and the special instructions, as laid down in occipitoposterior positions, are to be followed.

Second stage: One should wait for spontaneous delivery to occur. **Perineum should be protected with liberal mediolateral episiotomy**. In case of delay, forceps delivery is done.

MENTOPOSTERIOR

First stage: In uncomplicated cases, vaginal delivery is allowed with strict vigilance hoping for spontaneous anterior rotation of the chin.

Second stage: (1) **If anterior rotation of the chin occurs**, spontaneous or forceps delivery with episiotomy is all that is needed. (2) **In incomplete or malrotation:** Early decision for the method of delivery is to be taken soon after full dilatation of the cervix. **The following methods may be employed to expedite the delivery**.

- **Cesarean section** is the preferred method and is commonly done these days.
- **Manual rotation** of the chin anteriorly followed by immediate forceps extraction is rarely done these days. The principles and the methods are similar to those employed in unrotated occipitoposterior position.

BROW PRESENTATION

Brow is the rarest variety of cephalic presentation where the **presenting part** is the brow and the **attitude of the head** is short that of degree of extension necessary to produce face presentation, i.e. the head lies in between full flexion and full extension. The denominator is the fore head (frontum: Fr).

INCIDENCE: The incidence of brow is very rare, about 1 in 1,000 births. However, it may persist temporarily while a deflexed head tends to become extended to produce a face presentation. This happens especially in flat pelvis where the biparietal diameter is held in the sacrocotyloid diameter.

CAUSES: The causes of persistent brow are more or less the same as those of face presentation. **The position is commonly unstable and converts to either vertex or face presentation**.

DIAGNOSIS: Antenatal diagnosis is rarely made. The findings are more or less like those of face presentation. The cephalic prominence and the groove between it and the back are less prominent (Fig. 26.29). The head feels very big and is nonengaged.

Vaginal examination: The position is to be confirmed on vaginal examination by **palpating supraorbital ridges and anterior fontanel**. If the anterior fontanel is on mother's left, with the sagittal suture in transverse pelvic diameter, it is left frontum transverse position. In late labor, the landmarks may be obscured by caput formation.

Sonography (Fig. 26.30) is confirmatory and also helps in excluding bony congenital malformation of the fetus.

MECHANISM OF LABOR: Diameter of engagement is through the oblique diameter with the brow anterior or posterior. As the engaging diameter of the head is mentovertical (14 cm), **there is no mechanism of labor in an average size baby with normal pelvis**. However, if the baby is small and the pelvis is roomy with good uterine contractions, delivery can occur in mentoanterior brow position. The brow descends until it touches the pelvic floor. Internal rotation and

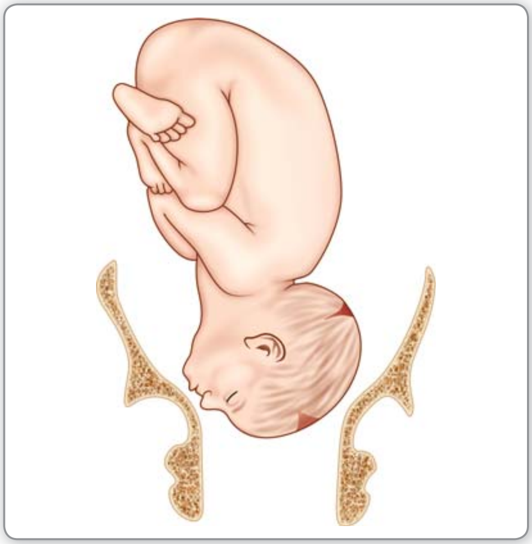


Fig. 26.29: Chief features to be recognized during abdominal examination in brow presentation

descent occur till the root of the nose hinges under the symphysis pubis. The brow and the vertex are delivered by flexion followed by extension to deliver the face. The mechanism is more or less the same as face-to-pubis delivery. Usual restitution and external rotation occur. **There is no mechanism in posterior brow position**.

TRIAL OF LABOR: Brow presentation when transitory, trial of labor may be permissible. Correction of brow with felexion to occiput presentation or complete extension to a face presentation occurs. In such a situation, though rare, trial of labor may be possible.

COURSE AND PROGNOSIS: In case of persistent brow presentation, there is a chance of obstructed labor. **It is an important cause of rupture of uterus in multiparae**. **On occasion (10%), there may be spontaneous conversion of brow** into face or vertex presentation.

MOLDING: Considerable overlapping of bones occurs if the labor lasts long. There is compression of submentovertical diameter with elongation of occipitofrontal diameter (see Fig. 9.5). There is associated marked bulging of the forehead due to caput formation.

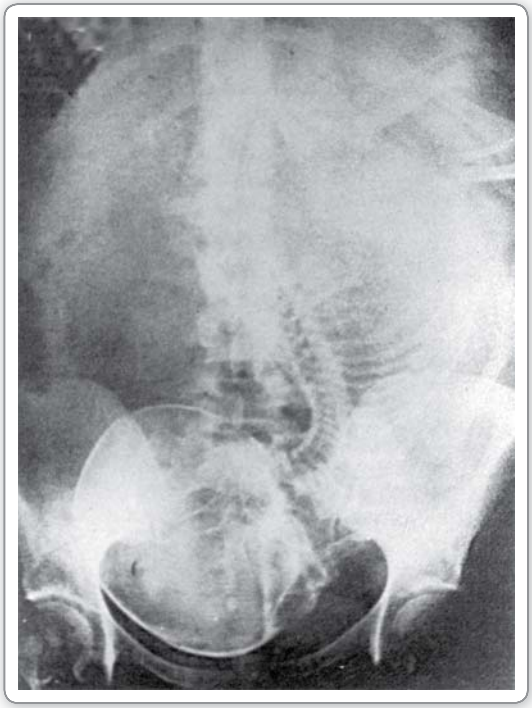


Fig. 26.30: Straight X-ray abdomen showing brow presentation. The head feels very big. Patient was delivered by cesarean section

MANAGEMENT

During pregnancy: If the presentation is diagnosed during pregnancy and there is no other contraindications for vaginal delivery, nothing is to be done. **Contracted pelvis and congenital malformation of the fetus are to be excluded**. Spontaneous correction into face is likely to occur.

Elective cesarean section: Cases with persistent brow presentation are delivered by elective cesarean section.

During labor: (1) **In uncomplicated cases**, if spontaneous correction to either vertex or face fails to occur early in labor, cesarean section is the best method of treatment.

(2) **Manual Correction** to face with full dilatation of cervix is seldom practiced nowadays.

(3) **Craniotomy—**If the labor becomes obstructed and the baby is dead, craniotomy is done. **Rupture of the uterus should be excluded**.

TRANSVERSE LIE

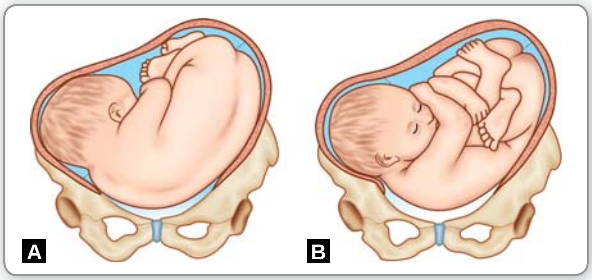
When the long axis of the fetus lies perpendicularly to the maternal spine or centralized uterine axis, it is called transverse lie. But more commonly, the fetal axis is placed oblique to the maternal spine and is then called **oblique lie.** In either of the conditions, the shoulder usually presents over the cervical opening during labor and as such both are collectively called shoulder presentations.

POSITION: The position is determined by the direction of the back, which is the denominator. **The position may be—**(1) dorsoanterior, which is the most common (60%). The flexor surface of the fetus is better adapted to the convexity of the maternal spine (Fig. 26.31), (2) dorsoposterior, (3) dorsosuperior or (4) dorsoinferior. The last two are rare. **In dorsoposterior, chance of fetal extension is common with increased risk of arm prolapse. According to the position of the head, the fetal position is termed**

right or left, the left one being more common than the right.

INCIDENCE: The incidence is about 1 in 200 births. It is common in premature and macerated fetuses, 5 times more common in multiparae than primigravidae. Transverse lie in twin pregnancy is found in 40% of cases.

ETIOLOGY: The causes are—(1) Multiparity—lax and pendulous abdomen, imperfect uterine tone and extreme uterine obliquity are the responsible factors. (2) Prematurity—center of the gravity lies almost in the middle of the body. (3) Twins—it is more common for the second baby than the first one to be in transverse position. (4) Hydramnios. (5) Contracted pelvis. (6) Placenta previa. (7) Pelvic tumors. (8) Congenital malformation of the uterus—arcuate or subseptate and (9) Intrauterine death.



Figs 26.31A and B: Positions of transverse lie—(A) Dorsoanterior and (B) Dorsoposterior

DIAGNOSIS

ABDOMINAL EXAMINATION

Inspection: The uterus looks broader and often asymmetrical, not maintaining the pyriform shape.

Palpation:

- **The fundal height** is less than the period of amenorrhea.
- **Fundal grip**—Fetal pole (breech or head) is not palpable.
- **Lateral grip**—(a) Soft, broad and irregular breech is felt to one side of the midline and smooth, hard and globular head is felt on the other side. The head is usually placed at a lower level on one iliac fossa. (b) The back is felt anteriorly across the long axis in dorsoanterior or the irregular small parts are felt anteriorly in dorsoposterior.
- **Pelvic grip**—The lower pole of the uterus is found empty. This, however, is evident only during pregnancy but during labor, it may be occupied by the shoulder.

Auscultation: FHS is heard easily much below the umbilicus in dorsoanterior position. FHS is, however, located at a higher level and often indistinct in dorsoposterior position.

Ultrasonography or radiography confirms the diagnosis (Fig. 26.32).

VAGINAL EXAMINATION

During pregnancy, the presenting part is so high that it cannot be identified properly but one can feel some soft parts.

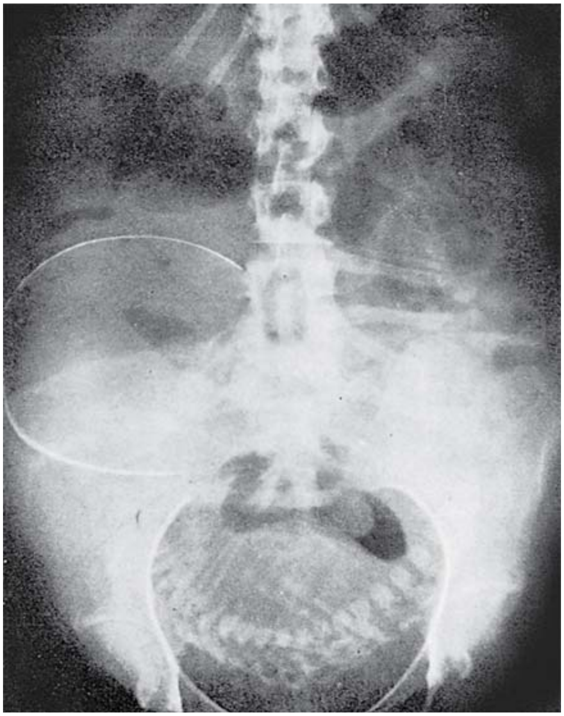


Fig. 26.32: Straight X-ray abdomen showing single fetus with transverse lie *[Courtesy: G & O Dept. Patna MCH]*

During labor—Elongated bag of the membranes can be felt if it does not rupture prematurely. **The shoulder is identified by palpating the following parts**—acromion process, the scapula, the clavicle and axilla (Fig. 26.33). The characteristic landmarks are the feeling of the ribs and intercostal spaces (grid iron feel). On occasion, the arm is found prolapsed. It should be remembered that the findings of **a prolapsed arm is confined not only to transverse lie but it may also be associated with compound presentation**. Rarely, a leg may be prolapsed. Frequently, a loop of cord may be found alongside the arm.

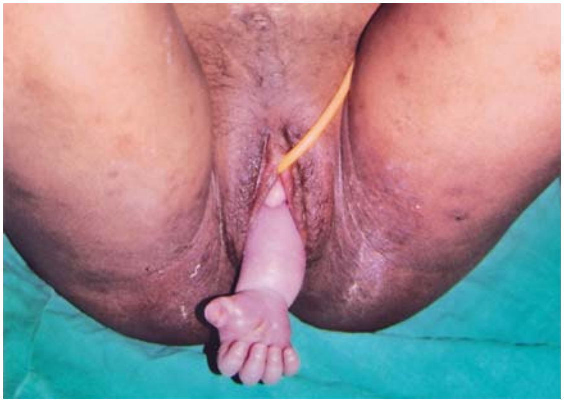


Fig. 26.33: Woman in labor with hand prolapse

Determination of position: Thumb of the prolapsed hand, when supinated, points toward the head, the palm corresponds to the ventral aspect. The angle of the scapula, if felt, indicates the position of the back. The side to which the prolapsed arm belongs, can be determined by shaking hands with the fetus. If the right hand is required for this, the prolapsed arm belongs to right side and vice-versa.

CLINICAL COURSE OF LABOR

There is no mechanism of labor in transverse lie and an average size baby fails to pass through an average size pelvis. If the lie remains uncorrected and the labor is left uncared for, the following sequence of events may occur.

UNFAVORABLE EVENTS (COMMON): There may be premature rupture of the membranes with escape of good amount of liquor because of absence of ball valve action of the presenting part. The hand of the corresponding shoulder may be prolapsed with or without a loop of cord. The cord may be prolapsed in isolation. There is increased chance of ascending infection from the lower genital tract. With increasing uterine contractions, the shoulder becomes wedged and impacted into the pelvis and the prolapsed arm becomes swollen and cyanosed. Gradually features of obstructed labor supervene.

The pathological anatomy of the uterus is like that of tonic uterine contraction and retraction (see Chapter 25). There is formation of a pathological retraction ring (Fig. 26.34).

The mother gets exhausted and features of dehydration and ketoacidosis develop; evidences of sepsis usually become apparent. **In primigravidae**, in response to obstruction, the uterus becomes inert and features of exhaustion and sepsis are only evident. **But in multiparae**,

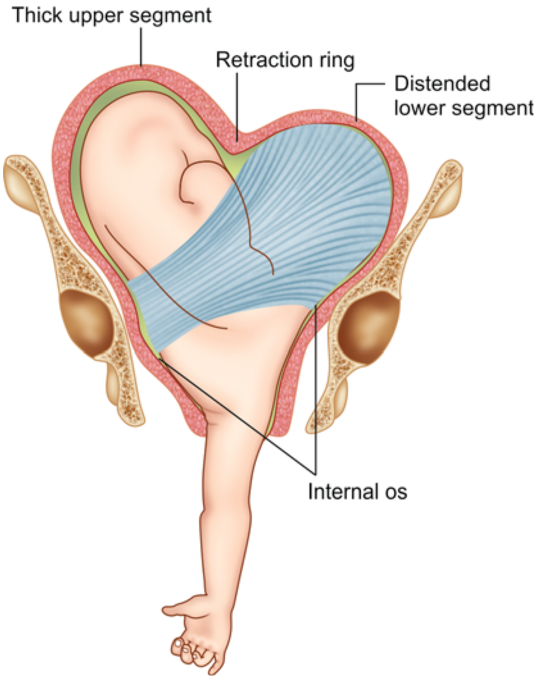


Fig. 26.34: Pathologic anatomy of the uterus in neglected shoulder presentation

the uterus reacts vigorously in response to obstruction and ultimately, the lower segment gives way as a result of marked thinning of its wall.

Neglected shoulder: By neglected shoulder, it means the series of complications that may arise out of shoulder presentation when the labor is left uncared. Such complications are impacted shoulder → obstructed labor → rupture of uterus with clinical evidences of dehydration, ketoacidosis, shock and sepsis. These put the mother and the fetus at risk. With proper intranatal supervision, the condition is avoidable but unfortunately, the condition is still rampant in rural areas of the developing countries.

FAVORABLE EVENTS (RARE): The following are the favorable events that may occur—(1) **spontaneous rectification or version,** (2) **spontaneous evolution** and (3) **spontaneous expulsion (conduplicato corpore)**. These events are very rare and occur only when the baby is premature or macerated.

- **Spontaneous rectification or version:** It usually occurs in early labor with good amount of liquor and the baby is small and movable. Contracting uterus forces the head or the breech lying in the iliac fossa to lie in alignment to the brim. Thus, the lie may be changed from oblique to longitudinal with vertex presentation, when it is called **rectification** or with breech presentation when it is called **version.** It is more frequent in multiparae.
- **Spontaneous evolution:** The arm is usually prolapsed; the head lies on one iliac fossa; the trunk and the breech are forced into the cavity; the neck is markedly elongated. Breech and the trunk are expelled first followed by delivery of the head. This requires very strong uterine contractions.
- **Spontaneous expulsion:** It is extremely rare and occurs only in premature and macerated fetus. Fetus is expelled doubled up, with chest and abdomen apposed. The head and the feet are delivered last.

PROGNOSIS: In a well supervised pregnancy and labor, the maternal and the fetal outlook is not much unfavorable with the increased use of cesarean section. However, increased maternal morbidity following early rupture of the membranes and increased operative delivery, is inevitable.

But in uncared pregnancy and labor, the outlook of the mother and the fetus is very much unpredictable. **The maternal risk is increased due to** dehydration, ketoacidosis, septicemia, ruptured uterus, hemorrhage, shock and peritonitis—sequences of neglected shoulder. **Marked increase of fetal loss is due to** cord prolapse, tonic contraction of the uterus and ruptured uterus. The overall perinatal mortality is as high as 25–50%.

MANAGEMENT OF SHOULDER PRESENTATION

ANTENATAL: External cephalic version should be done in all cases beyond 35 weeks provided there is no contraindication as mentioned in breech presentation (see p. 440). If the lie fails to stabilize even at 36th week, the case is to be managed as outlined in unstable lie.

If version fails or is contraindicated:

- **The patient is to be admitted at 37th week**, because risk of early rupture of the membranes and cord prolapse is very much there. **Elective cesarean section** is the preferred method of delivery.
- **Vaginal delivery may be allowed** in a dead or congenitally malformed (small size) fetus. The labor may be allowed to continue under supervision till full dilatation of the cervix, when the baby can be delivered by internal version.

PATIENT SEEN IN LABOR: The principles in management are as outlined below:

EARLY LABOR:

- **External cephalic version:** Provided there is good amount of liquor amnii and there is no contraindication. External cephalic version should be tried in all cases.
- **Cesarean section** is the preferred method of delivery if version fails or is contraindicated. Difficulties are faced during cesarean section as the lower uterine segment is poorly developed. However, the fetus can be manipulated to a polar (cephalic/breech) presentation before entering

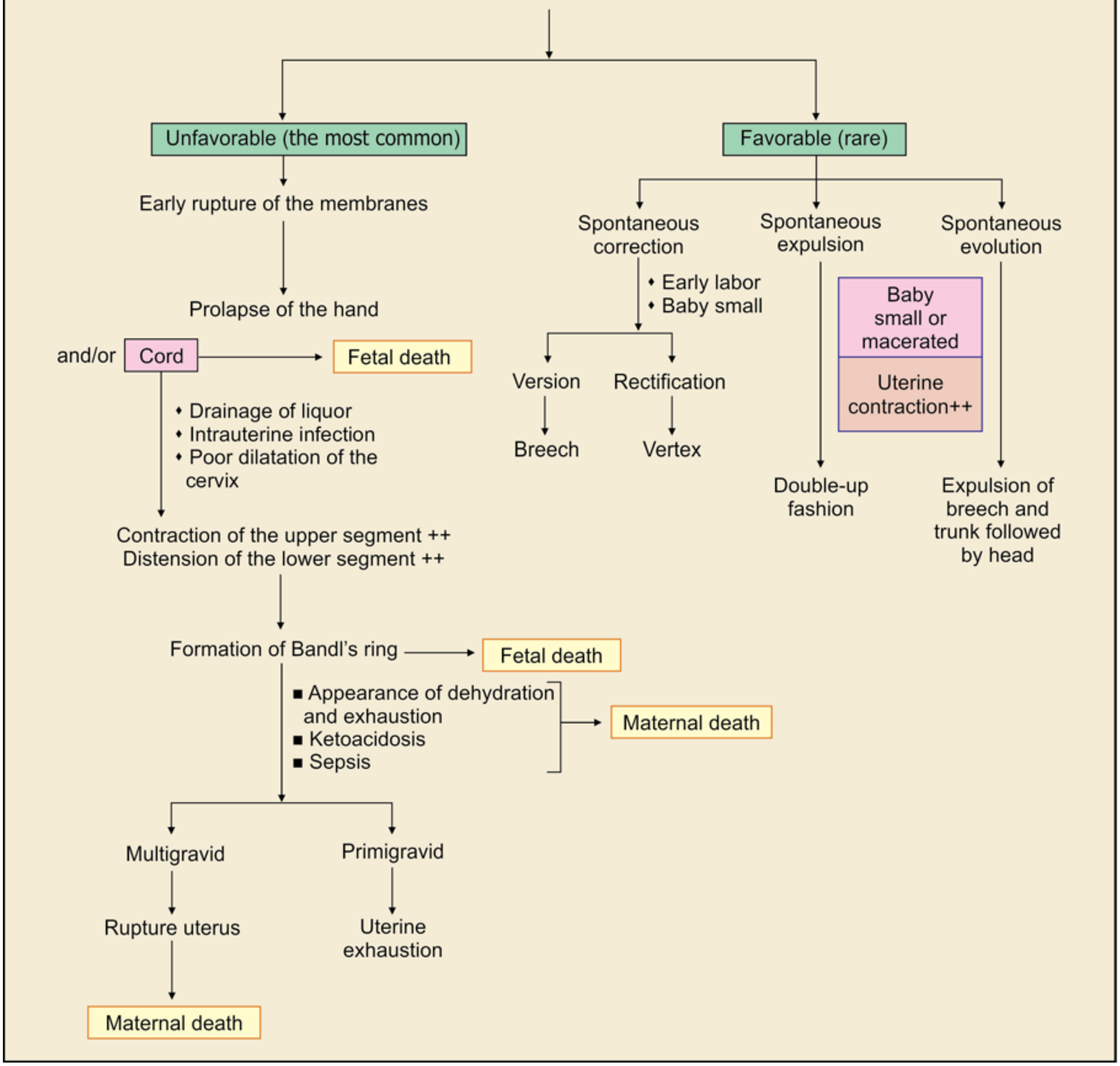
the uterus and a low transverse incision may be made. Otherwise lower segment vertical incision may be made.

LATE LABOR:

- **Baby alive**—There is hardly any scope of external version in late labor because of invariable rupture of the membranes and drainage of liquor. **If the baby is mature and the fetal condition is good, it is preferable to do cesarean section in all cases.**

  Internal version—In a singleton fetus, the risk of internal version is high. **Not only it might inflict injury to the uterus (rupture uterus) but also the fetal mortality is increased to the extent of about 50%. In modern obstetric practice, internal version is not recommended except in the case of second twin.**
- **Baby dead—Cesarean section** even in such cases, is much safer in the hands of those who are not conversant with destructive operations. Internal version should not be done.

CLINICAL COURSE OF LABOR IN TRANSVERSE LIE LEFT UNCARED FOR


Unfavorable (the most common)
Favorable (rare)
Early rupture of the membranes
Prolapse of the hand
and/or
Cord
Fetal death
• Drainage of liquor
• Intrauterine infection
• Poor dilatation of the cervix
Contraction of the upper segment ++
Distension of the lower segment ++
Formation of Bandl's ring
Fetal death
■ Appearance of dehydration and exhaustion
■ Ketoacidosis
■ Sepsis
Maternal death
Multigravid
Primigravid
Rupture uterus
Uterine exhaustion
Maternal death
Spontaneous correction
Spontaneous expulsion
Spontaneous evolution
• Early labor
• Baby small
Version
Rectification
Breech
Vertex
Baby small or macerated
Uterine contraction++
Double-up fashion
Expulsion of breech and trunk followed by head

UNSTABLE LIE

This is a condition where the presentation of the fetus is constantly changed even beyond 36th week of pregnancy when it should have been stabilized.

CAUSES: The causes are those which prevent the presenting part to remain fixed in the lower pole of the uterus. **Such conditions are**: (1) Grand multipara with lack of uterine tone and pendulous abdomen—the most common cause, (2) Hydramnios, (3) Contracted pelvis, (4) Placenta previa, (5) Pelvic tumor.

Complications: Cord entanglement is a possible risk. Risk of cord prolapse is there once the membranes rupture. Perinatal death is high.

MANAGEMENT: ANTENATAL: At each antenatal visit, the presentation and the lie are to be checked. If there is no contraindication, external version is to be done to correct the malpresentation. ***Hospitalization***: **The patient is to be admitted at 37th week**. Premature or early rupture of the membranes with cord prolapse is the real danger with the lie remaining oblique. **After admission, the investigation is directed to exclude** placenta previa, contracted pelvis or congenital malformation of the fetus with the help of sonography for localization of the placenta.

FORMULATION OF THE LINE OF TREATMENT: • **Elective cesarean section** is done in majority of the cases especially in the presence of complicating factors like preeclampsia, placenta previa, contracted pelvis, etc. • **Stabilizing induction of labor**: **External cephalic version** is done (if not contraindicated) after 37 weeks → oxytocin infusion is started to initiate effective uterine contractions. This is followed by low rupture of the membranes (amniotomy). Labor is monitored for successful vaginal delivery. This procedure may be done even after the spontaneous onset of labor.

COMPOUND PRESENTATION *(Syn: Complex presentation)*

When a cephalic presentation is complicated by the presence of a hand or a foot or both alongside the head or presence of one or both hands by the side of the breech, it is called compound presentation. The most common one being the head with hand (Fig. 26.35) and the rarest one being the presence of head, hand and a foot. The incidence is about 1 in 600.

ETIOLOGY: Conditions preventing engagement of the head can result in slipping of either upper or lower limbs by the side of the head. **Prematurity (the most common)**, contracted pelvis, pelvic tumors, multiple pregnancy, macerated fetus, high head with premature or early rupture of the membranes and hydramnios are the known etiological factors.

DIAGNOSIS: The diagnosis is not difficult when the cervical os is sufficiently dilated to feel the limb by the side of the presenting part, especially after rupture of the membranes. Premature or early rupture of the membranes occurs in about one-third of the cases. Cord prolapse is to be excluded because of its frequent association—10–15%.

MANAGEMENT: Factors to be considered are—(1) stage of labor, (2) maturity of the fetus, (3) singleton or twins, (4) pelvic adequacy and (5) associated cord prolapse. **The fetal risks** in compound presentation are birth trauma and cord prolapse.

Indication of cesarean section—Mature singleton live fetus associated with contracted pelvis or cord prolapse should be safely delivered by cesarean section.

Expectant treatment—In otherwise uncomplicated cases (vertex/hand), an attitude of wait and watch policy is preferable. The labor process needs to be monitored very carefully (preferably by electronic

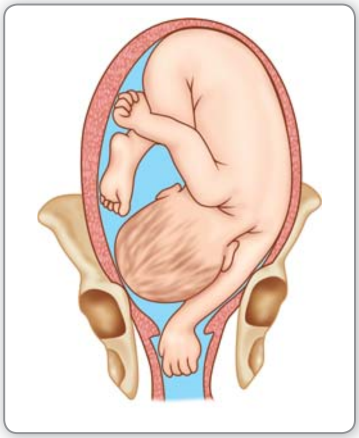


Fig. 26.35: Compound presentation—head with hand

fetal monitoring). **Elevation of the prolapsed limb with descent of the presenting part usually takes place spontaneously**. Slight elevation of the prolapsed limb during uterine contraction is a favorable sign. **Temptation to replace the limbs early is not only unnecessary but carries increased maternal and fetal risks**.

CORD PROLAPSE

There are three clinical types of abnormal descent of the umbilical cord by the side of the presenting part. All these are placed under the heading cord prolapse.

- **Occult prolapse**—The cord is placed by the side of the presenting part and is not felt by the fingers on internal examination. It could be seen on ultrasonography or during cesarean section.
- **Cord presentation—**The cord is slipped down below the presenting part and is felt lying in the intact bag of membranes.
- **Cord prolapse**—The cord is lying inside the vagina or outside the vulva following rupture of the membranes (Fig. 26.36).

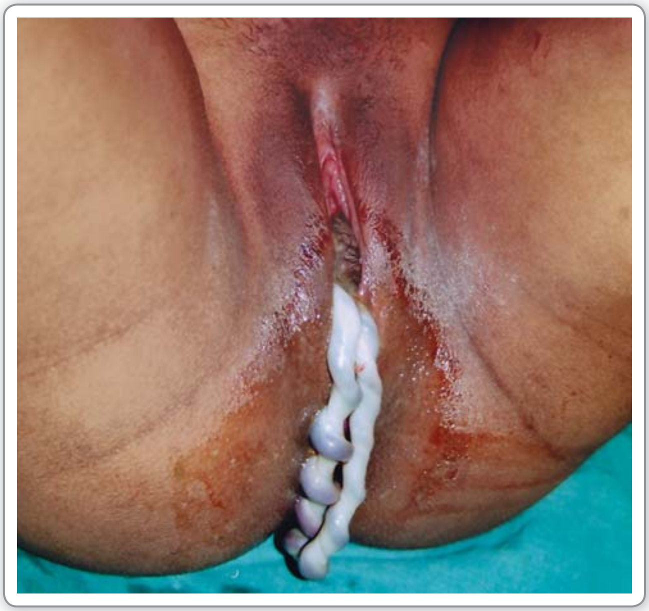


Fig. 26.36: Woman in labor wiht cord prolapse

INCIDENCE: The incidence of cord prolapse is about 1 in 300 deliveries. It is mostly confined to parous women. **Incidence is reduced with the increased use of elective CS in noncephalic presentations**.

ETIOLOGY: Anything which interferes with perfect adaptation of the presenting part to the lower uterine segment, disturbing the ball valve action may favor cord prolapse. Too often, more than one factor operates. **The following are the associated factors**: (1) Malpresentations—the most common being transverse (5–10%) and breech (3%) especially with flexed legs or footling and compound (10%) presentation, (2) Contracted pelvis, (3) Prematurity, (4) Twins, (5) Hydramnios, (6) Placental factor—minor degree placenta previa with marginal insertion of the cord or long cord, (7) Iatrogenic—low rupture of the membranes, manual rotation of the head, ECV, IPV (see p. 604, 605), (8) Stabilizing induction (see p. 397).

DIAGNOSIS: Occult prolapse—is difficult to diagnose. The possibility should be suspected if there is persistence of variable deceleration of fetal heart rate pattern **detected on continuous electronic fetal monitoring. Cord presentation—**The diagnosis is made by feeling the pulsation of the cord through the intact membranes. **Cord prolapse—**The cord is palpated directly by the fingers and its pulsation can be felt if the fetus is alive. Cord pulsation may cease during uterine contraction which, however, returns after the contraction passes off. **Temptation to pull down the loop for visualization or unnecessary handling is to be avoided to prevent vasospasm**. Fetus may be alive even in the absence of cord pulsation. Hence, prompt USG for cardiac movements or auscultation for FHS to be done before fetal death is declared.

PROGNOSIS: Fetal—The fetus is at risk of anoxia from the moment cord is prolapsed. The blood flow is occluded either due to mechanical compression by the presenting part or due to vasospasm of the umbilical vessels due to exposure to cold or irritation when exposed outside the vulva or as a result of handling. **The hazards to the fetus is more in vertex presentation** especially when the cord is prolapsed through the anterior segment of the pelvis or when the cervix is partially dilated. The prognosis is, however, related with the interval between its detection and delivery of the baby and if the delivery is completed, within 10–30 minutes the fetal mortality can be reduced to 5–10%. **The overall perinatal mortality is about 15–50%.**

Maternal—The maternal risks are incidental due to emergency operative delivery, especially through the vaginal route. Operative delivery involves the risk of anesthesia, blood loss and infection.

ANTICIPATION AND EARLY DETECTION: (1) **Internal examination** should be done whenever the membranes rupture prematurely or during labor in all cases of malpresentation, twins, hydramnios or vertex presentation where the head is not engaged. (2) **Surgical induction** should preferably be conducted in the operation theater keeping everything ready for cesarean section. The uterine contraction may be initiated by oxytocin, **if the head is not engaged prior to low rupture of the membranes. Internal examination both before and after amniotomy should be carried out with cord accident in mind.** (3) **One should exclude** cord presentation or occult prolapse, in unexplained fetal distress during labor.

MANAGEMENT

Cord presentation: The aim is to preserve the membranes and to expedite the delivery.

- **Once the diagnosis is made, no attempt should be made to replace the cord**, as it is not only ineffective but the membranes inevitably rupture leading to prolapse of the cord.
- **If immediate vaginal delivery is not possible or contraindicated, cesarean section is the best method of delivery**. During the time of preparing the patient for operative delivery, she is kept in exaggerated Sims' position to minimize cord compression.
- **A rare occasion** is a multipara with longitudinal lie having good uterine contractions with the cervix three-fourths (7–8 cm) dilated, without any evidence of fetal distress. Watchful expectancy can be adopted till full dilatation of the cervix, when the delivery can be completed by forceps or breech extraction.

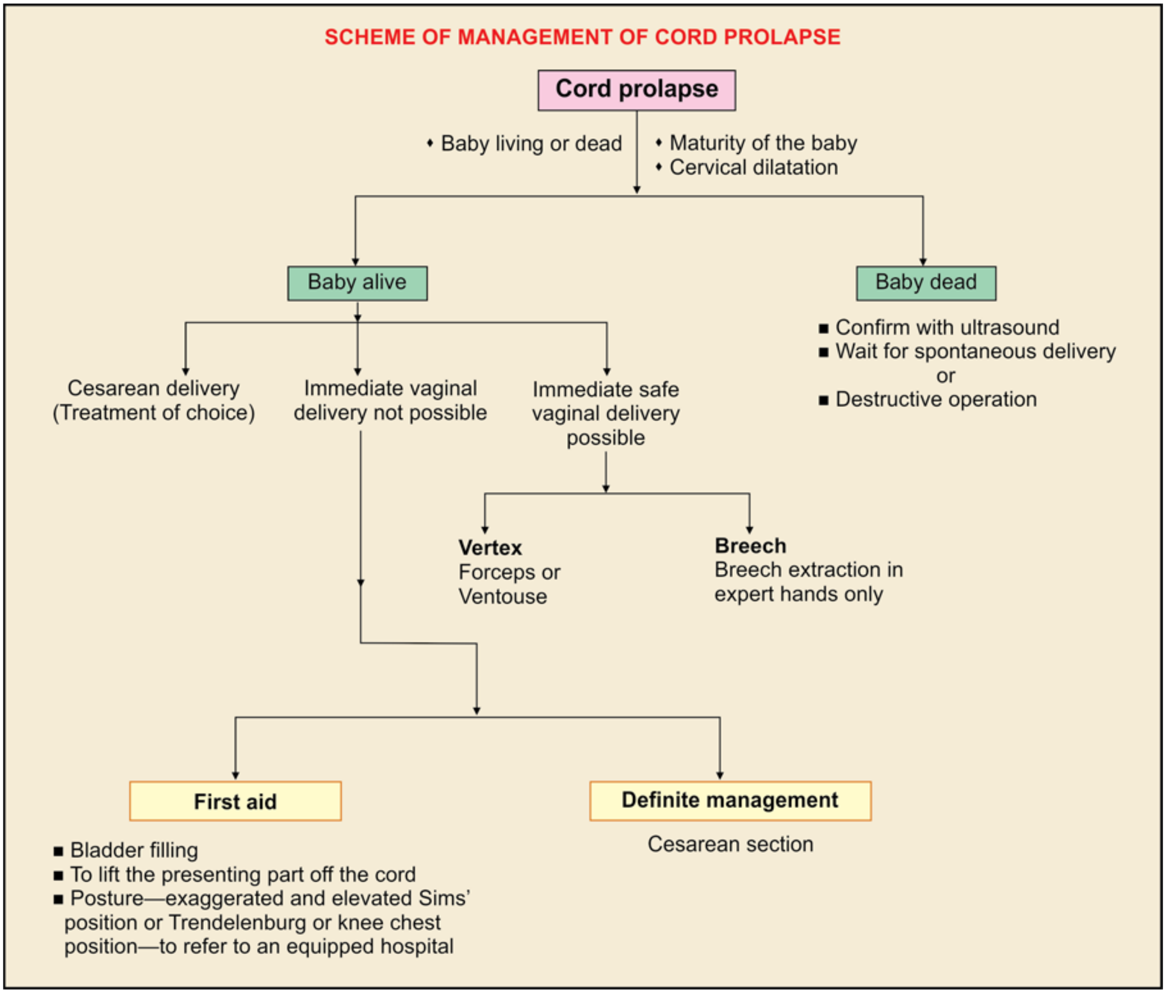
CORD PROLAPSE: Management protocol is to be guided by: (1) Baby living or dead, (2) Maturity of the baby and (3) Degree of dilatation of the cervix.

BABY LIVING: I. Definitive treatment: • **Cesarean section** is the best treatment when the baby is sufficiently mature and is alive. **Just prior to making the abdominal incision, the fetal heart should be auscultated once more to avoid unnecessary section on a dead baby**. The operation should be done quickly up to the delivery of the baby.

II. Immediate safe vaginal delivery is possible: • **If the head is engaged**, delivery is to be completed by forceps. Ventouse may not be ideal in such circumstances as it takes a longer time. • **If breech**, the delivery is to be completed by breech extraction and in transverse lie, it should be completed by internal version followed by breech extraction. The same also applies in cases where the head is not engaged **in second baby of twins**.

III. Immediate safe vaginal delivery is not possible: First aid management: The aim is to minimize pressure on the cord till such time when the patient is prepared for assisted delivery or is transferred to an equipped hospital. If an oxytocin infusion is on, this should be stopped. At this time intravenous fluid and O_2 by face mask is given.

- **Bladder filling** has been done to raise the presenting part off the compressed cord till such time that patient has delivered (either by CS or vaginally). Bladder is filled with 400–750 mL of normal saline with a Foley's catheter, the balloon is inflated and the catheter is clamped. Bladder is emptied before cesarean delivery.



SCHEME OF MANAGEMENT OF CORD PROLAPSE

Cord prolapse
• Baby living or dead
• Maturity of the baby
• Cervical dilatation
Baby alive
Baby dead
■ Confirm with ultrasound
■ Wait for spontaneous delivery
or
■ Destructive operation
Cesarean delivery (Treatment of choice)
Immediate vaginal delivery not possible
Immediate safe vaginal delivery possible
Vertex
Forceps or Ventouse
Breech
Breech extraction in expert hands only
First aid
■ Bladder filling
■ To lift the presenting part off the cord
■ Posture—exaggerated and elevated Sims' position or Trendelenburg or knee chest position—to refer to an equipped hospital
Definite management
Cesarean section

- **To lift the presenting part off the cord**, by the gloved fingers introduced into the vagina. The fingers should be placed inside the vagina till definitive treatment is instituted.
- **Postural treatment**—exaggerated and elevated **Sims' position** with a pillow or wedge under the hip or thigh; **Trendelenburg or knee-chest position** has been traditionally mentioned but may be tiring and irksome to the patient.
- **To replace the cord into the vagina** to minimize vasospasm due to irritation.

BABY DEAD: Labor is allowed to proceed awaiting spontaneous delivery.

QUESTIONS

Related theory questions (Long & Short), Obstetric Case Discussions, Viva table discussions, Postoperative word round discussions, and MCQs are discussed in author's books:

1. **Bedside Clinic and Viva Voce:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

For further reading:

2. **Master pass in Obstetrics and Gynecology:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

Chapter 27

Prolonged Labor, Obstructed Labor, Dystocia Caused by Fetal Anomalies

PROLONGED LABOR

DEFINITION: The labor is said to be prolonged when the combined duration of the first and second stage is more than the arbitrary time limit of 18 hours. The prolongation may be due to protracted cervical dilatation in the first stage and/or inadequate descent of the presenting part during the first or second stage of labor. Labor is considered prolonged when the cervical dilatation rate is less than 1 cm/h and descent of the presenting part is less than 1 cm/h for a period of minimum 4 hours observation (WHO-1994). **Prolonged labor is not synonymous with inefficient uterine contraction.** Inefficient uterine contraction can be a cause of prolonged labor, but labor may also be prolonged due to pelvic or fetal factor.

PROLONGED LATENT PHASE

Latent phase is the preparatory phase of the uterus and the cervix before the actual onset of labor. **Mean duration of latent phase** is about 8 hours in a primi and 4 hours in a multi. Whether prolonged latent phase has got any adverse effect on the mother or on the fetus, it is not clearly known. **A latent phase that exceeds 20 hours in primigravidae or 14 hours in multiparae is abnormal. The causes include:** (1) unripe cervix, (2) malposition and malpresentation, (3) cephalopelvic disproportion, (4) premature rupture of the membranes, (5) induction of labor and (6) early onset of regional anesthetic.

Prolonged latent phase may be worrisome to the patient but does not endanger the mother or fetus.

Management: Expectant management is usually done unless there is any indication (for the fetus or the mother) for expediting the delivery. Rest and analgesic are usually given. When augmentation is decided, medical methods (oxytocin or prostaglandins p. 573) are preferred. Amniotomy is usually avoided. **Prolonged latent phase is not an indication for cesarean delivery.**

CAUSES OF PROLONGED LABOR: Any one or combination of the factors in labor could be responsible.

***First stage*:** Failure to dilate the cervix is due to:

- **Fault in power:** Abnormal uterine contraction such as uterine inertia (common) or incoordinate uterine contraction
- **Fault in the passage:** Contracted pelvis, cervical dystocia, pelvic tumor or even full bladder
- **Fault in the passenger:** Malposition (OP) and malpresentation (face, brow), congenital anomalies of the fetus (hydrocephalus).

Too often deflexed head, minor degrees of pelvic contraction and disordered uterine action have got sinister effects in causing non-dilatation of the cervix.

- **Others:** Injudicious (early) administration of sedatives and analgesics before the active labor begins.

***Second stage*:** Sluggish or non-descent of the presenting part in the second stage is due to:

- **Fault in the power:** (1) Uterine inertia, (2) Inability to bear down, (3) Regional (epidural) analgesia, (4) Constriction ring.
- **Fault in the passage:** (1) Cephalopelvic disproportion, android pelvis, contracted pelvis, (2) Undue resistance of the pelvic floor or perineum due to spasm or old scarring, (3) Soft tissue pelvic tumor.
- **Fault in the passenger:** (1) Malposition (occipitoposterior), (2) Malpresentation, (3) Big baby (4) Congenital malformation of the baby.

DIAGNOSIS: Prolonged labor is not a diagnosis but it is the manifestation of an abnormality, the cause of which should be detected by a thorough abdominal and vaginal examination. During vaginal examination, if a finger is accommodated in between the cervix and the head during uterine contraction pelvic adequacy can be reasonably established. Intranatal imaging (radiography, CT or MRI) is of help in determining the fetal station and position as well as pelvic shape and size.

***First stage*: First stage of labor is considered prolonged** when the duration is more than 12 hours. The rate of cervical dilatation is <1 cm/h in a primi and <1.5 cm/h in a multi. The rate of descent of the presenting part is <1 cm/h in a primi and <2 cm/h in a multi. **In a partograph** (WHO-1994), the labor process is divided into: (i) **Latent phase** that ends when the cervix is 4 cm dilated. (ii) **Active phase**—starts with cervical dilatation of 4 cm or more. Cervix should dilate at least 1 cm/h in this active phase. Cervical dilatation rate (cervicograph) is plotted in relation to **alert line** and **action line (Fig. 27.2). Alert line** starts at the end of latent phase (4 cm cervical dilatation) and ends with full dilatation of the cervix (10 cm) in 6 hours (1 cm/h dilatation rate). The **action line** is drawn 4 hours to the right of the alert line. An interval of 4 hours is allowed to diagnose delay in active phase and then appropriate intervention is done. **Labor is considered abnormal when cervicograph crosses the alert line and falls on zone 2 and intervention is required when it crosses the action line and falls on zone 3 (Fig. 27.2). Partograph** can diagnose any dysfunctional labor early and help to initiate correct management.

Disorders of the active phase: Active phase disorders may be divided into: (A) protraction and (B) arrest disorders. (A) **Protracted active phase:** When the rate of cervical dilatation is <1.2 cm/h in a primipara and <1.5 cm/h in a multipara. **A protracted active phase may be due to:** (i) inadequate uterine contractions, (ii) cephalopelvic disproportion, (iii) malposition (OP) or malpresentation (brow) or (iv) regional (epidural) anesthesia.

(B) **Arrest disorder:** Arrest of dilatation is defined when no cervical dilatation occurs after 2 hours in the active phase of labor. It is commonly due to inefficient uterine contractions. No descent for a period of more than 2 hour is called arrest of descent. It is commonly due to CPD.

Secondary arrest (Fig. 27.1) is defined when the active phase of labor (cervical dilatation) commences normally but stops or slows significantly for 2 hours or more prior to full dilatation of the cervix. It is commonly due to malposition or CPD.

***Second stage*:** Mean duration of second stage is 50 minutes for nullipara and 20 minutes in

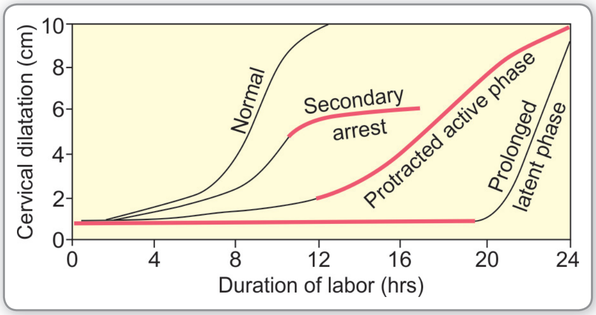


Fig. 27.1: Partographic analysis of labor to detect types of prolonged labor—protracted latent phase, protracted active phase and secondary arrest

multipara. Prolonged second stage is diagnosed if the duration exceeds 2 hours in nullipara and 1 hour in a multipara when no regional anesthesia is used. One hour or more is permitted in both the groups when regional anesthesia is used during labor (ACOG).

Disorders of the second stage: (i) **Protraction of descent** is defined when the descent of the presenting part (station) is at less than 1 cm/h in a nullipara or less than 2 cm/h in a multipara. (ii) **Arrest of descent** is diagnosed when no progress in descent (no change in station) is observed over a period of at least 2 hours. It may be due to one or a combination of several underlying abnormalities like CPD, malposition (OP), malpresentation, inadequate uterine contradictions or asynclitism.

DANGERS: *Fetal:* The fetal risk is increased due to the combined effects of:

(1) **Hypoxia** due to diminished uteroplacental circulation, especially after rupture of the membranes, (2) **Intrauterine infection,** (3) **Intracranial stress or hemorrhage** following prolonged stay in the perineum and/or supermoulding of the head, (4) **Increased operative delivery.** Prolonged second stage of labor is often associated with variable and delayed decelerations (see p. 695). Scalp blood pH estimations show fetal acidosis. All these result in increased perinatal morbidity and mortality.

***Maternal*: There is increased incidence of:** (1) distress (see p. 158) (2) chorioamnionitis, (3) Postpartum hemorrhage, (4) trauma to the genital tract—concealed (undue stretching of the perineal muscles which may be the cause of prolapse at a later period) or revealed such as cervical tear, rupture uterus, (5) increased operative delivery (vaginal instrumental or difficult cesarean), (6) puerperal sepsis, (7) subinvolution. The sum effects of all these lead to increased maternal morbidity and also increased maternal deaths.

TREATMENT

PREVENTION

- **Antenatal or early intranatal detection** of the factors likely to produce prolonged labor (big baby, small women, malpresentation or position).
- **Use of partograph (**Figs 27.2 and 34.4) helps early detection.

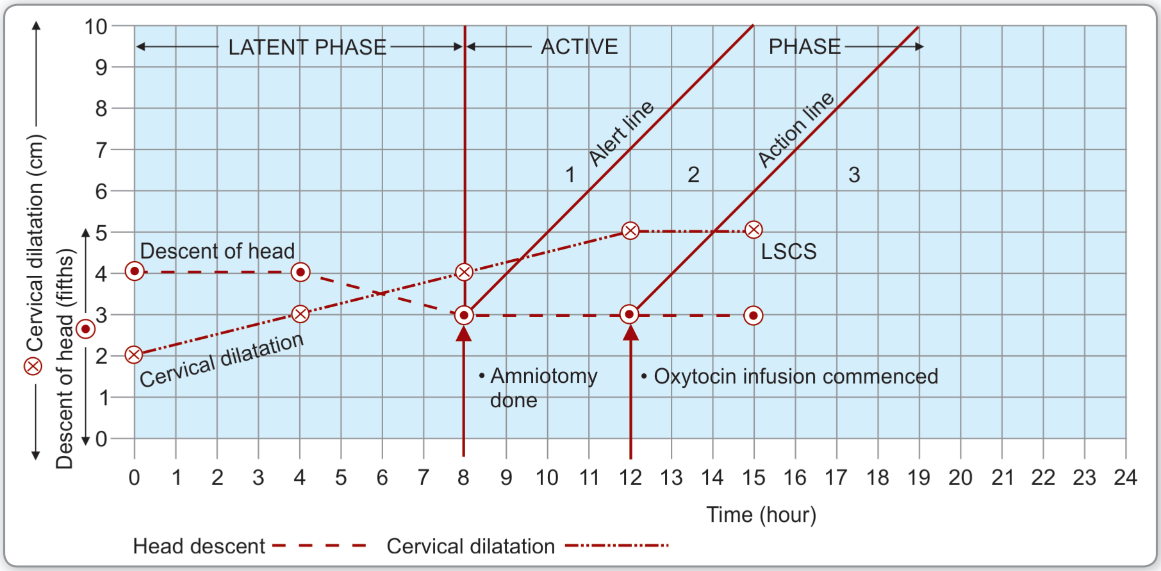


Fig. 27.2: Cervicograph showing slow (protracted) cervical dilatation and descent of the presenting part. Oxytocin infusion was started following amniotomy. Partograph showed arrest in the progress in spite of adequate contractions. Labor was terminated by cesarean section

- **Selective and judicious augmentation** of labor by low rupture of the membranes followed by oxytocin drip (see p. 575).
- **Change of posture in labor** other than supine to increase uterine contractions, **emotional support, avoidance of dehydration** in labor and use of **adequate analgesia** for pain relief.

ACTUAL TREATMENT: Careful evaluation is to be done to find out: (1) cause of prolonged labor (2) effect on the mother, (3) effect on the fetus. In a *nulliparous patient*, **inadequate uterine activity** is the most common cause of **primary dysfunctional labor.** Whereas in a *multiparous patient*, **cephalopelvic disproportion** (due to malposition) is the most common cause.

***Preliminaries*:** In an equipped labor ward, prolonged labor is unlikely to occur in modern obstetric practice. But cases of neglected prolonged labor with evidences of dehydration and ketoacidosis are admitted not infrequently to the referral hospitals in the developing countries. Correction of ketoacidosis should be made urgently by rapid intravenous infusion of Ringer's solution.

***Definitive treatment*: First stage delay:** Vaginal examination is done to verify the fetal presentation, position and station. Clinical pelvimetry is done. **If only uterine activity is suboptimal,** (1) amniotomy and/or oxytocin infusion is adequate, (2) effective pain relief is given by intramuscular pethidine or by regional (epidural) analgesia. **For the management of secondary arrest,** especially in multipara one should be very careful to use oxytocin, (3) cesarean section is done when vaginal delivery is unsafe (malpresentation, malposition, big baby or CPD).

Second stage delay—Short period of expectant management is reasonable provided the FHR (electronic monitoring) is reassuring and vaginal delivery is imminent. Otherwise appropriate assisted delivery, vaginal (forceps, ventouse) or abdominal (cesarean) should be done. Difficult instrumental delivery should be avoided.

KEY POINTS

- **Prolonged labor** is defined when the combined duration of first and second stage of labor is more than 18 hours.
- **Prolonged latent phase** is defined when the duration is more than 20 hours in a primigravida. Normally, it lasts for 8 hours in a primigravida. Latent phase ends when the cervix is 3 cm or more dilated.
- **Prolonged labor** may be due to abnormality of any one or combination of the factors, e.g. **passage** (pelvis, cervix); **passenger** (fetal size, presentation, position, congenital malformation) and the **power** (efficiency of the uterine contractions see p. 463).
- **Malpresentation and malposition** are associated with poor adaptation of the presenting part to the cervix. This causes poor progress of labor (see p. 424).
- **Detection of prolonged labor** in the first stage or in the second stage can be made on careful clinical examination and using a partograph (Figs 27.2 and 35.5).
- **Labor is considered abnormal** when cervicograph crosses the **alert line** and falls on zone 2 and intervention is required when it crosses the action line and falls on zone 3 (Fig. 27.2).
- In a primigravida, **inadequate uterine contraction is the most common cause** of primary dysfunctional labor. In a multigravida, **cephalopelvic disproportion** is the most common cause.
- In the **active phase of labor,** the cervix should dilate at least at the rate of 1 cm per hour.
- **Dangers of prolonged labor** are both to the mother and the fetus (see p. 465).
- **Management** is primarily aimed in prevention, early detection and appropriate intervention (see p. 465).
- **Maternal supine position and dehydration** in labor should be avoided. There should be adequate pain relief (see p. 466).
- **Common causes of prolonged labor are** abnormal uterine contractions, abnormal presentation and position of the fetus and cephalopelvic disproportion.
- **Dangers of prolonged labor are:** (A) **Fetal:** Hypoxia, intrauterine infection (see p. 405). (B) **Maternal:** Chorioamnionitis, increased operative delivery and PPH (see p. 465).

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- **Protraction disorders** are defined when the progress of labor is slower than normal, whereas **arrest disorders** are defined where there is complete cessation of progress.
- **Combined disorder** in active phase is defined as the arrest of dilatation of the cervix or descent of the fetal head occurring in a woman who has previously developed protracted labor. Women with combined disorder often have less favorable chance of vaginal delivery. Patient needs to be evaluated as regard uterine contractions, pelvic adequacy, fetal presentation, position, station and estimated fetal weight.

OBSTRUCTED LABOR

DEFINITION: Obstructed labor is one where in spite of good uterine contractions, the progressive descent of the presenting part is arrested due to mechanical obstruction. This may result either due to factors in the fetus or in the birth canal or both, so that further progress is almost impossible without assistance.

INCIDENCE: In the developing countries, the prevalence is about 1–2% in the referral hospitals.

CAUSES:

- ***Fault in the passage*:** (1) **Bony:** Cephalopelvic disproportion and contracted pelvis are the common causes. Secondary contracted pelvis may be encountered in multiparous women. (2) **Soft tissue obstructions:** This includes cervical dystocia due to prolapse or previous operative scarring, cervical or broad ligament fibroid, impacted ovarian tumor or the nongravid horn of a bicornuate uterus below the presenting part.
- ***Fault in the passenger*:** (1) Transverse lie, (2) Brow presentation, (3) Congenital malformations of the fetus—hydrocephalus (commonest), fetal ascites, double monsters, (4) Big baby, occipito-posterior position, (5) Compound presentation, (6) Locked twins.

MORBID ANATOMICAL CHANGES

***Uterus*:** The morbid anatomical changes in response to obstruction have already been described in relation to the formation of pathological retraction ring or Bandl's ring (see p. 420).

***Bladder*:** The bladder becomes an abdominal organ and due to compression of urethra between the presenting part and symphysis pubis, the patient fails to empty the bladder. The transverse depression at the junction of the superior border of the bladder and the distended lower segment is often confused with the Bandl's ring. The bladder walls get traumatized, which may lead to blood stained urine, a common finding in obstructed labor. **The base of the bladder and urethra, which are nipped in between the presenting part and symphysis pubis, may undergo pressure necrosis.** The devitalized tissue becomes infected and later on may slough off resulting in the development of **genitourinary fistula.**

EFFECTS ON THE MOTHER

Immediate: (1) **Exhaustion** is due to a constant agonizing pain and anxiety. (2) **Dehydration** is due to increased muscular activity without adequate fluid intake. (3) **Metabolic acidosis** is due to accumulation of lactic acid and ketones. (4) **Genital sepsis** is an invariable accompaniment, especially after rupture of the membranes with repeated vaginal examination or attempted manipulation outside. (5) **Injury to the genital tract includes rupture of the uterus** which may be spontaneous in multiparae or may be traumatic following instrumental delivery. (6) **Postpartum hemorrhage and shock** may be due to isolated or combined effects of atonic uterus or genital tract trauma. All these lead to an increased maternal morbidity and mortality. **The deaths are due to rupture of the uterus, shock and sepsis with metabolic changes.**

***Remote*:** Even if the patient survives, **the following legacies may be left behind:** (1) genitourinary fistula or rectovaginal fistula, (2) variable degree of vaginal atresia, (3) secondary amenorrhea following hysterectomy due to rupture or due to Sheehan's syndrome.

EFFECTS ON THE FETUS

(1) **Asphyxia** results from tonic uterine contraction that interferes with the uteroplacental circulation or due to cord prolapse, especially in shoulder presentation. (2) **Acidosis** due to fetal hypoxia and maternal acidosis. (3) **Intracranial hemorrhage** is due to supermoulding of the head leading to tentorial tear or due to traumatic delivery. (4) **Infection.** All these lead to **increased perinatal loss.**

CLINICAL FEATURES: The clinical features are like those mentioned in tonic uterine contraction and retraction (see p. 420).

PREVENTION

- **Antenatal** detection of the factors (see p. 465) likely to produce prolonged labor (big baby, small women, malpresentation and position).
- **Intranatal:** Continuous vigilance, **use of partograph** and timely intervention of a prolonged labor due to mechanical factors can prevent obstructed labor. Failure in progress of labor in spite of good uterine contractions for a reasonable period (2–4 hours) is an impending sign of obstructed labor.

ACTUAL TREATMENT: The underlying principles are: (1) **to relieve** the obstruction at the earliest by a safe delivery procedure, (2) **to combat** dehydration and ketoacidosis, (3) **to control sepsis.**

***Preliminaries*:** (1) **Fluid electrolyte balance and correction of dehydration and ketoacidosis** are done by rapid infusion of Ringer's solution; at least 1 liter is to be given in running drip. At least 3 liters of fluid is required to correct clinical dehydration. (2) **A vaginal swab** is taken and sent for culture and sensitivity test. (3) **Blood sample is sent** for group and cross matching and a bottle of blood should be at hand prior to any operative intervention. (4) **Antibiotic: ceftriaxone 1 g** IV is administered. (5) IV infusion, metronidazole is given for anaerobic infection.

***Obstetric management*: Before proceeding for definitive operative treatment, rupture of the uterus must be excluded.** A balanced decision should be taken about the best method of relieving the obstruction with least hazards to the mother. Frantic attempt to deliver a moribund baby by a method ignoring the risk involved to the mother is indeed bad obstetrics. **There is no place of "wait and watch", neither is any scope of using oxytocin to stimulate uterine contraction**.

***Vaginal delivery*:** The baby is invariably dead in most of the neglected cases and destructive operation is the best choice to relieve the obstruction. If, however, the head is low down and vaginal delivery is not risky, forceps extraction may be done in a living baby. **There is no place of internal version in obstructed labor.** After completion of the delivery and expulsion of the placenta, **exploration of the uterus and the lower genital tract should be done to exclude uterine rupture or tear**.

***Cesarean section*:** If the case is detected early with good fetal condition, **cesarean section gives the best result. But in late and neglected cases, even if the fetal heart sound is audible, desperate attempt to do a cesarean section to save the moribund baby more often leads to disastrous consequences**. Not infrequently, the baby is either delivered stillborn or dies due to neonatal sepsis. The postoperative period of the mother also becomes stormy and at times, ends fatally.

Symphysiotomy: The place of symphysiotomy has to be duly considered in the developing countries as an alternative to risky cesarean section. This can be done in a case of established obstruction due to outlet contraction with vertex presentation having good FHS. The details are mentioned on page 679.

DYSTOCIA CAUSED BY FETAL ANOMALIES

MACROSOMIA *(generalized fetal enlargement)*: Abnormally large size baby weighing more than 4 kg is considered macrosomic. **The causes are:** hereditary, race, size of the parents—particularly the mother (obesity), poorly controlled maternal diabetes and gestational diabetes, postmaturity, multiparity and male fetus. **Diagnosis** is suspected because of: (1) disproportionate increase in uterine size, (2) clinically, the fetus is felt big, (3) ultrasonographic measurements of fetal BPD, HC, FL and AC are done to predict the estimated fetal weight. **Dangers** involve both the fetus and the mother. **Fetal hazards are:** surprise dystocia due to cephalopelvic disproportion, shoulder dystocia, brachial plexus injury, asphyxia, birth trauma and meconium aspiration. Overall perinatal mortality and morbidity are high. **Maternal hazards** include: injury to the maternal soft tissues (vagina, perineum), PPH and puerperal sepsis. Maternal morbidity is high.

Management: (i) Prophylactic induction of labor (early) to reduce the risk of shoulder dystocia or (ii) Elective cesarean delivery, especially in diabetic women with big baby to reduce perinatal hazards (shoulder dystocia).

SHOULDER DYSTOCIA

***Definition*:** The term shoulder dystocia is defined to describe a wide range of additional obstetric maneuvers to deliver the fetus after the head has been born and gentle traction has failed to deliver the shoulder. **Shoulder dystocia** occurs when either the anterior or the posterior (rare) fetal shoulder impacts on the maternal symphysis or on the sacral promontory respectively. Overall incidence varies between 0.2% and 1%.

***Risk factors*:** (1) Previous shoulder dystocia, (2) Macrosomia (>4.5 kg), (3) Diabetes, (4) Obesity (BMI > 30 kg/m^2), (5) Induced labor, (6) Prolonged first stage or second stage of labor, (7) Secondary arrest of labor, (8) Postmaturity, (9) Multiparity, (10) Anencephaly, (11) Mid-pelvic instrumental delivery (more following ventouse than forceps), (12) Fetal ascites. **Complications:** (A) **Fetal:** asphyxia, brachial plexus injury (plexopathy) due to stretch, Erb, Klumpke palsy (see p. 537), humerus fracture, clavicle or sternomastoid hematoma during delivery. Perinatal morbidity and mortality are high. (B) **Maternal:** PPH (11%), cervical laceration, vaginal tear, perineal tear (3rd and 4th degree), rupture of uterus, bladder, sacroiliac joint dislocation and morbidity. **Prevention** of shoulder dystocia is not possible accurately even with antenatal ultrasonographic assessment. **Prediction:** Shoulder dystocia neither could be predicted accurately nor could be prevented entirely. Prolonged first or second stage of labor, secondary arrest of labor (see p. 404) and difficult mid-pelvic instrumental delivery are the important intrapartum observations to predict. Maneuvers to prevent shoulder dystocia may be used prophylactically in cases where it is anticipated.

***Diagnosis*:** (1) Definite recoil of the head back against the perineum (turtle neck sign), (2) Inadequate spontaneous restitution, (3) Fetal face becomes plethoric, (4) Failure of shoulder to descend.

***Management principles*:** Extra help is to be called (a) To clear infant's mouth and nose (b) Not to give traction over baby's head (c) Never to apply fundal pressure as it causes further impaction of the shoulder (Fig. 27.3) (d) To perform wide mediolateral episiotomy as it provides space posteriorly (e) To involve the anesthetist (as analgesia is ideal) and the pediatrician (for infant's resuscitation).

Considering the need of emergency management, shoulder dystocia drill should be practiced by the birth attendants.

***Management*:** The following maneuvers are commonly employed. There is no evidence that any method is superior to another in releasing the impacted shoulder or reducing the chance injury (ACOG-2002).

- Head and neck should be grasped and taken posteriorly while suprapubic pressure is applied by an assistant slightly toward the side of fetal chest. This will reduce the bisacromial diameter and rotate the anterior shoulder toward the oblique diameter. **This maneuver is simple as well as effective. It needs only one assistant.**

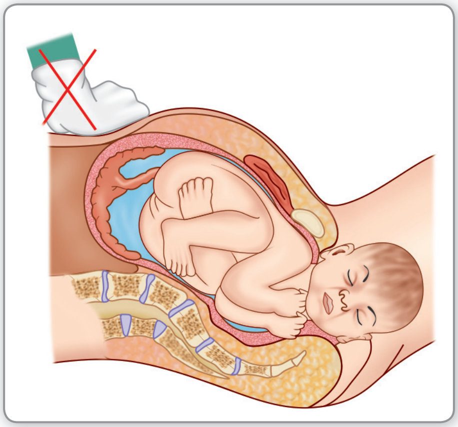


Fig. 27.3: Fundal pressure should not be used as it causes further shoulder impaction

- **McRoberts maneuver:** Abduct the maternal thighs and sharply hyperflex them onto her abdomen. There is rotation of symphysis pubis upward and decrease in angle of pelvic inclination. This straightens the lumbosacral angle, rotates the maternal pelvis upward and increases the anterior-posterior diameter of the pelvis. This maneuver is effective and is successful in about 90% of cases. Suprapubic pressure may be used together. This procedure when possible may be done first (RCOG-2012).

- **Wood's maneuver:** General anesthesia is administered. The posterior shoulder is rotated to anterior position (180°) by a corkscrew movement. This is done by inserting two fingers in the posterior vagina. Simultaneous suprapubic pressure is applied. This pushes the bisacromial diameter from the anteroposterior diameter to an oblique diameter. This helps easy entry of the bisacromial diameter into the pelvic inlet.
- **Extraction of the posterior arm:** The operator's hand is introduced into the vagina along the fetal posterior humerus in the sacral hollow. The arm is then swept across the chest and thereafter delivered by gentle traction. This procedure may cause either fracture clavicle or humerus or both.
- **"All Fours" Position:** Changing the mother on to all fours may increase the pelvic dimensions and allow the fetal position to shift. Downward traction on the posterior shoulder helps to free the impacted shoulder. This may be done for a mobile and slim woman in a community setting.

Other techniques may be used when all the above maneuvers have failed:

Deliberate fracture of the clavicle by finger pressure (fracture heals rapidly) or **cleidotomy:** one or both clavicles may be cut with scissors to reduce the shoulder girth. This is applicable to a living anencephalic baby as a first choice or in a dead fetus. **Zavanelli maneuver** (pushing the fetus back to the uterus and delivering by cesarean section) or **symphysiotomy** is done rarely.

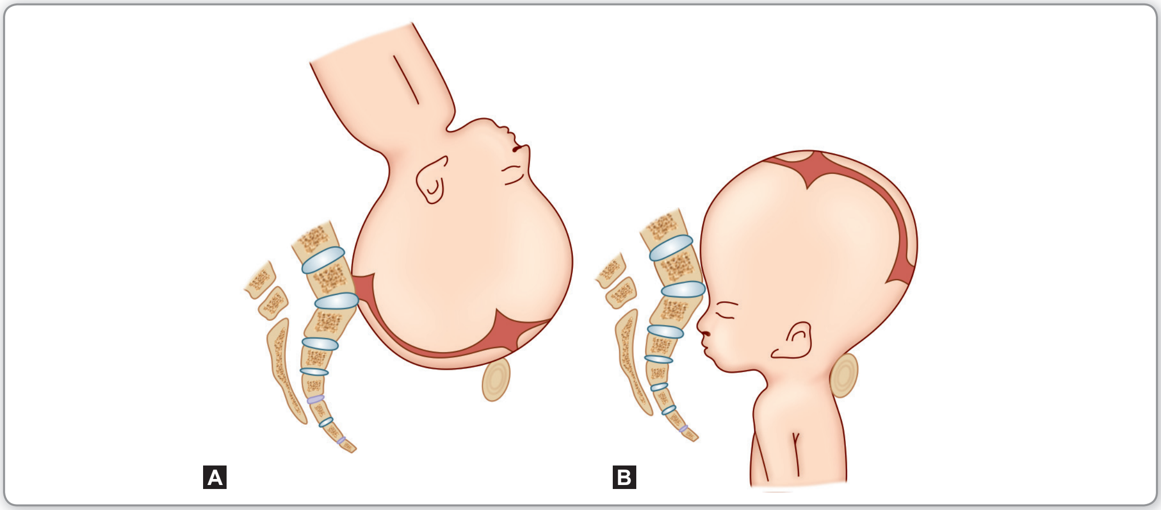
All maternity staff should have shoulder dystocia training. Skill drills using mannequins in simulation improves management outcome when applied in real life.

All the maneuvers employed must be documented correctly to avoid litigation.

HYDROCEPHALUS: Excessive accumulation of cerebrospinal fluid (0.5–1.5 L) in the ventricles with consequent thinning of the brain tissue and enlargement of the cranium occurs in 1 in 2,000 deliveries (Fig. 27.3A). It is associated with other congenital malformations (aneuploidy) in one-third of cases and neural tube defects. Recurrence rate is about 5%. Breech presentation occurs in about 30% cases.

***Diagnosis*: Antenatally, minor degree may escape attention but the severe degree presents with the following features:** (1) The head is felt larger (head circumference > 50 cm), globular and softer than the normal head. (2) The head is high-up and impossible to push down into the pelvis. (3) FHS is situated high-up above the umbilicus. (4) **Sonography:** (a) Cranial shadow is globular rather than normal ovoid, (b) Fontanels and sutures are wide, (c) Vault bones thinner, (d) The lateral and third ventricles are dilated with marked thinning of the cerebral cortex. (5) Often the dilatation is due to stenosis of the aqueduct of Sylvius, agenesis of corpus callosum or fetal TORCH infections. Isolated mild ventriculomegaly 10–12 mm) has a good prognosis (Fig. 27.4A). (6) Internal examination during labor reveals: (a) gaping sutures and fontanels and (b) crackling sensation on pressing the head (Fig. 27.4).

In breech presentation, however, the diagnosis is not made until the aftercoming head is arrested at the brim. Presence of open spina bifida points strongly toward hydrocephalus.



Figs 27.3A and B: Hydrocephalus: (A) Forecoming head; (B) Aftercoming head

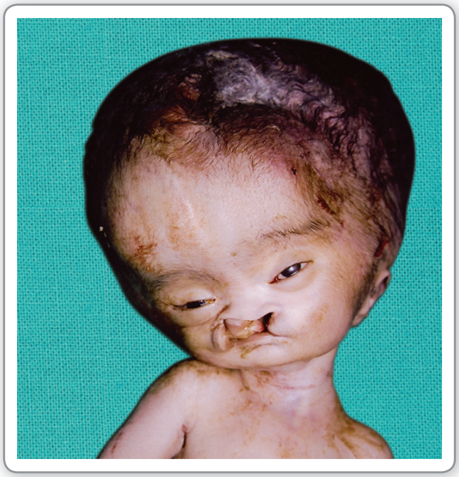


Fig. 27.4: Hydrocephalus with setting sun eyes, cleft lip is also present

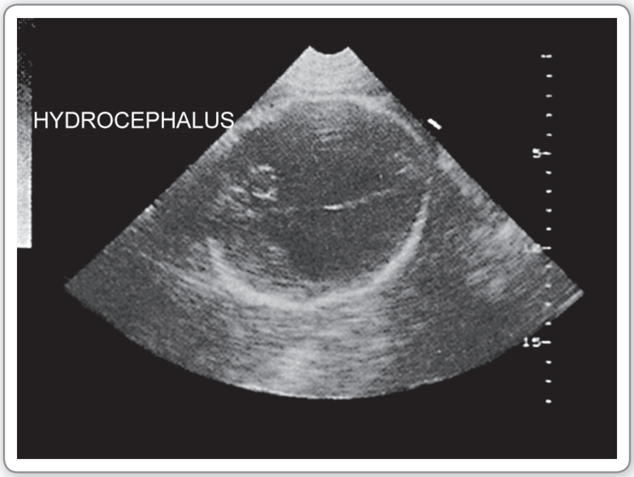


Fig. 27.4A: Sonogram showing hydrocephalus

***Prognosis*:** Fetal outlook is extremely poor except in mild variety. The place of ventriculoamniotic shunts is limited at present. The fetus is either delivered stillborn or dies in neonatal period. Babies, those survive often suffer developmental delay. **Maternal prognosis is not unfavorable in diagnosed cases but in undiagnosed cases and cases left uncared for, obstructed labor with its consequences may occur (see p. 438).** Rupture may occur even before the cervix is fully dilated because of too much stretching of the lower segment by the head.

***Management*:** Principle is to decompress the hydrocephalic head in labor either in vertex or in breech presentation. This is also done during cesarean delivery before incising the uterus. Bladder is evacuated before hand. Once the labor is established and the cervix is 3–4 cm dilated, decompression of the head is done by a sharp pointed scissors or with a wide bore (17 gauge) long needle.

In breech presentation, the arrested head can be decompressed by perforating the suboccipital region using a needle or a sharp pointed scissors under the guidance of two fingers of the left hand protecting the anterior vaginal wall. Exploration of the uterus must be done after delivery of the head. Decompression of the head (cephalocentesis) through the abdominal route using a large bore needle under ultrasound guidance may be done.

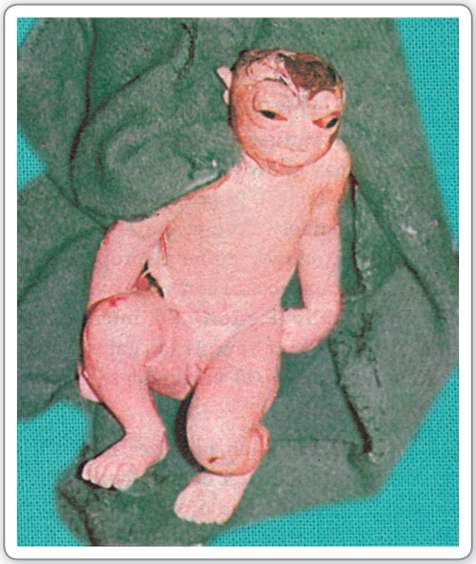


Fig. 27.5: Anencephaly
(*By courtesy:* Dr Mahesh Bharpilania)

NEURAL TUBE DEFECTS (NTD): Anencephaly and spina bifida comprise 95% of NTD and the remaining 5% is encephalocele. It is more common in lower socioeconomic group. Recurrence risk after one affected child is 4%.

ANENCEPHALY: The incidence of anencephaly is about 1 in 1,000 births. The anomaly results from deficient development of the vault of the skull and brain tissue, but the facial portion is normal (Fig. 27.5). The pituitary gland is often absent or hypoplastic. Typically, there is marked diminution of the size of the adrenal glands probably secondary to the absence of the pituitary gland.

About 70% of anencephalic fetuses are females. It is more prevalent in first birth and in young and elderly mothers. Genetic and environmental factors are probably involved (multifactorial).

***Diagnosis*: In the first half of pregnancy,** the diagnosis is made by elevated alpha-fetoprotein in amniotic fluid. Diagnosis is confirmed by sonography (see Chapter 12). The findings around 10 weeks are: (a) absence of cranial vault, (b) angiomatous, brain tissue. **In the latter half of pregnancy,** the diagnosis is difficult especially when associated with hydramnios. Inability to locate the fetal head on abdominal palpation arouses the suspicion. Even on internal examination, the diagnosis of face presentation is made. **Confirmation is done by sonography.**

Complications include: (1) Hydramnios (70%), (2) Malpresentation—face or breech, (3) Premature labor, especially when associated with hydramnios, (4) Tendency of postmaturity, (5) Shoulder dystocia, (6) Obstructed labor if the head and shoulders try to engage together because of short neck.

Management: If confirmed before 20 weeks, termination of pregnancy is to be done. When diagnosed in late pregnancy, termination is to be done. The couple is counseled in either situation. The uterus is most often refractory to oxytocin because of low level of estriol as a result of insufficient production of its precursor cortisol from fetal adrenals. Use of prostaglandin vaginal gel (PGE_2) has been proved to be effective in resistant cases. **During labor,** there is tendency of delay. **Shoulder dystocia should be managed by cleidotomy.**

Prevention: Prepregnancy counseling is essential (see p. 116). Folic acid supplementation beginning 1 month before conception to about 12 weeks of pregnancy has reduced the incidence of NTD significantly (85%). A dose of 4 mg daily is recommended. Risk of recurrence is about 2% in subsequent pregnancy.

Iniencephaly: There is failure of formation of cervical and upper thoracic vertebrae and base of the skull with abnormally formed brain tissue.

ENLARGEMENT OF FETAL ABDOMEN: The enlargement of fetal abdomen sufficient to produce dystocia may be due to ascites, distended bladder or enlargement of kidney by a tumor or an umbilical hernia. Antenatal diagnosis can be made by sonography (Figs 27.6, 27.7) which shows an appearance resembling that of the "Buddha position". **In an unbooked case, the diagnosis is made when there is difficulty in delivery of the trunk following birth of the head.** Confirmation is done by introducing the hand and palpating the hugely distended abdomen. **The decompression of the abdomen is done** by simple puncture with a wide bore needle which is soon followed by spontaneous delivery.

MONSTERS: The varieties of incomplete twinning result in development of groups of monsters (see p. 244). The condition is extremely rare and often causes surprise dystocia (Fig. 17.6).

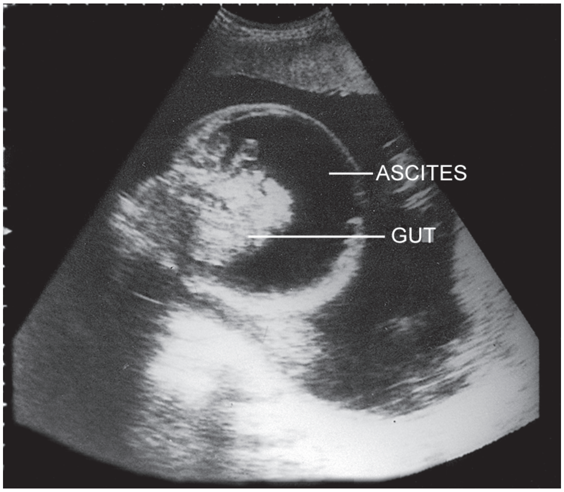


Fig. 27.6: Sonography showing fetal ascites

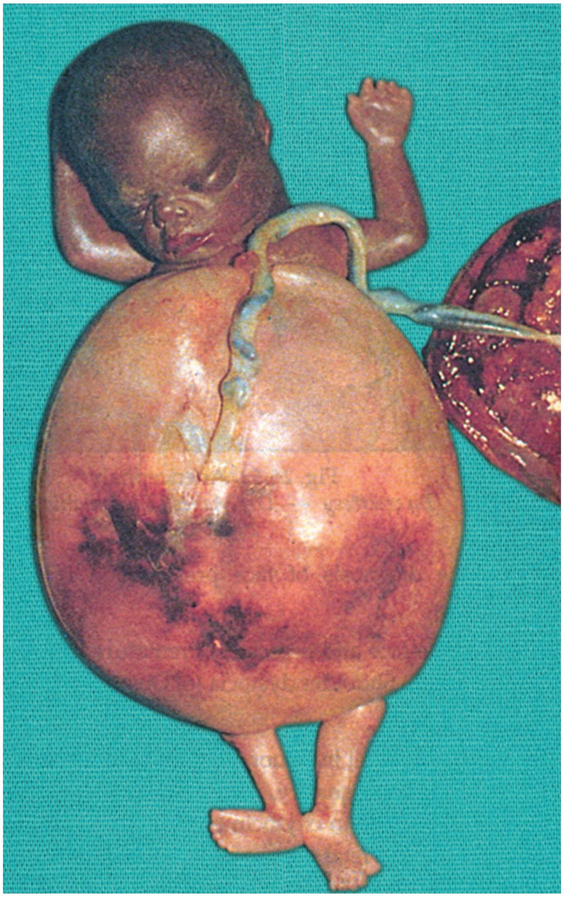


Fig. 27.7: Huge abdominal enlargement due to ascites

CONJOINED TWINS (see Figs 17.6 and 27.8)

Diagnosis: Ultrasonography: A thorough targeted USG is done. Demonstration of: (i) a continuous external skin contour, (ii) body parts of twins (heads) are on the same level, (iii) no change in relative positions of twins on successive scans, (iv) spines are in unusual close proximity and are extended and (v) single placenta. Unless there is bony fusion, **radiographic diagnosis** is unreliable.

Management: Cesarean section offers best chance of fetal survival as in few cases of conjoined twins can be surgically separated. It is commonly done when the diagnosis is made during pregnancy. **Preterm labor** often results in **vaginal delivery** as the fetuses are small and the point of union permits some mobility.

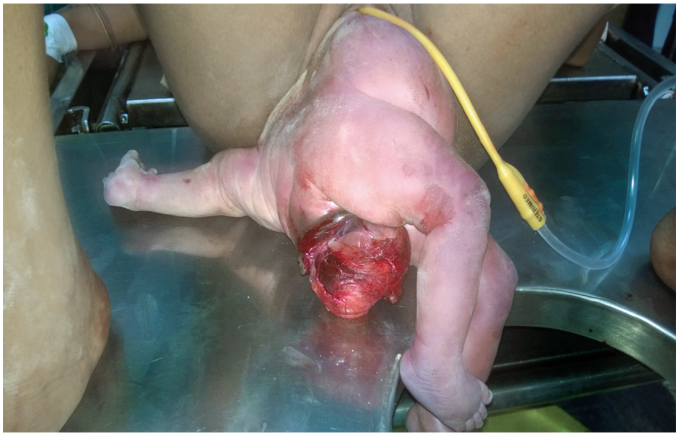


Fig. 27.8: Obstructed labor due to conjoint twins (ischiopagus with sacrococcygeal teratoma). *Courtesy:* Dept. Ob-Gyn; CNMCH, Kolkata

Destructive operation (evisceration and amputation of body parts) is an alternative when diagnosed in labor with dead fetuses.

KEY POINTS

OBSTRUCTED LABOR

- **Obstructed labor** is the arrest of descent of the presenting part despite good uterine contractions. This occurs due to mechanical obstruction.
- **Common causes are:** CPD, soft tissue obstruction (impacted ovarian tumor) or fetal malpresentation or position.
- **Effects on the mother or the fetus** are worse compared to prolonged labor (see p. 467, 468). Perinatal and maternal morbidity and mortality are high.
- **Actual management** includes: to correct maternal dehydration, ketoacidosis, sepsis and to deliver the woman. Cesarean delivery is commonly done when the fetal condition is good.
- **Management** is primarily aimed in prevention (see p. 468). The actual management is to relieve the obstruction and to deliver the fetus safely (see p. 468).
- **Dystocia** may also be due to macrosomia (see p. 468) or due to fetal anomalies [(hydrocephalus, anencephaly or conjoined twins (see p. 471, 473)].
- **Shoulder dystocia** (difficulties during delivery of the shoulders) may be due to several factors (see p. 469). Dangers are mainly to the fetus. Hazards could be minimized if management principles are strictly followed (see p. 469).

QUESTIONS

1. Define prolonged Labor? What are the causes of prolonged labor? Outline the management of case with prolonged first stage of labor? (p. 463-64)

Write Short Notes on:

A. Effects on the mother due to obstructed labor (p. 467)
B. Effects on the fetus due to prolonged labor (p. 468)

Chapter 28

Complications of the Third Stage of Labor

Of all the stages of labor, **third stage is the most crucial one for the mother**. Fatal complications may appear unexpectedly in an otherwise uneventful first or second stage. **The following are the important complications:** (1) Postpartum hemorrhage, (2) Retention of placenta, (3) Shock—hemorrhagic or non-hemorrhagic, (4) Pulmonary embolism either by amniotic fluid or by air, (5) Uterine inversion (rare).

POSTPARTUM HEMORRHAGE (PPH)

DEFINITION: Quantitative definition is arbitrary and is related to the amount of blood loss in excess of 500 mL following birth of the baby (WHO). It may be useful for statistical purposes. As the effect of the blood loss is important rather than the amount of blood lost, the **clinical definition, which is more practical states**, "any amount of bleeding from or into the genital tract following birth of the baby up to the end of the puerperium, which adversely affects the general condition of the patient evidenced by rise in pulse rate and falling blood pressure is called postpartum hemorrhage".

The average blood loss following vaginal delivery, cesarean delivery and cesarean hysterectomy is 500 mL, 1000 mL and 1500 mL respectively.

Depending upon the amount of blood loss, PPH can be ♦ Minor (< 1L), ♦ Major (> 1L) or ♦ Severe (> 2L).

INCIDENCE: The incidence widely varies mainly because of lack of uniformity in the criteria used in definition. The incidence is about 4–6% of all deliveries.

TYPES: ♦ **Primary** ♦ **Secondary**

***Primary*:** Hemorrhage occurs within 24 hours following the birth of the baby. In the majority, hemorrhage occurs within two hours following delivery. **These are of two types:**

- **Third stage hemorrhage**—Bleeding occurs before expulsion of placenta.
- **True postpartum hemorrhage—**Bleeding occurs subsequent to expulsion of placenta (majority).

***Secondary*:** Hemorrhage occurs beyond 24 hours and within puerperium, also called delayed or late puerperal hemorrhage.

PRIMARY POSTPARTUM HEMORRHAGE

CAUSES

Four basic pathologies are expressed as the four Ts' (RCOG): Tone (atonicity), Tissue (retained bits, blood clots), Trauma (genital tract injury) and Thrombin (coagulopathy).

- **Atonic**
- **Traumatic**
- **Retained tissues**
- **Blood coagulopathy (Thrombin)**

♦ **Atonic uterus (80%)**: **Atonicity of the uterus is the commonest cause** of postpartum hemorrhage. With the separation of the placenta, the uterine sinuses, which are torn, cannot be compressed effectively due to imperfect contraction and retraction of the uterine musculature and bleeding continues. The following are the conditions, which often interfere with the retraction of the uterus as a whole and of the placental site in particular.

— **Grand multipara**—Inadequate retraction and frequent adherent placenta contribute to it. Associated anemia may also probably play a role.

— **Overdistension of the uterus** as in multiple pregnancy, hydramnios and big baby (>4 kg). Imperfect retraction and a large placental site are responsible for excessive bleeding.

— **Malnutrition and anemia (<9.0 g/dL)**—Even slight amount of blood loss may develop clinical manifestations of postpartum hemorrhage.

— **Antepartum hemorrhage (Both placenta previa and abruption):** The causes of excessive bleeding are mentioned in Chapter 19 p. 282.

— **Prolonged labor (>12 hours):** Poor retraction, infection (amnionitis), dehydration are important factors (Tone).

— **Anesthesia:** Depth of anesthesia and the anesthetic agents (ether, halothane) may cause atonicity.

— **Initiation or augmentation of delivery by oxytocin:** Postdelivery uterine atonicity is likely unless the oxytocin is continued for at least one hour following delivery.

— **Malformation of the uterus:** Implantation of the placenta in the uterine septum of a septate uterus or in the cornual region of a bicornuate uterus may cause excessive bleeding.

— **Uterine fibroid** causes imperfect retraction mechanically.

— **Mismanaged third stage of labor:** This includes—(a) Too rapid delivery of the baby preventing the uterine wall to adapt to the diminishing contents, (b) Premature attempt to deliver the placenta before it is separated, (c) Kneading and fiddling the uterus, (d) Pulling the cord. All these produce irregular uterine contractions leading to partial separation of placenta and hemorrhage, (e) Manual separation of the placenta increases blood loss during cesarean delivery.

— **Placenta:** Morbidly adherent (accreta, percreta), partially or completely separated and/or retained (constriction ring uterus p. 486) cause PPH.

— **Precipitate labor:** In rapid delivery, separation of the placenta occurs following the birth of the baby. Bleeding continues before the onset of uterine retraction. Bleeding may be due to genital tract trauma also (see p. 420).

— **Other causes of atonic hemorrhage are:** ♦ Obesity (BMI > 35) ♦ Previous PPH ♦ Age (>40 yrs) ♦ Drugs: Use of tocolytic drugs (ritodrine), $MgSO_4$, Nifedipine.

— **Traumatic (20%)**: Trauma to the genital tract usually occurs following **operative delivery**; even after spontaneous delivery. Blood loss from the **episiotomy** wound is often underestimated. Similarly, blood loss in **cesarean section** amounting to 800–1000 mL is most often ignored. **Trauma** involves usually the cervix, vagina, perineum (episiotomy wound and lacerations), paraurethral region and rarely, rupture of the uterus occurs. The bleeding is usually revealed but can rarely be concealed (vulvovaginal or broad ligament hematoma).

- **Retained tissues**: Bits of placenta, blood clots cause PPH due to imperfect uterine retraction.
- **Combination of atonic and traumatic causes**.
- **Thrombin**: Blood coagulation disorders, acquired or congenital, are less common causes of postpartum hemorrhage. The blood coagulopathy may be due to diminished procoagulants (washout phenomenon) or increased fibrinolytic activity. The firmly retracted uterus can usually prevent bleeding. **The conditions where such disorders may occur** are abruptio placentae, jaundice in pregnancy, thrombocytopenic purpura, severe preeclampsia, HELLP syndrome or in IUD (see p. 712). Specific therapy following coagulation screen including recombinant activated factor VII (rF VIIa) may be given.

DIAGNOSIS AND CLINICAL EFFECTS: In the majority, the vaginal bleeding is visible outside, as a slow trickle. Rarely, the bleeding is totally concealed as either vulvovaginal or broad ligament hematoma. **The effect of blood loss depends on**—(a) Predelivery hemoglobin level, (b) degree of pregnancy induced hypervolemia and (c) speed at which blood loss occurs. Alteration of pulse, blood pressure and pulse pressure appears only after class 2 hemorrhage (20–25% loss of blood volume). On occasion, blood loss is so rapid and brisk that death may occur within a few minutes.

State of uterus, as felt per abdomen, gives a reliable clue as regards the cause of bleeding. **In traumatic hemorrhage**, the uterus is found well contracted. **In atonic hemorrhage**, the uterus is found flabby and becomes hard on massaging. However, both the atonic and traumatic cause may coexist. Even following massive blood loss from the injured area, a state of low general condition can make the uterus atonic.

PROGNOSIS: Postpartum hemorrhage is one of the life-threatening emergencies. It is one of the major causes of maternal deaths both in the developing and developed countries (see p. 685). Prevalence of malnutrition and anemia, inadequate antenatal and intranatal care and lack of blood transfusion facilities, substandard care are some of the important contributing factors. **There is also increased morbidity**. **These include** shock, transfusion reaction, puerperal sepsis, failing lactation, pulmonary embolism, thrombosis and thrombophlebitis. **Late sequelae include** Sheehan's syndrome (selective hypopituitarism) or rarely diabetes insipidus.

PREVENTION

Postpartum hemorrhage cannot always be prevented. However, the incidence and especially its magnitude can be reduced substantially by assessing the risk factors and following the guidelines as mentioned below:

However, most cases of PPH have no identifiable risk factors.

- **Antenatal**
  - **Improvement of the health status** of the woman and to keep the hemoglobin level normal (> 10 g/dL) so that the patient can withstand some amount of the blood loss.
  - **High-risk patients** who are likely to develop postpartum hemorrhage (such as twins, hydramnios, grand multipara, APH, history of previous PPH, severe anemia) are to be screened and delivered in a well-equipped hospital.
  - **Blood grouping** should be done for all women so that no time is wasted during emergency.
  - **Placental localization** must be done in all women with previous cesarean delivery (see p. 286) by USG or MRI to detect placenta accreta or percreta (see p. 486).
  - **All women with prior cesarean delivery** must have their placental site determined by ultrasound/MRI to determine morbid adherent placenta.
  - **Women with morbid adherent placenta** (see p. 486) are at high risk of PPH. Such a case should be delivered by a senior obstetrician. Availability of blood and or blood products must be ensured beforehand. Multidisciplinary team approach should be made in such a case.

- **Intranatal**
  - **Active management of the third stage**, **for all women in labor should be a routine as it reduces PPH by 60%.**
  - **Cases with induced or augmented labor by oxytocin**, the infusion should be continued for at least one hour after the delivery.
  - **Women delivered by cesarean section**, oxytocin 5 IU slow IV is to be given to reduce blood loss. **Carbetocin** (long-acting oxytocin) 100 µg is very useful to prevent PPH.
  - **Exploration of the uterovaginal canal** for evidence of trauma following difficult labor or instrumental delivery.
  - **Observation for about two hours** after delivery to make sure that the uterus is hard and well contracted before sending her to ward.
  - **Expert obstetric anesthetist** is needed when the delivery is conducted under general anesthesia. **Local or epidural anesthesia** is preferable to general anesthesia, in forceps, ventouse or breech delivery.
  - **During cesarean section** spontaneous separation and delivery of the placenta reduces blood loss (30%).
  - **Examination of the placenta** and membranes should be a routine to detect at the earliest any missing part.

All said and done, it is the **intelligent anticipation, skilled supervision, prompt detection and effective institution of therapy** that can prevent a normal case from undergoing disastrous consequences.

MANAGEMENT OF THIRD STAGE BLEEDING

The principles in the management are:

- **To empty the uterus** of its contents and to make it contract.
- **To replace the blood**. On occasion, patient may be in shock. In that case patient is managed for shock first (see p. 704).
- **To ensure effective hemostasis** in traumatic bleeding.

STEPS OF MANAGEMENT: ◆ **Placental site bleeding** ◆ **Traumatic bleeding**

Placental site bleeding

- **To palpate the fundus and massage** the uterus to make it hard. The massage is to be done by placing four fingers behind the uterus and thumb in front. However, if bleeding continues even after the uterus becomes hard, suggests, the presence of genital tract injury.
- **To start crystalloid solution** (Normal saline or Ringer's solution) with oxytocin (1 L with 20 units) at 60 drops per minute and to arrange for blood transfusion if necessary.
- **Oxytocin** 10 units IM or methergine 0.2 mg is given intravenously. **Carbetocin**, a longer acting oxytocin derivative is found (100 µg) as effective as oxytocin infusion.
- **To catheterize** the bladder.
- **To give antibiotics** (Ampicillin 2 g and Metronidazole 500 mg IV).

During this procedure, if features of placental separation are evident, expression of the placenta is to be done either by fundal pressure or controlled cord traction method. If the placenta is not separated, manual removal of placenta under general anesthesia is to be done. **However, if the patient is in shock, she is resuscitated first before undertaking manual removal. If the patient is delivered under general anesthesia**, quick manual removal of the placenta solves the problem. **In cases where oxytocin 10 units is given IM** with the delivery of the anterior shoulder, manual removal is done promptly when two attempts of controlled cord traction fail. **Crede's expression of the placenta is abandoned** as it is not only ineffective, but produces shock and rarely inversion.

***Management of traumatic bleeding*:** The uterovaginal canal is to be explored under general anesthesia after the placenta is expelled and hemostatic sutures are placed on the offending sites.

STEPS OF MANUAL REMOVAL OF PLACENTA

Step–I: The operation is done under general anesthesia. **In extreme urgency where anesthetist is not available**, the operation may have to be done under deep sedation with 10 mg diazepam given intravenously. The patient is placed in lithotomy position. With all aseptic measures, the bladder is catheterized.

Step–II: One hand is introduced into the uterus after smearing with the antiseptic solution in cone shaped manner following the cord, which is made taut by the other hand (Fig. 28.1). While introducing the hand, the labia are separated by the fingers of the other hand. The fingers of the uterine hand should locate the margin of the placenta.

Step–III: Counter pressure on the uterine fundus is applied by the other hand placed over the abdomen. The abdominal hand should steady the fundus and guide the movements of the fingers inside the uterine cavity until the placenta is completely separated.

Step–IV: As soon as the placental margin is reached, the fingers are insinuated between the placenta and the uterine wall with the back of the hand in contact with the uterine wall. The placenta is gradually separated with a sideways slicing movement of the fingers, until whole of the placenta is separated (Fig. 28.2).

Step–V: When the placenta is completely separated, it is extracted by traction of the cord by the other hand. The uterine hand is still inside the uterus for exploration of the cavity to be sure that nothing is left behind.

Step–VI: Intravenous methergine 0.2 mg is given and the uterine hand is gradually removed while massaging the uterus by the external hand to make it hard. **After the completion of manual removal, inspection of the cervicovaginal canal is to be made to exclude any injury.**

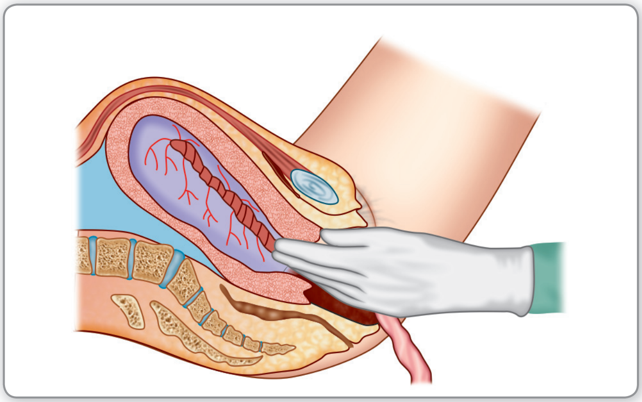
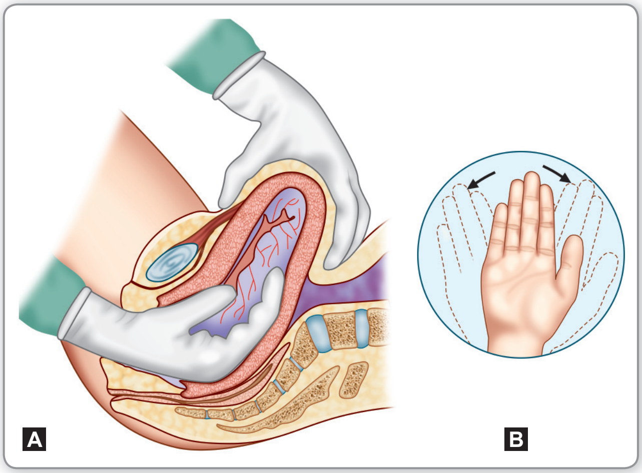


Fig. 28.1: Introduction of the hand into the uterus in a cone shaped manner following the taut umbilical cord

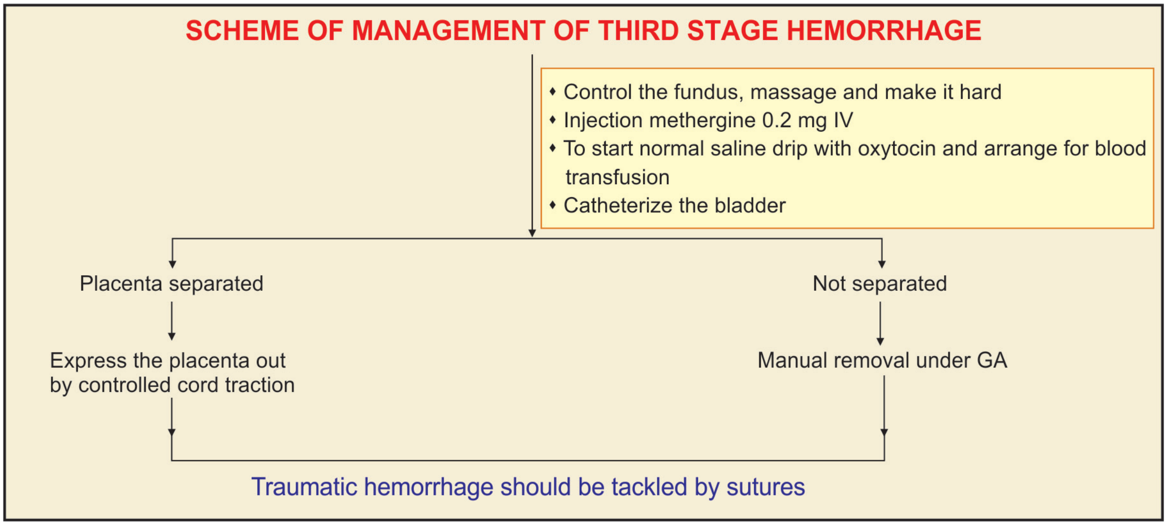


Figs 28.2A and B: The placenta is separated by (A) keeping the back of the hand in contact with the uterine wall (B) with slicing movements of the hand

Step–VII: The placenta and membranes are inspected for completeness and be sure that the uterus remains hard and contracted.

Difficulties: (1) **Hour-glass contraction** leading to difficulty in introducing the hand, (2) **Morbid adherent placenta** which may cause difficulty in getting to the plane of cleavage of placental separation. In such a case placenta is removed gently in fragments using an ovum forceps.

Complications: (1) Hemorrhage due to incomplete removal, (2) Shock, (3) Injury to the uterus, (4) Infection, (5) Inversion (rare), (6) Subinvolution, (7) Thrombophlebitis, (8) Embolism. In such cases placenta is removed in fragments using an ovum forceps or a flushing curette.


SCHEME OF MANAGEMENT OF THIRD STAGE HEMORRHAGE
• Control the fundus, massage and make it hard
• Injection methergine 0.2 mg IV
• To start normal saline drip with oxytocin and arrange for blood transfusion
• Catheterize the bladder
Placenta separated
Not separated
Express the placenta out by controlled cord traction
Manual removal under GA
Traumatic hemorrhage should be tackled by sutures

MANAGEMENT OF TRUE POSTPARTUM HEMORRHAGE

PRINCIPLES: Simultaneous approach

♦ **Communication** ♦ **Resuscitation** ♦ **Monitoring** ♦ **Arrest of bleeding**

It is essential in all cases of major PPH (blood loss > 1000 mL or clinical shock). (RCOG - 2009).

MANAGEMENT

Immediate measures are to be taken by the attending house officer (doctor/midwife).

- **Call for extra help—**involve the obstetric registrar (senior staff) on call.
- Put in two large bore (14-gauge) intravenous cannulas.
- Keep patient flat and warm.
- Send blood for full blood count, group, cross matching, diagnostic tests (RFT, LFT), coagulation screen including fibrinogen and ask for 2 units (at least) of blood.
- Infuse rapidly 2 liters of normal saline (crystalloids) or plasma substitutes like Haemaccel (colloids), an urea-linked gelatin, to reexpand the vascular bed. It does not interfere with cross matching.
- Give oxygen by mask 10–15 L/min.
- Start 20 units of oxytocin in 1 L of normal saline IV at the rate of 60 drops per minute. Transfuse blood as soon as possible.
- One midwife/rotating houseman should be assigned to monitor the following—(i) Pulse (ii) Blood pressure (iii) Temperature (iv) Respiratory rate and oximeter (v) Type and amount of fluids (blood, blood products) the patient has received (vi) Urine output (continuous catheterization) (vii) Drugs-type, dose and time (viii) Central venous pressure (when sited).

ACTUAL MANAGEMENT

♦ **Atonic** ♦ **Traumatic** ♦ **Retained tissues** ♦ **Coagulopathy (p. 711)**

The first step is to control the fundus and to note the feel of the uterus. If the uterus is flabby, the bleeding is likely to be from the atonic uterus. If the uterus is firm and contracted, the bleeding is likely of traumatic origin.

Atonic uterus: *Step*—I: (a) **Massage the uterus** to make it hard and express the blood clot, (b) **Methergine** 0.2 mg is given intravenously, (c) **Injection oxytocin** drip is started (10 units in 500 mL of normal saline) at the rate of 40–60 drops per minute, (d) Foley catheter to keep bladder empty and to monitor urine output, (e) **To examine the expelled placenta and membranes**, for evidence of missing cotyledon or piece of membranes. If the uterus fails to contract, proceed to the next step.

Step—II: **The uterus is to be explored under general anesthesia**. Simultaneous inspection of the cervix, vagina especially the paraurethral region is to be done to exclude coexistent bleeding sites from the injured area. **In refractory** cases:

- Injection 15 methyl $PGF_{2\alpha}$ 250 µg IM in the deltoid muscle every 15 minutes (up to maximum of 2 mg).

OR

- Misoprostol (PGE_1) 1000 µg per rectum is effective.
- When uterine atony is due to tocolytic drugs, calcium gluconate (1 g IV slowly) should be given to neutralize the calcium blocking effect of these drugs (see p. 583).

Step—III: **Uterine massage and bimanual compression**.

Procedures: (a) The whole hand is introduced into the vagina in cone shaped fashion after separating the labia with the fingers of the other hand, (b) The vaginal hand is clenched into a fist with the back of the hand directed posteriorly and the knuckles in the anterior fornix, (c) The other hand is placed over the abdomen behind the uterus to make it anteverted, (d) The uterus is firmly squeezed between the two hands (Fig. 28.3). It may be necessary to continue the compression for a prolonged period until the tone of the uterus is regained. **This is evidenced by absence of bleeding if the compression is released.**

During the period, the resuscitative measures are to be continued. If, in spite of therapy, the uterus remains refractory and the bleeding continues, the possibility of blood coagulation disorders should be kept in mind and massive fresh whole blood transfusion should be given until specific measures can be employed. However, with oxytocics and blood transfusion, almost all cases respond well. Uterine contraction and retraction regain and bleeding stops. **But in rare cases, when the uterus fails to contract**, the following may be tried desperately as an alternative to hysterectomy.

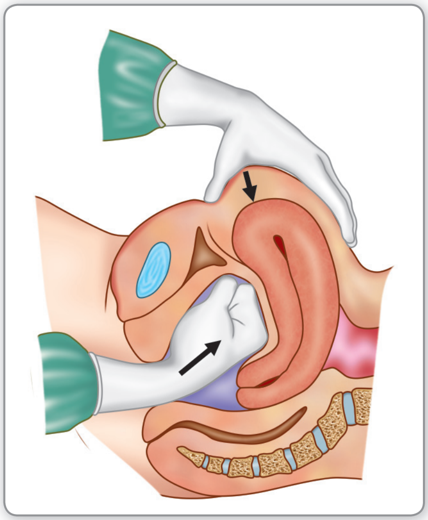


Fig. 28.3: Bimanual compression of the uterus

Step—*IV*: **Uterine tamponade**—

(a) **Tight intrauterine packing** is done uniformly under general anesthesia.

Procedure: A 5 meters long strip of gauze, 8 cm wide folded twice is required. The gauze should be soaked in antiseptic cream before introduction. The gauze is placed high up and packed into the fundal area first while the uterus is steadied by the external hand. Gradually, the rest of the cavity is packed so that no empty space is left behind. **A separate pack is used to fill the vagina.** An abdominal binder is placed. **Intrauterine plugging acts not only by stimulating uterine contraction but exerts direct hemostatic pressure (tamponade effect) to the open uterine sinuses.** Antibiotic should be given and **the plug should be removed after 24 hours.**

Intrauterine packing is useful in a case of uncontrolled postpartum hemorrhage where other methods have failed and the patient is being prepared for transport to a tertiary care center.

(b) Balloon tamponade (Fig. 28.4): Tamponade using various types of hydrostatic balloon catheter has mostly replaced uterine packing. Mechanism of action is similar to uterine packing. Foley catheter, Bakri balloon, Condom catheter or Sengstaken-Blakemore tube is inserted into the uterine cavity and the balloon is inflated with normal saline (200–500 mL). It is kept for 4–6 hours. It is successful in atonic

SCHEME OF MANAGEMENT OF TRUE PPH

Skill drill for PPH using mannequins for all labor attendants

Immediate measures

- Call for extra help (communication)
- Commence IV line with two wide bore cannulas
- Send blood for cross matching tests, coagulation screening including fibrinogen level and ask blood for 2 units (at least)
- Rapidly infuse normal saline/haemaccel 2 liters till blood is available
- To catheterize the bladder
- To monitor pulse, BP, temperature, output, oximeter every 15–30 minutes

To Feel the Uterus by Abdominal Palpation

Uterus atonic

- Massage the uterus to make it hard
- To add oxytocin 10–20 units in 500 mL of Normal saline, at the rate of 40 drops per minute
- Injection methergine 0.2 mg IV (slowly)
- To examine the expelled placenta

Uterus remains atonic

- Exploration of the uterus
- Blood transfusion
- To continue oxytocin drip

Uterus atonic

- 15 methyl $PGF_{2\alpha}$ 250 μg IM/intramyometrial

OR

- Misoprostol 1000 μg per rectum

OR

- Carbetocin 100 μg IM / IV

Uterus atonic

Uterine tamponade

(any method)

- Balloon tamponade (Fig. 28.4)
- Bimanual compression (Fig. 28.3)
- Tight intrauterine packing under anesthesia

Uterus atonic

Surgical methods

(any method)

Stepwise uterine devascularization procedure

- B-Lynch compression and multiple square sutures (Fig. 28.6)
- Ligation of uterine artery and utero-ovarian anastomotic vessels unilateral or bilateral (Fig. 28.5)
- Ligation of anterior division of internal iliac artery (unilateral or bilateral)
- Angiographic arterial embolization with gelatin sponge

→ Bleeding controlled

- Continuous observation in high dependency unit/ICU
- Documentation of procedures adopted in respect to time

Hysterectomy (rarely)

Uterus hard and contracted

Traumatic

Exploration

Hemostatic sutures on the tear sites

Commonly Used Oxytocics in the Management of PPH

Drug	Dose	Route	Dose frequency	Side effects	Contra-indications
Oxytocin	10–40 units in 1 L of crystalloid solution	First line: IV; Second line: IM (10 units)	Continuous IV	Nausea Water intoxication	Not as IV bolus, otherwise none
Methergine	0.2 mg	First line IM/IV; Second line PO	Every 2–4 hours	Nausea Vomiting Hypertension	Hypertension Pre-eclampsia
15 methyl $PGF_{2\alpha}$	0.25 mg	First line IM; Second line intra-uterine	Every 15–90 min. (8 doses maximum)	Nausea Vomiting Diarrhea Chills	Bronchial asthma Active cardiac, renal or hapatic disease
Misoprostol (PGE_1)	600–1000 mcg	First line PR second line PO	Single dose	Fever, Tachycardia	None

Guidelines for Management of PPH : RCOG, FIGO and ACOG

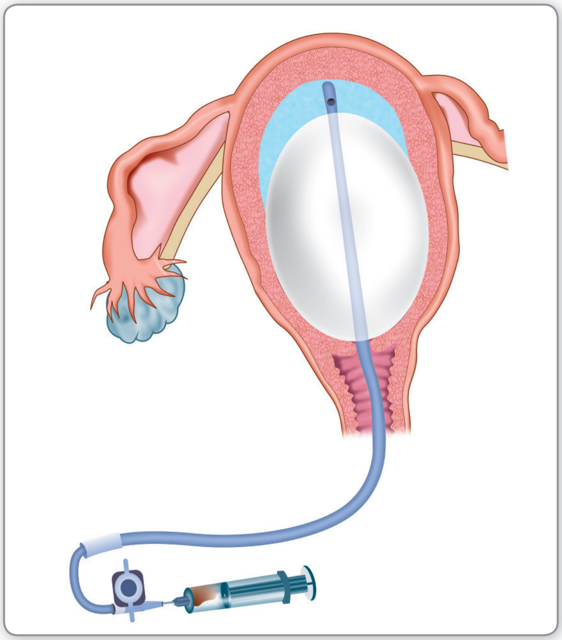


Fig. 28.4: Uterine tamponade using Bakri balloon

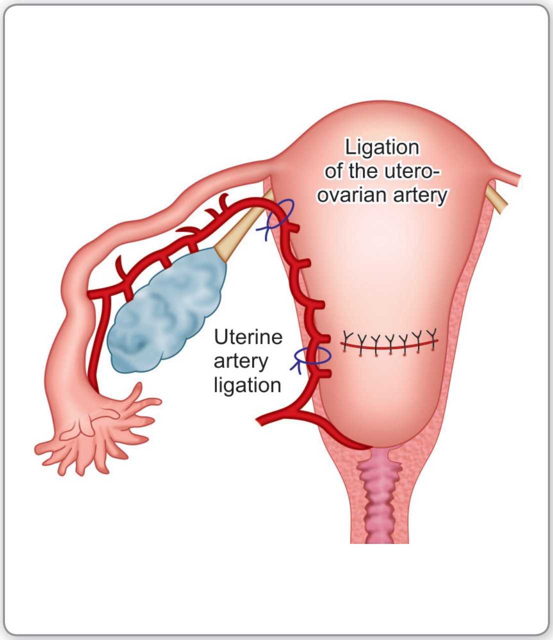


Fig. 28.5: Ligation of the utero-ovarian artery

PPH. This can avoid hysterectomy in 78% cases. **It is considered the first line surgical intervention for most women with atonic PPH.**

Other Measures: ♦ **A non-pneumatic antishock garment** may be used when patient is being transferred to a referral center. ♦ **Compression of the abdominal aorta** may be a temporary but effective measure. This allows time for resuscitation and volume replacement before any surgical intervention is done.

Step V: **Surgical methods to control PPH** are many. An outline of stepwise uterine devascularization procedures are given below:

(a) **B-Lynch compression suture (1997) and multiple square sutures:** Both these surgical methods work by tamponade (like bimanual compression) of the uterus (Fig. 28.6). Success rate is about 80% and it can avoid hysterectomy.

(b) **Ligation of uterine arteries**—the ascending branch of the uterine artery is ligated at the lateral border between upper and lower uterine segment. The suture (No. 1 chromic) is passed into the myometrium 2 cm medial to the artery (Fig. 28.5). In atonic hemorrhage, bilateral ligation is effective in about 75% of cases.

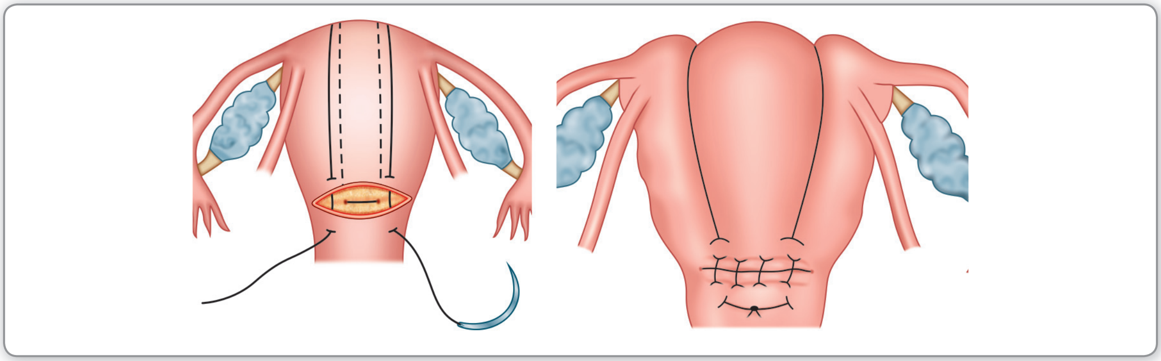


Fig. 28.6: B-Lynch brace suture for control of atonic PPH

(c) **Ligation of the ovarian and uterine artery anastomosis,** if bleeding continues, is done just below the ovarian ligament (Fig. 28.5). Rarely temporary occlusion of the ovarian vessels at the infundibulopelvic ligament may be done by rubber-sleeved clamps.

(d) **Ligation of anterior division of internal iliac artery** (unilateral or bilateral)—reduces the distal blood flow. It helps stable clot formation by reducing the pulse pressure up to 85%. Due to extensive collateral circulation, there is no pelvic tissue necrosis. Bilateral ligation (not division) can avoid hysterectomy in about 50% of the cases.

(e) **Angiographic selective arterial embolization** (bleeding vessel) under fluoroscopy (interventional radiology) can be done using gel foam. Success rate is more than 90% and it avoids hysterectomy.

Step VI: **Hysterectomy—**rarely uterus fails to contract and bleeding continues in spite of the above measures. Hysterectomy has to be considered involving a second consultant. Decision of hysterectomy should be taken earlier in a parous woman. Depending on the case, it may be subtotal or total.

TRAUMATIC PPH: The trauma to the perineum, vagina and the cervix is to be searched under good light by speculum examination and hemostasis is achieved by appropriate catgut sutures. The repair is done under general anesthesia, if necessary (see Chapter 29).

Skill drill for management of PPH management for all birth attendants is essential to improve outcome.

Documentation of all measures adopted in respect of time should be done.

SECONDARY POSTPARTUM HEMORRHAGE

CAUSES: The bleeding usually occurs between 8th and 14th day of delivery. **The causes of late postpartum hemorrhage are:** (1) Retained bits of cotyledon or membranes (most common), (2) Infection and separation of slough over a deep cervicovaginal laceration, (3) Endometritis and subinvolution of the placental site—due to delayed healing process, (4) Secondary hemorrhage from cesarean section wound usually occur between 10–14 days. **It is probably due to**—(a) separation of slough exposing a bleeding vessel or (b) from granulation tissue, (5) Withdrawal bleeding following estrogen therapy for suppression of lactation, (6) Other rare causes are: chorionepithelioma—occurs usually beyond 4 weeks of delivery; carcinoma cervix; placental polyp; infected fibroid or fibroid polyp and puerperal inversion of uterus.

DIAGNOSIS: The bleeding is bright red and of varying amount. Rarely it may be brisk. Varying degree of anemia and evidences of sepsis are present. Internal examination reveals evidences of sepsis, subinvolution of the uterus and often a patulous cervical os. **Ultrasonography** is useful in detecting the bits of placenta inside the uterine cavity.

MANAGEMENT

Principles:
- To assess the amount of blood loss and to replace it (blood transfusion).
- To find out the cause and to take appropriate steps to rectify it.

***Supportive therapy*:** (1) Blood transfusion, if necessary, (2) To administer methergine 0.2 mg intramuscularly, if the bleeding is uterine in origin, (3) To administer antibiotics (clindamycin and metronidazole) as a routine.

***Conservative*:** If the bleeding is slight and no apparent cause is detected, a careful watch for a period of 24 hours or so is done in the hospital.

***Active treatment*:** As the most common cause is due to retained bits of cotyledon or membranes, **it is preferable to explore the uterus urgently under general anesthesia**. One should not ignore the small amount of bleeding; as unexpected alarming hemorrhage may follow sooner or later. The products are removed by ovum forceps. Gentle curettage is done by using flushing curette. Methergine 0.2 mg is given intramuscularly. **The materials removed are to be sent for histological examination**.

Presence of bleeding from the sloughing wound of cervicovaginal canal should be controlled by hemostatic sutures. **Secondary hemorrhage following cesarean section** may at times require laparotomy. The bleeding from uterine wound can be controlled by hemostatic sutures; may rarely require ligation of the internal iliac artery or may end in hysterectomy.

KEY POINTS

- **Complications of 3rd stage of labor are:** a) PPH, b) Retained placenta, c) Shock, d) Pulmonary embolism and e) Inversion of uterus.
- **Obstetric hemorrhage:** The major cause of maternal death both in the developed and developing countries. Clinical definition of PPH is more important than the quantitative definition of blood loss >500 mL following birth of the baby.
- **Common causes of** PPH are (4Ts): a) Atonic (80%), b) Traumatic, c) Retained tissues and d) Coagulopathy (see p. 410).
- **Prediction and prevention of PPH** may be possible to some extent, though most cases of PPH have no identifiable risk factor (see p. 412).
- **Full management protocol** for major PPH (blood loss >1 L) includes: a) Communication b) Resuscitation c) Monitoring and investigations and d) Arrest of bleeding (see p. 479-483).
- **Volume replacement** should be quick and balanced. Crystalloid up to 2L; Colloid up to 1–2 L; Cross matched blood; FFP 4 units for every 6 units of red cells. Platelets (if count $<50 \times 10^9$/L) and cryoprecipitate (if fibrinogen <1 g/L).
- Therapeutic goals is to maintain: • Hemoglobin >8 g/dL • Platelet count 75×10^9/L • Fibrinogen >1 g/L • APT >1.5 × mean control
- **Cases with atonic PPH,** the following pharmacological agents are most helpful. These are: oxytocin (IV/IM), Ergometrine IV/IM; Carboprost IM/intramyometrial or misoprostol P/R.
- **The mechanical** and **conservative surgical measures** are: Bimanual uterine compressions; Balloon tamponade; Hemostatic breast sutures; Bilateral ligation of uterine arteries; Bilateral ligation of anterior division of internal iliac arteries or Selective arterial embolization.
- **Hysterectomy** is the last resort in a rare case. However decision for hysterectomy should not be delayed. A second consultant may be involved in decision-making.

RETAINED PLACENTA

DEFINITION: The placenta is said to be retained when it is not expelled out even 30 minutes after the birth of the baby (WHO 15 minutes).

CAUSES: There are three phases involved in the normal expulsion of placenta: (1) Separation through the spongy layer of the decidua, (2) Descent into the lower segment and vagina, (3) Finally its expulsion to outside.

Interference in any of these physiological processes, results in its retention.

- — **Placenta completely separated but retained** is due to poor voluntary expulsive efforts.
- — **Simple adherent placenta** is due to uterine atonicity in cases of grand multipara, over distension of uterus, prolonged labor and uterine malformation or due to bigger placental surface area. **The commonest cause of retention of non-separated placenta is atonic uterus**.
- — **Morbid adherent placenta—**partial or rarely, complete.
- — **Placenta incarcerated following** partial or complete separation due to constriction ring (hourglass contraction), premature attempts to deliver the placenta before it is separated.

DIAGNOSIS: The diagnosis of retained placenta is made by an arbitrary time (15 minutes) spent following delivery of the baby. Features of placental separation are assessed (see p. 143). The hourglass contraction or the nature of adherent placenta (simple or morbid) can only be diagnosed during manual removal.

DANGERS: The risks involved in prolonged retention of placenta are:

(1) Hemorrhage. (2) Shock is due to—(a) blood loss, (b) at times unrelated to blood loss, especially when retained more than one hour and (c) frequent attempts of abdominal manipulation to express the placenta out. (3) Puerperal sepsis. (4) Risk of its recurrence in next pregnancy.

MANAGEMENT

PERIOD OF WATCHFUL EXPECTANCY

- During the period of arbitrary time limit of half an hour, the patient is to be watched carefully for evidence of any bleeding, revealed or concealed and to note the signs of separation of placenta.
- The bladder should be emptied using a rubber catheter.
- Any bleeding during the period should be managed as outlined in third stage bleeding.

RETAINED PLACENTA: ♦ **Separated** ♦ **Unseparated** ♦ **Complicated**

***Placenta is separated and retained*—**To express the placenta out by controlled cord traction.

Unseparated retained placenta (apparently uncomplicated): Manual removal of placenta is to be done under general anesthesia as described earlier (see p. 478).

MANAGEMENT OF UNFORESEEN COMPLICATIONS DURING MANUAL REMOVAL

(i) ***Hour-glass contraction*—**The placenta, either unseparated or separated—partially or completely, may be trapped by a localized contraction of circular muscles of the uterus. This may be situated at the junction of the lower and upper segment or may be placed in one cornu. Administration of any oxytocic, especially ergometrine in the active management of third stage or undue irritability of the uterus by premature attempts to express the placenta is the important cause. **The diagnosis** is only made during attempted manual removal. **Management: The ring should be made to relax by** deepening the plane of anesthesia (halothane is useful in these cases), then the cone shaped hand is introduced and the separation of the placenta is preferably done from above downward to minimize bleeding.

(ii) ***Morbid adherent placenta***—In majority, the diagnosis is made only during attempted manual removal. On rare occasion, however, no cleavage between the placenta and the uterine wall is made possible and the diagnosis of a total placenta accreta is certain.

Complicated retained placenta: The following guidelines are formulated to manage the cases of retained placenta complicated by hemorrhage, shock or sepsis:

- **Retained placenta with shock but no hemorrhage:** To treat the shock and when the condition improves, manual removal of the placenta is to be done.
- **Retained placenta with hemorrhage:** The management protocol is similar to that mentioned in third stage hemorrhage.
- **Retained placenta with sepsis:** The patient is usually delivered outside and is admitted in the referral hospital after few hours or even days after confinement.

  Intrauterine swabs are taken for culture and sensitivity test and broad-spectrum antibiotic is given. Blood transfusion is helpful. As soon as the general condition permits, arrangement is made for manual removal. **The operation should be done by a senior person, as there is possibility of the presence of an adherent placenta.**
- **Retained placenta with an episiotomy wound:** The bleeding points of the episiotomy wound are to be secured by artery forceps. An early decision for manual removal should be taken followed by repair of the episiotomy wound.

PLACENTA ACCRETA

(Syn: Morbid adherent placenta)

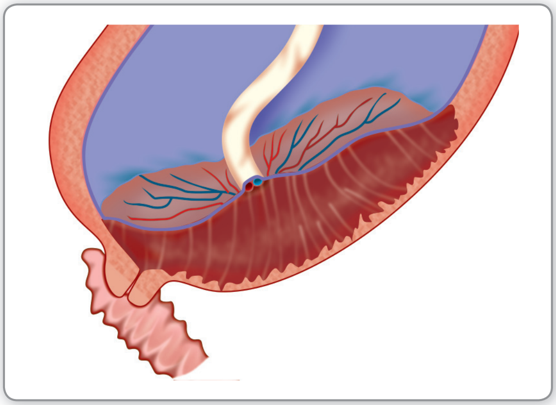


Fig. 28.7: Placenta increta

Placenta accreta is an extremely rare form in which the placenta is directly anchored to the myometrium partially or completely without any intervening decidua. The probable cause is due to absence of decidua basalis and poor development of fibrinoid layer. Overall incidence of placenta accreta or its variations is 1 in 550 deliveries.

Risk factors for placenta accreta: Most important are the placenta previa and prior cesarean delivery. Other risk factors include prior uterine surgery (dilatation and curettage, manual removal of placenta, synaecolysis or myomectomy) increasing maternal age and parity.

The risk of placenta accreta with placenta previa in an unscarred uterus is about 3%. The risk rises sharply with increasing number of cesarean delivery. Placenta previa with one prior cesarean section, the risk of being accreta is about 11%, whereas with two it is 40% and it is 67% with four or more cesarean sections.

The diagnosis is made only during attempted manual removal when the plane of cleavage between the placenta and the uterine wall cannot be made out. **Ultrasound imaging, color Doppler and MRI** have all been valuable in the diagnosis of placenta accreta, increta and percreta during pregnancy. **USG findings suggestive of placenta accreta are**: (i) loss of normal hypoechoic retroplacental myometrial zone, (ii) thinning and disruption of the uterine serosa-bladder interface and focal exophytic masses invading the bladder. Color flow Doppler study shows hypervascularity of serosa bladder interface. MRI reveals detour vessels and dark intraplacental bands on T2-weighted imaging. Unexplained rise of maternal serum αFP is observed with placenta accreta. **Pathological confirmation includes—** (a) absence of decidua basalis, (b) absence of Nitabuch's fibrinoid layer and (c) varying degree of penetration of the villi into the muscle bundles (increta) (Fig. 28.7) or up to the serosal layer (percreta). **The risks include** hemorrhage, shock, infection and rarely inversion of the uterus.

MANAGEMENT: ♦ Multidisciplinary team approach in management is to be done (see p. 291).

In partial placenta accreta (focal) → Remove the placental tissue as much as possible. Effective uterine contraction and hemostasis are achieved by oxytocic and if necessary by intrauterine plugging. In cases following cesarean delivery bleeding areas are oversewed. If the uterus fails to contract, an early decision of hysterectomy may have to be taken and this is preferable in multiparous women.

♦ **In total placenta accreta**, hysterectomy is indicated in parous women, while in patients desiring to have a child, conservative attitude may be taken. This consists of incising the uterus above the placental attachment and clamping and cutting the umbilical cord as close to its base as possible and leaving behind the placenta, which is expected to be autolyzed in due course of time. Appropriate antibiotics should be given. Any attempt of placental separation risks massive hemorrhage and ends in hysterectomy in 100% of cases. Uterine artery embolization or therapy with methotrexate has been done for conservation of the uterus.

♦ **In a rare case**, placenta accreta may invade the bladder. In that case, try to avoid placental removal. It may need hysterectomy and partial cystectomy.

INVERSION OF THE UTERUS

It is an extremely rare but a life-threatening complication in third stage in which the uterus is turned inside out partially or completely. The incidence is about 1 in 20,000 deliveries. The obstetric inversion is almost always an acute one and usually complete.

VARIETIES

- ***First degree***—There is dimpling of the fundus, which still remains above the level of internal os.
- ***Second degree***—The fundus passes through the cervix but lies inside the vagina.
- ***Third degree* (complete)—**The endometrium with or without the attached placenta is visible outside the vulva. The cervix and part of the vagina may also be involved in the process (Fig. 28.8).

It may occur before or after separation of placenta.

ETIOLOGY: The inversion may be spontaneous or more commonly induced.

***Spontaneous (40%)*:** This is brought about by localized atony on the placental site over the fundus associated with sharp rise of intraabdominal pressure as in coughing, sneezing or bearing down effort. Fundal attachment of the placenta (75%), short cord and placenta accreta weakness of uterine wall at the placental site are often associated.

***Iatrogenic*:** This is due to the mismanagement of third stage of labor.

- — **Pulling the cord** when the uterus is atonic especially when combined with fundal pressure
- — **Fundal pressure** while the uterus is relaxed—**Faulty technique in manual removal.**

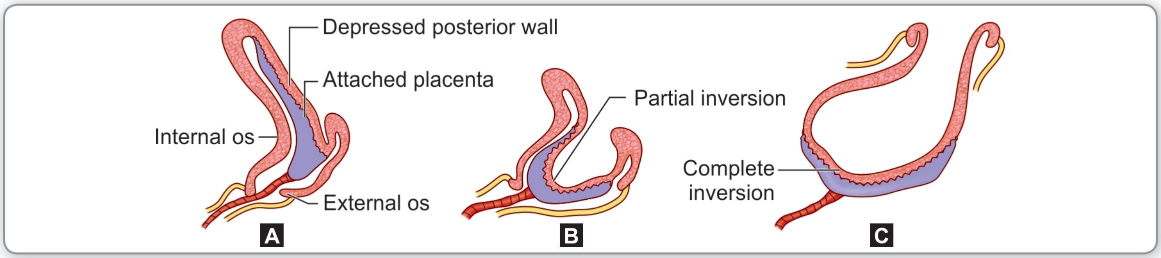
Common risk factors are uterine over enlargement, prolonged labor, fetal macrosomia, uterine malformations, morbid adherent placenta, short umbilical cord, tocolysis and manual removal of placenta. It is more common in women with collagen disease like Ehler-Danlos syndrome.

DANGERS: (1) **Shock** is extremely profound mainly of neurogenic origin due to—(a) tension on the nerves due to stretching of the infundibulopelvic ligament, (b) pressure on the ovaries as they are dragged with the fundus through the cervical ring and (c) peritoneal irritation.

(2) **Hemorrhage**, especially after detachment of placenta, (3) **Pulmonary embolism** (4) **If left uncared for**, **it may lead to**—(a) infection, (b) uterine sloughing and (c) a chronic one.

DIAGNOSIS: ***Symptoms***: Acute lower abdominal pain with bearing down sensation.

Signs: (1) Varying degree of shock is a constant feature, (2) Abdominal examination—(a) Cupping or dimpling of the fundal surface, (b) Bimanual examination not only helps to confirm the diagnosis but also the degree. In complete variety, a pear-shaped mass protrudes outside the vulva with the broad end pointing downward and looking reddish purple in color (Fig. 28.9). (c) **Sonography** can confirm the diagnosis when clinical examination is not clear.



Figs 28.8A to C: Inversion of the uterus—(A) First degree (B) Second degree (C) Third degree

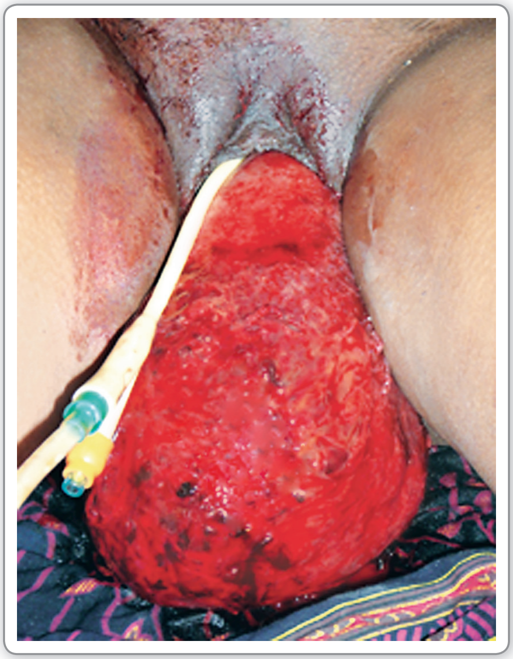


Fig. 28.9: Spontaneous acute and complete uterine inversion. *By courtesy:* Department of Obstetrics and Gynecology, NRS Medical College, Calcutta

PROGNOSIS: As it is commonly met in unfavorable surroundings, the prognosis is extremely gloomy. Even if the patient survives, infection, sloughing of the uterus and chronic inversion with ill health may occur.

PREVENTION: Do not employ any method to expel the placenta out when the uterus is relaxed. Pulling the cord simultaneous with fundal pressure should be avoided. Manual removal should be done in a manner, as it should be (see p. 478).

MANAGEMENT: • **Call for extra help** • **Before the shock develops**, urgent manual replacement even without anesthesia, if it is not readily available, is the essence of treatment for a skilled accoucheur.

Principal steps: The patient is under general anesthesia. (1) **To replace that part first, which is inverted last** with the placenta attached to the uterus by steady firm pressure exerted by the fingers. (2) To apply counter support by the other hand placed on the abdomen. (3) **After replacement, the hand should remain inside the uterus until the uterus becomes contracted by parenteral oxytocin or $PGF_{2\alpha}$**. (4) The placenta is to be removed manually only after the uterus becomes contracted. The placenta may however be removed prior to replacement—(a) to reduce the bulk which facilitates replacement or (b) if partially separated to minimize the blood loss, (5) **Usual treatment of shock** including blood transfusion should be arranged simultaneously.

- ***After the shock develops***

Principal Steps: (1) The treatment of shock should be instituted with an urgent normal saline infusion and blood transfusion (see p. 704). (2) The inverted fundus lies on the palm of the hand with the fingers placed near the uterocervical junction. When pressure is exerted on the fundus, it gradually returns into the vagina. The vagina is packed with antiseptic roller gauze. (3) **Foot end of the bed is raised** (4) **Replacement of the uterus** using hydrostatic method (O'Sullivan's) under general anesthesia is to be done along with resuscitative measures. Hydrostatic method is quite effective and less shock producing.

Hydrostatic method: The inverted uterus is replaced into the vagina. Warm sterile fluid (up to 5 liters) is gradually instilled into the vagina through a douche nozzle. The vaginal orifice is blocked by operator's palms supplemented by labial apposition around the palm by an assistant. Alternatively, a silicon cup (vacuum extraction cup) is placed into the vagina. The douche can be placed at a height of about 3 feet above the uterus. The water distends the vagina and the consequent increased intravaginal pressure leads to replacement of the uterus.

- ***Subacute stage***: (1) To improve the general condition by blood transfusion, (2) Antibiotics are given to control sepsis, (3) Reposition of the uterus either manually or by hydrostatic method may be tried, (4) If fails, reposition may be done by abdominal operation (Haultain's operation).

QUESTIONS

Related theory questions (Long & Short), Obstetric Case Discussions, Viva table discussions, Postoperative word round discussions, and MCQs are discussed in author's books:

1. **Bedside Clinic and Viva Voce:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

For further reading:

2. **Master pass in Obstetrics and Gynaecology:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

Chapter 29

Injuries to the Birth Canal

Maternal injuries following childbirth process are quite common and contribute significantly to maternal morbidity and even to death. Prevention, early detection and prompt and effective management not only minimize the morbidity but prevent many gynecological problems from developing later in life.

VULVA

Lacerations of the vulval skin posteriorly and the paraurethral tear on the inner aspect of the labia minora are the common sites. Paraurethral tear may be associated with brisk hemorrhage and should be repaired by interrupted catgut sutures, preferably after introduction of a rubber catheter into the bladder to prevent injury of the urethra.

PERINEUM

While minor injury is quite common especially during first birth, **gross injury (third and fourth degree) is invariably a result of mismanaged second stage of labor**. Overall risk is 1% of all vaginal deliveries.

CAUSES: Perineal injury (mainly the third and fourth degree) results from (i) over stretching and/or (ii) rapid stretching of the perineum especially when the perineum is inelastic (elderly primigravida, perineal scar).

PREVENTION: Proper conduct in the second stage of labor taking due care of the perineum when it is likely to be damaged is essential (see p. 160). The prevention of the perineal injuries in normal delivery has been outlined in p. 160.

Risk Factors for Third Degree Perineal Tear

- Big baby (weight ≥ 3 kg)
- Nulliparity
- Outlet contraction with narrow pubic arch
- Shoulder dystocia
- Forceps delivery
- Scar in the perineum (perineorrhaphy, episiotomy)
- Face to pubis delivery
- Midline episiotomy
- Precipitate labor

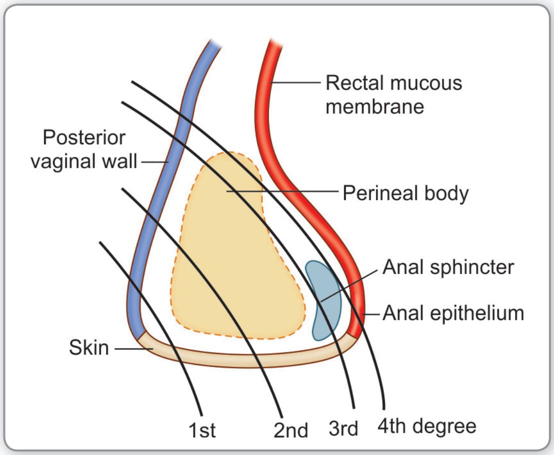


Fig. 29.1: Diagrammatic representation showing different degrees of perineal tear

MANAGEMENT

Recent tear should be repaired immediately following the delivery of the placenta. This reduces the chance of infection and minimizes the blood loss. **In cases of delay beyond 24 hours**, the repair is to be withheld. Antibiotics should be started to prevent infection. **The complete tear should be repaired after 3 months if delayed beyond 24 hours**. In case of any doubt to grade of 3rd degree tear, it is advisable to classify to the higher degree rather than lower degree.

Repair of complete perineal tear (for details see author's Textbook of Gynecology Chapter 26).

Classification of Obstetric Anal Sphincter Injury (RCOG–2007)

First degree: Injury to perineal skin only

Second degree: Injury to perineum involving perineal body (muscles) but not involving the anal sphincter

Third degree: Injury to perineum, involving the anal sphincter complex (both external and internal)

3a : <50% of EAS thickness torn
3b : >50% of EAS thickness torn
3c : Both EAS and IAS torn

Fourth degree: Injury to perineum involving the anal sphincter complex (EAS and IAS) and anal epithelium (Fig. 29.1)

Step I: Patient is put in lithotomy position. Antiseptic cleaning of the local area is done. Repair may be done with local infiltration of 1% lignocaine hydrochloride (10–15 mL) or with pudendal block or preferably under regional or general anesthesia.

Step II: Dissection is **not required** as in an old complete perineal tear. (a) **The rectal and anal mucosa** is first sutured from above downward. No. "3-0" vicryl or 3-0 PDS, atraumatic needle, interrupted stitches with knots inside the lumen are used. (b) **The rectal muscles including the pararectal fascia** are then sutured by interrupted sutures using the same suture material. (c) **The torn ends of the sphincter ani externus (EAS)** are then exposed by Allis's tissue forceps. The sphincter is then reconstructed with a figure of eight stitch, and it is supported by another layer of interrupted sutures. **For repair of EAS either an overlapping or end-to-end approximation method can be used with similar outcome**. IAS repair is done by interrupted suture.

Step III: Repair of perineal muscle is done by interrupted sutures using No. "0" PDS or dexon or polyglactin (vicryl). Surgical knots are buried under the superficial muscles.

Step IV: The vaginal wall and the perineal skin are apposed by interrupted sutures.

Suture material: For repair of EAS, monofilament sutures such as polydioxanone (PDS) or polyglactin (vicryl) can be used. Repair of IAS is done with fine suture size such as 3-0 PDS and 2-0 vicryl as they cause less irritation and discomfort.

AFTERCARE: The aftercare of the repaired perineal injuries is similar to that following episiotomy (see p. 570). **Special care following repair of complete tear**—(1) A low residual diet consisting of milk, bread, egg, biscuits, fish, sweets, etc. is given from third day onward. (2) Lactulose 8 mL twice daily beginning on the second day and increasing the dose to 15 mL on the third day is a satisfactory regime to soften the stool. (3) Any one of the broad-spectrum antibiotics (IV cefuroxime 1.5 g) is used during the intraoperative and the postoperative period. Metronidazole 400 mg thrice daily is to be continued for 5–7 days to cover the anaerobic contamination of fecal matter. The woman is advised physiotherapy and pelvic floor exercises and she is reviewed again 6–12 weeks postpartum. In case of persistent incontinence of flatus and feces, **endoanal USG and anorectal manometry** should be considered to detect any residual defects (20–30%). Consultation with a colorectal surgeon may be needed.

PLAN FOR FUTURE DELIVERY: All women need to have institutional delivery following repair of obstetric sphincter injury. Vaginal delivery may be allowed in a selected case with or without episiotomy. Women having symptoms or with abnormal endoanal USG and/or manometry should be delivered by elective cesarean birth.

VAGINA

Isolated vaginal tears or lacerations without involvement of the perineum or cervix are not uncommon. These are usually seen following instrumental or manipulative delivery. In such cases, the tears are extensive and often associated with brisk hemorrhage.

TREATMENT: Tears associated with brisk hemorrhage require exploration under general anesthesia with a good light. The tears are repaired by interrupted or continuous sutures using chromic catgut No. "0". In case of extensive lacerations, in addition to sutures, hemostasis may be achieved by intravaginal plugging by roller gauze, soaked with glycerin and acriflavine. **The plug should be removed after 24 hours**. Selective arterial embolization may also be done if bleeding persists.

COLPORRHEXIS: **Rupture of the vault of the vagina is called colporrhexis**. It may be primary where only the vault is involved or secondary when associated with cervical tear (common). It is said to be complete when the peritoneum is opened up. Posterior fornix usually ruptures, however, cervical tear is usually associated with tear of the lateral fornix.

Treatment—If the tear is limited to the vault close to the cervix, the repair is done from below. If, however, the cervical tear extends high up into the lower segment or major branches of uterine vessels are damaged, laparotomy is to be done simultaneously with resuscitative measures. Evacuation of hematoma and arterial ligation may be needed.

CERVIX

Minor degree of cervical tear is invariable during first delivery and requires no treatment. Extensive cervical tear is rare. **It is the commonest cause of traumatic postpartum hemorrhage**. Left lateral tear is the commonest.

CAUSES:

- ***Iatrogenic***—Attempted forceps delivery or breech extraction through incompletely dilated cervix.
- ***Rigid cervix***—This may be congenital or more commonly following scar from previous operations on the cervix like amputation, conization or presence of a lesion like carcinoma cervix.
- ***Strong uterine contractions*** as in precipitate labor or extremely vascular cervix as in placenta previa.
- ***Detachment***—Detachment of the cervix may be annular which involved the entire circumference of the cervix. This occurs following prolonged labor in primary cervical dystocia. It may, however, involve only the anterior lip when it is nipped between the head and the symphysis pubis in association with the sacral os. In both varieties, the bleeding is minimal and healing occurs through epithelialization.

DIAGNOSIS: **Excessive vaginal bleeding immediately following delivery in presence of a hard and contracted uterus—raises the suspicion of a traumatic bleeding**. Exploration of the uterovaginal canal under good light not only confirms the diagnosis but also helps to know the extent of the tear.

DANGERS:

Early — (1) Deep cervical tears involving the major vessels lead to severe postpartum hemorrhage. (2) Broad ligament hematoma. (3) Pelvic cellulitis. (4) Thrombophlebitis.

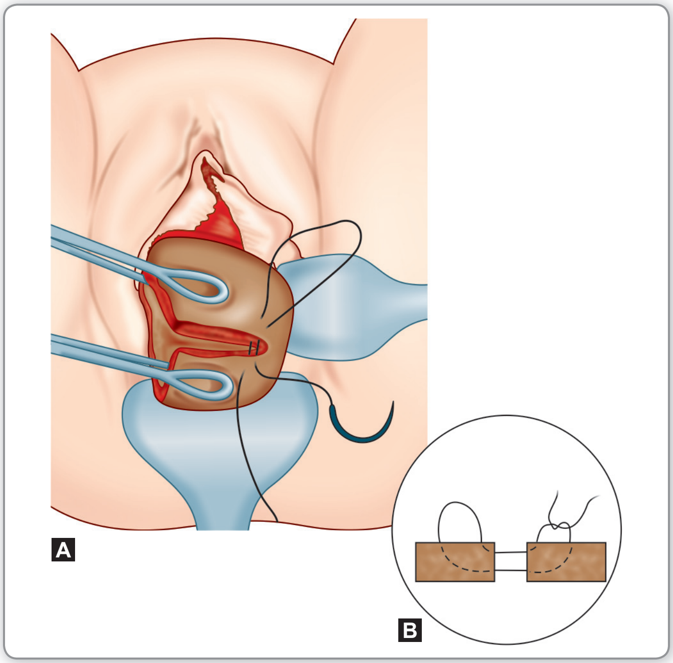
Late — (1) Ectropion. (2) Cervical incompetence with midtrimester abortion.

TREATMENT:

Only deep cervical tear associated with bleeding should be repaired soon after delivery of the placenta. **Repair should be done under general anesthesia**, **in lithotomy position with a good light**.

The prerequisites are — Sims' posterior vaginal speculum, vaginal wall retractors, at least two sponge holding forceps and an assistant.

***Procedures*:** The anterior and posterior margins of the torn cervix are grasped by the sponge holding forceps. Instead of giving traction to the forceps, it is better to push down the fundus gently by the assistant. This makes the tear more accessible for effective suturing. **The apex is to be identified first and the first vertical mattress suture is placed just above the apex** using polyglactin (vicryl) or chromic catgut No. "0" taking whole thickness of the cervix (Fig. 29.2). The bleeding stops immediately. The rest of the tear is repaired by similar mattress sutures. Mattress suture is preferable as it prevents rolling in of the edges. **A helpful guide for proper exposure** in such a case is to start suture at the proximal end and using the suture for traction, more distal tear area is exposed until the apex is in view and is repaired. The cervical tears extending to the lower segment or vault with broad ligament hematoma are managed as outlined in rupture uterus.



Figs 29.2A and B: Methods of repair of the cervical tear with vertical mattress suture

PELVIC HEMATOMA

DEFINITION: Collection of blood anywhere in the area between the pelvic peritoneum and the perineal skin is called pelvic hematoma.

ANATOMICAL TYPES: Depending upon the location of the hematoma, whether below or above the levator ani, it is termed as:

- Infralevator hematoma—common
- Supralevator hematoma—rare

INFRALEVATOR HEMATOMA:

The commonest one is the vulval hematoma.

Etiology: (1) Improper hemostasis during repair of vaginal or perineal tears or episiotomy wound—(a) Failure to take precaution while suturing the apex of the tear (b) Failure to obliterate the dead space while suturing the vaginal walls. (2) Rupture of paravaginal venous plexus either spontaneously or following instrumental delivery.

***Symptoms*:** (1) Persistent, severe pain on the perineal region. (2) There may be rectal tenesmus or bearing down efforts when extension occurs to the ischiorectal fossa. There may be even retention of urine.

Signs: (1) Variable degrees of shock may be evident. (2) Local examination reveals a tense swelling at the vulva which becomes dusky and purple in color and tender to touch (Fig. 29.3).

***Treatment*:** A small hematoma (<5 cm) may be treated conservatively with cold compress. Larger hematomas should be explored in the operation theater under general anesthesia. Simultaneous resuscitative measures are to be taken. The blood clots are to be scooped out and the bleeding points are to be secured. Usually, a generalized oozing surface is visible. The dead space is to be obliterated by deep mattress sutures and a closed suction drain may be kept in that place for 24 hours. A Foley catheter is inserted till the tissue edema subsides. Prophylactic antibiotic is to be administered.

SUPRALEVATOR HEMATOMA: ***Causes***—(1) Extension of cervical laceration or primary colporrhexis (vault rupture). (2) Lower uterine segment rupture (Fig. 29.4). (3) Spontaneous rupture of paravaginal venous plexus adjacent to the vault.

Diagnosis**:** The diagnosis is usually late as pain is not of a conspicuous nature and so also the vaginal bleeding. **Unexplained shock with features of internal hemorrhage following delivery raises the suspicion.** Abdominal examination reveals a swelling above the inguinal ligament pushing the uterus to the contralateral side. **Vaginal examination reveals** (a) occlusion of the vaginal canal by a bulge or (b) a boggy swelling felt through the fornix. **Rectal examination** corroborates the presence of the boggy mass. Ultrasonography may be needed for exact localization of the hematoma.

Management**:** Usual treatment of shock is to be instituted and arrangement is made for laparotomy. The anterior leaf of the broad ligament peritoneum is incised and the blood clot is scooped out. The bleeding points, if visible, are to be secured and ligated. **Random blind sutures should not be placed to prevent ureteric damage.** If the oozing continues, one may have to tie the anterior division of the internal iliac artery. The presence of associated rupture uterus may modify the treatment as mentioned later in the chapter.

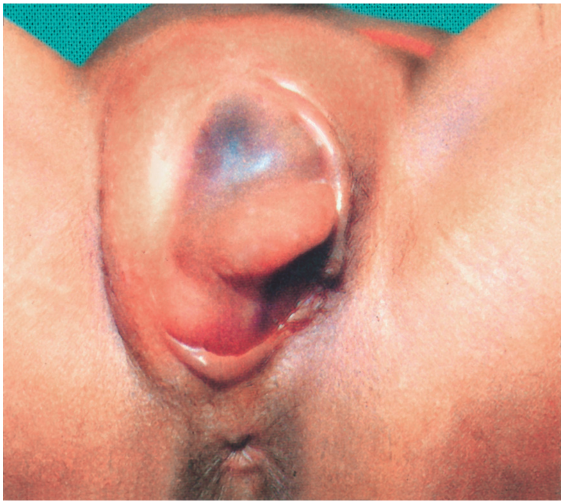


Fig. 29.3: Vulval hematoma following spontaneous delivery

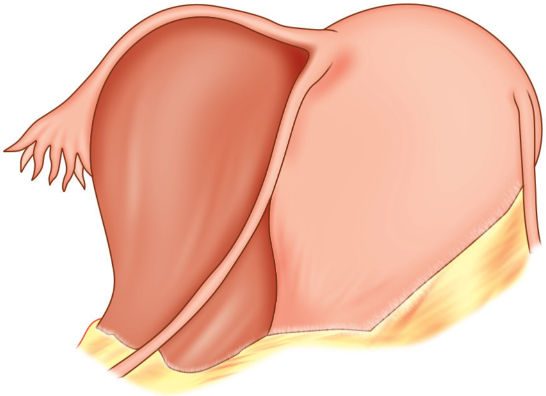


Fig. 29.4: Supralevator hematoma

RUPTURE OF THE UTERUS

DEFINITION: Disruption in the continuity of the all uterine layers (endometrium, myometrium and serosa) any time beyond 28 weeks of pregnancy is called rupture of the uterus. Small rupture to the wall of the uterus in early months is called perforation either instrumental or perforating hydatidiform mole. Rupture of a rudimentary pregnant horn has got a special clinical entity and is grouped in ectopic pregnancy.

INCIDENCE: The prevalence widely varies from 1 in 2,000 to 1 in 200 deliveries. During the past few decades, the prevalence has been found to be almost static. Whereas improved obstetric care reduces the rupture from obstructed labor but there has been increased prevalence of scar rupture following increased incidence of cesarean section over the years.

ETIOLOGY

The causes of rupture of the uterus are broadly divided into:

- **Spontaneous**
- **Scar Rupture**
- **Iatrogenic**

SPONTANEOUS

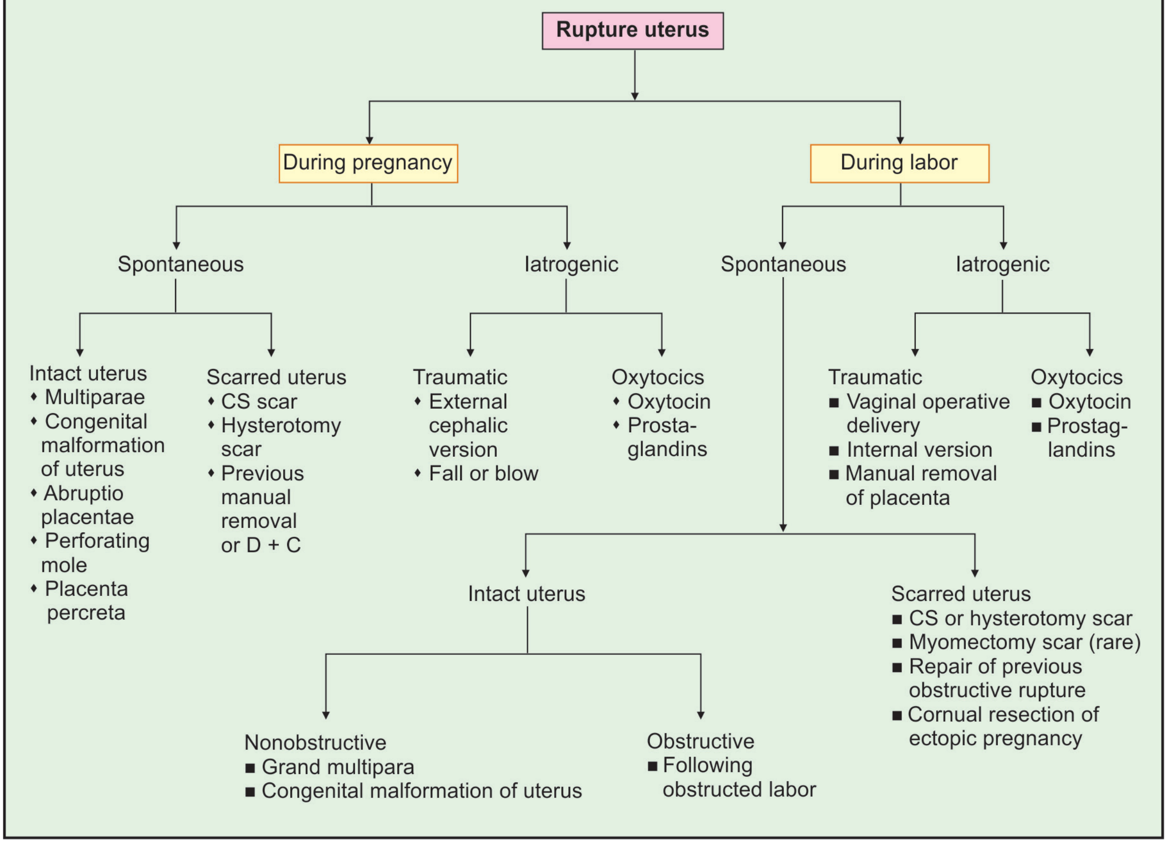
***During pregnancy*:** It is indeed rare for an apparently uninjured uterus to give way during pregnancy. **The causes are**: (1) Previous damage to the uterine walls following dilatation and curettage operation or manual removal of placenta. (2) Rarely in grand multiparae due to thin uterine walls. (3) Congenital malformation of the uterus (bicornuate variety) is a rare possibility. (4) In Couvelaire uterus (see p. 296).

Spontaneous rupture during pregnancy is usually complete, **involves the upper segment** and usually occurs in later months of pregnancy. On rare occasion, spontaneous rupture may occur even in early months.

***During labor*:** Spontaneous rupture which occurs predominantly in an otherwise intact uterus during labor is due to:

- **Obstructive rupture**—This is the end result of an obstructed labor. The mechanism of rupture has already been described in p. 494, 497. The rupture involves the lower segment and usually extends through one lateral side of the uterus to the upper segment.

SCHEME SHOWING ETIOLOGY OF RUPTURE UTERUS


Rupture uterus
During pregnancy
During labor
Spontaneous
Iatrogenic
Spontaneous
Iatrogenic
Intact uterus
• Multiparae
• Congenital malformation of uterus
• Abruptio placentae
• Perforating mole
• Placenta percreta
Scarred uterus
• CS scar
• Hysterotomy scar
• Previous manual removal or D + C
Traumatic
• External cephalic version
• Fall or blow
Oxytocics
• Oxytocin
• Prostaglandins
Traumatic
■ Vaginal operative delivery
■ Internal version
■ Manual removal of placenta
Oxytocics
■ Oxytocin
■ Prostaglandins
Intact uterus
Scarred uterus
■ CS or hysterotomy scar
■ Myomectomy scar (rare)
■ Repair of previous obstructive rupture
■ Cornual resection of ectopic pregnancy
Nonobstructive
■ Grand multipara
■ Congenital malformation of uterus
Obstructive
■ Following obstructed labor

- **Nonobstructive rupture**—Grand multiparae are usually affected and rupture usually occurs in early labor. Weakening of the walls due to repeated previous births as mentioned earlier may be the responsible factor. The rupture usually involves the fundal area and is complete.

SCAR RUPTURE: With the liberal use of primary cesarean section, scar rupture constitutes significantly to the overall incidence of uterine rupture. **The incidence of lower segment scar rupture is about 1–2%, while that following classical one is 5–10 times higher**. **Uterine scar, following operation on the nonpregnant uterus** such as myomectomy or metroplasty hardly rupture as the wound heals well because the uterus remains quiescent following operation. **Uterine scar following hysterotomy** behaves like that of a classical scar and is of growing concern.

***During pregnancy*: Classical cesarean or hysterotomy scar is likely to give way during later months of pregnancy**. The weakening of such scar is due to implantation of the placenta over the scar and consequent increased vascularity. Right angle stretching effect by the increased transverse diameter of the enlarging uterus puts an additional effect in disruption of the upper segment scar. **Lower segment scar rarely ruptures during pregnancy**.

***During labor*:** The classical or hysterotomy scar or cornual resection for ectopic pregnancy is more vulnerable to rupture during labor. Although rare, lower segment scar predominantly ruptures during labor.

IATROGENIC OR TRAUMATIC

***During pregnancy*:** (1) Injudicious administration of oxytocin. (2) Use of prostaglandins for induction of abortion or labor. (3) Forcible external version especially under general anesthesia. (4) Fall or blow on the abdomen.

***During labor*:** (1) Internal podalic version—especially following obstructed labor. (2) Destructive operation. (3) Manual removal of placenta. (4) Application of forceps or breech extraction through incompletely dilated cervix. (5) Injudicious administration of oxytocin for augmentation of labor.

PATHOLOGY

TYPES: Pathologically, it is customary to distinguish between complete and incomplete rupture depending on whether the peritoneal coat is involved or not. So far from the treatment point of view, it matters little. In incomplete rupture, the peritoneum remains intact.

Incomplete rupture usually results from rupture of the lower segment scar or extension of a cervical tear into the lower segment with formation of a broad ligament hematoma. **Complete rupture** usually occurs following disruption of the scar in upper segment. It may also be due to spontaneous rupture of both obstructive and nonobstructive type.

SITES: Spontaneous nonobstructive rupture usually involves the upper segment and often involves the fundus. Whereas, **in obstructive type**, the rupture involves the anterior lower segment transversely and often extends upward along the lateral uterine wall. The margins are ragged and necrosed (Fig. 29.5). On occasion, the

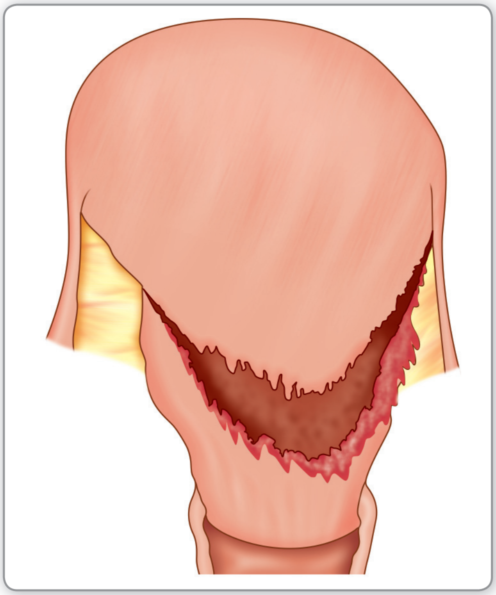


Fig. 29.5: Diagrammatic representation showing rupture of the uterus following obstructed labor. The rent is in the lower segment extending to one side of the uterus. The margins are ragged and necrosed

posterior wall may be involved due to friction with the sacral promontory.

Not infrequently, the tear extends downward to involve the cervix and the vaginal wall (colporrhexis). The bladder may be involved, at times. **Rupture over the previous scar** is almost always located at the site of the scar. The margins of the ruptured cesarean scar are usually clean and look fibrosed (Fig. 29.6). The rent over the lower segment scar may extend to one or both the sides to involve the major branches of uterine vessels.

The morbid pathology of traumatic rupture following destructive operation or internal version is almost similar to that met in spontaneous obstructive variety. This may at times be indistinguishable (Fig. 29.7).

Dehiscence and scar rupture

Scar dehiscence—(a) disruption of part of scar and not the entire length. (b) fetal membranes remain intact. (c) bleeding is almost nil or minimal.

Scar rupture—(a) disruption of the entire length of the scar. (b) complete separation of all the uterine layers including serosa. (c) rupture of the membranes with. (d) varying amount of bleeding from the margins or from its extension. (e) uterine cavity and peritoneal cavity become continuous.

FETUS AND PLACENTA: In incomplete rupture, both the fetus and placenta remain inside the uterine cavity or part of the fetus may occupy in between the layers of broad ligament. **In complete rupture**, the fetus with or without the placenta usually escapes out of the uterus. The uterus remains contracted. Blood loss is not much unless major vessels are affected.

PROGNOSIS: Prognosis depends upon the manner in which labor is managed prior to the accident, type of rupture, morbid pathological changes at the site of the rupture and the effective management. Lower segment scar rupture gives a comparatively better prognosis. But, rupture following obstructed labor either spontaneous or due to instrumentation gives a maternal death rate of about 20% or more. **The major causes of death** are hemorrhage, shock and sepsis. **Late sequelae include** intestinal obstruction and scar rupture in subsequent pregnancies if the uterine rent has been repaired.

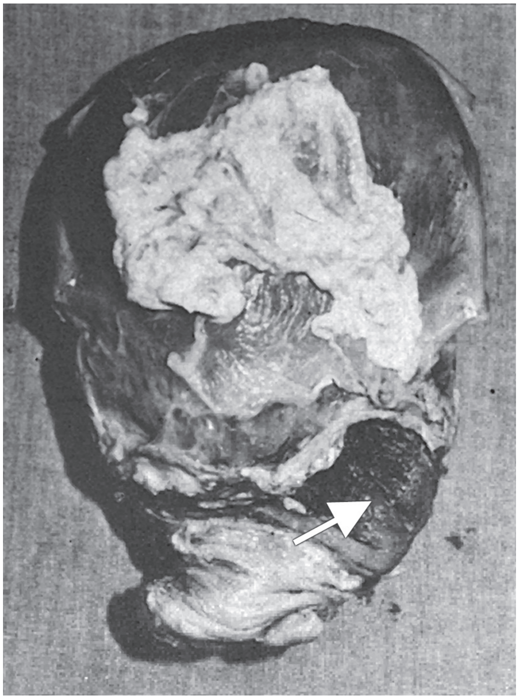


Fig. 29.6: Lower segment scar rupture (arrow). The margins are found clean and scarred. There is omental adhesion on the upper segment. Total hysterectomy was done (*Courtesy*: Museum, G & O Dept NRSMCH)

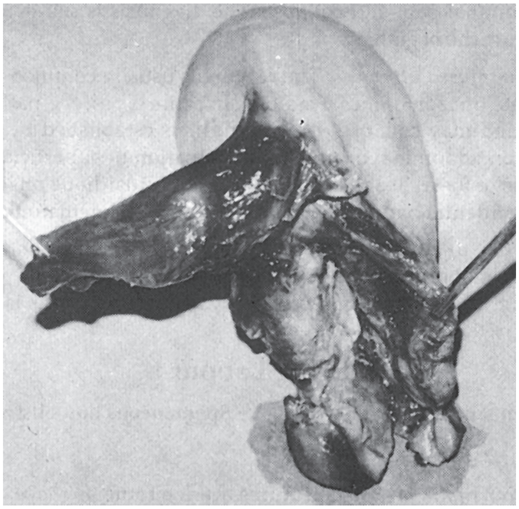


Fig. 29.7: Lower segment scar rupture. The margins are found clean and scarred. There is omental adhesion on the upper segment. Total hysterectomy was done (*Courtesy*: Museum, G & O Dept NRSMCH)

DIAGNOSIS OF RUPTURE UTERUS

It is indeed difficult to categorize a universal clinical feature applicable to all the varieties of uterine rupture. However, the salient diagnostic features of different varieties are described but **it should be remembered that one should be conscious of the entity for an early diagnosis**.

During Pregnancy: ♦ **Scar Rupture** ♦ **Spontaneous** ♦ **Iatrogenic**

***Scar rupture*: Classical or hysterotomy**—The patient complains of a dull abdominal pain over the scar area with slight vaginal bleeding. There is varying degrees of tenderness on uterine palpation. FHS may be irregular or absent. The features may not be always dramatic in nature (silent phase). Sooner or later, the rupture becomes complete. There is a sense of something giving way accompanied by acute abdominal pain and collapse. The diagnosis is self-evident. However, an acute dramatic onset may occur from the beginning.

Spontaneous rupture in uninjured uterus—The rupture is usually confined to the high parous women. The onset is usually acute but sometimes insidious. In acute types, the patient has acute pain abdomen with fainting attacks and may collapse. The diagnosis is established by the presence of features of shock, acute tenderness on abdominal examination, palpation of superficial fetal parts, if the rupture is complete and absence of fetal heart rate. **However, with insidious onset, the diagnosis is often confused with concealed accidental hemorrhage or rectus sheath hematoma**.

Rupture following fall, blow or external version or use of oxytocics—There is history of such an accident followed by acute pain abdomen and slight vaginal bleeding. Rapid pulse and tender uterus raise the suspicion of rupture. The confirmation is done by laparotomy. **This is too often confused with accidental hemorrhage**.

During Labor: ♦ **Scar Rupture** ♦ **Spontaneous Obstructive**
♦ **Spontaneous Nonobstructive** ♦ **Iatrogenic**

***Scar rupture*:** *Classical or hysterotomy scar rupture*—The features are the same as those occur during pregnancy. The onset is usually acute.

Lower segment scar rupture—The onset is insidious. There is no classical feature of lower segment scar rupture (details see p. 495). The confirmation is by laparotomy. The features of scar rupture are not as dramatic as those following obstructed labor (vide infra) and hence called "silent rupture".

***Spontaneous obstructive rupture*:** This type of spontaneous rupture has got a distinct premonitory phase prior to rupture.

Premonitory phase: **The patient is usually a multipara who is in labor with features of obstruction.** Initially, the pains become severe in an attempt to overcome the obstruction and come at quick intervals. Gradually, the pains become continuous and mainly confined to the suprapubic region. **On examination,** the patient is dehydrated and exhausted. The pulse rate and temperature rise. **Abdominal examination reveals** a distended tender lower segment. Bandl's ring may be visible and there are evidences of fetal distress or FHS may be absent. **On vaginal examination,** the presenting part is found jammed in the pelvis and the vagina becomes dry and edematous.

Phase of rupture: (1) **There is a sense of something giving way at the height of uterine contraction.** (2) The constant pain is changed to dull aching pain with cessation of uterine contractions. (3) **General examination reveals** features of exhaustion and shock. (4) **Abdominal examination reveals**—(i) superficial fetal parts, (ii) absence of FHS, (iii) absence of uterine contour and (iv) two separate swellings, one contracted uterus and the other—fetal ovoid. (5) **Vaginal examination reveals—**(i) recession of the presenting part and (ii) varying degrees of bleeding.

***Spontaneous nonobstructive rupture*:** This is rare and solely confined to high parous women. The patient at the height of uterine contraction is suddenly seized with an agonizing bursting pain followed by a relief, with cessation of contractions. **The diagnostic features of the catastrophe are**—presence of shock, evidences of internal hemorrhage, tenderness over the uterus and varying amount of vaginal bleeding.

Rupture following manipulative or instrumental delivery**: Sudden deterioration of the general condition of the patient** with varying amount of vaginal bleeding following manipulative or instrumental delivery raises the suspicion. Exploration of uterus to feel the rent confirms the diagnosis. Not infrequently, the diagnosis is not revealed until after varying intervals following development of shock or broad ligament hematoma or peritonitis. **Shortening of the cord immediately following a difficult vaginal delivery is pathognomonic of uterine rupture**, the placenta being extruded out into the abdominal cavity, through the rent in the uterus.

MANAGEMENT OF RUPTURE UTERUS

PROPHYLAXIS: The following guidelines are helpful to prevent or to detect at the earliest the tragic occurrence of rupture uterus:

- **The at-risk mothers, likely to rupture, should have mandatory hospital delivery. These are—** (a) Contracted pelvis. (b) Previous history of cesarean section, hysterotomy or myomectomy. (c) Uncorrected transverse lie. (d) Grand multiparity. (e) Known case of hydrocephalus.
- **General anesthesia should not be used** to give undue force in external version.
- **Undue delay in the progress of labor** in a multipara with previous uneventful delivery should be viewed with concern and the cause should be sought for.
- **Judicious selection of cases with previous history of cesarean sections** for vaginal delivery (VBAC) (see p. 383).
- **Judicious selection of cases** and careful watch are mandatory during oxytocin infusion either for induction or augmentation of labor.
- **There is hardly any place of internal podalic version** in singleton fetus in present day obstetrics. **It should never be done in obstructed labor** as an alternative to destructive operation or cesarean delivery.
- **Attempted forceps delivery or breech extraction** through incompletely dilated cervix should be avoided.
- **Destructive vaginal operations** should be performed by skilled personnel and **exploration of the uterus should be done as a routine** following delivery.
- **Manual removal in morbid adherent placenta**—should be done by a senior person (see p. 476).

TREATMENT: ♦ **Resuscitation** ♦ **Laparotomy**

Depending upon the state of the clinical condition, either resuscitation is to be done followed by laparotomy or in acute conditions, resuscitation and laparotomy are to be done simultaneously.

LAPAROTOMY: Any of the three procedures may be adopted following laparotomy.

- ***Hysterectomy*****: Hysterectomy is the surgery for rupture uterus unless there is sufficient reason to preserve it**. This is especially indicated in spontaneous obstructive rupture, so common in the developing countries. Considering the low general condition and disturbed morbid anatomical changes near the cervicovaginal region, it is preferable to perform a **quick subtotal hysterectomy, rather than total hysterectomy**. Chance of injury to the ureters or bladder is thereby minimized.

  However, if the condition permits and/or there is colporrhexis, a total hysterectomy may be done.
- ***Repair*****: This is mostly applicable to a scar rupture where the margins are clean**. Repair is done by excision of the fibrous tissue at the margins. One may have to repair a spontaneous obstructive rupture in odd circumstances (desirous of having child), if possible. In such cases, however, there is chance of peritonitis and septicemia. Remote prognosis during future pregnancy is very much unfavorable because of high risk of scar rupture.
- ***Repair and sterilization*****:** This is mostly done in patients with a clean cut scar rupture having desired number of children.

To tackle a broad ligament hematoma—To open up the anterior leaf of the broad ligament → Scoop out the blood clot → Secure the bleeding points → Replaced by ligature, **taking care not to injure the ureter**. Failing to secure the bleeding points → To tie the anterior division of the internal iliac artery.

VISCERAL INJURIES

BLADDER: Causes—Obstetrical injury to the bladder may be due to:

(A) **Traumatic**—(1) Instrumental vaginal delivery such as destructive operations or forceps delivery especially with Kielland (2) Abdominal operation such as hysterectomy for rupture uterus or cesarean section. (B) **Sloughing fistula**—It results from prolonged compression effect on the bladder between the head and symphysis pubis in obstructed labor (see p. 499).

***Diagnosis*:** (A) **Traumatic**—(1) Urine dribbles out soon following the operative delivery. Blood stained urine following cesarean section or hysterectomy is suggestive of bladder injury (2) Margins are clean cut with oozing surfaces. (B) **Sloughing fistula**—(1) History of prolonged labor. (2) Dribbling of urine occurs after varying interval following delivery (5–7 days). (3) **Margins devitalized and necrosed.** (4) Missing of a chunk of tissue.

Management: *Traumatic fistula*: **Immediate local repair is preferable**, if the local tissues are healthy.

In unfavorable condition, a self-retaining catheter is introduced and to be kept for 10–14 days or even longer. Urinary antiseptics are prescribed. In favorable condition, there may be spontaneous closure of the fistula. **If it fails, repair is to be done after 3 months**.

***Sloughing fistula*:** Repair should not be attempted as the conditions are not ideal (vide supra), instead, a self-retaining catheter is placed as outlined above. **Repair is to be done after 3 months**.

RECTUM: Rectal injury, other than that involved along with complete perineal tear is rare in obstetrics. **This is because**, the middle-third of the rectum is protected by the curved sacral hollow and the upper-third is protected by the peritoneal lining. Prolonged compression of the rectum by the head in midpelvic contraction with a flat sacrum predisposes to ischemic necrosis of the anterior rectal wall and results in rectovaginal fistula. **The repair in such cases should be postponed for at least 3 months**.

URETHRA: Urethral injury may be traumatic resulting from instrumental delivery or during pubiotomy; may be ischemic sloughing, the mechanism of which is similar to that of bladder necrosis. The principles in management are similar to those of bladder injury.

KEY POINTS

- **Fourth degree perineal (complete) tear** involves tear of the posterior vaginal wall, perineal body, anal sphincter complex with tear of the anal or rectal mucosa. Recent tear can be repaired immediately (see p. 490, 491).
- **Pelvic hematoma** is commonly due to vulvar hematoma (infralevator). Large vulval hematoma should be explored in the operation theater (see p. 492).
- **Rupture of uterus** may be—(i) spontaneous, (ii) scar rupture or (iii) iatrogenic (see p. 495). Rupture of uterus may occur either during pregnancy or during labor (see p. 495). Diagnosis is difficult (see p. 497). One should be conscious of the entity.
- **Management of rupture uterus** is resuscitation and laparotomy (see p. 498). Subtotal hysterectomy is commonly done. Repair may be done in cases where margins are clean. Repair and sterilization is done when the woman has completed her family.
- **Obstetric emergencies** are often very frightening though rare.
- **Obstetric emergency** drill training should be regularly practiced by the labor ward team to face such emergencies.
- **Rupture uterus**, cord prolapse, PPH, shoulder dystocia, eclampsia, inversion uterus are some of the many obstetric emergencies. All these need prompt and appropriate management (see p. 494).

QUESTIONS

1. Mention the different types of uterine rupture? How the diagnosis of a spontaneous rupture of the uterus can be made? (p. 494)
2. Outline the management of a case with spontaneous rupture of the uterus? (p. 498)
3. Write short notes on: Risk factors for complete perineal tear? (p. 489)

Chapter 30

Abnormalities of the Puerperium

PUERPERAL PYREXIA

DEFINITION: A rise of temperature reaching 100.4°F (38°C) or more (measured orally) on two separate occasions at 24 hours apart (excluding first 24 hours) within first 10 days following delivery is called puerperal pyrexia. In some countries, postabortal fever is also included.

Causes of Puerperal Pyrexia

- Puerperal sepsis
- Urinary tract infections: Cystitis, Pyelonephritis
- Mastitis, Breast abscess
- Wound infections: CS or Episiotomy
- Pulmonary infections : Atelectasis, Pneumonia
- Septic pelvic thrombophlebitis
- A recrudescence of malaria or pulmonary tuberculosis
- Others: Pharyngitis, Gastroenteritis

The pathology and prevention of child birth fever (puerperal pyrexia) are best known from the works of *Ignaz Semmelweis*, working in Vienna in the nineteenth century. He faced difficulties to establish his doctrine. Unfortunately, he died of an infection on his right hand that he contracted during an operation.

PUERPERAL SEPSIS

(Syn: Puerperal infection)

DEFINITION: An infection of the genital tract which occurs as a complication of delivery is termed puerperal sepsis. Puerperal pyrexia is considered to be due to genital tract infection unless proved otherwise.

There has been marked decline in puerperal sepsis during the past few years due to: (1) improved obstetric care, (2) availability of wider range of antibiotics.

Puerperal sepsis is commonly due to—(i) endometritis, (ii) endomyometritis, or (iii) endoparametritis or a combination of all these when it is called **pelvic cellulitis**.

***Vaginal flora*: The vaginal flora in late pregnancy and at the onset of labor consists of the following organisms:** (1) Doderlein's bacillus (60–70%), (2) Yeast-like fungus with increased prevalence of *Candida albicans* (25%), (3) *Staphylococcus albus* or *aureus*, (4) *Streptococcus*—anaerobic common; beta-hemolytic rare, (5) *Escherichia coli* and *Bacteroides* group, (6) *Clostridium welchii* on occasion. **These organisms remain dormant** and are harmless during normal delivery conducted in aseptic condition.

PREDISPOSING FACTORS OF PUERPERAL SEPSIS: The pathogenicity of the vaginal flora may be influenced by certain factors: (1) The cervicovaginal mucous membrane is damaged even in normal delivery, (2) The uterine surface too, especially the placental site, is converted into an open wound by the cleavage of the decidua which takes place during the third stage of labor, and (3) The blood clots present at the placental site are excellent media for the growth of the bacteria.

***Antepartum risk factors*:** (1) Malnutrition and anemia, (2) Preterm labor, (3) Premature rupture of the membranes, (4) Immunocompromised (HIV), (5) Prolonged rupture of membrane more than 18 hours, (6) Diabetes.

***Intrapartum risk factors*:** (1) Repeated vaginal examinations, (2) Dehydration and ketoacidosis during labor, (3) Traumatic vaginal delivery, (4) Hemorrhage—antepartum or postpartum, (5) Retained bits of placental tissue or membranes, (6) Prolonged labor, (7) Obstructed labor, (8) Cesarean delivery.

Due to the factors mentioned above, the organisms gain foothold either in the traumatized tissues of the uterovaginal canal or in the raw decidua left behind or in the blood clots, especially at the placental site.

Microorganisms responsible for puerperal sepsis and the major pathology

- ***Aerobic*—Group A beta-hemolytic *Streptococcus* (GAS)—**Toxic shock syndrome, necrotizing fasciitis in episiotomy or cesarean section wound. **Group B beta-hemolytic *Streptococcus* (GBS)** is a significant cause of neonatal deaths due to septicemia, respiratory disease and meningitis. Maternal risks are also high. Methicillin-resistant *S. aureus* (MRSA) causes severe infection. ***Others*—*Staphylococcus pyogenes*, *S. aureus*, *E. coli*, *Klebsiella*, *Pseudomonas*, *Proteus*, *Chlamydia*.**
- ***Anaerobic*—*Streptococcus, Peptococcus, Bacteroides* (*fragilis, bivius*), Fusobacteria, *Mobiluncus* and Clostridia.**

Most of the infections in the genital tract are polymicrobial with a mixture of aerobic and anaerobic organisms.

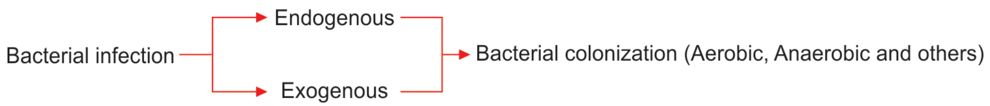
MODE OF INFECTION: Puerperal sepsis is essentially a wound infection. Placental site (being a raw surface), lacerations of the genital tract or cesarean section wounds may be infected in the following ways:

- Sources of infection may be **endogenous** where organisms are present in the genital tract before delivery. **Anaerobic *Streptococcus*** is the predominant pathogen. Infection may be **autogenous** where organisms present elsewhere (skin, throat) in the body and migrate to the genital organs by bloodstream or by the patient herself. **Beta-hemolytic *Streptococcus*, *E. coli*, *Staphylococcus*** are important. Infection may be **exogenous** where infection is contracted from sources outside the patient (from hospital or attendants). Beta-hemolytic *Streptococcus*, *Staphylococcus* and *E. coli* are important.

PATHOLOGY

The primary sites of infection are: (1) perineum, (2) vagina, (3) cervix, (4) uterus. The infection is either localized to the site or spreads to distant sites. The lacerations on the **perineum**, **vagina** and **the cervix** are often infected by the organisms due to the presence of blood clots or dead space. The wounds become red, swollen and there is associated seropurulent discharge. There may be disruption of the wound if repaired before control of infection. **Diabetes**, **obesity**, **immunocompromised state (HIV)** are the other high risk factors for wound infection.

PATHOGENESIS



Endometrium (placental implantation site), cervical lacerated wound, vaginal wound or perineal lacerated wound are the favorable sites for bacterial growth and multiplication. The devitalized tissue, blood clots, foreign body (retained cotton swabs), and surgical trauma favor polymicrobial growth, proliferation and spread of infection. This ultimately leads to metritis, parametritis and/or cellulitis.

Uterus: *Endomyometritis*—The incidence varies from 1–3% following vaginal delivery and about 10% following cesarean delivery. It is commonly polymicrobial (Group A or B Streptococci, Clostridia). **The decidua especially over the placental site is primarily affected**. The **risk factors for endometritis** are, retained products of conception, cesarean section, chorioamnionitis, prolonged rupture of membranes, preterm labor and repeated vaginal examinations in labor.

The necrosed decidua sloughs off. The discharge is offensive. A zone of leukocytic barrier prevents the infection to the deeper myometrium. Severe infection is rare nowadays.

SPREAD OF INFECTION

- **Pelvic cellulitis (parametritis)** is due to spread of infection to the pelvic cellular tissues **by direct or by lymphatic or hematogenous routes**. The infection causes exudation and formation of an indurated mass usually confined to one side of the uterus. The uterus in that case is pushed to the contralateral side.

Peritonitis is common following infection (metritis) after cesarean delivery. There may be necrosis of uterine incision wound and dehiscence. Patient presents with bowel distension and a dynamic ileus.

- **Salpingitis** may be interstitial (due to lymphatic spread) or perisalpingitis (following pelvic peritonitis). Endosalpingitis (tubal mucosa) is uncommon. **Pelvic abscess** following pelvic peritonitis may be due to spread of infection—(a) directly through the tubes, (b) lymphatic spread, or (c) bursting of parametrial abscess. Rarely, there may be generalized peritonitis. Pelvic abscess has become rare (<1%) with the use of antibiotics.
- **Septic pelvic thrombophlebitis**—may involve the ovarian veins, uterine veins, pelvic veins and rarely, the inferior vena cava. The infected thrombus may undergo complete resolution or suppuration. At times, emboli may occlude the microcirculation of the vital organs like lungs or kidney. The anaerobic pathogens are commonly involved (see p. 193).
- **Septicemia and septic shock—**may be due to hemolytic Streptococci (Streptococcal toxic shock syndrome) or anaerobic Streptococci. Septicemia may cause lung abscess, meningitis, pericarditis, endocarditis or multiorgan failure. Death occurs in about 30% of cases.

CLINICAL FEATURES

♦ **Local infection** ♦ **Uterine infection** ♦ **Spreading infection**

LOCAL INFECTION (WOUND INFECTION): (1) There is slight rise of temperature, generalized malaise or headache, (2) The local wound becomes red and swollen, (3) Pus may form which leads to disruption of the wound. When severe (acute), there is high rise of temperature with chills and rigor.

UTERINE INFECTION

Mild—(1) There is rise in temperature (>100.4°F) and pulse rate (>90), (2) Lochial discharge becomes offensive and copious, (3) The uterus is subinvoluted and tender.

Severe—(1) The onset is acute with high rise of temperature, often with chills and rigor, (2) Pulse rate is rapid, out of proportion to temperature, (3) Often there is breathlessness, coughs, abdominal pain and dysuria, (4) Lochia may be scanty and odorless, (5) Uterus may be subinvoluted, tender and softer. There may be associated wound infection (perineum, vagina or the cervix).

SPREADING INFECTION (EXTRAUTERINE SPREAD) is evident by presence of pelvic tenderness **(pelvic peritonitis)**, tenderness on the fornix **(parametritis)**, bulging fluctuant mass in the pouch of Douglas **(pelvic abscess)**.

Parametritis—The onset is usually about 7–10th day of puerperium. (1) Constant pelvic pain, (2) Tenderness on either side of the hypogastrium, (3) Vaginal examination reveals a unilateral tender indurated mass pushing the uterus to the contralateral side, (4) Rectal examination confirms the induration especially extending along the uterosacral ligament. It takes a few weeks to resolute completely. **If, however, suppuration occurs, the features are:** (a) steady rise of spiky temperature with chills and rigor, (b) intense pain, (c) gradual deterioration of the general condition, (d) a fluctuant point may be palpated according to the spread along the cellular plane, but usually above the medial aspect of Poupart's ligament, (e) leukocytosis.

***Pelvic peritonitis*—**(1) Pyrexia with increase in pulse rate, (2) Lower abdominal pain and tenderness, muscle guard may be absent, (3) Vaginal examination reveals tenderness on the fornix and with the movement of the cervix, (4) Collection of pus in the pouch of Douglas is evidenced by swinging temperature, diarrhea and a bulging fluctuant mass felt through the posterior fornix.

General peritonitis—(1) High fever with a rapid pulse, (2) Vomiting, (3) Generalized abdominal pain, (4) Patient looks very ill and dehydrated, (5) Abdomen is tender and distended. Rebound tenderness is often present.

***Thrombophlebitis*—**(1) The clinical features of pelvic thrombophlebitis are similar to those of uterine infection or parametritis, (2) There may be swinging temperature continued for a longer period with chills and rigor, (3) The features of pyemia are present according to the organs involved. It runs a protracted course. These cases are fortunately rare with the advent of wider range of antibiotics. Thrombophlebitis of the leg veins is discussed later in the chapter.

***Septicemia*—**(1) There is high rise of temperature usually associated with rigor. Pulse rate is usually rapid even after the temperature settles down to normal, (2) Blood culture is positive, (3) Symptoms and signs of metastatic infection in the lungs, meninges or joints may appear.

Bacteremia, endotoxic or septic shock is due to release of bacterial endotoxin (lipopolysaccharide) causing circulatory inadequacy and tissue hypoperfusion. It is manifested by hypotension, oliguria and adult respiratory distress syndrome (see p. 702).

INVESTIGATION OF PUERPERAL PYREXIA

The underlying **principles in investigations are:** (1) To locate the site of infection, (2) To identify the organisms, (3) To assess the severity of the disease.

A case of puerperal pyrexia is considered to be due to genital sepsis unless proved otherwise. The investigations should also be directed to find out any extragenital source of infection to account for the fever as well.

Investigations of Puerperal Pyrexia

History: Antenatal, intranatal and postnatal history of any high risk factor for infection like anemia, prolonged rupture of membranes or prolonged labor are to be taken.

Clinical examination includes thorough general, physical and systemic examinations. Abdominal and pelvic examinations are done to note the involution of genital organs and locate the specific site of infection. Legs should be examined for thrombophlebitis or thrombosis.

***Investigations include*:** (1) High vaginal and endocervical **swabs** for culture in aerobic and anaerobic media and sensitivity test to antibiotics. (2) "Clean catch" midstream specimen of **urine** for analysis and culture including sensitivity test. (3) **Blood** for total and differential white cell count, hemoglobin estimation. A low platelet count may indicate septicemia or DIC. **Thick blood film** should be examined for **malarial parasites**. (4) **Blood culture**, if fever is associated with chills and rigor. Other specific investigations as per the clinical condition are needed. (5) **Pelvic ultrasound** is helpful—(i) to detect any retained bits of conception within the uterus, (ii) to locate any abscess within the pelvis, (iii) to collect samples (pus or fluid) from the pelvis for culture and sensitivity, and (iv) for color flow Doppler study to detect venous thrombosis. **Use of CT and MRI** is needed especially when diagnosis is in doubt or there is pelvic vein thrombosis. (6) **X-ray chest (CXR)** should be taken in cases with suspected pulmonary Koch's lesion and also to detect any lung pathology like collapse and atelectasis (following inhalation anesthesia). (7) **Blood urea and electrolytes** may be done in a selected case to have a baseline record in the event that renal failure develops later in the course of the disease or laparotomy is needed.

PROPHYLAXIS

Puerperal sepsis is to a great extent preventable provided certain measures are undertaken before, during, and following labor.

Antenatal prophylaxis includes improvement of nutritional status (to raise hemoglobin level) of the pregnant woman and eradication of any septic focus (skin, throat, tonsils) in the body.

Intranatal prophylaxis includes—(a) *Full surgical asepsis* during delivery (see p. 156), (b) *Screening for Group B Streptococcus* in a high risk patient. Prophylactic use of antibiotic is not recommended as a routine, (c) *Prophylactic use of antibiotic at the time of cesarean section* (see p. 726) has significantly reduced the incidence of wound infection, endometritis, urinary tract infection and other serious infections.

Postpartum prophylaxis includes aseptic precautions for at least 1 week, following delivery until the open wounds in the uterus, perineum, vagina are healed up. Too many visitors are restricted. Sterilized sanitary pads are to be used. Infected babies and mothers should be in isolated room.

TREATMENT

General care: (i) **Isolation of the patient** is preferred especially when hemolytic *Streptococcus* is obtained on culture, (ii) **Adequate fluid and calorie** are maintained by intravenous infusion (IV), (iii) **Anemia** is corrected by oral iron or if needed by blood transfusion, (iv) **An indwelling catheter** is used to relieve any urine retention due to pelvic abscess. It also helps to record urinary output, (v) **A chart** is maintained by recording pulse, respiration, temperature, lochial discharge, and fluid intake and output.

(vi) **Antibiotics**: Ideal antibiotic regimen should depend on the culture and sensitivity report. Pending the report, gentamicin (2 mg/kg IV loading dose, followed by 1.5 mg/kg IV every 8 hours) and clindamycin (900 mg IV every 8 hours) should be started. Metronidazole 0.5 g IV is given at 8 hours interval to control the anaerobic group. The treatment is continued until the infection is controlled for at least 7–10 days.

ANTIBIOTIC REGIMENS: Severe sepsis. A combination of either piperacillin-tazobactam or carbapenem plus clindamycin has broadest range of antimicrobial coverage. Women with MRSA infection should be treated with vancomycin or teicoplanin.

***Surgical treatment*:** There is little role of major surgery in the treatment of puerperal sepsis.

- **Perineal wound**—The stitches of the perineal wound may have to be removed to facilitate drainage of pus and relieve pain. The wound is to be cleaned with **sitz bath** several times a day and is dressed with an antiseptic ointment or powder. After the infection is controlled, secondary suture may be given.
- **Retained uterine products** with a diameter of 3 cm or less may be disregarded and left alone. Otherwise surgical evacuation after antibiotic coverage for 24 hours should be done to avoid the risk of septicemia. Cases with **septic pelvic thrombophlebitis** are treated with IV heparin for 7–10 days (see p. 509).
- **Pelvic abscess** should be drained by colpotomy under ultrasound guidance.
- **Wound dehiscence**: Dehiscence of episiotomy or abdominal wound following cesarean section is managed by scrubbing the wound twice daily, debridement of all necrotic tissue and then closing the wound with secondary suture. Appropriate antimicrobials are used following culture and sensitivity.
- **Laparotomy has got limited indications**. Maintenance of electrolyte balance by intravenous fluids along with appropriate antibiotic therapy usually controls the peritonitis. However, in unresponsive peritonitis, laparotomy is indicated. Even if no palpable pathology is found, drainage of pus may be effective. **Hysterectomy is indicated** in cases with rupture or perforation, having multiple abscesses, gangrenous uterus or gas gangrene infection. Ruptured tubo-ovarian abscess should be removed.
- **Necrotizing fasciitis** (Fig. 30.1) is rare but fatal complication of wound infection (abdominal, perineal, vaginal), involving muscle and fascia. **Risk factors are diabetes, obesity and hypertension**. Infection is caused by Group A beta-hemolytic *Streptococcus* and often it is polymicrobial. Tissue necrosis is the significant pathology. **Treatment includes**: Rehydration, wound scrubbing, debridement of all necrotic tissues, and use of high dose broad-spectrum (IV) antibiotics.

Indications of intensive care unit management: (1) Hypotension, (2) Oliguria, (3) Raised serum creatinine, (4) Raised serum lactate (≥4 mmol/L), (5) Thrombocytopenia, (6) ARDS, (7) Hypothermia.

- **Management of bacteremic or septic shock** (see p. 705) includes: fluid and electrolyte balance (to monitor CVP), respiratory supports (to maintain arterial PO_2 and PCO_2), circulatory support (dopamine or dobutamine), infection control (intensive antibiotic therapy, surgical removal of septic foci) and specific management (as hemodialysis for renal failure).

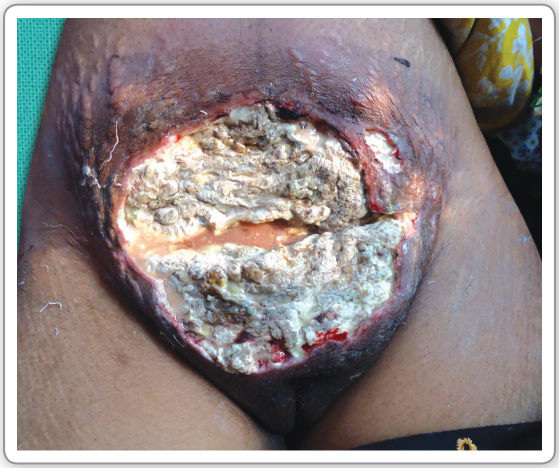


Fig. 30.1: Necrotizing fasciitis involving the skin, subcutaneous tissues, rectus sheath and the muscles (myofasciitis) in a cesarean section wound

SUBINVOLUTION

DEFINITION: When the involution is impaired or retarded, it is called subinvolution. The uterus is the most common organ affected in subinvolution. As it is the most accessible organ to be measured per abdomen, the uterine involution is considered clinically as an index to assess subinvolution.

CAUSES: Predisposing factors are—(1) Grand multiparity, (2) Overdistension of uterus as in twins and hydramnios, (3) Maternal ill-health, (4) Cesarean section, (5) Prolapse of the uterus, (6) Retroversion after the uterus becomes pelvic organ, (7) Uterine fibroid.

Aggravating factors are: (1) Retained products of conception, (2) Uterine sepsis (endometritis).

SYMPTOMS: The condition may be asymptomatic. The predominant symptoms are: (1) abnormal lochial discharge, either excessive or prolonged, (2) irregular or at times excessive uterine bleeding, (3) irregular cramp-like pain in cases of retained products or rise of temperature in sepsis.

SIGNS: (1) The uterine height is greater than the normal for the particular day of puerperium. Normal puerperal uterus may be displaced by a full bladder or a loaded rectum. It feels boggy and softer. (2) Presence of features responsible for subinvolution may be evident.

MANAGEMENT: Mere size of the uterus is not important and provided there is absence of features, such as excessive lochia or irregular bleeding or sepsis, the size of the uterus can be safely ignored. **Appropriate therapy is to be instituted only when subinvolution is found to be a mere sign of some local pathology**: (1) Antibiotics in endometritis, (2) Exploration of the uterus in retained products, (3) Pessary in prolapse or retroversion. Methergine, so often prescribed to enhance the involution process, is of little value in prophylaxis.

URINARY COMPLICATIONS IN PUERPERIUM

URINARY TRACT INFECTION: It is one of the common causes of puerperal pyrexia, the incidence being 1–5% of all deliveries. **The infection may be the consequence of any of the following**: (1) Recurrence of previous cystitis or pyelitis, (2) Asymptomatic bacteriuria becomes overt, (3) **Infection contracted for the first time during puerperium is due to**— (a) effect of frequent catheterization, either during labor or in early puerperium to relieve retention of urine, (b) stasis of urine during early puerperium due to lack of bladder tone and less desire to pass urine.

The organisms responsible are—*E. coli*, *Klebsiella*, *Proteus* and *S. aureus*. The clinical features, diagnosis and management have been described in Chapter 19.

RETENTION OF URINE: This is a common complication in early puerperium. **The causes are**—(1) Bruising and edema of the bladder neck, (2) Reflex from the perineal injury, (3) Unaccustomed position.

Treatment: If simple measure fails to initiate micturition, an indwelling catheter is to be kept in situ for about 48 hours. This not only empties the bladder but helps in regaining the normal bladder tone and sensation of fullness. Following removal of catheter, the amount of residual urine is to be measured. If it is found to be more than 100 mL, continuous drainage is resumed. Appropriate urinary antiseptics should be administered for about 5–7 days.

INCONTINENCE OF URINE: This is not a common symptom following birth. **The incontinence may be:** (1) overflow incontinence, (2) stress incontinence, (3) true incontinence. **Overflow incontinence** following retention of urine should first be excluded before proceeding to differentiate between the other two. **Stress incontinence** usually manifests in late puerperium whereas, **true incontinence** in the form of genitourinary fistula usually appears soon following delivery or within first week of puerperium (see p. 499). Diagnosis of **stress incontinence** is established by noting the escape of urine through the urethral opening during stress. The exact nature of urinary fistula is established by noting the fistula site by examining the patient in Sims' position, using Sims' speculum or by three swab test, if the fistula is tiny.

SUPPRESSION OF URINE: One should differentiate suppression from retention of urine. **If the 24 hours urine excretion is less than 400 mL or less, suppression of urine is diagnosed**; the cause is to be sought for and appropriate management is instituted (see Chapter 39).

BREAST COMPLICATIONS

The common breast complications in puerperium are: (1) breast engorgement, (2) cracked and retracted nipple leading to difficulty in breastfeeding, (3) mastitis and breast abscess, (4) lactation failure. Breast engorgement and infection are responsible for puerperal pyrexia.

BREAST ENGORGEMENT

***Cause*: Breast engorgement is due** to exaggerated normal venous and lymphatic engorgement of the breasts which precedes lactation. This in turn prevents escape of milk from the lacteal system. The primiparous patient and the patient with inelastic breasts are likely to be involved. Engorgement is an indication that the baby is not in step with the stage of lactation.

Onset: It usually manifests after the milk secretion starts (third or fourth day postpartum).

Symptoms include—(a) Considerable pain and feeling of tenseness or heaviness in both the breasts, (b) Generalized malaise or even transient rise of temperature and (c) Painful breastfeeding.

Prevention includes**—**(i) To avoid prelacteal feeds (see p. 522), (ii) To initiate breastfeeding early and unrestricted, (iii) exclusive breastfeeding on demand, (iv) Feeding in correct position, (v) Correct latch on (see p. 521).

Treatment: (1) To support the breasts with a binder or brassiere, (2) Frequent suckling, (3) Manual expression of any remaining milk after each feed, (4) To administer analgesics for pain, (5) The baby should be put to the breast regularly at frequent intervals, (6) In a severe case, gentle use of a breast pump may be helpful. This will reduce the tension in the breast without causing excess milk production.

CRACKED AND RETRACTED NIPPLE

***Cracked nipple*: The nipple may become painful due to—**(1) Loss of surface epithelium with the formation of a raw area on the nipple, or (2) Due to a fissure situated either at the tip or the base of the nipple. These two conditions frequently coexist and are referred to as cracked nipple. It is caused by—(a) unclean hygiene resulting in formation of a crust over the nipple, (b) retracted nipple, and (c) trauma from baby's mouth due to incorrect attachment to the breast, (d) infection with *Candida albicans* and *S. aureus* is often present. The condition may remain asymptomatic but becomes painful when the infant sucks. When infected, the infection may spread to the deeper tissue producing mastitis. **Prophylaxis includes** local cleanliness during pregnancy and in the puerperium before and after each breastfeeding to prevent crust formation over the nipple. ***Treatment*:** Correct attachment (latch on) will provide immediate relief from pain and rapid healing. Fresh human milk and saliva have got healing properties. Purified lanolin with the mother's milk is applied three or four times a day to hasten healing. When it is severe, mother should use a breast pump and the infant is fed with the expressed milk. Inflamed nipple and areola may be due to thrush also. Miconazole lotion is applied over the nipple as well as in the baby's mouth if there is oral thrush. *If it fails to heal up*, rest is given to the affected nipple using a breast pump while the nipples heal. Nipple shields (thin latex) can be used. **The persistence of a nipple ulcer, in spite of therapy mentioned, needs biopsy to exclude malignancy**.

***Retracted and flat nipple*:** It is commonly met in primigravidae. It is usually acquired. Babies are able to attach to the breast correctly and are able to suck adequately. In difficult cases, manual expression of milk can initiate lactation. Gradually breast tissue becomes soft and more protractile, so that feeding is possible (see p. 524).

ACUTE MASTITIS

The **incidence** of mastitis is 2–5% in lactating and less than 1% in non-lactating women. The **common organisms** involved are *S. aureus*, *Staphylococcus epidermidis* and Streptococci viridans. **Risk factors** for mastitis are poor nursing, maternal fatigue and cracked nipple.

***Mode of infection*—There are two different types of mastitis** depending upon the site of infection. (1) Infection that involves the **breast parenchymal tissues** leading to cellulitis. The lacteal system remains unaffected. (2) Infection gains access through the lactiferous duct leading to development of primary mammary adenitis. The source of organisms is the infant's nose and throat.

Noninfective mastitis may be due to milk stasis. Feeding from the affected breast solves the problem.

***Onset*:** In superficial cellulitis, the onset is acute during first 2–4 weeks postpartum. However, acute mastitis may occur even several weeks after the delivery.

***Clinical features:* Symptoms include—**(a) Generalized malaise and headache, nausea, vomiting, (b) Fever (102°F or more) with chills, and (c) Severe pain and tender swelling in one quadrant of the breast. **Signs include**—(a) Presence of toxic features, and (b) Presence of a swelling on the breast. The overlying skin is red, hot and flushed and feels tense and tender.

***Diagnosis*:** Microscopic examination of breast milk, showing leucocytes more than 10^6/mL and bacterial count more than 10^3/mL, supports the diagnosis of mastitis.

***Complications*:** Due to variable destruction of breast tissues, it leads to the formation of a **breast abscess**.

PROPHYLAXIS: Thorough hand washing before each feed, cleaning the nipples before and after each feed, and keeping them dry, reduce the nosocomial infection rates.

***Management*—**(a) Breast support, (b) Plenty of oral fluids, (c) Breastfeeding is continued with good attachment. Nursing is initiated on the uninfected side first to establish let down, (d) The infected side is emptied manually with each feed, (e) Dicloxacillin (penicillinase-resistant penicillin) is the drug of choice. A dose of 500 mg every 6 hours orally is started till the sensitivity report available. Erythromycin is an alternative to patients who are allergic to penicillin. Antibiotic therapy is continued for at least 7 days, (f) Analgesics (ibuprofen) are given for pain, (g) Milk flow is maintained by breastfeeding the infant. This prevents proliferation of *Staphylococcus* in the stagnant milk. The ingested *Staphylococcus* will be digested without any harm.

BREAST ABSCESS: *Features are*—(1) Flushed breasts not responding to antibiotics promptly, (2) Brawny edema of the overlying skin, (3) Marked tenderness with fluctuation, (4) Swinging temperature.

If an abscess is formed, it is to be drained under general anesthesia by a deep radial incision extending from near the areolar margin to prevent injury of the lactiferous ducts. Incision perpendicular to the lactiferous ducts increases the risk of fistula formation and ductal occlusion. Finger exploration is done to break up the walls of the loculi. The cavity is loosely packed with gauze which should be replaced after 24 hours by a smaller pack. The procedure is continued till it heals up. The abscess can also be drained by serial percutaneous needle aspiration under ultrasound guidance. Surgical drainage is commonly done.

Breastfeeding is continued in the uninvolved side. The infected breast is mechanically pumped every 2 hours and with every let down. Recurrence risk is about 10%. Once cellulitis has resolved, breastfeeding from the involved side may be resumed.

Antibiotics to be continued depending upon the culture report of pus.

Breast pain may be due to engorgement, infection (*C. albicans*), nipple trauma, mastitis or occasionally with latching on or let down reflex.

Management: Appropriate nursing technique, positioning and breast care can reduce pain significantly when it is due to nipple trauma, engorgement or mastitis. Use of miconazole oral lotion or gel into both the nipples and into infant's mouth thrice daily for 2 weeks is helpful.

LACTATION FAILURE (INADEQUATE MILK PRODUCTION): The causes are: (1) Infrequent suckling, (2) Depression or anxiety state in the puerperium, (3) Reluctance or apprehension to nursing, (4) Ill development of the nipples, (5) Painful breast lesion, (6) Endogenous suppression of prolactin (retained placental bits), (7) Prolactin inhibition (ergot preparations, diuretics, pyridoxine).

Treatment: For maintenance of effective lactation in an otherwise healthy individual, the following guidelines are helpful.

- **Antenatal:** (1) To counsel the mother regarding the advantages of nursing her baby with breast milk, (2) To take care of any breast abnormality especially a retracted nipple and to maintain adequate breast hygiene especially in the last 2 months of pregnancy.
- **Puerperium:** (1) To encourage adequate fluid intake, (2) To nurse the baby regularly, (3) Painful local lesion is to be treated to prevent development of nursing phobia, (4) Metoclopramide, intranasal oxytocin and sulpiride (selective dopamine antagonist) have been found to increase milk production. They act by stimulating prolactin secretion. Metoclopramide given in a dose of 10 mg thrice daily is found helpful.

PUERPERAL VENOUS THROMBOSIS AND PULMONARY EMBOLISM

Thrombosis of the leg veins and pelvic veins is one of the common and important complications in puerperium especially in the Western countries. The prevalence is, however, low in Asian and African countries.

Basic pathology for venous thrombosis are—(i) Vascular stasis, (ii) Hypercoagulability of blood (pregnancy), and (iii) Vascular endothelial trauma (Virchow's triad 1856). Other pregnancy-specific risk factors are as mentioned below:

Venous thromboembolic diseases include:

♦ **Deep vein thrombosis (iliofemoral)** ♦ **Thrombophlebitis (superficial and deep veins)** ♦ **Pulmonary embolus**

Pathophysiology: (1) In a **normal pregnancy** there is rise in concentration of coagulation factors I, II, VII, VIII, IX, X, XII. Plasma fibrinolytic inhibitors are produced by the placenta and the level of protein S is markedly (40%) decreased. (2) **Alteration in blood constituents**—increased number of young platelets and their adhesiveness. (3) **Venous stasis** is increased due to compression of gravid uterus to the inferior vena cava and iliac veins. This stasis causes damage to endothelial cells. (4) **Thrombophilias** are hypercoagulable states in pregnancy that increase the risk of venous thrombosis. It may be inherited or acquired. **Inherited thrombophilias** are the genetic conditions associated with the deficiencies of antithrombin III, protein C, protein S and prothrombin gene mutation. Others are factor V Leiden mutation and hyperhomocysteinemia. **Acquired thrombophilias** are due to the presence lupus anticoagulant and antiphospholipid antibodies.

Risk factors for VTE—(1) **High risk:** Previous VTE, thrombophilia; (2) **Intermediate risk:** (a) Heart disease, (b) SLE, (c) Surgical procedures (LSCS); (3) **Low risk:** Presence of less than three 3 from any of these risk factors mentioned: [(a) age >35 years, (b) Obesity (BMI >35), (c) Parity ≥3, (d) Immobility, (e) Dehydration, (f) Hyperemesis, (g) Multiple pregnancy]. N.B. Risk Factors more than three 3 make the patient as intermediate risk.

DEEP VEIN THROMBOSIS—Diagnosis: Clinical diagnosis is unreliable. In majority, it remains asymptomatic.

Symptoms include pain in the calf muscles, edema legs and rise in skin temperature. **On examination** asymmetric leg edema (difference in circumference between the affected and the normal leg more than 2 cm) is **significant (Fig. 30.2)**. **A positive Homan's sign**—pain in the calf on dorsiflexion of the foot may be present.

Investigations: The following biophysical tests are employed to confirm the diagnosis:

(1) **Doppler ultrasound** to detect the changes in the velocity of blood flow in the femoral vein by noting the alteration of the characteristic "whoosh" sound which is audible from a patent's vein. **Venous ultrasonography (VUS):** It is done by placing the transducer over the femoral vein and then gradually it is moved to the great saphenous vein, the popliteal vein and to its branches with the deep veins of the calf. **Doppler USG:** The most accurate ultrasound criteria for diagnosis of venous thrombosis is—(a) Soft tissue mass within the venous lumen, (b) Noncompressibility of the venous lumen in a transverse plane under gentle probe pressure. The overall sensitivity and specificity of VUS using duplex and color flow Doppler are at 90–100% for proximal vein thrombosis.

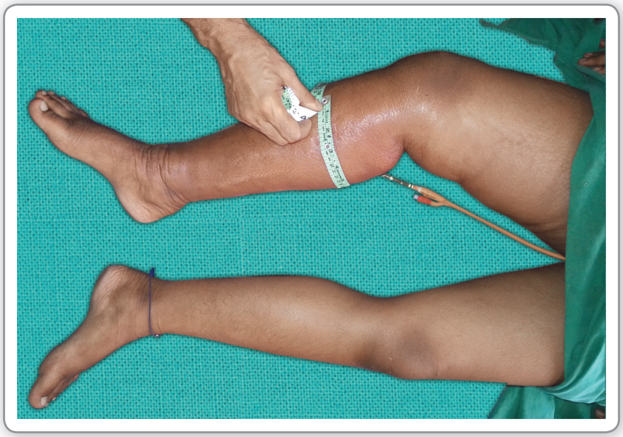


Fig. 30.2: Deep vein thrombosis of the lower limb (right) showing massive edema of both the leg and the thigh. Compression ultrasonography was positive

(2) **Venography** by injecting nonionic water soluble radiopaque dye to note the filling defect in the venous lumen is a reliable method, if carefully interpreted. Venogram is restricted in pregnancy due to the risk of radiation and contrast allergy.

(3) **Magnetic resonance imaging (MRI)** is found superior to VUS and equivalent to contrast venography in the diagnosis of DVT. MRI is helpful to detect thrombosis in pelvic, iliac or femoral veins. The sensitivity and specificity of MRI in the diagnosis of DVT are 100% and the accuracy is 96%.

D-dimer assays: D-dimer is a product of degradation of fibrin by plasmin. The test is of limited value because of false positive results in pregnancy.

PELVIC THROMBOPHLEBITIS: Postpartum thrombophlebitis originates in the thrombosed veins at the placental site by organisms such as anaerobic Streptococci or *Bacteroides* (*fragilis*). **When localized in the pelvis, it is called pelvic thrombophlebitis.** There is no specific clinical feature of pelvic thrombophlebitis, but **it should be suspected in cases where the pyrexia continues for more than a week in spite of antibiotic therapy.**

Extrapelvic spread: (1) Through the right ovarian vein into inferior vena cava and thence to the lungs, (2) Through the left ovarian vein to the left renal vein and thence to the left kidney, (3) Retrograde extension to **iliofemoral** veins to produce the clinicopathological entity of *"phlegmasia alba dolens"* or white leg.

Phlegmasia alba dolens (Syn: White leg): **It is a clinicopathological condition usually caused by retrograde extension of pelvic thrombophlebitis to involve the iliofemoral vein.** The femoral vein may be directly affected from adjacent cellulitis. The condition is seldom met nowadays.

Clinical features: (1) It usually develops on the second week of puerperium. (2) Mild pyrexia is common prior to the dramatic local manifestations. At times, the fever may be high with chills and rigor. (3) Evidences of constitutional disturbances such as headache, malaise and rising pulse rate or features of toxemia may be present. (4) The affected leg is swollen, painful, white and cold. **The pain is due to arterial spasm** as a result of irritation from the nearby thrombosed vein. (5) Blood count shows polymorphonuclear leukocytosis. **The diagnosis** may be made by venous ultrasound, computed tomography (CT) scan or by magnetic resonance imaging (MRI). A trial of heparin therapy be considered. When the symptoms improve with heparin therapy, diagnosis is confirmed.

PROPHYLAXIS AND MANAGEMENT FOR VENOUS THROMBOEMBOLISM (VTE) IN PREGNANCY AND PUERPERIUM

Preventive measures include:

- **Prevention of** trauma, sepsis, anemia in pregnancy and labor. Dehydration during delivery should be avoided.

- **Use of** elastic compression stocking and intermittent pneumatic compression devices during surgery.
- **Leg exercises, early ambulation** are encouraged following operative delivery.

Women at risk of venous thromboembolism during pregnancy have been grouped into different categories depending on the presence of risk factors (see above). **Thromboprophylaxis** to such a woman depends on the specific risk factor and the category.

(1) **A low risk woman** has no personal or family history of VTE and is heterozygous for factor V Leiden mutation. Such a woman needs no thromboprophylaxis, early mobilization and adequate hydration to be maintained. (2) A **high risk woman** needs low-molecular-weight heparin (LMWH) prophylaxis throughout pregnancy and postpartum 6 weeks. (3) **Intermediate risk women** with three or more risk factors are considered for antenatal prophylaxis with LMWH up to 7 days of puerperium.

Management: (1) The patient is put to bed rest with the foot end raised above the heart level. (2) Pain on the affected area may be relieved with analgesics. (3) Appropriate antibiotics are to be administered. (4) **Anticoagulants—** (a) **Heparin** 15,000 units are administered intravenously, followed by 10,000 units 4–6 hourly for 4–6 injections when the blood coagulation is likely to be depressed to the therapeutic level. Heparin is continued for at least 7–10 days or even longer if thrombosis is severe. Prolongation of activated partial thromboplastin time (APTT) to 1.5–2.5 times indicates effective and safe anticoagulation. Serum heparin level should be of 0.1–0.2 U/mL. **LMWH** can be used safely in pregnancy. Enoxaparin 20 mg (wt <50 kg) or 40 mg (wt 50–90 kg) daily is given. It does not cross the placenta. **Fondaparinux** a synthetic pentasaccharide can inhibit factor Xa but not thrombin. It has limited transplacental passage. It can be used in cases with heparin-induced thrombocytopenia or heparin allergies. (b) A drug of **coumarin series**—warfarin is commonly used orally with an overlap of at least 3 days with heparin. The initial daily single dose of 7 mg for 2 days is adequate for induction. Subsequent maintenance dose depends upon international normalized ratio (INR) which should be within the range of 2.0–3.0. The daily maintenance dose of warfarin is usually 5–9 mg, to be taken at the same time each day. The anticoagulant therapy should be continued till all evidences of the disease have disappeared which generally take 3–6 months. **The anticoagulant (warfarin, LMWH or unfractionated heparin) is safe for breastfeeding.** (5) As soon as the pain subsides, gentle movement is allowed on bed by the end of first week. High quality elastic stockings are fitted on the affected leg before mobilization. (6) **Inferior vena cava filters** are used for patients with recurrent pulmonary embolism or where anticoagulant therapy is contraindicated. Vena cava may be completely ligated by teflon clips. (7) **Fibrinolytic agents** like streptokinase produce rapid resolution of pulmonary emboli. (8) **Venous thrombectomy** is needed for massive iliofemoral vein thrombosis or for massive pulmonary embolus.

PULMONARY EMBOLISM (PE)

Pulmonary embolism is the leading cause of **maternal deaths** in many centers especially in the developed countries after the sharp decline of maternal mortality due to hemorrhage, hypertension and sepsis. While deep venous thrombosis in the leg or in the pelvis is most likely the cause of pulmonary embolism, but in about 80–90%, it occurs without any previous clinical manifestations of deep vein thrombosis. The predisposing factors are those already mentioned in venous thrombosis. The clinical features depend on the size of the embolus and on the preceding health status of the patient. The classical symptoms of massive pulmonary embolism are sudden collapse with acute chest pain and air hunger. **Death usually occurs within short time from shock and vagal inhibition**.

The important signs and symptoms of pulmonary embolism are: tachypnea (>20 breaths/min), dyspnea, pleuritic chest pain, cough, tachycardia (>100 bpm), hemoptysis and rise in temperature more than 37°C.

DIAGNOSIS: X-ray of the chest shows diminished vascular marking in areas of infarction, elevation of the dome of the diaphragm and often pleural effusion. It is useful to rule out pneumonia, pulmonary infiltrates and atelectasis.

- **ECG**: Tachycardia, right axis shift, nonspecific ST change, right bundle branch block.
- **Arterial blood gas:** PO_2 more than 85 mm Hg on room air is reassuring but does not rule out PE. Oxygen saturation less than 95% on room air needs further investigation.
- **D-Dimer:** A negative D-Dimer value may rule out the diagnosis of PE. It has a high negative predictive value (Ch. 41).
- **Doppler ultrasound** can identify a DVT. When the test is positive for DVT, anticoagulation therapy should be started.

Lung scans (Ventilation/Perfusion scan or V/Q scan): Perfusion scan will detect areas of diminished blood flow whereas a reduction in perfusion with maintenance of ventilation indicates pulmonary embolism. **V/Q** scanning is the method of choice for patients with suspected PE and with normal chest radiograph. High probability V/Q scan suggests PE. **Magnetic Resonance Imaging (MRI)** can be used in pregnancy as the risk of ionizing radiation is absent.

Pulmonary angiography is accurate to the diagnosis but has got high risks of complications. Mortality rate is 0.5% and overall complication rate is 3%.

Spiral Computed Tomographic Pulmonary Angiography (CTPA): It requires an IV contrast and simultaneous imaging. CTPA is found to be less precise in pregnant as compared to a nonpregnant woman.

Magnetic resonance angiography (MRA) with IV gadolinium: It has got sensitivity of 100% and specificity of 95% in the diagnosis of PE.

Management: ***Prophylaxis*** (as mentioned in page 509).

Active treatment includes: (1) **Resuscitation**—cardiac massage, oxygen therapy, intravenous heparin bolus dose of 5,000 IU and morphine 15 mg (IV) are started. Heparin remains the mainstay of therapy for VTE. Therapeutic doses of LMWH [enoxaparin 1 mg/kg subcutaneous (SC) twice daily] may be used. Antifactor Xa levels of 0.6-1 U/mL are to be maintained. Heparin therapy (IV) should be continued for 5–10 days until patient improves clinically. Thereafter, it is changed to SC injections. Anticoagulation may need to be continued for 6 weeks to 6 months depending upon the case. Heparin level is maintained at 0.2–0.4 U/mL or the activated partial thromboplastin time (APTT) about twice the normal (1.5–2.5 times). (2) **IV fluid support** is continued and blood pressure is maintained, if needed by dopamine or adrenalin. (3) **Tachycardia** is counteracted by digitalis. (4) **Recurrent attacks** of pulmonary embolism necessitate surgical treatment like embolectomy, placement of inferior caval filter or ligation of inferior vena cava and ovarian veins. Surgical treatment is done following pulmonary angiography.

Indications of inferior vena cava filters are: (a) absolute contraindication to medical anticoagulation, (b) failure of anticoagulation, (c) heparin-induced thrombocytopenia, (d) allergy to heparin.

Contraindications of heparin therapy are: ♦ Women with active antenatal or postpartum bleeding, ♦ Risk of major hemorrhage (placenta previa), ♦ Coagulopathy, ♦ Thrombocytopenia.

OBSTETRIC PALSIES *(Syn: Postpartum traumatic neuritis)*

The commonest form of obstetric palsy encountered in puerperium is foot drop. It is usually unilateral and appears shortly after delivery or during first day postpartum or so. **It is thought to be due to stretching of the lumbosacral trunk by the prolapsed intervertebral disk between L_5 and S_1.** Backward rotation of the sacrum during labor may also be a contributing factor. Direct pressure either by the fetal head or by forceps blade on the lumbosacral cord or sacral plexus as a causative factor is no longer tenable.

The condition is usually mild and may pass unnoticed unless there is disability. Neurological examination reveals lower motor neuron type of lesion with flaccidity and wasting of the muscles in areas supplied by the femoral nerve or lumbosacral plexus. Sensory loss is often present. **Management** of the damaged lumbosacral nerve roots is the same as that of the prolapsed intervertebral disk in consultation with an orthopedist.

Paraplegia due to epidural hematoma or abscess (arachnoiditis) following regional anesthesia is extremely rare.

PUERPERAL EMERGENCIES

There are many acute complications that may occur during the puerperium. The majority of the alarming complications, however, arise immediately following delivery, except pulmonary embolism, as a consequence of thromboembolic phenomenon; the late complications are relatively less risky. **The complications are:**

(a) ***Immediate***—(1) **Postpartum hemorrhage,** (2) **Shock**—hypovolemic, endotoxic or idiopathic, (3) **Postpartum eclampsia,** (4) **Pulmonary embolism**—liquor amnii or air, (5) **Inversion**.

(b) ***Early (within one week)***—(1) Acute retention of urine, (2) Urinary tract infection, (3) Puerperal sepsis, (4) Breast engorgement, (5) Mastitis and breast abscess, (6) Pulmonary infection (atelectasis), (7) Anuria following abruptio placentae, mismatched blood transfusion or eclampsia.

(c) ***Delayed***—(1) Secondary postpartum hemorrhage, (2) Thromboembolic manifestation—pulmonary embolism, thrombophlebitis, (3) Psychosis, (4) Postpartum cardiomyopathy, (5) Postpartum hemolytic uremic syndrome (Ch. 39).

PSYCHIATRIC DISORDERS DURING PUERPERIUM

In the first 3 months after delivery, the incidence of mental illness is high. Overall incidence is about 15–20%. Sleep deprivation, hormone elevation near the end of gestation and massive postpartum withdrawal contribute to the risk.

HIGH RISK FACTORS FOR POSTPARTUM MENTAL ILLNESS:

- **Past history**: Psychiatric illness, puerperal psychiatric illness.
- **Family history**: Major psychiatric illness, marital conflict, poor social situation.
- **Present pregnancy**: Young age, cesarean delivery, difficult labor, neonatal complications.
- **Others**: Unmet expectations.

PUERPERAL BLUES

- It is a transient state of mental illness observed 4–5 days after delivery and it lasts for a few days.
- Nearly 50% of the postpartum women suffer from the problem.
- Manifestations are—depression, anxiety, tearfulness, insomnia, helplessness and negative feelings toward the infant.
- No specific metabolic or endocrine abnormalities have been detected. But lowered tryptophan level is observed. It suggests altered neurotransmitter function.
- **Treatment** is reassurance and psychological support by the family members.

POSTPARTUM DEPRESSION

- It is observed in 10–20% of mothers.
- It is more gradual in onset over the first 4–6 months following delivery or abortion.
- Changes in the hypothalamo–pituitary–adrenal axis may be a cause.
- Manifested by loss of energy and appetite, insomnia, social withdrawal, irritability and even suicidal attitude.
- Risk of recurrence is high (50–100%) in subsequent pregnancies.

Treatment: Treatment is started early. Fluoxetine or paroxetine (serotonin reuptake inhibitors) is effective and has fewer side effects. It is safe for breastfeeding also. Estrogen patch has also been used. General supportive measures are essential as in blues. If no prompt response with medication, psychiatric consultation is sought for. The overall prognosis is good.

POSTPARTUM PSYCHOSIS (SCHIZOPHRENIA)

- Observed in about 0.14–0.26% of mothers. Commonly seen in women with past history of psychosis or with a positive family history.
- Onset is relatively sudden usually within 4 days of delivery.
- Manifested by fear, restlessness, confusion followed by hallucinations, delusions and disorientation (usually manic or depressive). Psychotic women may have delusions. Suicidal, infanticidal impulses may be present. In that case temporary separation and nursing supervision are needed.
- Risk of recurrence in the subsequent pregnancy is 20–25% and there is increased risk of psychotic illness outside pregnancy also.

Management: A psychiatrist must be consulted urgently. Hospitalization is needed. Chlorpromazine 150 mg stat and 50–150 mg three times a day is started. Sublingual estradiol (1 mg thrice daily) results in significant improvement. Electroconvulsive therapy is considered if it remains unresponsive or in depressive psychosis. Lithium is indicated in manic depressive psychosis. In that case breastfeeding is contraindicated.

PSYCHOLOGICAL RESPONSE TO PERINATAL DEATHS AND MANAGEMENT

Most perinatal events are joyful. But when a fetal or neonatal death occurs, special attention must be given to the grieving patient and her family. Perinatal grieving may also be due to unexpected hysterectomy, birth of a malformed or critically ill infant. Prolonged separation from a critically ill newborn can also provoke grief reaction. Physician, nurse and attending staff must understand the patient's reaction. The common maternal somatic symptoms are: insomnia, fatigue and sighing respirations, feeling of guilt, hostility and anger.

***Management of perinatal grieving*:** Facilitating the grieving process with consolation, support and sympathy is important. Others are: supporting the couple in seeing or holding or taking photographs of the infant; autopsy requests, planning investigations, follow-up visit and plan for subsequent pregnancy.

KEY POINTS

- **The common causes of puerperal pyrexia** are: (i) Puerperal sepsis, (ii) Urinary tract infection, (iii) Mastitis, (iv) Infection of the cesarean section wound, (v) Pulmonary infection, (vi) Septic thrombophlebitis, (vii) Recrudescence of malaria, tuberculosis or (viii) Unknown.
- **The common causes of puerperal sepsis** are the infections in the genital tract. This includes: (i) Endometritis, (ii) Endomyometritis, or (iii) Episiotomy wound infection.
- **Pathogens** commonly responsible for female genital infections are: (A) Aerobes (Gram-positive—Streptococci and Staphylococci, Gram-negative—*E. coli*, *Klebsiella* or Gram-variable—*Gardnerella*). (B) Anaerobes (Peptostreptococci, *Fusobacterium*, *Clostridium*) and (C) Others (*Mycoplasma*, *Chlamydia*).
- **Common causes of subinvolution are:** (a) Excess enlargement of the uterus (twins), (b) Anemia, (c) Retained bits of tissues, (d) Endometritis (see p. 502).
- **Common breast complications in the puerperium are:** (a) Breast engorgement (b) Cracked and retracted nipple, (c) Mastitis and breast abscess (see p. 506).
- **Puerperal emergencies** are often immediate (third stage complications). There may be some complications that are relatively delayed but acute and alarming. These are—(i) Mastitis and breast abscess (see p. 506), (ii) thromboembolism (see p. 508), (iii) Psychiatric disorders and (iv) Postpartum cardiomyopathy (see p. 511).

Workup for Women with Suspected Pulmonary Embolus

- D-dimer, CXR, VUS, V/Q scan, CTPA are to be done depending upon individual woman's signs and symptoms. Woman with positive observation in VUS, CXR, V/Q scan and CTPA need therapy. Anticoagulation is started in the absence of contraindications.
- **Risk factors for VTE are:** (a) Previous VTE, (b) Heart disease (see p. 510), (c) SLE, (d) Surgical procedure (LSCS), (e) Obesity, (f) Immobility (see p. 508).
- **Thromboprophylaxis** against VTE for women with high risk and intermediate risk factors are: LMWH throughout pregnancy and postpartum 7 days to 6 weeks.

QUESTIONS

Related theory questions (Long and Short), Obstetric Case Discussions, Viva table discussions, Postoperative word round discussions and MCQs are discussed in author's books:

1. **Bedside Clinic and Viva Voce:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

For further reading:

2. **Master pass in Obstetrics and Gynecology:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

Chapter 31

The Term Newborn Infant

DEFINITION: A healthy infant born at term (between 38 weeks and 42 weeks) should have an average birth weight for the country (usually exceeds 2,500 g), cries immediately following birth, establishes independent rhythmic respiration and quickly adapts to the changed environment.

The weight is variable from country to country but usually exceeds 2,500 g. In India, the weight varies between 2.7 kg and 3.1 kg with a mean of 2.9 kg (Fig. 31.1). The length (crown to foot) is 50–52 cm. **The length is a more reliable criterion of gestational age than the weight.** Occipitofrontal circumference measures about 32–37 cm and the biparietal diameter measures about 9.5 cm.

PHYSICAL FEATURES OF THE NEWBORN

The newborn must be examined thoroughly within 24 hours of birth. Before the actual examination, the important maternal and perinatal history should be reviewed (p. 128). **Maternal history** (age, parity, medical disorders, etc.), **Pregnancy problems**—present and past (drugs, IUFD, preeclampsia, IUGR, prematurity), **Labor and Delivery history** (duration, anesthesia, duration of PROM, Apgar score) should be obtained. Assessment of gestational age is done (see Table).

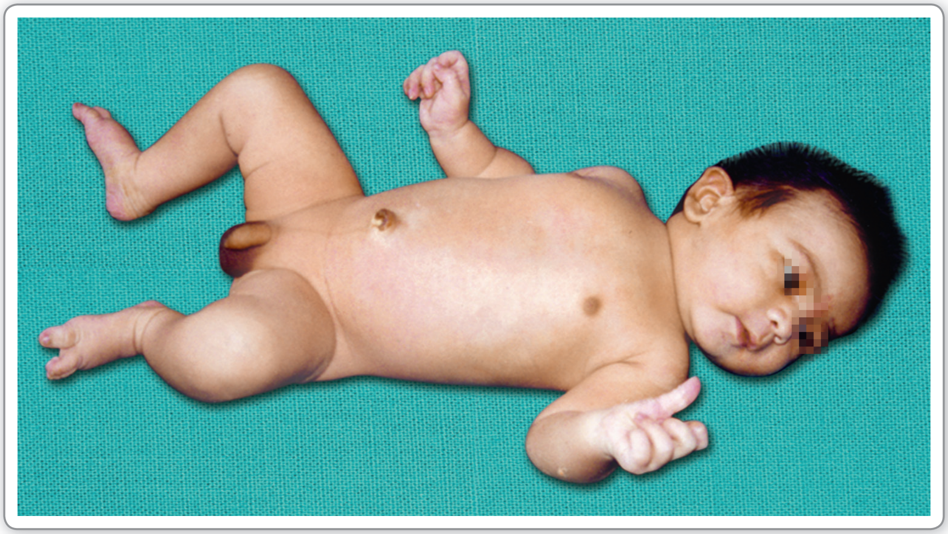


Fig. 31.1: A healthy term baby weighing 3.3 kg

1. **Examination of vital signs:**
   - i. ***Temperature*** is recorded and the site (e.g. rectal, oral or axillary) is mentioned.
   - ii. ***Respiration:*** Normal, 30–60 breaths/min. May need screening with pulse oximetry (>95% and ≤3% difference between right hand and foot).
   - iii. ***Pulse:*** Normal, 100–160 beats per min (bpm) and when asleep it is around 70–80 bpm.
   - iv. ***Blood pressure:*** Normal range 45–60/25–40 mm Hg. BP is directly related to gestational age and birth weight of the infant.
2. **General examination:**

   Skin color: It is the single most important parameter of cardiorespiratory function.
   - a. **Pallor** may be due to anemia, birth asphyxia, or shock.
   - b. **Cyanosis:**
     - *Central cyanosis* (bluish skin, including the tongue and lips) is caused by low oxygen saturation. It may be due to congenital heart or lung disease. Desaturation of hemoglobin should be >3–5 g/dL.
     - *Peripheral cyanosis* (bluish skin with pink lips and tongue) may be due to drugs (nitrates or nitrites) or hereditary. It is often associated with **methemoglobinemia** (hemoglobin oxidizes from ferrous to ferric form)
     - *Acrocyanosis* (bluish hands and feet only) may be normal immediately following birth. It may be due to cold stress
   - c. **Plethora** commonly seen in infants with polycythemia. It may be seen in an overheated or over oxygenated infant. Hematocrit value may be done.
   - d. **Jaundice:** Bilirubin level > 5 mg/dL (details see p. 551).
   - e. **Extensive bruising** may be due to difficult or traumatic delivery.

   Skin rashes:
   - a. **Milia** seen on the nose, cheeks and forehead are due to plugged sweat glands.
   - b. **Mongolian spots** are bluish, often large, commonly seen on the back, buttocks or thighs. Usually present in Blacks and Asians (90%). They disappear by 4 years of age.
   - c. **Erythema toxicum:** These are papular lesions with an erythematous base. Commonly seen after 48 hours of birth. They resolve spontaneously.
   - d. **Diaper rash** usually the skinfolds are involved. It appears as erythematous plaques and the edges are well demarcated. It is a form of irritant contact dermatitis. It may be infected with *Candida albicans.*
3. **Head: Fontanels (see p. 95):**
   - a. **Large fontanels are** associated with hypothyroidism, osteogenesis imperfecta or chromosomal anomalies (Down syndrome). **Bulging fontanel** may be due to increased intracranial pressure, meningitis or hydrocephalus. **Depressed fontanels** are seen with dehydration. **A small fontanel** may be due to hyperthyroidism, microcephaly or craniosynostosis.
   - b. **Caput succedaneum** (see p. 98) should be differentiated from cephalhematoma (see p. 558).
   - c. **Molding** (see p. 97) seen with prolonged labor. Usually molding subsides within 5 days.
   - d. **Cephalhematoma** is due to subperiosteal hemorrhage resulting from a traumatic delivery (see p. 558). It never extends beyond the suture line. X-ray and CT scans should be taken to exclude skull fracture. Hematocrit and bilirubin levels should be estimated. Aspiration of hematoma is rarely needed as they often resolve in 4–6 weeks' time.

e. **Raised intracranial pressure** is diagnosed by the following signs: (i) Bulging anterior fontanel; (ii) Separation of suture lines; (iii) Paralysis of upward gaze; (iv) Prominent veins of the scalp.

Craniosynostosis is the premature closure of one or more of sutures of the skull. On palpation, a bony ridge is felt over the suture line and the cranial bones cannot be moved. X-ray studies of the skull should be done.

4. **Neck:** It is checked for movements, goiter, thyroglossal cysts, sternomastoid hematoma (sternomastoid tumor) or short neck, webbed neck (Turner's syndrome).
5. **Face and Mouth:** Face is looked for hypertelorism (eyes widely separated) or low-set ears (trisomy 9, 18, triploidy) or facial nerve injury. Mouth is checked for clefts (palate, lips), natal teeth, lingual frenulum (tongue tie), macroglossia (Beckwith syndrome) or oral thrush (see p. 567). Thrush is treated with nystatin suspension.
6. **Eyes** are examined for congenital cataract, Brushfield's spots in the iris (Down's syndrome) or subconjunctival hemorrhage (traumatic delivery) and conjunctivitis.
7. **Chest** is examined for any asymmetry (tension **pneumothorax**), tachypnea, grunting, intercostal retractions (respiratory distress), pectus excavatum and the breath sounds. The newborn's breasts may be enlarged (normal 1 cm in diameter) due to maternal estrogen. The white discharge from nipple is common known as "Witch's milk".
8. **Heart** is examined for rate (normal 120–160 bpm), rhythm, the quality of heart sounds and presence of any murmur. **Murmurs** may be associated with VSD, PDA, ASD, transposition of great vessels, tetralogy of Fallot, coarctation of aorta and others. **Fetal echocardiography** at 18–20 weeks gestation can make the antenatal diagnosis in utero. Fetal cardiac intervention **in utero** is a new and promising method of treatment.
9. **Abdomen** is examined for any defect, e.g. omphalocele (see p. 736), hepatomegaly (sepsis), splenomegaly (CMV, rubella infection) or any other mass. A scaphoid abdomen may be due to diaphragmatic hernia.
10. **Umbilicus** is examined for any discharge, redness or infection. A greenish-yellow colored cord suggests meconium staining (fetal distress). *Single umbilical artery* (more in twin births) indicates genetic (trisomy 18) and congenital **anomalies** (40%), and IUGR (see p. 533).
11. **Genitalia** should be examined carefully before gender assignment. **Male** is examined for penis (normal > 2 cm), testes within the scrotum, any hydrocele or hypospadias. Prepuce is normally long and phimosis is present. **Female** is examined for any clitorial enlargement (maternal drug), fused labia with clitorial enlargement (adrenal hyperplasia). Blood stained vaginal discharge may be due to maternal estrogen withdrawal. Normally labia majora cover the labia minora and clitoris.
12. **Anus and Rectum** is checked to rule out imperforation and their position. Meconium should be passed within 48 hours of birth.
13. **Extremities, spine and joints** are examined for syndactyly (fusion of digits), polydactyly, simian crease (Down's syndrome), talipes equinovarus, hip dislocation (Ortolani and Barlow maneuvers).
14. **Nervous system** is examined for any **irritability**, abnormal muscle tone, reflexes, cranial and peripheral nerves (Erb's paralysis see p. 562). Neurological development is dependent on gestational age. The reflexes including *Moro reflex* are present at birth.

Reflex behaviors: (A) **Muscle tone: Hypotonia** (floppiness) or hypertonia (increased resistance) is examined.

(B) **Reflexes:** (1) **Rooting reflex:** Stroke the corner of the cheek with a finger and the infant will turn in that direction and open her mouth; (2) **Glabellar reflex:** To tap gently over the forehead and the eyes will blink; (3) **Grasp reflex (Palmer grasp):** Place a finger in the open palm of the infant's hand and the infant will grasp the finger; (4) **Moro reflex:** The infant is supported from behind the upper back with one hand and then the baby is allowed to drop back ≥1 cm but not on the mattress. The baby will symmetrically abduct, extend the arms and fingers. This is followed by flexion and adduction of the arms. Asymmetry may signify a fractured clavicle, hemiparesis or brachial plexus injury. An absent Moro reflex may signify CNS pathology; (5) **Sucking and swallowing reflexes:** A normal infant starts sucking when something (nipple and the areola) touches the palate. Baby swallows when the mouth is filled with milk.

(C) **Gestational age:** See Table below:

Table 31.1: Assessment of Gestational Age at Birth

Character	< 36 Weeks	37–38 Weeks	> 39 Weeks
Sole creases	1–2 Transverse creases on anterior 1/3rd of sole	Multiple creases on anterior 2/3rd of sole	Entire sole covered with creases
Breast nodule	2 mm	4 mm	7 mm
Scalp hair	Fine, Wooly, Fuzzy	Fine, Wooly, Fuzzy	Coarse, Silky
Ear lobe	No cartilage	Moderate amount of cartilage	Stiff ear lobe, thick cartilage
Testes and scrotum	Testes partially descended, scrotum small and few rugae	—	Testes fully descended, scrotum normal size, prominent rugae

Hematological findings: Blood volume soon after birth is about 80 mL/kg body weight if immediate cord clamping is carried out. RBC—6–8 million/cu mm, Hb%—18–20 g%, WBC—10,000–17,000/cu mm, Platelets —3,50,000/cu mm, Nucleated red cells 500/ cu mm, Sedimentation rate is elevated. Clotting power may be poor because of deficient vitamin K which is necessary for the production of prothrombin from the liver. Reticulocyte count ranges from 3% to 7%. In a healthy term infant, hemoglobin values reaches a nadir of 11 g/dL at 8–12 weeks of birth. This is known as **physiological anemia of infancy.** In preterm infants, the decline (7–9 g/dL) is more at 4–8 weeks.

IMMEDIATE CARE OF THE NEWBORN

- **Care at Birth**
- **Care in Nursery**

CARE AT BIRTH: This has already been described in p. 160.

CARE IN NURSERY:

Admission in Nursery—All healthy newborns are kept in the delivery room with their mother to promote immediate breastfeeding and early bonding. **Common indications** for admission of the newborn in the nursery are: prematurity, respiratory distress, poor perfusion or presence of pallor or cyanosis, malformation and need for O_2 therapy.

Routine Nursery Care—The newborn is examined systematically (see p. 162) and assessment of the gestational age is done.

Infant's weight, fronto-occipital circumference (FOC) and length are recorded. On these basis, the newborn is classified as average for gestational age (AGA), small for gestational age (SGA) or large for gestational age (LGA).

The newborn must be kept under a **neutral thermal condition.** This is defined as the external temperature range where metabolic rate and oxygen consumption are at minimum. The normal skin temperature in the neonate is 36.0–36.5°C (96.8–97.7°F). Normal core (rectal) temperature is 36.5–37.5°C (97.7–99.5°F). Axillary temperature may be 0.5–1.0°C lower.

Mechanisms of heat loss are: (i) Radiation, (ii) Conduction from the infant to the surface in direct contact, (iii) Convection from the infant to the surrounding area, and (iv) Evaporation of water from the skin.

Consequences of excessive heat loss: (i) Compensatory heat production through increase in metabolic rate, (ii) Insufficient oxygen supply → Hypoxia → anaerobic metabolism, (iii) Hypoglycemia, (iv) Metabolic acidosis, (v) Apnea; and (vi) Pulmonary hypertension.

Consequences of hypothermia are: (a) DIC, (b) Pulmonary hemorrhage, (c) Shock, (d) IVH, (e) Increased mortality.

The measures to prevent heat loss are: (i) Place the baby under a preheated (36.5°C) radiant warmer (servo-control) immediately following delivery, (ii) Dry baby immediately after birth, (iii) Cover baby (including the head) with a pre-warm towel, (iv) Put baby close to mother's breast (**Kangaroo method**), (v) Wrap the mother and baby together, and (vi) Commence early breastfeeding.

DAILY OBSERVATION AND CARE

Rooming-in: Soon after birth, if mother is fit, baby is kept in a cot by the bedside of mother. This establishes mother-child relationship. Mother also learns the art of baby care.

Baby bath: Routine bath is delayed until the baby is able to maintain the body temperature and has started breastfeeding. The excess vernix, blood or meconium are wiped off from the skin using sterile moist swabs and then make the skin dry by using a soft towel. The water for baby bath should be at body temperature (> 97.5°F) and a separate bathtub should be earmarked for each baby.

Umbilical cord care: It is kept exposed to air and allowed to dry to promote early detachment. Topical antiseptics or antibiotics such as triple dye or neosporin powder may be applied to reduce bacterial colonization.

Routine medications: A single intramuscular dose of 0.5–1 mg of vitamin K_1 (phytonadione) is given to all newborns within 6 hours of birth. This prevents vitamin K deficient bleeding.

Eyes are kept clean with cotton wool soaked with sterile normal saline as a prophylaxis against ophthalmia neonatorum (chlamydia, gonococcus). Erythromycin ointment (0.5%) bilaterally in the conjunctival sac or tetracycline (1%) ointment may be used.

Immunization and vaccines: Hepatitis B vaccine is given at birth. Other vaccine information is given to the parents (p. 526).

Screening of the newborn: Commonly done screening tests are: (A) Glucose screening and detect hypoglycemia especially for infants of diabetic mothers, SGA and LGA infants; (B) Bilirubin screening; (C) Other metabolic screen depending on need (e.g. galactosemia).

Assessment of vital signs: Respiratory rate, heart rate, axillary temperature are recorded every 6–8 hours in baby's chart. Each urine and stool output is recorded. Most of the newborns pass urine by 24 hours and meconium by 48 hours of life. Daily weights are recorded. Weight loss in excess of 7% is often due to inadequate calorie intake.

Feedings: The frequency, duration and volume of each feed is important for newborn's growth and development. The infant should be put to breast as soon as possible after delivery in the delivery room. Feeding is allowed on demand (demand feeding). Usually it is 8–12 times per day.

Discharge: Each infant is evaluated carefully to decide the optimal time of discharge. Considering the huge number of institutional deliveries in a developing country set up, early discharge of mother and infant may be done to avoid overcrowding in the postnatal ward and in the nursery.

The following infants may be discharged by 48 hours of age: Vaginal delivery, gestational age > 38 weeks, singleton birth, birth weight—AGA, normal vital signs, passed urine and stool, initial immunization done, successful feedings and normal on physical examination.

Follow-up: Follow-up of newborns should be organized depending upon the risks of feeding problems, infections, hyperbilirubinemia or other issues. During follow-up, the newborn is assessed for weight, hydration, infection and for any new problem. Parental education and immunization schedule are discussed (see p. 526).

INFANT GROWTH ASSESSMENT: Serial measurement of weight, length and head circumference allow for evaluation of infant growth.

WEIGHT: There is weight loss of 7–10% in the first week of life. Weight gain generally begins by the second week. Average daily weight gain is 20–30 g/day. The infant should be weighed daily.

LENGTH: Normal weekly length gain is 0.8–1.0 cm for first 8–12 weeks.

HEAD CIRCUMFERENCE: Intrauterine growth is 0.5–0.8 cm/week.

INFANT FEEDING

The rate of growth of the infants during the first 6 months of life is greater and faster than any other period of life. Its weight is doubled by the age of 5 months and tripled by the end of one year. Keeping this in mind, the baby should be nursed adequately (both quantitatively and qualitatively) which allows easy digestion and absorption.

NUTRITIONAL REQUIREMENTS IN THE NEONATE

- **The infant should get sufficient fluid.** Fluid intake should be 150–175 mL/kg body weight per day
- **The infant should get adequate calorie.** A term healthy infant needs 100–110 kcal/kg of body weight per day. Low birth weight infant needs about 105–130 kcal/kg/day. Each 30 mL (1 oz) of breast milk gives 20 calories. Calorie needs are primarily dependent on oxygen consumption
- **The food should have a balanced composition** of protein (2–4 g/kg/day), fat (4–6 g/kg/day), carbohydrate (10–15 g/kg/day), minerals and vitamins and it should be easily digestible.

TYPES OF FEEDING: ♦ **Breastfeeding** ♦ **Artificial feeding**

BREASTFEEDING

The two vital considerations for the infants in tropical countries are breastfeeding and avoidance of infection. Artificial feeding may be required in a very rare situation, but where the mothers have an inadequate knowledge of the technical details of artificial feeding, gastroenteritis and malnutrition of the neonates are inevitable consequences. All the babies, regardless of the type of delivery, should be given **early and exclusive breastfeeding up to 6 months of age. Exclusive breastfeeding** means giving nothing orally other than colostrum and breast milk. Medicines and vitamins are allowed.

Breastfeeding is the "**Gold standard**" for infant feeding. There are several areas of biological superiority of breastfeeding and breast milk over artificial (formula) milk. Obstetricians and midwives should educate the mother during prenatal and postnatal care for the usefulness of breastfeeding.

BABY FRIENDLY HOSPITAL INITIATIVE: Baby Friendly Hospital Initiative with ten steps to successful breastfeeding (WHO/UNICEF 1992: Protecting, Promoting and Supporting breastfeeding). These are: (i) There must be a written breastfeeding policy; (ii) All health care staff must be trained to implement this policy; (iii) All pregnant women must be informed about the benefits of breastfeeding; (iv) Mothers should be helped to initiate breastfeeding within half an hour of birth; (v) Mothers are shown the best way to breastfeed; (vi) Unless medically indicated, the newborn should be given no food or drink other than breast milk; (vii) To practice 'rooming-in' by allowing mothers and babies to remain together 24 hours a day; (viii) To encourage demand breastfeeding; (ix) No artificial teats to babies should be given; and (x) Breastfeeding support groups are established and mothers are referred to them on discharge.

A baby friendly hospital should also provide other preventive health cares, e.g. infant immunization, rehydration salts against diarrheal dehydration and child's growth and development surveillance.

ADVANTAGES OF BREASTFEEDING:

A. **Composition:** Breast milk is an **ideal food** with easy digestion and low osmotic load.
   - **Carbohydrate:** Mainly lactose, stimulates growth of intestinal flora, produces organic acids needed for synthesis of vitamin B
   - **Fat:** Smaller fat globules, better emulsified and digested
   - **Protein:** Rich in lactalbumin and lactoglobulin, less in casein
   - **Minerals:** Low osmotic load (K^+, Ca^{2+}, Na^+, Cl^-), less burden on the kidney.

B. **Protection against infection and deficiency states:**
   1. **Vitamin D** promotes bone growth, protects the baby against rickets
   2. **Leukocytes, lactoperoxidase** prevents growth of infective agents
   3. **Lysozyme, lactoferrin, interferon** protect against infection
   4. **Long-chain omega-3 fatty acids** essential for neurological development
   5. **Immunoglobulins IgA (secretory), IgM, IgG** protect against infection
   6. **Supply of nutrients and vitamins.**

C. **Breast milk** is a readily available food to the newborn at body temperature and without any cost.

D. **Breastfeeding** acts as a natural contraception to the mother (see p. 172, 614).

C. **Additional advantages are:** (i) It has laxative action; (ii) No risk of allergy; (iii) Psychological benefit of mother-child bonding; (iv) Helps involution of the uterus (see p. 173); and (v) Lessens the incidence of sore buttocks, gastrointestinal infection and atopic eczema. The incidence of scurvy and rickets is significantly reduced.

Long-term risks of exclusive artificial (bottle) feeding: (a) Type I diabetes; (b) Sudden infant death; (c) Adult type 2 diabetes; (d) Childhood obesity; (e) Adult obesity; (f) Crohn's disease; (g) Ulcerative colitis; (h) Atopic dermatitis; and (i) Reduced Intelligence Quotient (IQ).

PREPARATIONS FOR BREASTFEEDING: The preparations for breastfeeding should actually be started from the middle of pregnancy. Any abnormality in the nipple, like cracked or depressed nipple should be adequately treated (see p. 507). Massaging the breasts, expression of the colostrum and maintenance of cleanliness should be carried out during the last four weeks of pregnancy.

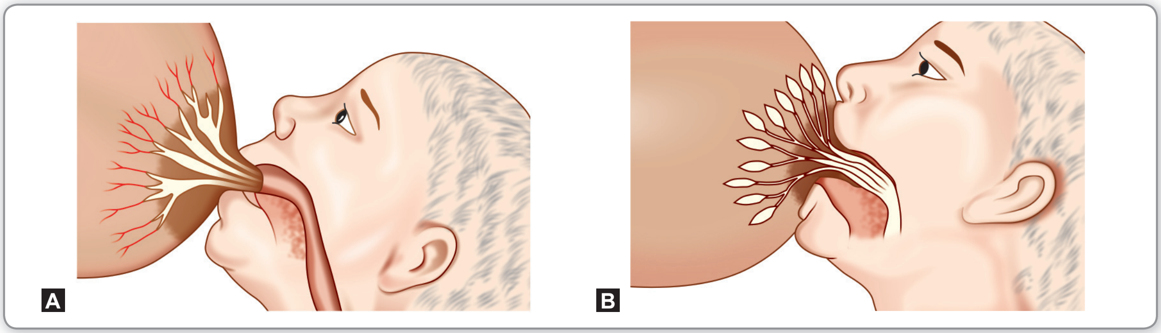
MANAGEMENT OF BREASTFEEDING: The modern practice is to reduce nipple cleansing to a minimum and to wash the breasts once daily. A clean, soft supporting brassiere should be worn. The mother should wash her hands prior to feeding. Mother and the baby should be in a comfortable position during feeding (see Fig. 31.3). Frequent feedings, 8–12 feeds/24 hours are encouraged.

***First feed*—**In the absence of anatomical or medical complications, a healthy baby is put to the breast immediately or at most 1/2–1 hour following normal delivery. Following cesarean delivery a period of 4–6 hours may be sufficient for the mother to feed her baby.

***Milk transfer*—**Milk transfer to infant is a physiological process. It starts with **good latch on.** The nipple is tilted slightly downward using a "C-hold". The milk is extracted by the infant not by negative pressure but by a peristaltic action from the tip of the tongue to the base. The latent period between latch on to milk ejection is about 2 minutes. Nearly 90% of the milk is obtained in the first 5 minutes. **The calorie rich hind milk is obtained at the end part of suckling**. The inflexible artificial nipple resists the undulating motion of infant's tongue and mouth.

Frequency of feeding:

- **Time schedule—**During the first 24 hours, the mother should feed the baby at an interval of 2–3 hours. Gradually, the regularity becomes established at 3–4 hours pattern by the end of first week. Baby should be fed more on demand.



Figs 31.2A and B: Technique of breastfeeding. (A) Poor attachment; (B) Good attachment

- **Demand feeding—**The baby is put to the breast as soon as the baby becomes hungry. There is no restriction of the number of feeds and duration of suckling time.

Duration of feed**—**The initial feeding should last for 5–10 minutes at each breast. This helps to condition the letdown reflex. Thereafter, the time spent is gradually increased. Baby is fed from one breast completely so that baby gets both the **foremilk** and the **hind milk**. Then the baby is put to the other breast if required. **Hind milk** is richer in fat and supplies more calories and satiety to the infant. The next feed should start with the other breast.

Night feed**—**In the initial period, a night feed is required to avoid long interval between feeds of over 5 hours. It not only eliminates excessive filling and hardening of the breasts but also quietens and ensures sound sleep for the baby. However, as the days progress, the baby becomes satisfied with the rhythmic 3–4 hourly feeding.

Amount of food**—**The average requirement of milk is about 60 mL/kg/24 hours on the first day, 100 mL/kg/24 hours on the third day and is increased to 150 mL/kg/24 hours on the 10th day. However, the baby can take as much as he wants.

Technique**—**The mother and the baby should be in a comfortable position. Feeding in the sitting position, the mother holds the baby in an inclined upright position on her lap; the baby's head on her forearm on the same side close to her breasts, the neck is slightly extended. **Good attachment** means the infant's mouth is wide open and chin touches the breast (Figs 31.2A and B). The mother should guide **the nipple and areola into the baby's mouth** for effective milk transfer. **The milk transfer to the infant begins with good latch on and by a peristaltic action of the tip of the tongue to the base.** The proper position for milk transfer is chest to chest contact of the infant and mother. The infant's ear, shoulder and hip are in one line (see Fig. 31.3). Baby sucks the areola (lactiferous sinuses) and the nipple holding between the tongue and the palate. **Feeding in lateral position** following cesarean delivery or with painful perineum is carried out by placing the baby along her side between the trunk and the arm.

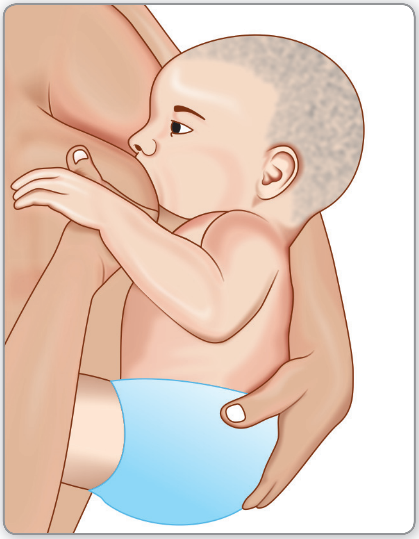


Fig. 31.3: Technique of breastfeeding—proper way of holding the baby

The failure to develop good milk transfer is the major cause of lactation failure and breast pain. Inhibition of let down reflex and failure to empty breasts leads to ductal distortion, parenchymal swelling and breast engorgement. Normally breast is washed with clean water and allowed to air dry.

Nipple confusion: If the baby is fed with an artificial nipple of a bottle, he cannot suck the mother's nipple effectively due to nipple confusion. In case of artificial nipple, he has to press the nipple only. But in case of mother's nipple, he has to press the areola and suck the nipple. **The baby is confused between these two procedures and lactation failure develops.** So artificial nipple is strictly discouraged. If at all needed, the artificial feed is given by spoon or *jhinuk.*

Breaking the wind (Burping)—All babies swallow varied amount of air during sucking. To breakup the wind, the baby should be held upright against the chest and the back is gently patted till the baby belches out the air. It is better to breakup the wind in the middle of sucking so as to make the stomach empty, enabling the baby to take more food and at the end of sucking to prevent hiccough and abdominal colic.

FACTORS FOR SUCCESSFUL LACTATION: (i) Positioning (Fig. 31.3), (ii) Attachment to breast (Fig. 31.2), (iii) Nursing technique (to avoid breast pain, nipple trauma, incomplete emptying), and (iv) A rotation of positions is helpful to reduce focal pressure on the nipple and to ensure complete emptying. To break the suction, a finger is inserted between the baby's lips and the breast. Otherwise it can injure nipple by forceful disengagement.

DIFFICULTIES IN BREASTFEEDING AND THE MANAGEMENT: At times, breastfeeding poses some problems and if it is not promptly detected and rectified, it may lead to adverse consequences.

The causes may be classified as those: ♦ **Due to mother** ♦ **Due to infant**

Due to mother:

- **Reluctance or dislike to breastfeeding**—careful listening to mother and intelligent counseling can solve the problem.
- **Infant's attachment to breast (Fig. 31.2)**—when poor, it leads to quick shallow sucks instead of slow and deep. Areola remains outside the lips. This causes nipple pain. Skilled support from health care provider can improve the technique of breastfeeding. Prelacteal feeds (e.g. honey, milk) inhibit lactation process and should be avoided.
- **Anxiety and Stress,** previous history of failed lactation or elderly primipara—the mother fails to relax during feeding and as such, the baby refuses to suck. Reassurance and practical support is helpful.
- **Following operative delivery** such as cesarean section or following prolonged and exhaustive labor often there is a delay. So mother should be helped to feed the baby in a comfortable position as early as possible.
- **Milk secretion is inadequate**—unrestricted feeding, well positioned infant, practical and emotional support to mother—all are important. Dopamine antagonist (metoclopramide) may be useful.
- **Breast ailments** such as engorgement of breast, cracked nipple, depressed nipple and mastitis (see p. 507) need treatment. Previous breast surgery, circumareolar incision have unsuccessful breastfeeding. Loss of breast sensation may be the cause.

Due to infant:

- **Low birth weight baby—**The baby is too small or feeble to suck (see p. 531).
- **Temporary illness** such as respiratory tract infection, nasal obstruction due to congestion, lethargy due to jaundice and oral thrush. All these conditions lead to imperfect suckling and is managed appropriately (see p. 567).
- **Overdistension of the stomach with swallowed air**—The problem can be overcome by breaking the wind of the baby several times during feeding.
- **Congenital malformation** such as cleft palate needs surgical correction.

CONTRAINDICATIONS OF BREASTFEEDING (Table 31.2): Contraindications are very few (Table 31.2). **In cases of temporary contraindications, the baby should be put to the breasts as soon as the condition permits. HIV positive mothers** are counseled as regard the risks and benefits (see p. 353). She is helped to make an informed choice.

Table 31.2: Contraindications of Breastfeeding

	Temporary	Permanent
Maternal	1. Acute puerperal illness	1. Chronic medical illness such as decompensated organic heart lesion, active untreated pulmonary tuberculosis
	2. Acute breast complications such as cracked nipples, mastitis or breast abscess	2. Puerperal psychosis
	3. Herpes simplex lesion of the breast	3. Mother having high doses of antiepileptic, antithyroid, antipsychotic or anticancer drugs
Neonatal	1. Very low birth weight baby	1. Severe degree of cleft palate
	2. Asphyxia and intracranial stress	2. Galactosemia
	3. Acute illness	

DRUGS AND BREASTFEEDING: Most drugs taken by the mother appear in the breast milk. Fortunately drug level in the breastfed infant ranges from 0.001% to 5% of the therapeutic doses. The infant tolerates the drug without any toxicity. Very few drugs are absolutely contraindicated. These are: anticancer drugs, chloramphenicol, radioactive materials, phenylbutazone and atropine.

MATERNAL NUTRITION DURING LACTATION: A healthy mother while breastfeeding will produce about 500–900 mL breast milk per day. This will give her baby about 75 kcal/dL. **This requires additional 750 kcal/day for the mother.** This amount is either to be supplemented through her diet or is made up from her body stores. A store of 5 kg of fat throughout pregnancy is adequate to make up the nutritional deficit.

The daily allowances of nutrients recommended for a lactating woman is given in Table 10.1. There is additional need (increased by 50%) of folic acid, iron, calcium and protein during pregnancy. Mother should drink at least 1 extra liter of fluid per day to make up the fluid loss through milk. Bone mineral density decreases in the breastfed women and it returns to normal after 12 months of stoppage of breastfeeding.

ASSESSMENT OF WELL-BEING OF THE INFANT: **Whether the baby with the feeding schedule is progressing normally is evidenced by:** (1) General condition—The baby is happy, sleeps between feeds and at night, does not vomit and passes urine at least six times in 24 hours; (2) Good vigor which is manifested by movements of the limbs and cry; (3) Infant has stopped losing weight; (4) Has yellow seedy stools and no more meconium stools; and (5) Expected level of weight curve.

UNDERFEEDING: It is commonly seen in artificially–fed babies. **The features are:** (1) Failure of the infant to gain weight as per schedule, evidenced from the weight curve; (2) The infant appears dissatisfied with the feeds evidenced by cry in between feeds and at night disturbing the sleep; (3) The baby has constipation; (4) The urinary output (normally> 6 times) becomes scanty and high colored; and (5) Test feeding is the only reliable method of diagnosis (vide infra).

Management—The deficient amount of milk should be substituted by artificial milk. The required deficit of 24 hours as calculated from test feeding is to be divided by the number of feeds to be given in 24 hours. The amount of deficit for each feed, so calculated, should be given after each feed. As soon as sufficient milk comes to the breast, the supplementary feed is withdrawn.

CARE OF THE BREASTS: Daily washing of the breasts with clean water is essential. The nipple should be cleaned with clean water before and after each feed. Brassieres are to be worn for support and comfort.

FEEDING DIFFICULTIES DUE TO NIPPLE ABNORMALITIES

Breast engorgement usually occurs on day 3–5 postpartum. There is copious milk production. Breasts are swollen and hard. There is difficulty to latch on for the infant. **Treatment options are:** (i) Gentle hand expression of milk to make the breasts soft so that the infant can latch on; (ii) Application of moist heat and cold compress to relieve edema; (iii) Gentle breast massage during feeding or milk expression; and (iv) Pain relief and to reduce inflammation (Ibuprofen).

Long nipples may cause poor feeding due to improper latch on to the nipple without the areola. Mother has to help the baby to draw the areola also.

Short nipples usually cause no problem. Mother is reassured.

Inverted and flat nipples attachment to the breasts is possible and babies are able to feed adequately. In difficult cases, lactation is initiated by expression. Baby is then attached to breast as breast tissue become soft and protractile gradually. It can be corrected by suction with a syringe or breast pump.

Expression of breast milk or artificial removal of breast milk is not generally needed where breastfeeding is normal. **The indications of expressing breast milk are:** (i) Where the baby is separated from the mother due to prematurity or illness; (ii) Where there are difficulties in breastfeeding as in attaching the baby to the breast (see p. 522), e.g. cleft palate; (iii) When the mother is separated from the baby because of work; and (iv) Colostrum should always be expressed and given to the babies if they cannot suck properly.

Methods of milk expression: (a) *Manual expression* is advantageous over the mechanical pumping. It increases the level of prolactin that helps to maintain lactation for longer period. It can be practiced anywhere and costs nothing. (b) *Breast pumps* may be electrical or manually controlled.

Donor Breast Milk: Historically, it has been used for centuries. Currently its use is limited. Transmission of infection (HIV, CMV, Hepatitis B, TB) is the concern for its safety. If the donor breast milk or milk banks are used, donor screening, pasteurization of milk and parental counseling are recommended.

Breast milk can be stored frozen at – 20°C for up to 6 months, refrigerated at 4°C for 24 hours and at room temperature for 4 hours. Fresh, unrefrigerated milk can be used within 4 hours of expression.

METHODS OF ESTABLISHMENT OF LACTATION: The following methods may be employed with varying success to establish lactation after it has been temporarily withheld.

For the baby: (1) To discontinue bottle feedings; (2) To put the baby to the breast at frequent intervals; (3) Baby should suck in a well-attached manner.

For the mothers: (1) To encourage plenty of fluid (1 L extra) and milk intake; (2) Drugs like metoclopramide or oxytocin (nasal spray) are of help (see p. 522).

ARTIFICIAL FEEDING

When the infant is fed by any preparation other than human milk or drug or vitamin, it is called artificial feeding. As artificial feeding is commonly accomplished by using a bottle, it is often called as bottle feeding. But an artificial feeding can be given without a bottle.

Indications

- Contraindications of breastfeeding either temporary or permanent (mentioned earlier)
- Changing lifestyle of women or pressurized under changed socioeconomic conditions (expressed breast milk may be an alternative).

FOOD USED: There is no perfect substitute for breast milk. Bottle fed infants are at much higher risk of diarrhea. Breastfeeding is continued even if the baby is unwell. In general, boiled diluted cow milk, various dried milk formulas are commonly used as artificial feeds. In some countries, goat milk or buffalo milk is used.

Composition: The principal compositions of the breast milk and the cow milk are given in the Table 31.3.

Table 31.3: Composition of Human and Cow milk

	Lactose (g/100 mL)	Fat (g/100 mL)	Protein (g/100 mL)	Sodium (mmol/L)	Water	Calories (kcal/100 mL)
Human	7	3.5	1.2	7	89	75
Cow milk	4.5	3.5	3.4	22	88	67

Qualitative differences between human and cow milk: The **sugar** in both is lactose. Breast milk is sweeter due to its **high lactose concentration.** The **fat globules** in cow milk are coarser and hence difficult to digest. The caseinogen (protein) in cow milk causes indigestion. **Sodium content** in cow milk is about four times higher.

Humanization of cow milk: It is indeed impossible to change the composition of the cow milk to that of human milk, no matter how the amounts of protein, fat, carbohydrate and minerals are altered. As such, so called humanization of the cow milk is an inappropriate usage.

- **Quantitative changes in the constituents can be done by dilution followed by addition.** One part of milk is added to one part of water. To bring about readjustment, about 4% of sugar and 2% of fat are to be added to the diluted cow milk. The sugar can be added as cane sugar or glucose in the proportion of a quarter of teaspoonful to each ounce of milk. The fat is added as cream (30–60%). However, in the tropical countries, addition of cream may be omitted.
- **The qualitative alteration is principally directed to change in the caseinogen to make it easily digestible by boiling.**

Sterilization: Sterilization of the milk should be done by boiling followed by rapid cooling or pasteurized by heating to 160°F (73°C) for 20 minutes followed by rapid cooling.

Container—Babies may be fed either by spoon from the bowl (*katori*) or by feeding bottle. It is easy to clean the former. The feeding bottle and the teat should be cleaned prior to and after each feeding. The rubber teat and the bottle should be boiled after each feeding. **Spoon feeding is always preferred to bottle feeding to avoid nipple confusion.**

PRINCIPLES TO FOLLOW IN BOTTLE FEEDING

♦ **The baby is to be held** in comfortable position during feeding. ♦ **The hole of the teat** should be of such size that 20–30 drops of milk are suckled by the baby per minute. ♦ **Burping of the baby should be done in the middle and at the end of each feed.** ♦ Not more than 20 minutes should be spent for each feed. ♦ **All utensils** including the bottle and teat are to be cleaned before and after each feed and to be sterilized by boiling.

SUCCESSFUL FEEDING: The most satisfactory guide to successful feeding is the regular weight gain of the baby after 10 days which should be at the rate of 25–30 grams per day up to 3 months. If the baby fails to attain the weight gain evidenced by weekly weighing or appears unsatisfied, the feeding is to be increased until the baby gains weight. Figure 31.4 shows the normal weight chart

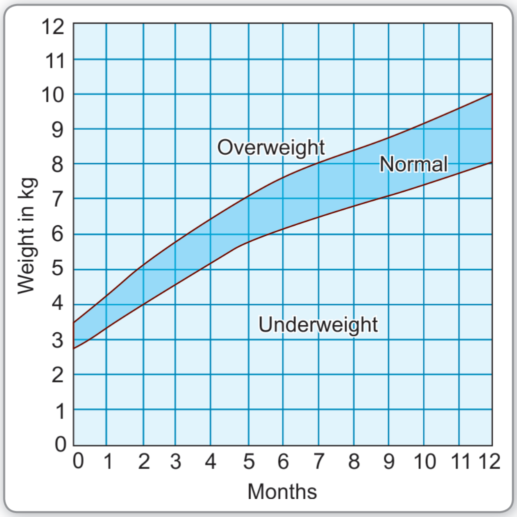


Fig. 31.4 : Normal Weight Chart

WEANING: It is the process during which the baby gets accustomed to food other than its mother's milk. **This period extends from 6th month to 1 year.** The infant requires—110–125 calories/kg body weight per day and its fluid requirement is about 150–175 mL/kg body weight per day. During the period of 3–4 months, the baby may weigh as much as 5–5.5 kg and as such, its demand is more. The breast milk cannot supply the necessary baby's need and as such additional foods are required by 6 months of age. Semi-solid foods such as rice, dal, boiled fish, egg are gradually incorporated in the tropical countries. These also prevent the baby from becoming anemic. Breastfeeding supports the development of neurological and immunological system up to 4–6 years of age. **The dangers of the weaning period are:** (a) Nutritional disturbances, (b) Weaning diarrhea due to altered composition of the food or contaminated with pathogens, and (c) Psychological trauma to the baby when weaning is abrupt.

CHILDHOOD IMMUNIZATION PROGRAM

Vaccine Schedule as Recommended by Indian Academy of Pediatrics (IAP – 2012)

At Birth	BCG, OPV0, Hep B1
6 weeks	DTwP1/DTaP1, IPV1, Hib1, Hep B2, RV1, PCV1
10 weeks	DTwP2/DTaP2, IPV2, Hib2, RV2, PCV2
14 weeks	DTwP3/DTaP3, IPV3, Hib3, RV3, PCV3
6 months	OPV1, Hep B3
9 months	OPV2, Measles
12 Months	Hep A1
15 Months	MMR1, Varicella 1, PCV booster
16–18 Months	DTwP B1/DTaP B1, IPV B1, Hib B1

BCG	Bacillus Calmette–Guérin
Hep B	Hepatitis B
DTaP	Diphtheria, Tetanus, Acellular Pertussis
Hib	*Haemophilus influenzae* type B
MMR	Measles, Mumps, Rubella
TdaP	Tetanus, low dose Diphtheria, low dose Acellular Pertussis
OPV	Oral Poliovirus Vaccine
DTwP	Diphtheria, Tetanus, Whole Cell Pertussis
IPV	Injectable Polio Vaccine
B_1	Booster 1
B_2	Booster 2
Td	Tetanus, low dose Diphtheria
PCV	Pneumococcal conjugate vaccine
RV	Rotavirus vaccine

QUESTIONS

1. Discuss in brief the immediate care of a newborn? (p. 517)

Write Short Notes on:

A. Assessment of gestational age of a newborn (infant at birth) (p. 517)
B. Baby friendly Hospital Initiative (p. 519)
C. Advantages of breastfeeding (p. 520)
D. Immunization schedule of the infant (p. 526)

Chapter 32

Low Birth Weight Baby

Previously, the birth weight of ≤ 2500 g was taken as the index of prematurity without taking any consideration of the gestational period or any other factors. But infants born at term or post-term may weigh ≤ 2500 g and occasionally a baby of diabetic mother may weigh much more than 2500 g even before 37 weeks. Therefore, survival outcome of an infant depends both on the gestational age as well as on the birth weight. Gestational age and birth weight are related by the following terms:

(a) **Small for Gestational Age (SGA):** Birth weight less than 10th percentile for gestational age (b) **Appropriate for Gestational Age (AGA):** Birth weight lies between the 10th and 90th percentiles for gestational age (c) **Large for Gestational Age (LGA):** Infant's birth weight above the 90th percentile for gestational age (Fig. 32.1).

- **Low birth weight (LBW) infant is defined as one whose birth weight is less than 2500 g irrespective of the gestational age**. *Very-low birth weight (VLBW)* infants weigh 1500 g or less and *extremely-low birth weight (ELBW)* infants weigh 1000 g or less (WHO).
- **Preterm**—Preterm Birth (PTB) is defined as one when birth occurs before completion of 37 menstrual weeks of gestation regardless of birth weight. The growth potential may be normal and appropriate for the gestational period (10 th to 90th percentile).
- **Small for gestational age (SGA)**—About 70% of infants with a birth weight below the 10th percentile are found normally grown. **They are constitutionally small and not at any increased risk for adverse outcome**. They present at the end of the normal spectrum for growth. The remaining 30% are truly growth restricted. The neonates are at

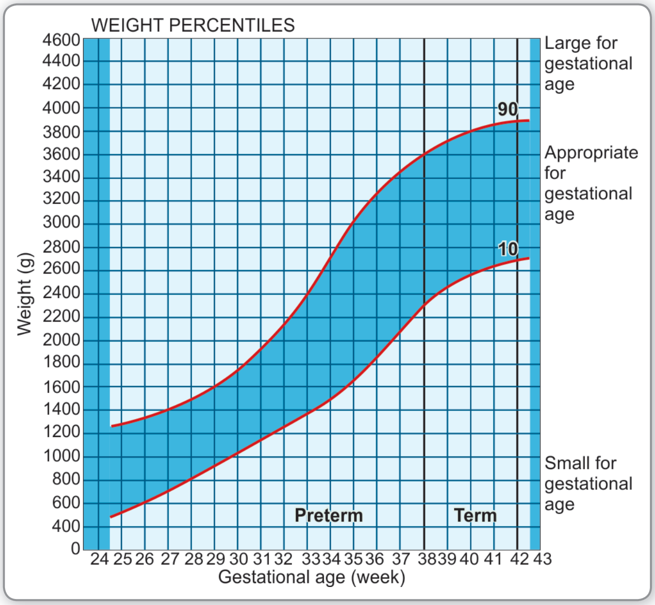


Fig. 32.1: Graph showing—correlation of birth weight and gestational age in percentile

increased risk for perinatal morbidity and mortality. The percentile cut off values to define IUGR is a matter of debate.

The identification of these two distinct clinical entities is important from both prognostic and management points of view.

INCIDENCE: The incidence of low birth weight is generally highest in those countries where the mean birth weight is low and as such varies from about 5–40% of live births. In India, about a third of the infants weigh less than 2500 g. **The factors influencing the low birth weight of the baby**, apart from the preterm birth period, are socioeconomic status, nutritional and intrauterine environment. Ethnic background and genetic control are also important. Thus, **it is logical to correlate birth weight and gestational age with risks of neonatal morbidity and mortality of the individual countries or population groups**.

PRETERM BABY

(Syn. Prematurity, premature baby)

DEFINITION: **A baby born before 37 completed weeks of gestation calculating from the first day of last menstrual period is arbitrarily defined as preterm baby**. Babies born before 37 completed weeks usually weigh 2500 g or less. However, in less than 5%, the babies may weigh more than 2500 g even when born before 37 completed weeks. Preterm baby's weight corresponds to average weight (above 10th percentile) for its gestational age.

INCIDENCE: **Preterm baby constitutes two-thirds of low birth weight babies.** The incidence of low birth weight baby is about 30–40% in the developing countries, as such the incidence of preterm baby is about 20–25%. In affluent societies and in the developed countries, the incidence of the former is less than 10%.

ETIOLOGY: This has been discussed in preterm labor (Ch. 22).

MANIFESTATIONS OF PREMATURITY: The clinical manifestations differ with the degree of prematurity.

***Anatomical*:** The weight is 2500 g or less and the length is usually less than 44 cm. The head and abdomen are relatively large; the skull bones are soft with wide sutures and posterior fontanel. **The head circumference disproportionately exceeds that of the chest.** (Normally, the head circumference is greater than the chest circumference at birth and the difference is about 1.5 cm). Pinnae of ears are soft and flat. The eyes are kept closed (Fig. 32.2).

The skin is thin, red and shiny, due to lack of subcutaneous fat and covered by plentiful lanugo and vernix caseosa. Muscle tone is poor. **Plantar deep creases are not visible before 34 weeks.** The testicles are undescended; the labia minora are exposed because the labia majora are not in contact. There is a tendency of herniation. The nails are not grown right up to the finger tips.

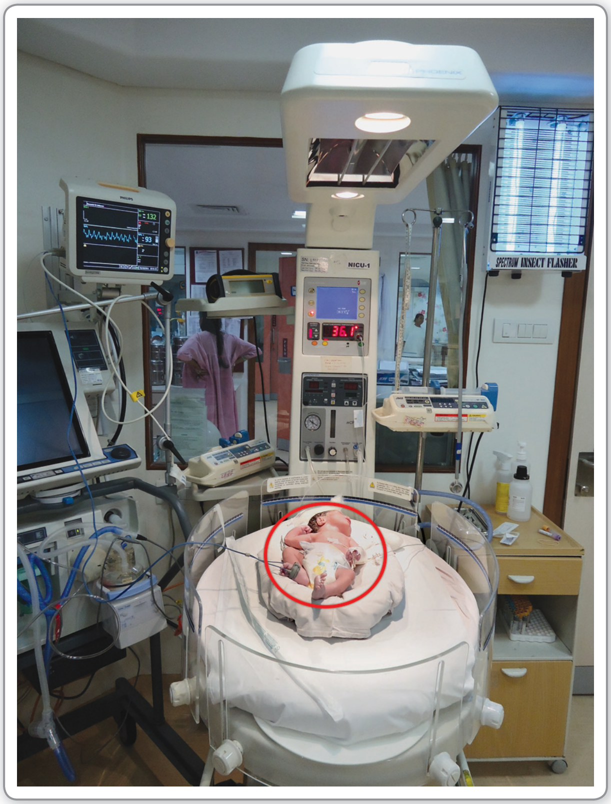


Fig. 32.2: Neonatal intensive care unit management of a preterm newborn weighing 1.1 kg. *Courtesy*: Neonatal Care Unit—NRS Medical College and Hospital, Kolkata

COMPLICATIONS OF A PRETERM NEONATE

Preterm infants are at risk of many complications due to immaturity of various organs and also for the cause of preterm birth. Late preterm infants (born between 34 and 37 weeks) though they appear equivalent to term infants, they have some short-term and long-term (behavioral and learning) difficulties.

- ***Asphyxia***—The babies are likely to be asphyxiated because of anatomical and functional immaturity. Even minor degree of anoxia may produce subserosal hemorrhages especially in the heart, lungs and liver. In addition, it may produce intense congestion of the choroid plexus leading to intraventricular hemorrhage (IVH).
- ***Hypothermia***—A low birth weight baby has reduced subcutaneous as well as brown fat and increased surface area. Very often the newborn fails to maintain the thermoneutral range of temperature (see p. 517).
- ***Pulmonary syndrome (23%)***—**This includes:** (a) Pulmonary edema (b) Intra-alveolar hemorrhage (c) Idiopathic respiratory distress syndrome (RDS) (d) Bronchopulmonary dysplasia. The first two are the effects of hypoxia; RDS is one of the major causes of death in preterm babies born before 34 weeks. The deficient lung surfactant is the principal factor responsible for pulmonary atelectasis leading to hypoxia and acidosis (details see p. 549). Surfactant therapy is effective in reducing RDS (see p. 549).
- ***Cerebral hemorrhage***—The causes are: (a) Soft skull bones allow dangerous degree of moulding leading to subdural or subarachnoid hemorrhage (b) Fragile subependymal capillaries cannot withstand minor degree of hypoxia leading to intraventricular hemorrhage (c) Associated hypoprothombinemia.
- ***Fetal shock***—Apart from the shock sustained during delivery, it may appear following improper resuscitative manipulation during the first day or two.
- ***Hypoglycemia*** (blood glucose < 40 mg/dL) is observed in about 15% of infants due to lack of glycogen stores in the liver. Cold stress, hyperinsulinemia and poor feeding, are the causes.
- ***Heart failure***—It may be precipitated by asphyxia with rapid development of pulmonary edema which in turn impairs pulmonary aeration. There may be patent ductus arteriosus.
- ***Oliguria, anuria***—as the immature kidneys are unable to handle water, solute and acid loads.
- ***Infection***—Protective passive immunity is usually obtained from the mother during the later months of pregnancy. As the transfer of protective immunoglobulins from the mother to a preterm baby is less, the incidence of infection is increased by 3–10 folds. **Both the humoral and cellular immune response is poor. The common types of infection are bronchopneumonia, meningitis and necrotizing enterocolitis**. Respiratory syncytial virus (RSV) infection is common to cause RSV bronchiolitis.
- ***Jaundice***—Because of hepatic immaturity, the bilirubin produced by the excessive hemolysis cannot be conjugated adequately for excretion as bile, leading to rise in unconjugated bilirubin which is responsible for exaggerated physiological jaundice (see p. 551).
- ***Patent Ductus Arteriosus (PDA)***—Persistant PDA is inversely related to gestational age. Up to 30% of PDA close spontaneously. Overhydration should be avoided.
- ***Dehydration and acidemia*** due to immature renal function may occur abruptly.
- ***Anemia***—Lack of stored iron, hypofunction of the bone marrow and excessive hemolysis all contribute to anemia.
- ***Apnea and Sudden Infant Death Syndrome (SIDS)*** is due to immaturity of the autonomic nervous system. The risks of bradycardia, apnea and SIDS are increased.

- ***Retinopathy of prematurity*** is a multifactorial disorder of the retina caused by abnormal neovascularization. It is an important cause of blindness for the children under 6 years. The cause is mostly related to the liberal administration of high concentration of oxygen above 40% for a prolonged period (1–2 days) following birth. Many other factors like extreme prematurity, hypoxia, lactic acidosis, vitamin E deficiency and bright light have been implicated. **The blindness is due to the formation of an opaque membrane behind the lens.**
- ***Length of stay***—Increased length of hospital stay especially for the neonates who are early preterm (<34 weeks).

PROGNOSIS: The chance of survival is directly related to the birth weight. **But vigor at birth is more important than birth weight.** A baby weighing more than 1500 g is most likely (95%) to survive. With intensive neonatal care the survival rate of the baby weighing 751–1000 g is to the extent of 80%. With gestational age < 23 weeks, mortality is > 97%. **The deaths are due to complications already mentioned and increased incidence of congenital malformations.** Most of the deaths (two-thirds) occur within 48 hours.

LONG-TERM PROGNOSIS: Major handicaps (cerebral palsy), hearing loss, chronic lung disease and poor growth are observed. Infants <2500 g are more likely to suffer attention deficit hyperactivity disorder (ADHD).

MANAGEMENT

- **Prevention of prematurity**
- **Management of preterm labor**
- **Care of preterm neonate**

The prevention of prematurity and the management of preterm labor has been discussed in page 366.

CARE OF A PRETERM NEONATE

IMMEDIATE MANAGEMENT FOLLOWING BIRTH

- **The cord is to be clamped quickly** to prevent hypervolemia and development of hyperbilirubinemia.
- **The cord length is kept long** (about 10–12 cm) in case exchange transfusion is required (see p. 396).
- **The air passage should be cleared of mucus** promptly and gently using a mucus sucker.
- **Adequate oxygenation** through mask or nasal catheter in concentration not exceeding 35%.
- **The baby should be wrapped including head in a sterile warm towel (normal temperature 36.5–37.5°C). Hypothermia and its sequelae:** Hypoxia → Hypoglycemia → Anaerobic metabolism → Metabolic acidosis (see p. 517).
- **Aqueous solution of vitamin K** 1 mg is to be injected intramuscularly to prevent hemorrhagic manifestations.

INTENSIVE CARE PROTOCOL: Preterm babies are functionally immature and "special care" is needed for their survival. **Those requiring "special care" are judged by:** (i) Inability to suckle the breast and to swallow. (ii) Incapacity to regulate the temperature within limited range from 96°–99°F (35.6°–37.2°C). (iii) Inability to control the cardiorespiratory function without cyanotic attacks.

The principles that are to be taken for the babies requiring special care are:

- To maintain a relatively stable thermoneutral condition—keep delivery room warm, dry and then wrap the baby with a warm towel, keep the baby with mother—skin-to-skin contact.
- Adequate humidification to counter balance increased insensible water loss.

— Oxygen therapy and adequate ventilation.

— To prevent infection.

— To maintain nutrition and adequate nursing care.

***To maintain body temperature*:** As the premature babies are extremely thermolabile, they can easily develop hyperpyrexia or hypothermia. The axillary temperature should be between 36.0°C and 36.5°C.

The ELBW babies are best placed in prewarmed double-walled incubators where temperature and humidity can be better stabilized. Alternatively, the baby could be managed under radiant warmer with protective plastic covers. The babies are to be placed naked. The skin temperature should be maintained at 36–36.5°C with surrounding humidity 80%.

Slow warming should be done for infants who become hypothermic.

Fluid Electrolytes: These infants need increased fluid replacement because of immature renal function and high insensible water loss. IV fluid therapy is needed and 50–70 mL/kg/day is given when the infant is in an incubator. Serum electrolytes should be tested at 12 hourly intervals.

***Respiratory support*:** To tide over the initial cyanotic phase, measures are taken to clear the air passage and to administer oxygen. The baby is placed in the incubator with oxygen running, alternatively baby's head is kept in an oxygen head box for prolonged oxygen therapy. Some of the neonates may initially require endotracheal intubation and mechanical ventilation. Others may need CPAP or high flow nasal cannula (HFNC). Ventilatory status is monitored by blood gas sampling at regular intervals. Continuous oxygen monitoring is done by pulse oximeter. Desirable level of arterial blood gas values should be (i) PaO_2 55–65 mm Hg (ii) $PaCO_2$ 35–45 mm Hg and (iii) pH 7.35–7.45 and pulse oximeter reading should be 90–92% oxygen saturation. **Surfactant** replacement therapy is indicated in HMD (see p. 549).

Hyperbilirubinemia: Serum bilirubin should be maintained <10 mg/dL. Infant may need phototherapy or exchange transfusion.

***Infection*:** **The main sites of infection** are respiratory tract, gastrointestinal tract, skin and the umbilicus. The poor defensive power of the neonates along with low leukocyte count and poor phagocytic activity make the baby more vulnerable to infection. **Every precaution should be taken to prevent or minimize infection**.

Prophylactic antibiotic therapy is to be given when the babies are born following premature rupture of the membranes. The common antibiotics used are Ampicillin 100 mg/kg per day and Amikacin 10 mg–15 mg/kg per day to be given intravenously in two divided doses for 5–7 days (see p. 564). GBS is an important pathogen.

***Nutrition*:** Preterm infants are often unable to suck and swallow. Enteral feeding may be possible depending on gestation age and vigor. Babies may require gavage feeding or parenteral nutrition. **Human milk is the first choice of nutrition for all low birth weight babies**. Colostrum, foremilk, hindmilk and preterm milk (see p. 172, 521) help faster growth of the baby.

Commencement—Early feeding within 1/2 –1 hour of birth is now widely recommended. It eliminates hypoglycemia, lowers serum bilirubin and neurological sequelae.

Intervals—Depending upon the birth weight, the interval of feeding ranges from hourly in extreme prematurity to 3 hourly feeds in babies born after 36 weeks.

Methods—The methods used depend on the size and vigor of the infant and his ability to suck and swallow. Thus, while a comparatively bigger baby with vigor can be put to the breast right from the beginning, **the smaller one should be fed by any of the following methods**.

- **Tube (Gavage)**
- **Pipette, dropper, katori and spoon**
- **Bottle**
- **Intravenous**

General guidelines are to start intravenous fluids to a **baby weighing < 1200 g (< 30 weeks)** and gradually to initiate tube (gavage) feeding after 1–3 days and spoon after 2–4 weeks and thereafter breastfeeding after some more time. Baby weighing 1200–1800 g (30–34 weeks) may be started with tube feeding and gradually to move on to spoon and breastfeeding. Whereas **babies weighing >1800 g (>34 weeks)** generally have no difficulty to start with breastfeeding.

- *Tube or Gavage*: A fine polythene tube of about 0.5 mm internal diameter is used. It should be passed through the nose down to esophagus. Expressed breast milk is started with a small volume and is gradually built up. It may be safely continued for about 7 days. Calculated amount of fluid is delivered with a syringe by gravitation (gavage feeding).
- *Pipette, dropper, katori and spoon*: This is used where the baby can swallow but fails to suck.
- *Bottle*—It is used when the baby can suck and swallow but cannot manage to express the milk out from the breast.
- *Intravenous fluid therapy*: Neonates within the incubator or under radiant heaters have 10% increased fluid requirement to counter balance the increased insensible water loss.

Fluid requirement varies from 60–80 mL/kg/day of 10% dextrose water or breast milk 10-20 ml/Kg/day on first day and to increase by 15 mL/kg/day. Amount should be 10% more, if phototherapy is used. Monitoring of fluid is done by measuring body weight, urine output, its specific gravity and serum sodium level.

Position—The baby, when fed in a cot, should be placed on right side with the head raised a little to prevent regurgitation.

Nature of food: Undiluted breast milk expressed from the mother or pooled (donor breast milk) is ideal. Breast milk (maternal or donor pooled) can promote gut development, villous growth, digestive enzyme secretion and gut motility. This approach is called **trophic feeding**. Incidence of infection, NEC, retinopathy of prematurity is decreased when breast milk is used. Mother should be encouraged to pump their breasts regularly. Alternatively, premature formulas can be used.

Calorie requirement—It is a paradox that the premature infants require more calories than their mature counterpart because of relatively greater loss of heat from the body surface. The calorie intake of 60 calories per kg per day on 7th day is to be stepped up gradually to 100 on 14th day and about 120–150 on 21st day.

Food volume—To meet the calorie requirements, the amount of milk to be given is slowly but progressively increased. Requirement on 1st day is 80 mL/kg. Gradually increased by 15 mL/kg/day to reach 200 mL/kg/day by 8th to 10th day. This is expected to be achieved by 2 weeks. Because of small stomach capacity, weak cardiac sphincter and poor cough reflex, the feeds should be small and are to be given at shorter intervals. Thus, initially a much smaller volume is required.

Additional supplements—All premature babies should receive additional supplement of vitamins and minerals which should be started after 2 weeks. The daily requirement consists of vitamin A 2500 IU, vitamin D 400 IU, vitamin C 50 mg, folic acid 65 µg and vitamin B_1-0.5 mg. Supplementation of calcium and phosphate is also essential. In addition, iron supplement should be given in the second or third week. A liquid preparation containing 2–4 mg/kg/day of elemental iron should be given in two divided doses. Intravenous gamma globulin therapy (400 mg/kg/dose) may be given to prevent infections in selected cases. For very low birth weight (< 1200 g) babies parenteral nutrition with amino acids, lipids along with dextrose and multivitamins are given.

***Adequate nursing care*: The single most important factor is high standard of nursing** and one trained nurse can adequately take care of two or three infants. (1) The temperature should be taken twice daily and the baby should be weighed daily to know whether over or underhydrated (2) Constant supervision especially during the crucial first 48 hours is imperative (3) Mother should be allowed to care her baby in the nursery (4) Mother is taught for the general care of the baby and manual expression of breast milk by pressing over the areola and the nipple. **Intelligent observation**, **prompt recognition of the abnormality and adequate measures to rectify the defect can be life saving in many occasions**.

FAVORABLE SIGNS OF PROGRESS: The following are the favorable signs: (1) The color of the skin remains pink all the time. (2) Smooth and regular breathing. (3) Increasing vigor evidenced by—(a) movements of the limbs and (b) cry. (4) Progressive gain in weight. Baby loses 1–2% weight every day for the first 5–7 days. Thereafter baby gains 1–1.5% of birth weight daily. Baby regains birth weight by 10–14 days.

WHEN TO DISCHARGE? The premature babies are discharged: (1) When they attain sufficient weight. (2) Attain good vigor. (3) Able to suckle the breast successfully.

ADVICES ON DISCHARGE: If possible, the supervision is to be continued at home by public health nurses or health visitors. Parental education is given for care of the baby at home. **The following advices are given on discharge:**

- Advice about feeding schedule.
- Prescribe a suitable multivitamin and oral iron preparation as mentioned earlier.
- To attend the well baby clinic for subsequent check up, immunization and guidance.

FOLLOW UP VISIT: Assessment is done for infants general health, weight, hydration and degree of jaundice. Immunization schedule is verified (see p. 526). Any new problem need to be identified. Pattern of feeding, its adequacy are explored. Screening test if any to be done. Guidance for infant care is given to mother.

FETAL GROWTH RESTRICTION (FGR)

Syn: Intrauterine Growth Restriction (IUGR), Chronic placental insufficiency

DEFINITION: Fetal Growth Restriction (FGR) is said to be present in those babies whose birth weight is below the 10th percentile of the average for the gestational age. Growth restriction can occur in preterm, term or post-term babies.

INCIDENCE: FGR comprises about one-third of low birth weight babies. In developed countries, its overall incidence is about 2–8%. The incidence among the term babies is about 5% and that among the post-term babies is about 15%.

NOMENCLATURE: SGA and IUGR are too often used synonymously although there is a degree of overlap. **SGA fetuses** constitute 70% of the babies with birthweight <10th percentile. These fetuses fulfill the growth potential and are not growth restricted (p. 527). They are constitutionally small but anatomically normal. They have no increased obstetric or neonatal risks. They grow parallel to the lower percentiles throughout pregnancy. These babies are small due to constitutional reasons and said as '*small mother small baby*' (p. 534). On the other hand, late onset of pathological cessation of growth may produce a baby with typical features of IUGR. This group has increased perinatal mortality and morbidity.

Normal fetal growth is characterized by cellular hyperplasia followed by hyperplasia and hypertrophy and lastly by hypertrophy alone. Most of the fetal weight gain (two-thirds) occurs beyond 24th week of pregnancy.

TYPES: Based on the clinical evaluation and ultrasound examination the small fetuses are divided into:

1. Fetuses those are **small and healthy**. The birth weight is less than 10th percentile for their gestational age. They have normal **ponderal index**, normal subcutaneous fat and usually have uneventful neonatal course.
2. Fetuses where growth is restricted by **pathological** process (true IUGR). Depending upon the relative size of their head, abdomen and femur, the fetuses are subdivided into: (a) Symmetrical or Type I (b) Asymmetrical or Type II.

Symmetrical (20%): The fetus is affected from the noxious effect very early in the phase of cellular hyperplasia. The total cell number is less. This form of growth retardation is most often caused by structural or chromosomal abnormalities or congenital infection (TORCH). **The pathological process is intrinsic to the fetus and involves all the organs including the head** (Table 32.1).

Asymmetrical (80%): The fetus is affected in later months during the phase of cellular hypertrophy. The total cell number remains the same but size is smaller than normal. The pathological processes that too often result in asymmetric growth retardation are maternal diseases extrinsic to the fetus. These diseases alter the fetal size by reducing uteroplacental blood flow or by restricting the oxygen and nutrient transfer or by reducing the placental size.

Table 32.1: Features of Symmetrical and Asymmetrical IUGR Fetuses

Symmetrical	Asymmetrical
Uniformly small	Head larger than abdomen
Ponderal index (Birth weight/Crown-heel length^3)—normal	Low
HC: AC and FL: AC ratios—normal	Elevated
Etiology: Genetic disease or infection—(Intrinsic to fetus)	Chronic placental insufficiency—(Extrinsic to fetus)
Total cell number—less Cell size—normal	Normal Smaller
Neonatal course—complicated with poor prognosis	Usually uncomplicated having good prognosis

ETIOLOGY: The causes of fetal growth restriction can be divided into four groups:

- **Maternal**
- **Fetal**
- **Placental**
- **Unknown**

Maternal:

- **Constitutional**—Small women, slim, low body mass index, maternal genetic and racial background are associated with small babies. These babies are not at increased risk. Prepregnancy maternal weight and weight gain during pregnancy are the two most important factors for fetal birth weight.
- **Maternal nutrition before and during pregnancy**—Critical substrate requirement for fetal growth such as glucose, amino acids and oxygen are deficient during pregnancy. This is an important cause of IUGR in women with undernutrition.

  Maternal diseases: Anemia, hypertension, thrombotic diseases, heart disease, chronic renal disease, collagen vascular disease are the important causes.
- **Toxins**—Alcohol, smoking, cocaine, heroin, drugs.

Fetal: There is enough substrate in the maternal blood and also crosses the placenta but is not utilized by the fetus. **The failure of nonutilization may be due to**—(1) **Structural anomaly** either cardiovascular, renal or others. (2) **Chromosomal abnormality** is associated with 8–12% of growth retarded infants. The common abnormalities are triploidy and aneuploidy. Trisomies (13, 18, 21) and Turner's syndrome are commonly observed. (3) **Infection** TORCH agents (toxoplasmosis, rubella, cytomegalovirus and herpes simplex) and malaria. (4) **Multiple pregnancy**—There is mechanical hindrance to growth and excessive fetal demand.

Placental: **The causes include cases of poor uterine blood flow to the placental site for a long time**. This leads to chronic placental insufficiency with inadequate substrate transfer. The placental pathology includes: Placenta previa, Abruption, Circumvallate, Infarction and Mosaicism.

Unknown: The cause remains unknown in about 40%.

Predictive factors for IUGR: (1) Presence of high risk factors (Obstetrics, Medical). (2) A low level of 1st trimester PAPP-1 value. (3) Abnormal uterine artery Doppler value (notching) at 20–24 weeks of pregnancy. (4) Fetal echogenic bowel on USG.

Pathophysiology: Basic pathology in small for gestational age is due to reduced *availability* of nutrients in the mother or its reduced *transfer* by the placenta to the fetus. It may also be due to reduced *ultilization* by the fetus. Brain cell size (asymmetric–SGA) as well as cell numbers (symmetric-SGA) are reduced. Liver glycogen content is reduced. There is oligohydramnios as the renal and pulmonary contribution to amniotic fluid is diminished due to reduction in blood flow to these organs. The SGA fetus is at risk of intrauterine hypoxia and acidosis, which if severe may lead to intrauterine fetal death.

DIAGNOSIS: Significant improvements have been made by clinical and biophysical methods in detecting a growth restricted fetus.

Clinical:

- **Clinical palpation** of the uterus for the fundal height, liquor volume and fetal mass may be used for screening. But it is less sensitive.
- **Symphysis Fundal Height** (SFH) measurement in centimeters closely correlates with gestational age after 24 weeks (see p. 80). A lag of 3 cm or more suggests growth restriction. It is a fairly sensitive parameter (30–80%). Serial measurement is important.
- **Maternal weight gain** remains stationary or at times falling (see p. 57) during the second half of pregnancy.
- **Measurement of the abdominal girth** showing stationary or falling values (see p. 111).

Biophysical: The first examination should confirm the clinical estimation of gestational age. USG is extremely useful not only to diagnose the growth retardation but also to identify a fetus of symmetrical or asymmetrical one. Sonographic predictive values that are commonly used are:

- **Head circumference (HC) and abdominal circumference (AC) ratios** (see p. 735): In a normally growing fetus the HC/AC ratio exceeds 1.0 before 32 weeks. It is approximately 1.0 at 32 to 34 weeks. After 34 weeks, it falls below 1.0. **If the fetus is affected by asymmetric IUGR, the HC remains larger. The HC/AC is then elevated.** In symmetric IUGR, both the HC and AC are reduced. The HC/AC ratio remains normal. Using HC/AC ratio, 85% of IUGR fetuses are detected. Transcerebellar diameter correlates well with the gestational age. **AC is the single most sensitive parameter to detect IUGR** (Fig. 41.8). Serial measurements of AC and estimation of fetal weight are more diagnostic to fetal growth restriction (see p. 84).
- **Femur length** (FL) is not affected in asymmetric IUGR. The FL/AC ratio is 22 at all gestational ages from 21 weeks to term. FL/AC ratio greater than 23.5 suggests IUGR.
- **Amniotic fluid volume**—The reduced amniotic fluid volume is too often associated with asymmetrical IUGR. Single deepest vertical pocket (SDVP) of amniotic fluid < 1 cm suggests IUGR in 96% of fetuses. The four quadrant technique consists of measuring the vertical diameter of the largest pockets of the fluid found in each of the four quadrant of the uterus (see Fig. 3.12). **The sum of the results is the amniotic fluid index (AFI).** An AFI between 5 and 24 cm is normal and an AFI less than 5 indicates oligohydramnios (see p. 250).
- **Anatomical survey:** To exclude fetal anomalies by sonography (Aneuploidy, structural defects).

Ultrasound Doppler Parameters:

- **Doppler Velocimetry:** Elevated systolic/diastolic (S/D) ratio, the resistance index and the pulsatility index indicate increased blood flow resistance and decrease in end-diastolic velocity. These are associated with FGR and intrauterine fetal hypoxia.
- **Uterine artery:** The presence of **diastolic notch** suggests incomplete invasion of placental trophoblasts to the uterine spiral arteries (see p. 36). This also predicts the possible development of **preeclampsia**. Normally, the diastolic flow increases as pregnancy progresses (see p. 123).
- **Umbilical artery (UA)** decreased endiastolic velocity indicates increased placental vascular resistance. There is progressive decrease in the umbilical artery end-diastolic velocity → reduced fetomaternal O_2 and nutrient exchange (see p. 123).
- **Umbilical artery Doppler study** should be the primary surveillance tool in the FGR fetus (RCOG). UA Doppler study can predict moderate acidosis and recommends delivery when there is presence of AREDV.
- **Reduced or absent or reversed end diastolic velocity (AREDV)** in the **umbilical artery** indicates fetal jeopardy and poor perinatal outcome (see p. 264).

- **Umbilical venous pulsations** indicate inefficient cardiac output with rise in central venous pressure → impending cardiac failure. Abnormal venous Doppler parameters (ductus venosus) are the important predictors of stillbirth.
- **Middle cerebral artery (MCA):** Increased diastolic velocity (brain sparing effect) is observed in a compromised fetus. This is due to cerebral vasodilatation in response to hypoxemia.
- **Ductus venosus Doppler study** can predict fetal acidemia and adverse perinatal outcome. It is used when UA Doppler study is abnormal and also to decide the time of delivery.
- **Ponderal index (PI)** — The degree of fetal wasting is judged by fetal PI. The index is determined by dividing the estimated fetal weight (g) by the third power of crown-heel length (cm) [(weight (g)/length cm^3) × 100]. PI below 10th percentile is taken as IUGR. It is highly accurate. Estimation of PI by fetal sonography has been made. Reduction in fetal facial fat stores has been associated with IUGR.

Biochemical markers: A low level of PAPP - A in maternal serum in the first trimester of pregnancy is considered a marker of major risk factor for FGR.

It is to be borne in mind that accurate prediction of fetal growth restriction using sonography has not been achieved as yet. However, the high rate of negative predictive value proves the value of the test.

Physical Features of an Infant with FGR at Birth

- **Weight** deficit at birth is about 600 g below the minimum in percentile standard. **Every hospital should have its own birth weight-gestational age chart**.
- **Length** is unaffected.
- **Head circumference is relatively larger than the body in asymmetric variety**.
- **Physical features** show dry and wrinkled skin because of less subcutaneous fat, scaphoid abdomen, thin meconium stained vernix caseosa and thin umbilical cord. **All these give the baby an "old-man look"**. Pinna of ear has cartilaginous ridges. **Plantar creases are well defined (Fig. 32.3, see Fig. 32.2 below)**.
- **The baby is alert, active and having normal cry**. Eyes are open.
- **Reflexes are normal** including Moro-reflex.

COMPLICATIONS: Fetal: (a) Antenatal—Chronic fetal distress, fetal death (b) Intranatal—Hypoxia and acidosis (c) After birth:

Immediate: (1) Asphyxia, bronchopulmonary dysplasia and RDS. (2) Hypoglycemia due to shortage of glycogen reserve in the liver. (3) Meconium aspiration syndrome (see p. 550). (4) Microcoagulation leading

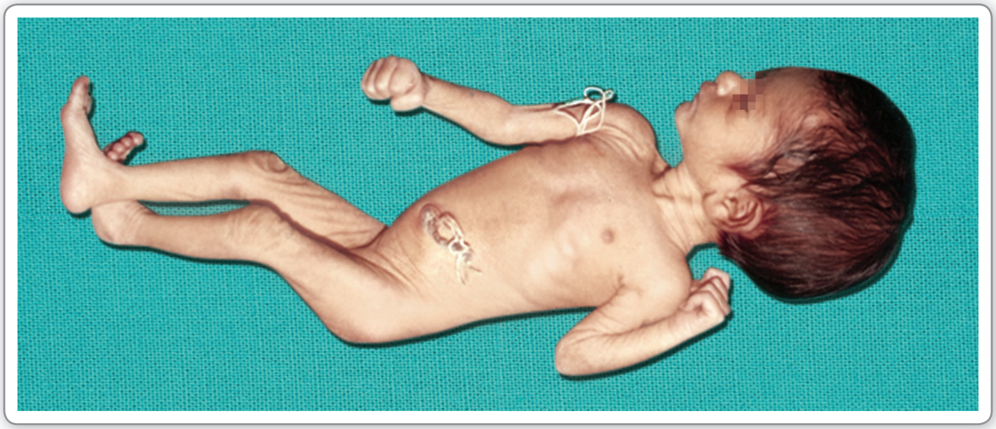


Fig. 32.3: Pregnancy complicated with chronic hypertension, delivered by CS at 38 weeks. The baby weighed 1.9 kg with features of asymmetrical IUGR. Physical features (wrinkled skin, scaphoid abdomen, head circumference > abdominal circumference) give the baby an "**old-man look**"

to DIC. (5) Hypothermia. (6) Pulmonary hemorrhage. (7) Polycythemia, anemia, thrombocytopenia. (8) Hyperviscosity-thrombosis. (9) Necrotizing enterocolitis due to reduced intestinal blood flow. (10) Intraventricular hemorrhage (IVH). (11) Electrolyte abnormalities: hypocalcemia, hyper hyperphosphatemia, hypokalemia due to impaired renal function. (12) Multiorgan failure. (13) Increased perinatal morbidity and mortality.

***Late*:** Asymmetrical IUGR babies tend to catch up growth in early infancy. The fetuses are likely to have: (1) retarded neurological and intellectual development in infancy. The worst prognosis is for IUGR caused by congenital infection, congenital abnormalities and chromosomal defects. **Other long-term complications** are: (2) Increased risk of metabolic syndrome in adult life: obesity, hypertension, diabetes and coronary heart disease (CHD). (3) LBW infants have an altered orexigenic mechanism that causes increased appetite and reduced satiety. (4) Reduced number of nephrons—causes renal vascular hypertension.

Maternal: Per se fetal growth restriction does not cause any harm to the mother. But underlying disease process like preeclampsia, heart disease, malnutrition may be life threatening. Unfortunately, for a woman with a growth retarded infant, risk of having another is two-fold.

MORTALITY: The immediate neonatal mortality is about 6 times more than the normal newborn. However, it is lower than premature AGA infants of the same birth weight. Most of the babies die within 24 hours. The morbidity rate rises to about 50%. They are at higher risk for poor postnatal growth and adverse cognitive outcome.

MANAGEMENT

Management is based upon the comprehensive diagnostic workup (discussed before). **Fetuses that are constitutionally small (70%) require no intervention. The fetuses that are symmetrically growth restricted (15%), should be investigated to exclude fetal anomalies, infections and genetic syndromes. Unfortunately there is no effective therapy for this group. Finally the growth restricted fetus owing to placental disease or reduced placental blood flow (chronic placental insufficiency), may be given some treatment. However, assessment of fetal well-being is more critical in the management as in majority there is no definitive therapy**.

Perinatal outcome is poor for women with early onset of IUGR (<34 weeks) compared to late onset of FGR (>34 weeks). Decision for early delivery has to balance the risk of neonatal deaths due to complications, on the other hand delay in delivery that may increase the risk of IUFD. Majority of fetal deaths occur after the 36th week of gestation. So, correct diagnosis and timed intervention are essential.

General: At present, there is no proven therapy for reversing growth restriction once it is established. However, the following may be tried with some success: (1) Adequate bedrest, especially in left lateral position; (2) To correct malnutrition by balanced diet: 300 extra calories per day are to be taken; (3) To institute appropriate therapy for the associated complicating factors likely to produce growth restriction; (4) Avoidance of smoking, tobacco and alcohol; (5) **Maternal hyperoxygenation** at the rate of 2.5 L/min by nasal prong, for short-term prolongation of pregnancy; (6) Low dose aspirin (50 mg daily) may be helpful in very selected cases with history of thrombotic disease, hypertension, preeclampsia, or recurrent IUGR; (7) **Maternal hyperalimentation** by amino acids can improve fetal growth if it was due to maternal malnutrition. It is not helpful when placental function is deficient; (8) **Maternal volume expansion** may be helpful in improving placental perfusion.

Antepartum evaluation: Serial evaluations of fetal growth and assessment of well-being should be done once the diagnosis is made.

- **Ultrasound examination** should be done at an interval of 3–4 weeks for the assessment of BPD, HC/AC, fetal weight and AFI.

- **Fetal well-being** is assessed by Kick count, NST, biophysical profile, amniotic fluid volume and cordocentesis for blood gases (see p. 130).
- Doppler ultrasound parameters are to be studied (see p. 123).

TIMING OF DELIVERY: The factors to be considered are: (1) Presence of fetal abnormality; (2) Duration of pregnancy; (3) Degree of FGR; (4) Associated complicating factor; (5) Underlying pathology (if known) (6) Results of antenatal fetal surveillance and (7) Availability of neonatal intensive care unit (NICU).

- **Optimum time of delivery** *for a growth restricted* fetus may be between 34 weeks and 37 weeks depending upon the presence of any additional risk factor(s) (e.g. Oligohydramnios, preeclampsia, abnormal Doppler study).

A. Pregnancy ≥ 37 weeks: Delivery should be done.

B. Pregnancy < 37 weeks (a) **Uncomplicated mild IUGR:** Fortunately, the majority falls in this group. Usual treatment as outlined above to improve the placental function may be employed. Pregnancy is continued at least 37 weeks. Thereafter delivery is done.

C. Severe degree of IUGR:

- **Delivery should be planned on the basis of fetal surveillance report** (see p. 539).
- **If the lung maturation is achieved** as evidenced by presence of phosphatidylglycerol and L: S ratio of ≥2 from the amniotic fluid study (amniocentesis), delivery is done.
- **If the lung maturation has not yet been achieved**, intrauterine transport to an equipped center is ideal in such a case. Betamethasone therapy (see p. 367) is given to accelerate pulmonary maturation when gestational age is less than 34 weeks. Corticosteroids reduce the risk of neonatal HMD and intraventricular hemorrhage (IVH) (see p. 560).
- Delivery to be done **at 34 0/7 weeks** of gestation in cases of FGR **with additional risk factors** for adverse perinatal outcome (Preeclampsia, oligohydramnios, AREDV).
- When delivery is to be done preterm, **antenatal corticosteroids** should be given.
- When delivery is to be done **before 32 weeks, magnesium sulfate** should be given to the mother for fetal and neonatal neuroprotection.
- **Fetuses with aneuploidy or congenital infection** have poor outcome irrespective of gestational age and timing of delivery.

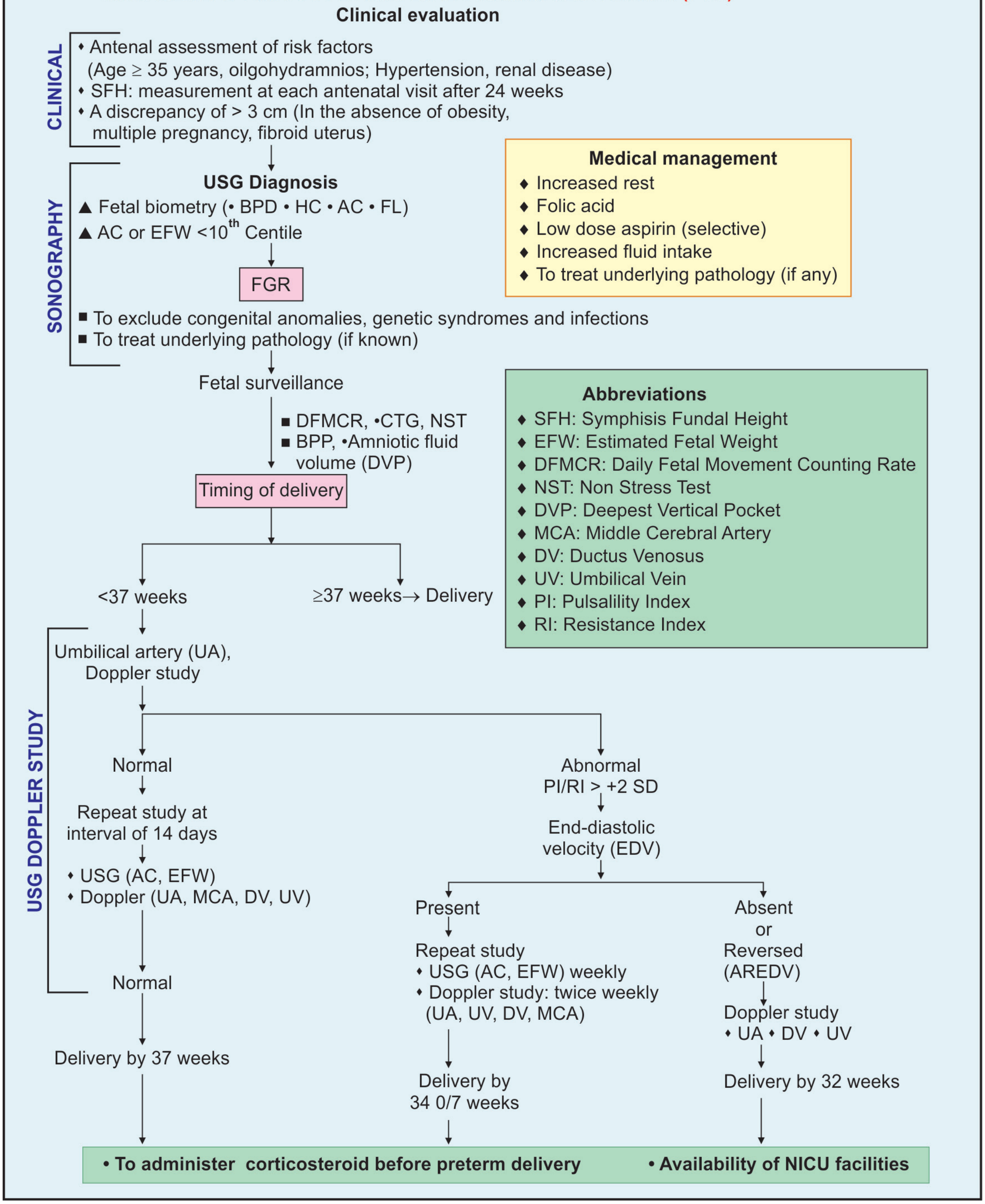
METHODS OF DELIVERY:

- **Low rupture of the membranes followed by oxytocin** is employed in cases such as pregnancy beyond 34 weeks with favorable cervix and the head is deep in the pelvis. Prostaglandin (PGE_2) gel could be used when the cervix is unfavorable. The color of the liquor could be a guide for further management.
- **Intrapartum monitoring** by clinical, continuous electronic and scalp blood sampling is needed as the risk of intrapartum asphyxia is high.
- **Cesarean delivery without a trial of labor** is done when the risks of vaginal delivery are more (presence of fetal acidemia, absent or reversed diastolic flow in umbilical artery or unfavorable cervix).

CARE DURING VAGINAL DELIVERY

- The delivery should be in an equipped institution where intensive intranatal monitoring (clinical and electronic) is possible and having facilities for neonatal intensive care unit.
- The precautions during labor are those required for preterm delivery (see p. 367).

MANAGEMENT PROTOCOL FOR FETAL GROWTH RESTRICTION (FGR)


Clinical evaluation
CLINICAL
• Antenal assessment of risk factors (Age ≥ 35 years, oilgohydramnios; Hypertension, renal disease)
• SFH: measurement at each antenatal visit after 24 weeks
• A discrepancy of > 3 cm (In the absence of obesity, multiple pregnancy, fibroid uterus)
SONOGRAPHY
USG Diagnosis
▲ Fetal biometry (• BPD • HC • AC • FL)
▲ AC or EFW <10th Centile
FGR
■ To exclude congenital anomalies, genetic syndromes and infections
■ To treat underlying pathology (if known)
Fetal surveillance
■ DFMCR, •CTG, NST
■ BPP, •Amniotic fluid volume (DVP)
Timing of delivery
<37 weeks
≥37 weeks→ Delivery
USG DOPPLER STUDY
Umbilical artery (UA), Doppler study
Normal
Repeat study at interval of 14 days
• USG (AC, EFW)
• Doppler (UA, MCA, DV, UV)
Normal
Delivery by 37 weeks
Abnormal PI/RI > +2 SD
End-diastolic velocity (EDV)
Present
Repeat study
• USG (AC, EFW) weekly
• Doppler study: twice weekly (UA, UV, DV, MCA)
Delivery by 34 0/7 weeks
Absent or Reversed (AREDV)
Doppler study
• UA • DV • UV
Delivery by 32 weeks
• To administer corticosteroid before preterm delivery
• Availability of NICU facilities

Medical management

- Increased rest
- Folic acid
- Low dose aspirin (selective)
- Increased fluid intake
- To treat underlying pathology (if any)

Abbreviations

- SFH: Symphisis Fundal Height
- EFW: Estimated Fetal Weight
- DFMCR: Daily Fetal Movement Counting Rate
- NST: Non Stress Test
- DVP: Deepest Vertical Pocket
- MCA: Middle Cerebral Artery
- DV: Ductus Venosus
- UV: Umbilical Vein
- PI: Pulsalility Index
- RI: Resistance Index

IMMEDIATE CARE OF THE BABY AFTER BIRTH

- A pediatrician should be available at the time of delivery.
- The same precautions as outlined in the preterm delivery are to be taken (see p. 367).
- The baby should be placed preferably in the neonatal intensive care unit.

***Intensive care protocols*:** The same protocols as conducted in the management of preterm babies are to be followed. Special precaution is to be taken to prevent and treat complications (see p. 530).

KEY POINTS

- **A low birth weight** infant is one whose birth weight is less than 2500 g irrespective of gestational age (see p. 527). It is a major cause of perinatal morbidity and mortality.
- **FGR** is a major cause of perinatal morbidity and mortality.
- **Etiology of FGR** is many (see p. 534). Symmetrical FGR infants face more complications (p. 536) and have got poor prognosis compared to asymmetrical ones (see p. 534).
- **Serial measurement of Symphysis Fundal Height (SFH)** should be done at each antenatal visit from 24 weeks onwards. It can predict the FGR well.
- When SFH revealed slow or static fetal growth (>3 cm), the woman should have **ultrasound evaluation** of fetal growth. **USG diagnosis of FGR**, is made from four biometric measures: (1) BPD (2) HC (3) AC and (4) FL. The estimated fetal weight can be derived from these values.
- Fetal **AC** or **EFW < 10th centile** is used for **diagnosis of FGR**.
- **Amniotic fluid volume** assessment should be done measuring the **single deepest vertical pocket**.
- CTG, amniotic fluid volume, BPP should be considered together in evaluation of FGR.
- With ultrasound evidence of FGR the woman should be **referred to a fetal medicine unit** for fetal anatomy survey and uterine artery Doppler study. Serological screening may be done when congenital infection is suspected.
- **Antiplatelet agent (aspirin)** is given in cases with preeclampsia.
- **Management of FGR** depends on its type, severity and duration of pregnancy. Serial assessment of fetal growth and surveillance is needed (see p. 537). **Timing of delivery** is based on the **fetal surveillance** of fetal hypoxia and acidemia.
- **In high risk women, use of umbilical artery Dopper velocity (UADV) study is the primary surveillance tool for a growth restricted fetus (RCOG)**. It reduces perinatal morbidity and mortality.
- **When UADV indices are abnormal** (PI or RI > 2SDs above the mean for gestational age) → Repeat surveillance is done.
- **Abnormal ductus venosus (DV) Doppler study, predicts fetal academia and poor outcome.**
- **Optimum time for delivery** for a woman with FGR is between 34 and 37 weeks.
- **Woman with FGR showing AREDV** in the umbilical artery – needs to be delivered (Cesarean section).
- **Presence of abnormal ductus venosus (DV) Doppler study** or pulsations in umbilical vein (UV) – delivery is recommended (provided the fetus is viable).
- **Delivery for FGR** should be organized in a **center with NICU facilities**.
- **The neonatal morbidities in FGR are**: Birth asphyxia, meconium aspiration, hypothermia and others (see p. 537).
- **FGR can cause** short-term as well as long-term morbidities (see p. 537).

QUESTIONS

1. Mention the important causes of Fetal Growth Restriction (FGR)? Outline the management of a case with FGR? (p. 534 and 537)

Related theory questions (Long and Short), Obstetric case discussions, Viva table discussions, Postoperative word round discussions, and MCQs are discussed in author's books:

1. **Bedside Clinic and Viva Voce:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

For further reading:

2. **Master Pass in Obstetrics and Gynaecology:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

Chapter 33

Disease of the Fetus and the Newborn

PERINATAL ASPHYXIA

DEFINITION: In common clinical parlance, asphyxia neonatorum means **nonestablishment of satisfactory pulmonary respiration** at birth. Its literal meaning is "stopping of the pulse".

Perinatal asphyxia is a condition of impaired blood gas exchange that, if persists, leads to progressive hypoxemia, hypercapnia and metabolic acidosis. The essential characteristics for the diagnosis of perinatal asphyxia are: (i) Profound acidemia (pH < 7.0) on umbilical cord arterial blood sample; (ii) Persistence of an Apgar score 0–3 for > 5 minutes; (iii) Neurological manifestations (hypotonia, coma, seizures) in the immediate neonatal period; (iv) Evidence of multiorgan system dysfunction. Often it is the continuation of antepartum or intrapartum event. **Perinatal asphyxia is a significant cause of perinatal death (50%)**. Incidence of asphyxia varies depending on the gestational age.

FETAL RESPIRATION

Human fetal breathing activity is observed at 11 weeks of intrauterine life. Initially these are rapid and small amplitude movements (60–90 per minute). Fetal breathing occurs during the periods of low-voltage electrocortical activity, e.g. rapid eye movement (REM) sleep. During high voltage electrocortical activity (non-REM sleep) occasional breaths are observed. **Increased fetal breathing movements are seen with increased fetal oxygen tension and hyperglycemia and it is decreased in hypoxia**. Following birth the fluid filled lung becomes the organ of gas exchange. **The essential requirements are:** (i) Aeration of the lungs; (ii) Establishment of pulmonary circulation; (iii) Establishment of ventilation and (iv) Diffusion of O_2 and CO_2 through the alveolar—capillary membranes.

PULMONARY DEVELOPMENT AND BIOCHEMICAL CHANGES

During 4th week of gestation the lungs develop as a ventral diverticulum from the foregut. By 16 weeks, the tracheobronchial tree up to the terminal bronchiole and vasculature is developed. In the human fetus the important stages of lung development are:

(1) Foregut ventral diverticulum formation—4th week; (2) Pseudoglandular period 8th–16th weeks; (3) Canalicular period 17th–27th weeks; (4) Saccular period—24th–38th weeks and (5) Alveolar period at or after 36 weeks.

The respiratory epithelial cells in the alveoli are of two types. **Type I cells** contain less subcellular organelles. **Type II cells** contain abundant mitochondria, endoplasmic reticulum, Golgi apparatus and osmiophilic lamellar bodies that contain **surfactant**. **Surfactant reduces the surface tension of the alveolus**. Natural surfactant contains 84% phospholipids, 8% neutral lipids and 8% protein. The important phospholipids are shown in Figure 33.1. All are synthesized and secreted by type II alveolar cells. **The hormones and growth factors that augment surfactant synthesis are:** glucocorticoids, thyroid hormone, thyrotropin releasing hormone (TRH), prolactin, cyclic adenosine monophosphate (cAMP) and epidermal growth factor.

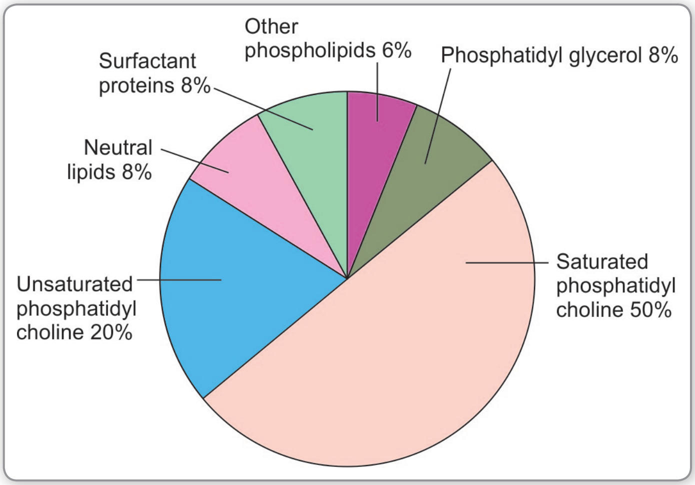


Fig. 33.1: Composition of pulmonary surfactant

Antenatal administration of glucocorticoids to the mother before 34 weeks of gestation, reduces the incidence of neonatal respiratory distress syndrome (RDS) significantly (70%). The effect starts after 24 hours and lasts for 7 days. **Other beneficial effects** of antenatal corticosteroids are: maturation of cardiovascular, gastrointestinal and CNS of the fetus. It reduces the risk of **periventricular/intraventricular hemorrhage (PVH/IVH)** and **necrotizing enterocolitis (NEC)**.

INITIATION OF RESPIRATION: With the first breath the neonate overcomes the following **resistances for expansion of the lungs.** These are (i) Viscosity of the lung fluid; (ii) Lung tissue resistance; (iii) The forces of surface tension at the air-liquid interface. During delivery of the chest, intrathoracic pressure increases up to 200 cm of H_2O due to vaginal squeeze. Following delivery of the head about 5–30 mL of tracheal fluid is squeezed out. With the delivery of the thorax, the elastic recoil of the chest initiates the passive inspiration. **Initial respiration is a short inspiration followed by a more prolonged expiration.** A pressure of about 25 cm H_2O is necessary to overcome the surface tension of the airways and the alveoli. Viscosity of the lung fluid is a major factor for normal neonatal lung expansion and aeration. Diaphragmatic contraction and chest wall expansion create a negative intrathoracic pressure. The volume of first breath is 40–70 mL.

The first breath (short inspiration followed by long expiration) establishes a functional residual capacity (16–20 mL) and brings about a huge increase in pulmonary perfusion and subsequent normal pattern of breathing. A negative intrathoracic pressure of 15 cm water is needed to establish regular respiration. This also is sufficient to overcome the surface tension and is helped immensely by **pulmonary surfactant**. Within the uterus, fetal lung alveoli are filled with fluid. This fluid is derived from the ultrafiltration of pulmonary capillary blood, secretion of alveolar cells and the amniotic fluid. Once labor is initiated this fluid is absorbed by the lung epithelium. Part of it is removed by mechanical drainage during labor by chest squeeze.

Important Factors That Operate to Overcome the Lung Resistance

1. Increased fluid absorption and less fluid secretion by the alveolar cells with the onset of labor.
2. Thoracic squeeze during delivery.
3. Marked increase in pulmonary lymph flow.
4. Removal of fluid via pulmonary circulation.

In a normal birth the process is completed within 2 hours. Babies born by cesarean, and premature infants have delayed lung fluid absorption.

ETIOPATHOLOGY OF PERINATAL ASPHYXIA: Ninety percent of asphyxial events occur in the antepartum or intrapartum periods as a result of placental insufficiency. The rests are postnatal. Asphyxia can be classified broadly into the following groups:

A. Continuation of intrauterine hypoxia (placental insufficiency)

- **The placenta, as a respiratory organ of the fetus, fails functionally** either due to anatomical changes or due to inadequacy of uteroplacental circulation (such as premature placental separation, circumvallate placenta, hypertensive disorders in pregnancy, abnormal labor, cord compression, vascular anomalies in cord, etc.).
- **Maternal hypoxic states:** The maternal diseases such as anemia, eclampsia, cyanotic cardiovascular disorders, status asthmaticus, dehydration and hypotension.

B. Prenatal and intranatal medication to the mother

Morphine, pethidine and anesthetic agents depress the respiratory centers directly and the chance of development of asphyxia is increased.

C. Birth trauma to the neonate

Malpresentation such as breech, oblique lie, occipitoposterior often requires manipulative and operative vaginal delivery (forceps or ventouse). Prolonged second stage of labor in contracted pelvis, often causes asphyxia. Increased intracranial tension → cerebral edema and congestion → increased intracranial pressure → asphyxia.

D. Postnatal factors

Postnatal asphyxia is secondary to pulmonary, cardiovascular and neurological abnormalities of the neonate.

These often overlap, making isolation of a single causative factor difficult.

CLINICAL FEATURES

The clinical features depend upon the etiology, intensity and duration of oxygen lack, plasma carbon dioxide excess and subsequent acidosis. The fetus and neonate are more resistant to asphyxia than the adults. In response to asphyxia, a mature fetus redistributes the blood flow to the heart, brain and adrenals to ensure adequate oxygen and substrate delivery to these **vital organs**.

But at present, according to the parameters denoted by Apgar, (Dr Virginia Apgar—1953) a scoring procedure has been designed for better understanding of the clinical state. **Long-term neurological correlation is obtained at the 5-minute score which is of more value**. In cases where the score remains significantly depressed at 5 minutes, it should be evaluated again after 15 minutes. This scoring is done in a newborn baby at 1 minute, 5 minutes and 15 minutes and can be tabulated as in follows (Table 33.1).

Majority of infants born with an Apgar score ≥4 by 10 minutes, >99% do not develop cerebral palsy (CP). 75% children who develop CP have normal Apgar score at birth. Apgar score alone should not be taken as an evidence of neurological damage. Cord blood pH can assess fetal oxygenation status better.

Table 33.1: Apgar Scoring

	Score		
Signs	**0**	**1**	**2**
Respiratory effort	Absent	Slow, irregular	Good, crying
Heart rate	Absent	< 100 bpm	> 100 bpm
Muscle tone	Flaccid	Flexion of extremities	Active body movements
Reflex irritability	No response	Grimace	Cough or sneeze
Color	Blue, pale	Body pink, extremities blue	Complete pink

- Total score = 10
- Mild depression = 5–7
- Severe depression = 0–2
- No depression = 8–10
- Moderate depression = 3–4

Normal range of arterial blood gas values for a term newborn are: PaO_2 50–95 mm Hg; $PaCO_2$ 35–45 mm Hg; HCO_3 24–26 mEq/L and pH 7.35–7.45.

Clinical sequelae of birth asphyxia: The variable clinical signs of CNS injury with HIE are: hypoxia, seizures, apnea, respiratory failure, hypotension, NEC, thrombocytopenia, metabolic acidosis and hypoglycemia. Initial response is hyperapnea and hypertension → primary apnea → gasping attempt to breathe → (if unresolved) → secondary apnea → bradycardia and shock → diminished cerebral blood flow → cerebral hemorrhage → hypoxic ischemic encephalopathy (HIE) → (if severe) → either death or disability (if the baby survives).

Neonatal diagnosis: (i) Electroencephalography may help to detect severity of asphyxial injury and to predict long-term neurodevelopmental outcome due to HIE; (ii) CT can detect cortical neuronal injury several weeks after asphyxial insults; (iii) USG is the method of choice for routine screening to detect IVH, necrosis of basal ganglia and thalamus. It is superior to CT; (iv) MRI can localize the area of brain injury due to the presence of cerebral edema. Results of MRI correlates with the neurodevelopmental outcome of infants with HIE.

- Magnetic resonance spectroscopy (MRS) can detect the biochemical change in the brain secondary to HIE.
- Diffusion weighted imaging (DWI) and diffusion tensor imaging (DTI) are designed to assess the magnitude of water diffusion in the brain.

MANAGEMENT

Management of perinatal asphyxia can be divided into two:

- **Prophylactic**
- **Definitive**

PROPHYLACTIC: (1) Antenatal detection of high-risk patients; (2) Scrupulous fetal monitoring to ensure early detection of fetal distress and timely delivery; (3) Intrapartum use of electronic fetal monitoring and scalp blood pH assessment when indicated. Scalp blood pH < 7.0 is a substantial evidence of prolonged intrauterine asphyxia; (4) Judicious administration of anesthetic agents and sedatives during labor; (5) Cooperation between obstetric and pediatric staff since delivery and (6) Avoidance of difficult or traumatic delivery.

DEFINITIVE: Apgar rating—classically, the evaluation of the cardiopulmonary status in the newborn has been assessed by Apgar rating at 1 minute and 5 minutes after birth. **But it must be emphasized that in certain circumstances, it is inappropriate to delay resuscitative efforts until the 1 minute Apgar score is obtained**. Heart rate, skin color and respiratory activity provide the most accurate evaluation and the need of resuscitation. However, most infants born with Apgar scores of 7–10 are essentially normal.

1. BABIES WITH APGAR SCORE 8–10: (Pink, breathing spontaneously, HR > 100 bpm).

- The oropharynx and the nasopharynx are to be cleared off any mucus by suction.
- Dry the infant and place under radiant heat source.
- Oxygen is administered only when required.
- The condition is reassessed at 5 minutes and if found normal, the infant should be given to mother.

2. BABIES WITH APGAR SCORE 5–7: Peripheral cyanosis, breathing spontaneously, HR > 100 bpm.

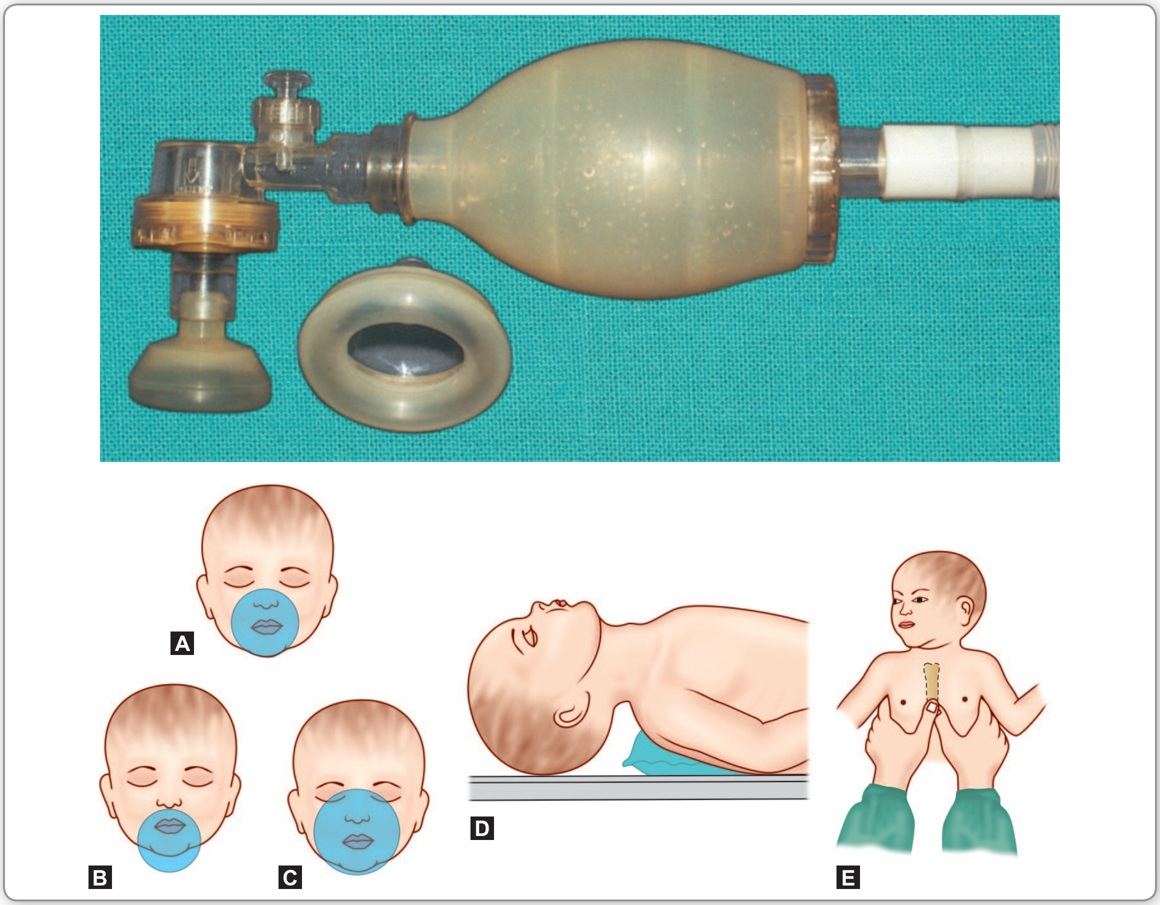
- Baby may follow primary apnea.
- Place under a radiant heater, dry the baby.
- A pulse oximeter placed on the right hand.
- The baby is put flat, head in midline with slight extension position.
- Immediate suction of the oropharynx and nasopharynx is done.
- Stimulus to back and sole (gentle rubbing).
- Oxygen (100%) is administered at a rate of 5 L/min by bag and mask at a pressure range of 30–40 cm H_2O (Fig. 33.2).
- CPAP may be given if necessary.
- Support should be continued until respirations are spontaneous, color improves and the heart rate is > 100 bpm. Such an infant may be acidotic but it is corrected spontaneously after respiration is established.

In majority of cases, the baby takes independent respiration with these simple measures. The Apgar rating is done at 5 minutes and if found satisfactory, the baby is returned to the mother.

Almost all newborns respond to ventilation with 100% O_2. Failure to respond to intubation and ventilation can result from mechanical causes (equipment failure, malposition of ET tube), pulmonary hypoplasia or severe asphyxia.

3. BABY IS APNEIC DESPITE TACTILE STIMULATION: Central cyanosis or HR < 100 bpm (Apgar score 3–4)

- Baby may develop secondary apnea : call for assistance.
- A bag (750 mL volume) and a mask ventilation is started. O_2 is administered at the rate of 5–8 L/min. Positive pressure of 25–30 cm H_2O may be needed for appropriate chest rise.



Figs 33.2A to E: ***Top*** —Infant resuscitation bag and mask (Ambu bag). ***Bottom***—Use of face mask: (A) Correct; (B) and (C)—Incorrect; (D) Correct positioning (neck slightly extended); (E) Chest compression (thumb method)

- If not effective intratracheal intubation and IPPV is started. A rate of 40–60 breaths/minute should be used. Baby is reassessed in next 15–30 seconds. Support is continued until respirations are spontaneous, HR is >100 bpm. These infants will be acidotic but are able to correct themselves once spontaneous respiration is established.

4. Baby is apneic, HR < 100 bpm despite 30 seconds of assisted ventilation (Apgar Score 0–2)

- HR > 60 bpm, to continue positive pressure ventilation. The heart rate is rechecked in 30 seconds ventilation.
- Increase the oxygen concentration to 100% if resuscitation was started using an air-oxygen blend.
- Failure to increase HR, poor status of oxygen saturation, persistent cyanosis, intubation is done rapidly by a skilled person.
- Cardiac massage is given to maintain circulation if HR < 60 bpm.

Drugs used for resuscitation: Drugs are needed for a persistent HR < 60 bpm even after ventilation and chest compression. Drug of choice is epinephrine (see p. 546). Other drugs are given as needed (see p. 546).

Meconium aspiration syndrome (MAS): Endotracheal intubation and suctioning is performed with a negative pressure of 80–100 mm Hg. This procedure may be repeated.

RESUSCITATION OF THE NEWBORN IN THE DELIVERY ROOM

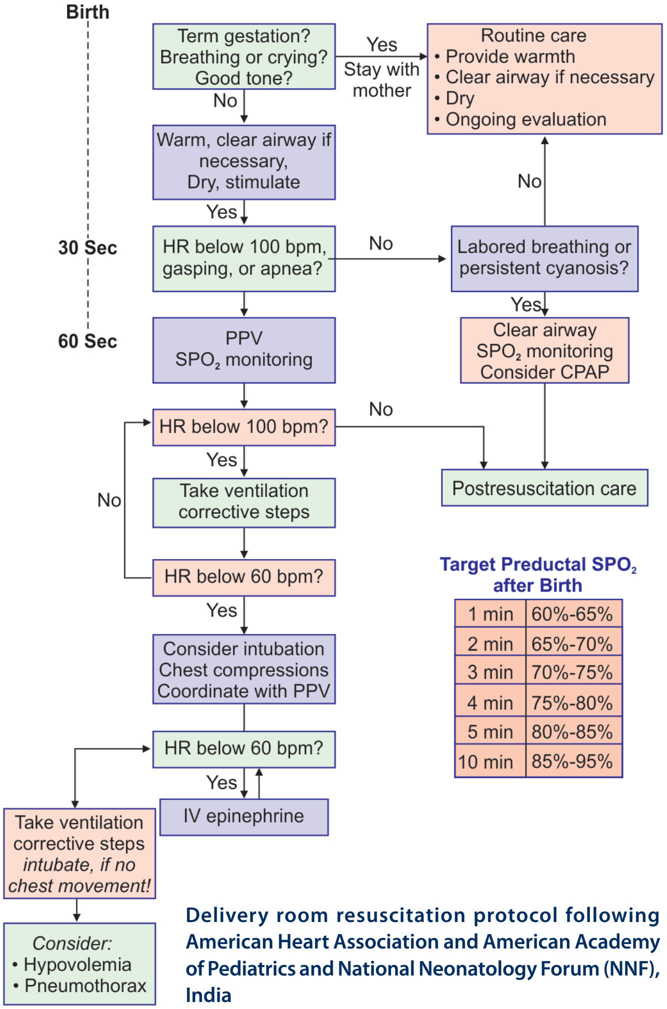
EQUIPMENTS NEEDED FOR NEONATAL RESUSCITATION

Ventilatory resuscitation:

- Dry the infant to place under the radiant heater.
- Place the infant with head in midline position, neck with slight extension.
- Suction of mouth, oropharynx with a suction bulb.
- Assess the infant's condition: respiratory effort, (apnea or regular breathing) and heart rate.
- Infants with regular breathing and heart rate > 100 bpm need no further intervention; if cyanotic, provide O_2 supplementation.
- Infants HR < 100 bpm, apnea or irregular respiration: bag and mask ventilation (100% O_2) to be given. A soft mask that seals around the mouth and nose is to be used.
- Most neonates can be effectively managed with a bag and face mask. If no improvement by another 30–40 seconds – intubation is proceeded.

Chest compression: The sternum is compressed about one-third the diameter of the chest at a regular rate of 90 compressions/min while ventilating (PPV) the infant at 30 breaths/min (3:1). The HR is checked periodically and chest compression is discontinued when the HR is > 60 bpm. The thumbs are placed together over the lower third of the sternum. The palms encircle the torso and support the back.

Medications: **Epinephrine**: 0.1–0.3 mL/kg in 1:10,000 dilution is given IV or endotracheal, when there is persistent bradycardia. It may be repeated at every 5 minutes. **Sodium bicarbonate** to treat metabolic acidosis (pH < 7.2) IV (1–2 mEq/kg of 0.5 mEq/mL, 4.2% solution) is given. **Reversal of narcotic drug** is needed when mother has been given pethidine or morphine within 3 hours of delivery. **Naloxone** 0.1–0.2 mg/kg is given to the baby by IV, IM or endotracheal. **Volume expansion** is needed when blood pressure is low and tissue perfusion is poor. Normal saline, 5% albumin or whole blood (10 mL/kg) IV is given. **Dopamine** infusion may be given for hypotension.


Birth
Term gestation? Breathing or crying? Good tone?
Yes
Stay with mother
Routine care
• Provide warmth
• Clear airway if necessary
• Dry
• Ongoing evaluation
No
Warm, clear airway if necessary, Dry, stimulate
Yes
No
30 Sec
HR below 100 bpm, gasping, or apnea?
No
Labored breathing or persistent cyanosis?
Yes
60 Sec
PPV
SPO_2 monitoring
Clear airway
SPO_2 monitoring
Consider CPAP
HR below 100 bpm?
No
Yes
Postresuscitation care
No
Take ventilation corrective steps
HR below 60 bpm?
Yes
Consider intubation
Chest compressions
Coordinate with PPV
HR below 60 bpm?
Yes
IV epinephrine
Take ventilation corrective steps
intubate, if no chest movement!
Consider:
• Hypovolemia
• Pneumothorax

Delivery room resuscitation protocol following American Heart Association and American Academy of Pediatrics and National Neonatology Forum (NNF), India

Target Preductal SPO_2 after Birth

1 min	60%-65%
2 min	65%-70%
3 min	70%-75%
4 min	75%-80%
5 min	80%-85%
10 min	85%-95%

Airway management

- Suction
- Stethoscope
- Ventilation
- Bag and mask
- Oral airways
- O_2 source
- Laryngoscope
- Endotracheal tubes

Thermoregulations

- Radiant warmer
- Warm blankets
- Servo control heating
- Temperature probe

Gastric decompression

- Nasogastric tube
- Suction

PROGNOSIS: **The prognosis is dependent on:** (1) Maturity of the baby; (2) Duration and intensity of hypoxia and acidosis as evidenced by Apgar score and blood pH—higher the score, normal the pH, better is the prognosis; (3) Facilities for immediate and competent management of a compromised baby. Most survivors of perinatal asphyxia do not have any major sequelae. Factors for increased risk of neurological sequelae are: (i) Apgar score of 0–3 at 20 minutes of age; (ii) Presence of multiorgan failure (oliguria > 24 hours of life); (iii) Severity of the neonatal neurological syndrome. Severe HIE carries mortality about 80% and (iv) Presence of neonatal seizure.

COMPLICATIONS: ***Immediate:*** (a) **Cardiovascular**–hypotension, cardiac failure; (b) **Renal**–acute cortical necrosis, renal failure; (c) **Liver function**–compromised; (d) **Gastrointestinal**–ulcers and necrotizing enterocolitis; (e) **Lungs**–persistent pulmonary hypertension; (f) **Brain**–cerebral edema, seizures.

Delayed: (a) Retarded mental and physical growth; (b) Epilepsy—up to 30% in severe asphyxia; (c) Minimal brain dysfunction.

RESPIRATORY DISTRESS IN THE NEWBORN

Increased alveolar fluid content, inadequate clearance of lung fluid, lack or inhibition of surfactant function, or reduced surface area for gas exchange is the basic pathology for respiratory distress. The important clinical causes are:

Pulmonary	Cardiovascular	Noncardiopulmonary
♦ Hyaline membrane disease (HMD) or RDS ♦ Meconium aspiration ♦ Clear fluid aspiration ♦ Pulmonary hypoplasia ♦ Bronchopulmonary dysplasia ♦ Bronchopneumonia ♦ Airway obstruction ♦ Transient tachypnea ♦ Pneumothorax ♦ Pulmonary edema	♦ **Congenital heart disease** Aortic stenosis Coarctation of aorta Cyanotic—Transposition of great vessels – Tetralogy of Fallot – PDA – VSD ♦ Heart failure ♦ Persistent pulmonary hypertension of newborn (PPHN)	♦ Metabolic acidosis ♦ Hypo- or hyperthermia ♦ Hypoglycemia ♦ Asphyxia ♦ Drugs (Pethidine) ♦ Birth trauma ♦ Intracranial injury

IDIOPATHIC RESPIRATORY DISTRESS SYNDROME

Syn: Hyaline Membrane Disease, Respiratory Distress Syndrome (RDS)

Respiratory distress syndrome is defined as the persistence of arterial O_2 tension (PaO_2) < 50 mm Hg and central cyanosis in room air (oxford network). Supplemental oxygen supply is required to maintain PaO_2 > 50 mm Hg or pulse oximeter saturation > 85%.

INCIDENCE: It ranges from 75% at around 28 weeks to 52% at 30 weeks of gestation. Use of exogenous surfactant has significantly reduced the risk of neonatal death by <10%.

PATHOGENESIS: The primary cause is inadequate pulmonary surfactant. Deficiency of surfactant (see p. 124) in the lung alveoli increases alveolar surface tension. It is seen within first 24 hours of birth. Surfactant is a surface active material. It is produced by alveolar epithelial cells called Type II pneumocytes at 24–28 weeks gestation. Antenatal corticosteroids enhances but fetal hyperinsulinemia delays surfactant synthesis. Other factors that enhance maturity of type II cells are: chronic stress, PIH, FGR, twins and placental insufficiency. There is poor lung compliance, reduction in ventilation—perfusion ratio and progressive atelectasis. Hyaline membrane disease (HMD) is further complicated by the weak respiratory muscles of the newborn.

The consequent finding is widespread atelectasis. A homogenous eosinophilic membrane (hyaline membrane) plastering the alveolar ducts and terminal bronchioles is found.

Blood biochemical changes: The infant develops both metabolic and respiratory acidosis. $PaO_2 < 50$ mm Hg and PCO_2 may rise to even 80 mm Hg in a severe case.

Normal blood gas values are: Arterial O_2 tension of 50–70 mm Hg, arterial CO_2 tension of 45–60 mm Hg, pH at or above 7.25, arterial O_2 saturation at 88–95%. Hypocalcemia and hypoglycemia can cause respiratory distress and tachypnea.

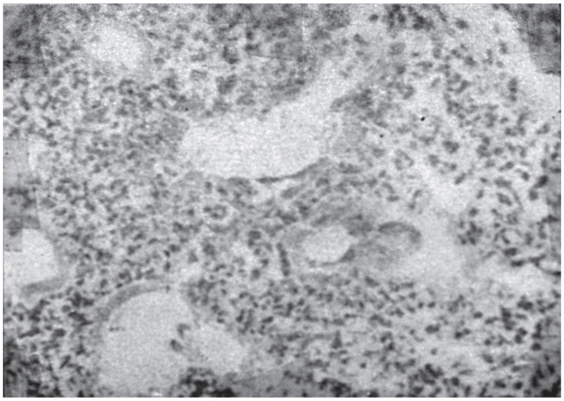
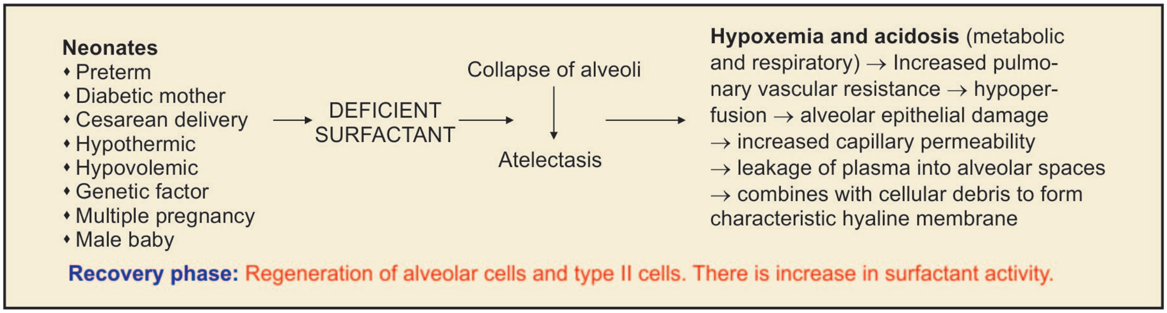


Fig. 33.3: Hyaline membrane atelectasis

OTHER INVESTIGATIONS: ♦ Sepsis workup: CBC, absolute neutrophil count (ANC), band cell, micro-ESR, CRP, blood culture are done to detect early onset of sepsis (Gr. B Streptococcus). ♦ Blood glucose, calcium levels. ♦ Serum electrolyte levels. ♦ Echocardiography to exclude PDA, congenital heart disease.

CLINICAL FEATURES: There is a wide spectrum of severity ranging from very mild transient distress to rapidly progresssing fatal illness causing death within few hours. Excess negative pressures generated to open the collapsed airways cause retraction and deformation of chest wall.


Neonates
• Preterm
• Diabetic mother
• Cesarean delivery
• Hypothermic
• Hypovolemic
• Genetic factor
• Multiple pregnancy
• Male baby
DEFICIENT SURFACTANT
Collapse of alveoli
Atelectasis
Hypoxemia and acidosis (metabolic and respiratory) → Increased pulmonary vascular resistance → hypoperfusion → alveolar epithelial damage → increased capillary permeability → leakage of plasma into alveolar spaces → combines with cellular debris to form characteristic hyaline membrane
Recovery phase: Regeneration of alveolar cells and type II cells. There is increase in surfactant activity.

The clinical manifestations usually appear abruptly, 4–6 hours after birth. **Two or more of the following features if found at examinations more than an hour apart, are enough to diagnose RDS:** (1) Respiratory rate more than 60 breaths/minute; (2) Nasal flaring; (3) Rib retraction; (4) Expiratory grunt and (5) Central cyanosis.

Chest X-ray shows uniform reticulogranular pattern known as ground glass mottling due to extensive atelectatic process.

DIFFERENTIAL DIAGNOSIS: (1) Aspiration pneumonia (liquor amnii or meconium); (2) Pneumothorax (3) Diaphragmatic hernia; (4) Congenital heart disease.

PREVENTION: (1) **Administration of betamethasone** (12 mg) to the mother two doses IM 24 hours apart especially before 34 weeks (p. 367). Cortisol acts on type II pneumocytes to stimulate phospholipid synthesis. **Benefits are obtained after 24 hours of therapy and continue for 7 days (see p. 367)**. Fetal hyperinsulinism blocks cortisol action. (2) **Assessment of lung maturity** before premature induction of labor and to delay the induction as much as possible without any risk to the fetus; (3) **Prevent fetal hypoxia** in diabetic mothers.

Decreased risk factors are: ♦ Vaginal delivery ♦ Corticosteroid ♦ Thyroid hormones ♦ Prolonged rupture of membranes ♦ Female baby

TREATMENT:

Principles of management in HMD are: (i) Prevent hypoxia and acidosis; (ii) Maintain fluid and electrolyte balance; (iii) Prevent atelectasis and pulmonary edema and (iv) Avoid lung injury (barotrauma) and infection.

- **The baby should be placed in neonatal intensive care unit** and nursed in a warm incubator with high humidity (neutral thermal condition). Air passage is cleaned periodically through endotracheal suction.
- **Adequate warmed and humidified oxygen** therapy in concentration of 35–40% under positive pressure is to be administered through endotracheal tube to relieve hypoxia and acidosis. If the arterial oxygen tension (PO_2) cannot be maintained above 50 mm Hg, application of continuous positive airway pressure (CPAP) at 5–8 cm of water is indicated.
- **Correction of hypovolemia** with albumin or other colloid solution (see p. 544).
- **Correction of anemia**, **electrolyte imbalance** if any and prevention of infection.
- **Frequent monitoring** of the arterial PO_2, PCO_2, pH and base excess are to be determined to diagnose metabolic and respiratory acidosis (see p. 548). Higher than necessary FiO_2 may cause lung injury and retinopathy of prematurity.
- **Acidosis should be corrected by** intravenous administration of sodium bicarbonate 4.2% (0.5 mEq/mL) in amount 1 mEq/kg or 2 mL/kg body weight in 1 : 1 dilution with distilled water and in minimum dosage.
- **Continuous positive airway pressure (CPAP):** Nasal (NCPAP) or nasopharyngeal (NPCPAP) is used early to delay or prevent the need for mechanical ventilation and tracheal intubation.
- **Surfactant replacement therapy** has significantly improved the outcome of the infants with HMD. Surfactant is composed chiefly of phospholipids 80% and protein 10%. It is produced and stored in the characteristic lamellar bodies of type II pneumocyte (see p. 541). Reduction of surface tension and stabilization of alveolar air-water interface is its basic function. **Surfactants** of human, bovine (survanta), porcine (poractant) or synthetic preparations have been used. Prophylactic surfactant replacement has reduced the risk of BPD, CLD and deaths. Natural surfactants are the treatment of choice. Mechanical ventilation is less often needed when the technique "Intubate-Surfactant-Extubate to CPAP" is done. **Prophylactic therapy** is given (within 15 minutes of birth) in very premature infants. Direct tracheal instillation is done through a feeding tube. Changes in positioning of the infant during therapy are done to facilitate distribution. Oxygen tension (PaO_2) is adequately maintained. Mechanical ventilation is less often

Table 33.2: The Sequelae of Perinatal Asphyxia

System	Manifestations	
	Acute	**Long-term**
Cardiac	Cardiogenic shock Papillary muscle dysfunction	
CNS	Cerebral edema Seizures Hemorrhage HIE (see p. 543)	Cerebral palsy (CP) Epilepsy Mental retardation (most CP, however is not related to birth asphyxia)
Pulmonary	Aspiration syndromes Pulmonary hemorrhage HMD	Chronic lung disease (following HMD)
Renal	Acute tubular necrosis, oliguria	Retinopathy of prematurity (see p. 530)
Adrenal	Hemorrhage	
GI system	Necrotizing enterocolitis	
Metabolic	Hypoglycemia	
Hematologic	DIC, Thrombocytopenia	

needed. **Early rescue** (within 2 hours of age) is preferable rather than delayed treatment. The dose of survanta is 4 mL (100 mg/kg phospholipid) per kg of body weight. Surfactant replacement therapy has reduced neonatal mortality and pulmonary morbidity. **Complication:** Pulmonary hemorrhage is a rare complication of surfactant therapy.

- **Mechanical Ventilation**—**Ventilator therapy—**indications are: Respiratory acidosis with a $PaCO_2 > 50$ mm Hg, a $PaO_2 < 50$ mm Hg or O_2 saturation <90% or severe apnea. Synchronized intermittent mechanical ventilation is preferred.
- Hypocapnia is associated with increased risk of BPD and CLD and periventricular leukomalacia. It should be avoided.
- **Fluid and nutrition—**Intragastric feeding, if possible, is the preferred method. If there is risk of vomiting and aspiration, intravenous administration of 10% glucose in amount of 60 mL/kg body weight per day may be given to a term baby on 1st day through a catheter inserted into peripheral or umbilical vein.
- Antibiotic therapy against common neonatal infections should be started initially.

COMPLICATIONS: Acute complications of RDS include—(i) Infection; (ii) Air leak (pneumothorax); (iii) Pneumomediastinum; (iv) Persistent patent ductus arteriosus. Other complications are: (a) intraventricular hemorrhage, (b) chronic lung disease (CLD), (c) bronchopulmonary dysplasia (BPD), (d) intracranial hemorrhage, (e) retionpathy of prematurity, (f) pulmonary hemorrhage, (g) barotrauma—pneumothorax, (h) retrolental fibroplasia and (i) neurological abnormalities.

PROGNOSIS: About one-third of the babies die. In mild affection with good vigor, the baby may survive, if acidosis and biochemical abnormalities are corrected effectively. The long-term sequelae of the infants in terms of respiratory and neurological development is dependent on the birth weight and gestational age. The major morbidities (BPD, CLD, NEC and IVH) are high for the smallest infants (Table 33.2).

MECONIUM ASPIRATION SYNDROME (MAS)

Meconium aspiration syndrome (MAS) usually occurs in **term or post-term babies who are small for gestational age (IUGR).** Overall incidence of meconium stained amniotic fluid (MSAF) varies between 8% and 20%. Chronic placental insufficiency leads to intrauterine hypoxia with passage of meconium. The meconium stained liquor may be aspirated by the fetus-in-utero or during first breath. **Pathophysiology** includes (a) airway obstruction, lung atelectasis causing hypoxia and increased pulmonary vascular resistance (PVR); (b) chemical pneumonitis; (c) pulmonary inflammation due to release of cytokines. This causes airway edema and hypoxia; (d) surfactant dysfunction and (e) development of persistent pulmonary hypertension (PPHN). Not all the infants with meconium aspiration will develop MAS. Features of respiratory distress develop immediately after birth in only 5–10% infants. The infant manifests with tachypnea, nasal flaring, intercostal retractions and cyanosis.

Diagnosis is mainly based on: (a) Aspiration of meconium from the trachea at birth; (b) Signs of respiratory distress; (c) Radiologically hyperinflated lung fields, flattened diaphragm with coarse and patchy infiltration and (d) Cyanosis.

Management: ♦ Proper intrapartum monitoring and care; ♦ Amnio infusion in oligohydramnios—may reduce cord compression, grasping and intrapartum aspiration; ♦ Maintenance of (a) Thermoneutral environment; (b) Minimum handling; (c) To correct metabolic abnormalities; (d) Circulatory support (N. Saline or whole blood) and (e) Airway and oral suctioning may be needed; ♦ Liberal oxygen supply; ♦ Antibiotic coverage, as meconium invites infection; ♦ In a severe case arterial blood gas analysis should be done; ♦ Inhaled nitric oxide or surfactant therapy may be beneficial; ♦ General management includes correction of hypoxia, acidosis, hypoglycemia and hypocalcemia. Mechanical ventilation is required where PO_2 is less than 50 mm Hg and PCO_2 is above 50 mm of Hg. **Complications** like airleak (pneumothorax), PPHN, bronchopulmonary dysplasia or chronic lung diseases are common. New modalities of therapy have reduced mortality to < 5%.

Prognosis: MAS may be associated with neurodevelopmental delay, cerebral palsy and mental retardation. Infants need long-term follow-up.

JAUNDICE OF THE NEWBORN

Yellow discoloration of the skin and the mucosa is caused by accumulation of excess of bilirubin in the tissue and plasma (serum bilirubin level should be in excess 7 mg/dL). A value > 15 mg/dL is considered severe. About 80% of term newborn and most of preterm newborns develop clinical jaundice.

Bilirubin source and metabolism: RBC hemoglobin → breakdown in RE system → biliverdin, CO (excreted by lungs) and iron (reutilized). Biliverdin is reduced to bilirubin by the enzyme biliverdin reductase. 1 g hemoglobin produces 35 mg of bilirubin. Bilirubin bound to serum albumin is transported to the liver cells → carried to the smooth endoplasmic reticulum by cytoplasmic ligandin (Y protein). **Unconjugated (indirect) bilirubin is converted to conjugated (direct) bilirubin (CB) by Uridine Diphosphate Glucuronyl Transferase (UDPG-T).** CB excreted in GI tract, is eliminated by stool. When CB is acted upon by β glucuronidase it is converted to UCB which is reabsorbed back to the liver **(enterohepatic circulation)** for reconjugation. Sulfonamides, free fatty acids can displace bilirubin from albumin. Phenobarbitone can induce the enzyme UDPG-T and catalyze the conjugation process. **Conjugated bilirubin is water soluble and nontoxic. Unconjugated bilirubin is toxic and causes neuronal dysfunction and death.**

CAUSES OF NEONATAL JAUNDICE

- **Physiological**
- **Nonphysiological**

PHYSIOLOGICAL: **The jaundice usually appears on 2nd and 3rd day and disappears by 7th–10th day, a little later in premature neonates.** In a term infant the level may be 6–8 mg/dL on 3rd day. A rise of unconjugated serum bilirubin to 12 mg/dL is in the physiological range. In a premature infant the peak level of 12–15 mg/dL in the 1st week may be without any abnormality.

Causes of excessive bilirubin production are: (1) Increased red cell volume per kg and increased red cell destruction due to shorter life span (90 days compared to 120 days in adult) in the neonate; (2) Transient decreased conjugation of bilirubin due to decreased UDPG-T activity; (3) Increased enterohepatic circulation due to decreased gut motility and high level of intestinal β glucuronidase; (4) Decreased hepatic excretion of bilirubin and (5) Decreased liver cell uptake of bilirubin due to decreased ligandin (transport protein).

Treatment: No specific treatment is required. The baby is given more frequent feeds. In premature babies, careful observation is required and evidences of rising bilirubin near critical level need exchange transfusion. However, use of phenobarbitone or phototherapy is quite useful (see p. 554) in such cases.

PATHOLOGICAL

CAUSES OF NONPHYSIOLOGIC JAUNDICE

A. Excessive red cell hemolysis

(i) **Hemolytic disease of the newborn:**

- Fetomaternal blood group incompatibilities: Rh (most common), ABO (rare), immunization against Kell antigen (rarest).
- Increased red cell fragility—Congenital spherocytosis.
- Deficient red cell enzyme—Glucose-6-phosphate dehydrogenase (G6PD deficiency and drugs).

(ii) **Sepsis**: Intrauterine (*Toxoplasma, Rubella*), Neonatal (*E. coli*), Omphalitis.

(iii) **Blood extravasation** (cephalhematoma, intraventricular hemorrhage).

Absolute Features of Nonphysiologic Jaundice

- Jaundice appearing **within 24 hours of birth**.
- **Bilirubin level** increasing at the rate of > 5 mg/dL/day or >0.2 mg/dL/hour.
- Conjugated bilirubin > 2 mg/dL.
- **Clinical jaundice** persisting > **1 week** in a term infant or > 2 weeks in a preterm infant.
- Jaundice requiring phototherapy
- Sick baby

B. Defective conjugation of bilirubin

(i) **Congenital deficiency of glucuronyl transferase**

- Crigler-Najjar syndrome (autosomal recessive), Gilbert syndrome (autosomal dominant), Preterm babies with impaired liver function.

C. Breast milk jaundice: The activity of the enzyme-glucuronyl transferase is inhibited by a specific steroid 3α, 20β–pregnanediol and increased fatty acids of breast milk. The bilirubin level rises from the 7th day after birth to a maximum of 20–30 mg/dL by 14th day. Jaundice is usually mild and it takes a time (4–12 weeks) to disappear. It rarely causes kernicterus. It requires no treatment. If the bilirubin level is more, temporary withdrawal of breastfeeding cures jaundice. Breast milk jaundice may recur in 70% of future pregnancies. **Breastfeeding jaundice** is due to decreased intake of milk that leads to increased enterohepatic circulation.

D. Metabolic and endocrine disorders: Galactosemia, hypothyroidism (unconjugated hyperbilirubinemia).

Galactosemia—There is hereditary deficiency of an enzyme—**galactose-1-phosphate uridyl transferase** which converts galactose derived from the milk into glucose-1-phosphate. As a result there is increased accumulation of galactose that leads to periportal fibrosis and cirrhosis of liver. The biliary canaliculi are blocked by inspissated bile and obstructive jaundice results. Baby develops jaundice, hepatomegaly and feeding intolerance. A reducing substance (lactose) is detected in the urine by clinistix. **Breastfeeding is contraindicated** because of high lactose content. **Lactose free milk feeding should be recommended**.

E. Increased enterohepatic circulation of unconjugated bilirubin: Duodenal atresia, pyloric stenosis, less frequent feeding.

F. Substances and disorders that affect binding of bilirubin to albumin: Aspirin, sulfonamides, fatty acids and asphyxia, acidosis, sepsis, or hypothermia increases free unconjugated bilirubin level.

G. Miscellaneous: Congenital obstruction (atresia or stricture of biliary canaliculi), asphyxia, polycythemia and thalassemia.

HYPERBILIRUBINEMIA OF THE NEWBORN

When the bilirubin (unconjugated) level rises more than the arbitrary cut-off point of 12 mg/dL, in a term infant the condition is called "hyperbilirubinemia of the newborn". The face and chest of the infant are usually stained yellow at this level of serum bilirubin.

- ***Unconjugated:*** Hemolytic disease due to Rh (common) or ABO (rare) incompatibility, increased red cell fragility (spherocytosis), prematurity, glucose-6-phosphate-dehydrogenase deficiency, sepsis, iatrogenic (drugs), breast milk jaundice, cephalhematoma, hemoglobinopathy, infant of diabetic mother, hypothyroidism, idiopathic, etc.
- ***Conjugated:*** Neonatal hepatitis, bacterial infection, intrauterine TORCH infection, Trisomy 21, 18; galactosemia, cystic fibrosis, biliary atresia, etc.

DIAGNOSIS OF NEONATAL HYPERBILIRUBINEMIA

A. **Clinical:** Evaluation of jaundice is done by blanching the skin with digital pressure. Clinical jaundice in a neonate indicates serum bilirubin of more than 5 mg/dL. Cephalocaudal progression of dermal icterus is a useful clinical tool (see Fig. 33.4).

Dermal icterus zone and serum bilirubin (indirect) level in a term infant (Kramer—1969).

Dermal zone	Bilirubin (mg/dL)
1	5
2	10
3	12
4	15
5	> 15

A thorough physical examination of the infant is done. Abnormal neurologic signs are: lethargy, poor feeding, vomiting, hypotonia and seizures.

B. **Laboratory studies:**

- **Serum bilirubin level > 12 mg/dL, requires further investigations.**
  1. **Blood group (ABO, Rh) status**—mother and infant. Antibody screen of the mother.

2. **Direct Coombs' test** (infant)—for alloimmunization disorder. Positive → Antibody study (Rh, ABO, Kell)
3. **Total bilirubin, conjugated bilirubin and unconjugated bilirubin.**
4. **Complete hemogram including reticulocyte count:**
    - **Hemolytic anemia:** ↓ Hb%, ↑ Reticulocyte count, presence of nucleated red cells.
    - **Sepsis:** WBC count (total and differential) ↓, Neutrophil ↓ Band cell, micro ESR↑, CRP↑
    - **Polycythemia:** Hematocrit (> 65%) ↑
    - **Hereditary spherocytosis**: Peripheral blood film ↑ reticulocyte count
5. **Serum albumin** to detect total bilirubin binding sites and to assess the need of albumin infusion.
6. **Other laboratory tests:**
    - **Urine** for reducing substance (galactosemia), culture for infection.
    - **Hemoglobin electrophoresis**
    - **Osmotic fragility tests**
    - **Thyroid and liver function tests**
    - **G6 PD screening**
    - **LFT: (AST, ALT, PT)**

C. **Radiology and Ultrasonography** to detect intestinal obstruction, intraventricular hemorrhage and tumor.

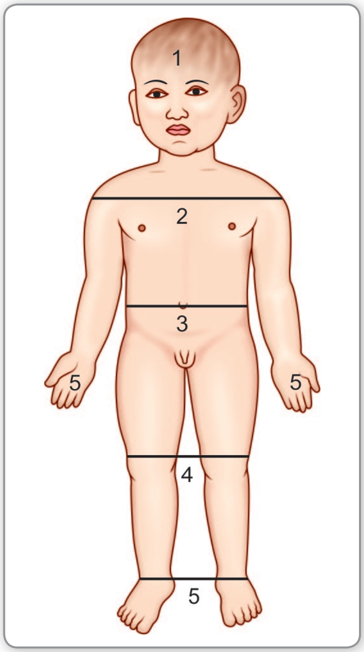


Fig. 33.4: Dermal icterus zone and serum bilirubin (indirect) level in a term infant (Kramer-1969)

COMPLICATION (unconjugated)— **Kernicterus** is the important complication which is more often fatal, if not promptly detected and adequately treated.

KERNICTERUS

Kernicterus is a pathological condition characterized by yellow staining of the brain by unconjugated bilirubin resulting, in **neuronal injury**. Basal ganglia, cranial nerve nuclei, hippocampus, brainstem nuclei and anterior horn cells of the spinal cord are commonly affected. The critical level of bilirubin causing kernicterus in a term infant is more than 20 mg/dL (340 µmol/L). **Bilirubin enters the brain in free (unbound) form.** Risk of bilirubin encephalopathy is unlikely, if the total bilirubin level is < 20 mg/dL.

Hypoxia, acidosis, hypoglycemia, hypothermia, sepsis or prematurity enhances the pathogenesis so that affection may occur even at a low level of bilirubin. **Excess rise of conjugated bilirubin cannot produce kernicterus.**

It is clinically characterized by lethargy, hypotonia, poor feeding and loss of neonatal reflexes. Gradually hypertonia, severe illness is manifested by prostration, respiratory distress and finally opisthotonos, hyperpyrexia, convulsions, enlarged liver, spleen and chronic bilirubin encephalopathy. The jaundice is intense along with anemia in babies of hemolytic disease.

Prevention and management: Regular and periodic estimation of serum bilirubin level in susceptible babies and the tendency to rise should be effectively tackled by double surface phototherapy and exchange transfusion. Use of barbiturate and phototherapy gives encouraging results (see below).

Prognosis: Neonates with overt neurological features usually die (75%). Those who survive often suffer from mental retardation and choreoathetoid cerebral palsy.

MANAGEMENT OF JAUNDICE IN THE NEWBORN

Three methods of treatment are used to reduce the level of **unconjugated bilirubin: (1) Phototherapy (2) Pharmacologic therapy** and **(3) Exchange transfusion**.

(1) **Phototherapy**—(see p. 397). It is best when used in moderate cases where the bilirubin level rises above 12 mg%. Skin color is not a reliable guide to assess the response and hence periodic bilirubin estimation should be done. Phototherapy is discontinued when serum bilirubin level is <13mg/dL in term and <11 mg/dL in preterm neonates. A rebound increase in serum bilirubin may occur once phototherapy is stopped. Adequate hydration of the neonate has to be maintained. **Special blue lamps** with an output of 420–480 nm wavelengths are the most effective. **Double Phototherapy** (over head light-plus light from below or fiberoptic blanket) is twice as effective as single phototherapy. Bilirubin (indirect) absorbs light maximally at that range and undergoes photoisomerization and is converted to less toxic polar isomer (4Z, 15E) which is excreted into the bile. Phototherapy also converts bilirubin to lumibilirubin by structural isomerization. Lumibilirubin is excreted in the bile and urine without conjugation.

Phototherapy should be started early, exposing the maximum surface area and shielding the eyes. It may be continuous or interrupted for breastfeeding. Phototherapy causes increased insensible fluid loss of the neonate. **Oral hydration** with frequent breast milk is encouraged. IV fluid therapy or nasogastric feeding may be needed. Phototherapy blankets protect the infants. **Complications of phototherapy are:** Watery diarrhea, skin rashes, dehydration, bronze baby syndrome (dark brown discoloration of the skin), low calcium levels and retinal damage.

Prophylactic phototherapy in LBW or severely bruised infants or with hemolytic disease may be indicated. Phototherapy is contraindicated in infants with direct hyperbilirubinemia caused by liver disease or obstructive jaundice.

(2) **Phenobarbital therapy** induces hepatic microsomal enzymes and increases bilirubin conjugation and excretion. A loading dose of 10 mg/kg on day 1 and maintenance dose of 5–8 mg/kg/day for next 4 days is given. It takes 3–7 days to be effective. However, as a prophylaxis, it may be used in the mother for 2 weeks prior to delivery in the dose of 90 mg/day. Phenobarbital is used to treat indirect hyperbilirubinemia of Crigler-Najjar syndrome type II.

(3) **Metalloporphyrins** (Sn MP, Zn MP) decreases bilirubin production by inhibiting heme oxygenase. Tin and zinc metalloporphyrins are currently used. By competitive inhibition, metalloporphyrins decrease the production of bilirubin.

(4) **Exchange transfusion** is used to prevent kernicterus. **Double-volume exchange** replaces 85% of circulating red blood cells and reduces bilirubin level by 50%.

(5) **Oral agar** significantly increases the efficiency and shortens the duration of phototherapy by decreasing enterohepatic circulation.

Indications of Exchange Transfusion

- When there is progressive rise of bilirubin (>1 mg/dL/hour) in spite of phototherapy
- Rate of bilirubin rise >0.5mg/dL/hour despite phototherapy when Hb is between 11–13 g/dL
- To improve anemia and congestive cardiac failure of the neonate
- The serum bilirubin level of the infant is >12 mg/dL in first 24 hours and >20 mg/dL in neonatal period
- Cord blood hemoglobin is <11 g/dL and bilirubin level is >5 mg/dL
- Progressive anemia of the neonate.
- When phototherapy fails to prevent the rise in bilirubin to toxic levels.

In nonimmune hyperbilirubinemia blood is typed and crossmatched with the infant. **Two-volume exchange** is usually done. If the newborn's blood volume is 80 mL/kg then 160 mL/kg of blood is used for exchange transfusion.

Complications of exchange transfusion: Air embolism, thrombosis, hypervolemia, RDS, hypothermia, acidosis, infection, hyperkalemia, hypocalcemia, hypoglycemia, cardiac arrhythmias, thrombocytopenia, coagulopathies and necrotizing enterocolitis.

HEMOLYTIC DISEASE OF THE NEWBORN

This is due to hemolysis of the fetal red blood cells (RBCs) due to passively acquired maternal antibodies. Hemolysis is manifested by a decreased hematocrit, increased reticulocyte count and an increased bilirubin level. The common causes are:

A. Immune hemolysis	B. Inherited RBC disorders	C. Acquired hemolysis
Rh incompatibility (p. 388)	RBC membrane defects (Spherocytosis, elliptocytosis)	Infection: bacterial, viral, parasitic (Rubella, syphilis)
ABO incompatibility		
Other blood group incompatibility (C, E, Kell, Duffy)	Metabolic: G6PD deficiency Systemic diseases (Galactosemia)	DIC Acute transfusion hemolysis
Maternal diseases (lupus); drugs	Hemoglobinopathies (α- and β-thalassemia syndrome)	Vitamin E deficiency Drugs (Vitamin K, nitrofurantoin)

Management is to treat this underlying primary disorder, simple transfusion, exchange transfusion and nutritional support (Fe, Folate, Vitamin E).

ABO GROUP INCOMPATIBILITY

Principle: The mother with blood group O has got naturally occurring anti-A and anti-B agglutinins. These antibodies are mainly IgM and do not cross the placenta. If the fetus happens to be blood group A or B corresponding to that of father, the immune antibodies are formed in response to the entry of A or B antigen bearing fetal red cells, into the maternal circulation. As these are mainly IgG, they can cross across the placenta into the fetal circulation and cause a variable amount of hemolysis due to antigen-antibody reaction. **Although 15% of the babies have got ABO blood group incompatibility only in <1% hemolysis occurs.** Positive direct Coombs' test occurs in only 3–4% cases.

In contrast to Rh-incompatibility, the first baby is affected (50%). As intrapartum 'boosting' of immune anti-A and anti-B antibodies does not occur, the progressive severe affection in successive babies is unlikely. The jaundice is usually mild appearing within 24 hours. The affection is less because the antibodies have got other tissue binding sites apart from the fetal red cells. The diagnosis is made only after birth. The baby is either blood group A or B, while the mother is group O. Specific anti-A or anti-B antibody can be detected from the maternal serum. Positive direct Coombs' test occurs in only 3–4% cases. Microspherocytes on blood smear is characteristic. Reticulocyte count is increased. RBCs have increased osmotic fragility. No treatment is usually required. Adequate hydration is maintained and sepsis is avoided. Phototherapy may be required in few (10%) cases. Need of exchange transfusion is extremely rare. Overall prognosis is excellent.

BLEEDING DISORDERS IN THE NEWBORN

It is a syndrome characterized by spontaneous internal or external bleeding. In neonates, there is decreased activity of several clotting factors and diminished platelet function. Hence, the causes may be different:

(A) **Abnormalities of clotting factors:** (i) Deficiencies of vitamin K dependent factors-II, VII, IX, X. It usually occurs between D2 and D5 and common in preterm and breastfed babies. Deficiency of protein C is also responsible (ii) **Drugs**—received by mother during pregnancy—phenytoin, warfarin compounds, rifampicin, salicylates (affect vitamin K function).

(B) **Abnormality in clotting:** DIC, due to infection, anoxia, shock and NEC.

(C) **Platelet problems:** Qualitative (thrombasthenia) or quantitative (thrombocytopenia).

(D) **Inherited abnormalities of blood coagulation:** Hemophilia A (Factor VIII levels are decreased), hemophilia B (Christmas disease-deficiency of factor IX), Von Wille-brand disease (VWD) due to decreased levels and functional activity of Von Wille-brand factor (VWF).

(E) **Trauma:** Obstetric trauma causing cephalhematoma, visceral (rupture of liver, spleen) injury, umbilical cord rupture or slipping of cord ligature.

(F) **Others:** Liver dysfunction, Vitamin K deficiency (breastfed babies).

Diagnosis—Evaluation is done by: **A. History; B. Clinical examination** and **C. Laboratory tests**

- **Laboratory tests** complete blood count, blood smear, platelet count and reticulocyte count. Significant bleeding is usually observed with platelet counts under 20,000–30,000/mm^3 or less.
- Prothrombin time (PT), partial thromboplastin time (PTT), Fibrinogen, d-Dimer test (normal level < 0.5 μg/mL, raised level in DIC), and specific assays and Von Wille-brand panels are done where family history is positive.

Treatment depends upon the underlying cause.

(i) Vitamin K_1 (Aquamephyton)—1 mg IV or IM may be repeated weekly

(ii) Fresh frozen plasma 10 mL/kg IV, may have to be repeated. FFP replaces clotting factors immediately

(iii) Platelets concentrate to raise the count from 50,000/mm^3 to 100,000/mm^3

(iv) Fresh whole blood—10 mL/kg, or more may be used for single transfusion (see p. 396)

(v) Clotting factor concentrates may be given when there is a known deficiency (e.g. factor VIII, IX or VMD), to stop bleeding

(vi) Treatment of specific problems, e.g. sepsis, DIC

Prognosis is favorable if the blood loss is less and the treatment is promptly initiated.

Management of cord bleeding: (1) Slipping ligature is to be tackled by religation; (2) If bleeding occurs following separation of the cord, hemostasis may be achieved by pressure or by a ligature after securing the bleeding points with a pair of artery forceps. Even a small amount of blood loss may be lethal and should be replaced by blood transfusion, if necessary.

ANEMIA IN THE NEWBORN

At birth normal values of hemoglobin (central venous) in infants > 34 weeks gestation are 14–20 g/dL with an average value of 17 g/dL.

DEFINITION: Central venous hemoglobin level < 13 g/dL in an infant of > 34 weeks gestation is considered anemia.

PATHOPHYSIOLOGY: Anemia in the newborn infant may be due to any one of the three pathologies: (a) Physiologic anemia of infancy is due to shorter life span of RBCs and less erythropoietin production; (b) Loss of RBCs (**Hemorrhagic anemia**); (c) Destruction of RBCs (**Hemolytic anemia**) (p. 554) or (d) Under production of RBCs (**Hypoplastic anemia**).

CAUSES: (1) **Hemorrhagic anemia: A. Obstetric causes:** (a) Abruptio placenta; (b) Placenta previa; (c) Traumatic rupture of umbilical cord; (d) Obstetric trauma (difficult delivery with visceral or intracranial hemorrhage); (e) Twin-twin transfusion; (f) Delivery of the baby by cesarean section after cutting through the placenta in anterior placenta previa; (g) Ruptured vasa previa; (h) Excessive fetomaternal bleed; (i) Anemia of prematurity; (j) Hereditary RBC disorders—hemoglobinopathies. **B. Neonatal causes:** (a) Caput succedaneum; (b) Cephalhematoma; (c) Intracranial hemorrhage; (d) Visceral hemorrhage (spleen, kidneys and adrenals); (e) DIC; (f) Thrombocytopenia and (g) Hemorrhage due to deficient vitamin K dependent factors (II, VII, IX and X). (2) **Hemolytic anemia** (see p. 554). (3) **Under production of RBCs:** (a) Congenital hypoplastic anemia, (b) Leukemia and (c) Infections (Rubella, Syphilis).

Diagnosis: (1) **History:** (a) Family history of bleeding and (b) Maternal medications (phenytoin, warfarin), (2) **Clinical examination** (jaundice, spleenomegaly, skin bruises) and (3) **Laboratory tests:** Complete blood count, RBC indices, blood smear, reticulocyte count, Kleihauer Betke test, coagulation profile (p. 744), intrinsic RBC defect, TORCH study and ultrasound of abdomen and head.

TREATMENT: Treatment of anemia in neonates, involves treatment of the underlying primary disorder along with blood transfusion, exchange transfusion and nutritional support. (1) Replacement transfusion in neonates with hemorrhagic anemia (hematocrit < 35%); (2) Exchange transfusion; (3) Oral iron in suspension (2–4 mg elemental iron/kg) and folic acid 50 mcg/day vitamin E 25 IU/day have to be continued for a longer period; (4) Recombinant human erythropoietin (rh-EPO) for anemia of prematurity; (5) **Exchange transfusion**—in neonates with (i) hemolytic or hemorrhagic anemia with raised CVP (ii) Rh incompatibility. **Treatment** of selected disorders (e.g. consumption coagulopathy, immune thrombocytopenia).

Prognosis depends on the basic underlying pathology and its severity.

SEIZURES IN NEWBORN

A seizure is a paroxysmal manifestation of neurological dysfunction (i.e. behaviors, motor or autonomic function). Overall incidence ranges from 2 in 1,000 to 14 in 1,000 live births. **Pathophysiology:** The basic mechanism is excessive depolarization (excitation) of neurons within the CNS. **Three possible reasons** for excessive depolarization are proposed: (A) Failure of sodium-potassium pump operation; (B) Relative excess of excitatory neurotransmitter compared to the inhibitory ones. (C) Relative deficiency of inhibitory neurotransmitters. The neurons within the CNS undergo depolarization due to inward migration of sodium and repolarization due to efflux of potassium.

Neonatal convulsion is usually a visible manifestation of some underlying pathology. Rarely, a baby may have convulsion where the cause cannot be detected. **The common causes of convulsion in the newborn are:**

Traumatic	Metabolic	Infective	Iatrogenic	Others (congenital)
♦ Perinatal asphyxia ♦ Hypoxic—ischemic encephalopathy ♦ Intracranial hemorrhage (Subarachnoid, peri- or intraventricular or subdural hemorrhage)	♦ Hypoglycemia ♦ Kernicterus ♦ Hypocalcemia ♦ Hypercalcemia ♦ Hyponatremia ♦ Hypomagnesemia ♦ Pyridoxin dependence ♦ Inborn errors of metabolism	♦ High fever ♦ CNS infection due to: ♦ Group B *Streptococcus* ♦ *E. coli* ♦ TORCH infection ♦ Tetanus	♦ Narcotic withdrawal ♦ Drug toxicity–theophylline ♦ Respiratory stimulants	♦ Cerebral malformation ♦ Neonatal epileptic syndromes ♦ Chromosomal syndromes ♦ Unknown

Risk factors: (a) Prematurity, (b) Birth weight <2.5 kg, (c) Maternal age ≥40 years, (d) Traumatic delivery (forceps).

DIAGNOSIS

*History***:** Details of delivery, Apgar score at birth, birth weight, gestational age, breastfeeding or not, maternal drug history, family history of seizures, inborn errors of metabolism, withdrawal of narcotic drugs.

INVESTIGATIONS: Laboratory studies

- Full blood count • Blood, Urine and CSF cultures • Serum IgM and IgG— specific TORCH titers
- Blood biochemical—estimation for glucose, ammonia, calcium, magnesium, bilirubin and electrolytes, if needed
- Blood gas levels to detect acidosis and hypoxia

Imaging studies: Ultrasonogrpahy and CT scan of the head—to detect intraventricular and/or subarachnoid hemorrhage. They are also useful to detect any congenital malformation. **MRI** – congenital abnormalities (lissencephaly, IVH, HIE); **EEG** – is informative both for diagnosis and prognosis.

TREATMENT: The principles of treatment are: (1) To control convulsions; (2) To stabilize the vital functions; (3) To treat the underlying pathology and (4) To maintain supportive therapy—nutrition, ventilation, cardiac output, serum electrolytes and pH. Neurologic consultation should be done.

To control convulsions: Intravenous administration of phenobarbitone 20 mg/kg body weight slowly over a period of 20 minutes is effective. A maintenance dose of 3–4 mg/kg body weight per day administered orally or IV for at least a period of 2 weeks or even longer. In resistant cases IV phenytoin (Dilantin), 15–20 mg/kg at the rate of 1 mg/kg/min is administered. Maintenance dose of 3–4 mg/kg/day is divided 12 hourly. Fosphenytoin is preferred.

To treat the underlying pathology:

Hypoglycemia: Glucose infusion, 2 mL/kg of 10% glucose, through an intravenous line is given over 2–3 minutes. Glucose infusion is continued at a rate of 6–8 mg/kg/min. Blood glucose should be maintained at 70–100 mg/dL.

Hypomagnesemia: Magnesium sulfate (0.4–0.8 mq/kg) is given IV every 12 hours until magnesium level is normal.

Infection: Appropriate antibiotic therapy following complete septic work-up (see p. 564).

Hypocalcemia: Intravenous administration of 2 mL/kg of 10% calcium gluconate taken over 5 minutes. This is to be followed by oral calcium 40–50 mg/kg/day for few days.

Pyridoxine deficiency: Intravenous administration of 100 mg pyridoxine is effective. **To relieve intracranial tension**—0.5–1 mL/kg of 20% mannitol is given intravenously over 30–60 minutes.

Prognosis varies with the etiology. Convulsions due to transient or metabolic disorders (hypocalcemia) have an excellent prognosis whereas seizures secondary to congenital malformations, HIE, have poor outcome. The overall mortality rate has decreased but neurological sequelae (neurodevelopmental impairment, CP, epilepsy) are still around 30–40%.

BIRTH INJURIES OF THE NEWBORN

Birth injuries is an impairment of the infant's body function or structure due to adverse influences that occurred at birth. Injury commonly occurs during labor or delivery. Birth injuries may be severe enough to cause neonatal deaths, still births or number of morbidities.

BIRTH INJURIES

High-risk Factors

- Prolonged or obstructed labor (p. 463)
- Fetal macrosomia (p. 329)
- Cephalopelvic disproportion (p. 409)
- Very low birth weight infant
- Abnormal presentation (breech) (p. 434)
- Instrumental delivery (forceps or ventouse)
- Difficult labor
- Shoulder dystocia (p. 469)
- Inadequate maternal pelvis
- Oligohydramnios
- Fetal anomalies (p. 468)
- Precipitate labor (p. 420)
- Manipulative delivery (IPV) (p. 665)

Types of Injury	Organ(s) Affected
Soft tissue	Skin—Lacerations, abrasions, fat necrosis, petechiae
Muscle	Sternocleidomastoid (p. 561)
Nerve	Facial nerve, Brachial plexus, Duchenne Erb (C_5, C_6), Klumpke (C_7, C_8, T_1), Spinal cord, Phrenic nerve (C_3, C_4 or C_5), Horner's syndrome, recurrent laryngeal nerve
Eye	Hemorrhages: Subconjunctiva, vitreous, retina
Viscera	Rupture of liver, adrenal gland, spleen testicular injury
Scalp	Laceration, abscess, hemorrhage, caput succedaneum
Dislocation	Hip, shoulder, cervical vertebrae
Skull	Cephalhematoma, subgaleal hematoma, fractures
Intracranial	Hemorrhages—Intraventricular, Subdural, subarachnoid
Bones	Fractures—Mandible, Clavicle, Humerus, Femur, Skull and Nasal bones

Injuries may involve (i) soft tissue (most common) and/or (ii) Bones (rare)

INJURIES TO THE HEAD

CEPHALHEMATOMA: It is a collection of blood in between the pericranium and the flat bone (subperiosteal) of the skull, usually unilateral and over a parietal bone (Fig. 33.5). It is due to rupture of a small emissary vein from the skull and may be associated with fracture of the skull bone. This may be caused by forceps delivery but may also be met with following a normal labor. Ventouse application does not increase the incidence of cephalhematoma. **It is never present at birth** but gradually develops after 12–24 hours. **The swelling is limited by the suture** lines of the skull as the pericranium is fixed to the margins of the bone (Fig. 33.5). It is circumscribed, soft, fluctuant and incompressible. There may be underlying fracture of the skull. In course of time, a hard sharp edge can be felt surrounding the swelling due to organization of the blood. It can cause hyperbilirubinemia when extensive and the infant may need blood transfusion. CT scan should be done if neurologic symptoms are present. Anemia and hyperbilirubinemia may be treated if needed. The condition may be confused with caput succedaneum (p. 98) or meningocele. **Meningocele always lies over a suture line or fontanel and there is impulse on crying**. The blood is absorbed in course of time (6–8 weeks) leaving an entirely normal skull. **Prognosis is good**. Rarely, suppuration occurs. No active treatment is necessary. Prevention of infection and avoidance of trauma are important. A head CT should be obtained if neurological symptoms are present.

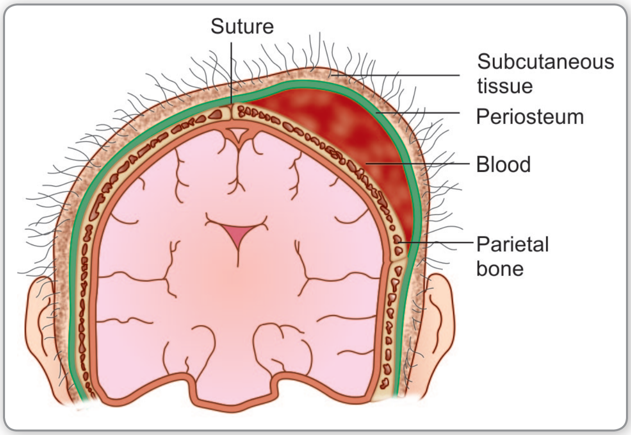


Fig. 33.5: Cephalhematoma

SCALP INJURIES: ***Minor injuries*** of the scalp such as abrasion in forceps delivery (tip of the blades), incised wound inflicted during cesarean section, scalp-electrode placement or episiotomy may be met with. On occasion, the incised wound may cause brisk hemorrhage and requires stitches. The wound should be dressed with an antiseptic solution like 2% mercurochrome.

Fracture skull: Fracture of the vault of the skull (frontal or anterior part of the parietal bone) may be of linear or depressed type. **Fractures are due to:** (1) Effect of difficult forceps delivery in disproportion or due to wrong application of the forceps (blades not placed over the biparietal diameter); (2) Projected sacral promontory of the flat pelvis may produce depressed fracture even though the delivery is spontaneous.

The fracture may be associated with cephalhematoma, extradural or subdural hemorrhage or a hematoma or brain contusions. Linear fracture if uncomplicated is usually symptomless. Depressed fracture may occasionally cause pressure effect. Neurological manifestations may occur later on due to effect of compression. Treatment is conservative in symptomless cases. Neurosurgical consultation should be obtained in presence of symptoms and X-ray or CT studies are needed. The depressed bone has to be elevated or subdural hematoma may have to be aspirated or excised surgically.

INTRACRANIAL HEMORRHAGE (ICH)

♦ **Traumatic** ♦ **Anoxic** ♦ **Primary hemorrhagic disease (p. 555)**

Intracranial hemorrhage (ICH) may be—(a) External to the brain (epidural, subdural or subarachnoid spaces); (b) in the parenchyma of brain (cerebrum or cerebellum) and (c) into the ventricles from subependymal germinal matrix or choroid plexus.

Very common is subdural hemorrhage (70%), then subarachnoid (20%) → intracerebral (20%) → intraventricular and epidural hemorrhage. The most common ICH in preterm infants is bleeding from the germinal matrix and that may result in intraventricular or periventricular hemorrhage.

Traumatic

- **In epidural hemorrhage** blood collects between inner skull and the dura mater. It is rare in newborn. Usually associated with fracture skull bone (described earlier).
- **Subdural hemorrhage (SDH)** is the accumulation of blood between the dura and arachnoid membrane.
    - **Slight hemorrhage may occur following:** (i) fracture of skull bone; (ii) rupture of the inferior sagittal sinus or; (iii) rupture of small veins leaving the cortex. The hemorrhage, so occurring, produces hematoma which may remain stationary or increase in size. **Neurological symptoms** may appear acutely or may have insidious onset, like vomiting, irritability and failure to gain weight. **Hydrocephalus and mental retardation may be a late sequelae.**

- **Massive hemorrhage:** Massive subdural hemorrhage usually results from—(1) Tear of the tentorium cerebelli thereby opening up the straight sinus or rupture of the vein of Galen or its tributaries (2) Injury to the superior sagittal sinus. **Clinical presentation:** Nuchal rigidity, coma, apnea, bulging fontanel (increased intracranial pressure) nonreactive pupils, seizures may be present.

Mechanism of tentorial tear: Normally, the falx cerebri is attached to the tentorium cerebelli and both help in anchoring the base of the skull to the vault. During excessive molding, there is compression of the diameter of engagement (occipitofrontal in deflexed head) with elongation of the diameter at right angle to it (mento-vertical). This results in upward movement of the vault from the base. As a result, too much strain is put on the vertical fibers of tentorium cerebelli—called stress fibers. If the molding is excessive or applied suddenly, these fibers are torn. As a result, it allows excessive elongation of the vault until the tear extends to involve the straight sinus or vein of Galen or its tributaries. The resulting hemorrhage may be supratentorial or subtentorial (Fig. 33.6).

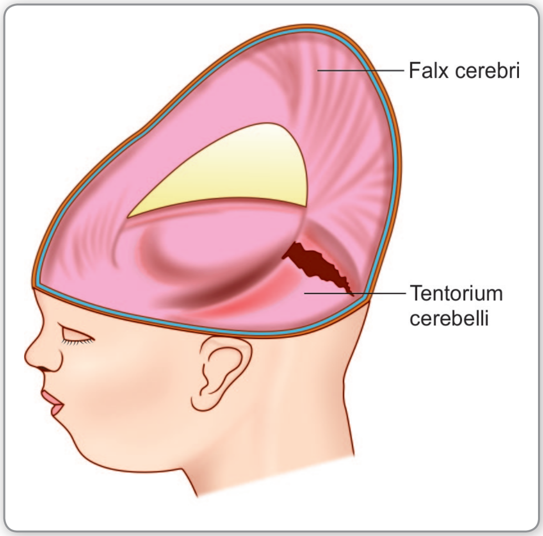


Fig. 33.6: Excessive molding of the head with marked elongation of the mentovertical diameter resulting in tear of the tentorium cerebelli

Causes: (i) Excessive molding in deflexed vertex with gross disproportion; (ii) Rapid compression of the head during delivery of the aftercoming head of breech or in precipitate labor and (iii) Forcible forceps traction following wrong application of the blades (other than biparietal diameter).

Clinical features: The hemorrhage may be fatal and the baby is delivered stillborn or with severe respiratory depression. In lesser affection, the baby recovers from the respiratory depression. Gradually, the features of cerebral irritation appear, such as, frequent high pitch cry, neck retraction, incoordinate ocular movements, convulsion, vomiting and bulging of the anterior fontanel.

Subarachnoid hemorrhage (SAH) is an accumulation of blood between the arachnoid and the pia mater. It is due to the rupture of small vessels due to birth trauma or birth asphyxia. It may be idiopathic and significant at times. The symptoms may appear late (1 week). Clinical presentations are: seizures, irritability and lethargy with focal neurological signs.

Anoxic

- **Intracerebral intraparenchymal hemorrhage:** It is also known as periventricular hemorrhagic infarction (PVHI). It is due to venous thrombosis and/or stasis. PVHI is seen following perinatal hypoxic ischemic event.
- **Intraventricular Hemorrhage (IVH)/Germinal Matrix Hemorrhage (GMH)**—The pathogenesis of IVH in the term infant is more likely due to trauma (difficult delivery) or perinatal asphyxia. In the preterm infant GMH/IVH is mainly due to ischemia/reperfusion. Clinical presentation is extremely diverse: clinically silent, seizures, apnea, irritability, lethargy, vomiting or a full fontanel.

  Diagnosis is made invariably by neuroimaging studies: Real time portable cranial ultrasonography (CUS) is the procedure of choice in the term newborn. IVH is diagnosed by head CT or CUS. MRI is also helpful.

Risk factors for GMH/IVH: Extreme prematurity, birth asphyxia, the need for vigorous resuscitation at birth, presence of neonatal seizures and sudden elevation of blood pressure. PVH, IVH and PVL are the most common neurologic complications of prematurity. GMH/IVH originates from the fragile involuting vessels of the subependymal germinal matrix.

PREVENTION: Comprehensive antenatal and intranatal care is the key to success in the reduction of intracranial injuries.

Antenatal prevention of IVH/GMH: (1) Tocolysis with indomethacin should be avoided; (2) *In utero* transfer of preterm labor to a center with NICU; (3) Cesarean delivery before active phase of labor in preterm infants; (4) Antenatal steroids can reduce the risk by threefold; (5) **To prevent or to detect at the earliest**, intrauterine fetal asphyxia by intensive fetal monitoring; (6) **To avoid traumatic vaginal delivery** in preference to cesarean section. Difficult forceps should be avoided and (7) **Administration of vitamin K 1 mg intramuscularly** soon after birth in susceptible babies.

Postnatal prevention: Avoid birth asphyxia, fluctuation of blood pressure and correct acid base abnormalities. Surfactant therapy is found helpful.

INVESTIGATIONS: (1) Ultrasonography is used to detect intraventricular hemorrhage; (2) Doppler ultrasonography can detect any change in cerebral circulation; (3) CT scan is useful to detect cortical neuronal injury; (4) Magnetic resonance imaging (MRI) is used to evaluate any hypoxic ischemic brain injury and (5) CSF—Elevated RBCs, WBCs and protein.

MANAGEMENT: **Prevention**: Antenatal glucocorticoids reduce GMH/IVH.

Supportive care: To maintain normal circulatory volume, cerebral perfusion, serum electrolytes and blood gases. Packed red blood cells transfusion may be needed where IVH is large. Thrombocytopenia and coagulation parameters should be corrected, seizures should be treated.

TREATMENT:

- Follow-up with serial neuroimaging cranial ultrasound (CUS or CT) to detect any progressive hydrocephalus.
- **Anticonvulsant**—Any of the following may be useful—(a) Phenobarbitone—20 mg/kg IV as loading dose, followed by 3–4 mg/kg/day in divided doses at 12 hourly intervals IV or orally; (b) Phenytoin 20 mg/kg intravenously as loading dose at the rate of 0.5 mg/kg/min followed by maintenance dose of 3–4 mg/kg/day with cardiac monitoring.
- **Subdural hematoma**—(a) Subdural tap—Aspiration of the blood through lateral angles of the anterior fontanel may be required which may have to be repeated; (b) Open surgical evacuation—Serial CT is indicated before surgical intervention. The infant should be monitored for any hydrocephalus. Surgical removal of the clot including the capsule may have to be done to prevent development of neurological sequelae and (c) Rarely subdural-peritoneal shunting may be needed. Neurosurgeon is consulted.

PROGNOSIS: **Prognosis** of GMH/IVH depends on the severity of IVH, brain lesions, birth weight and gestational age of the infant. The surviving infants usually behave normally in later life. There is, however, some correlation with mental retardation and neurological disorders. Epilepsy may develop later in life.

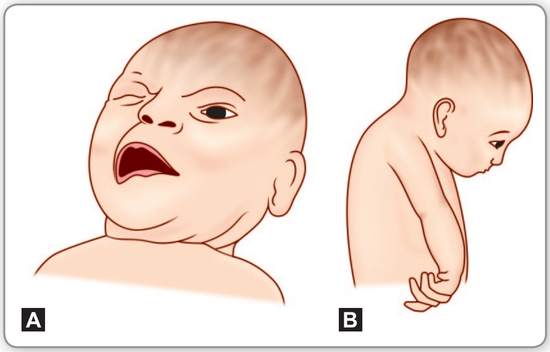
OTHER INJURIES

SKIN AND SUBCUTANEOUS TISSUES: Bruises and lacerations on the face are usually caused by forceps blades. These are treated with application of 1% lotion mercurochrome. Buttocks in breech presentation, or eyelids, lips or nose in face presentation, similarly become edematous and congested. No treatment is required. Scalpel cut or laceration injury may occur during cesarean section. They usually occur on the buttocks, scalp or thigh. Small cut heals spontaneously. Laceration injury may need repair by stitches with 7-0 nylon. Healing is usually rapid.

MUSCLES:

- **Sternocleidomastoid (SCM)** injury (congenital torticollis) is characterized by a well circumscribed immobile mass in the midpoint of the SCM. The head tilts towards the involved side. The patient cannot move the head normally.
- **Sternomastoid hematoma** usually appears about 7–10 days after birth and is usually situated at the midposition of the muscle. **It is caused by** rupture of the muscle fibers and blood vessels, followed by a hematoma and cicatrical contraction. **It may be associated with** difficult breech delivery or attempted delivery following shoulder dystocia or excessive lateral flexion of the neck even during normal delivery. There is transient torticollis and it is wise not to massage. **Treatment** is conservative. Stretching of the involved muscle should be done several times a day. Recovery is rapid (3–4 months) in majority of cases. Surgery is needed if it persists after 6 months of physical therapy.
- **Necrosis of the subcutaneous tissue** may occur while the superficial skin remains intact. After a few days, a small hard subcutaneous nodule appears. It is the result of the fat necrosis due to pressure, and takes many weeks to disappear. No treatment is required and it has no clinical importance.

NERVE INJURIES: ***Facial palsy (peripheral):*** The facial nerve remains unprotected after its exit through the stylomastoid foramen. It is involved by direct pressure of the forceps blades or by hemorrhage and edema around the nerve. It may even be involved in spontaneous delivery when too much pressure is applied on the ramus of the mandible where the nerve crosses superficially. Diagnosis is made by noting the eye of the affected side which remains open and eyelids are immobile. On crying , the angle of the mouth is drawn over to the unaffected side. No nasolabial fold is present. Sucking remains unaffected. **Treatment** aims at protecting the eye, which remains open even during sleep, with synthetic tears (1% methyl cellulose drops). The condition usually disappears within weeks unless complicated by intracranial damage (Fig. 33.7).



Figs 33.7A and B: (A) Facial palsy; (B) Erb's palsy

Brachial palsy: Either the nerve roots or the trunk of the brachial plexus are involved. The damage of the nerve is due to stretching (common) or effusion or hemorrhage inside the sheath. Tearing of the fibers is rare.

The cause is undue traction on the neck during attempted delivery of the **shoulder. The affection is due to hyperextension** of neck to one side with forcible digital extension and abduction of the arm in an attempt to deliver the shoulders. Unilateral involvement is common. Two clinical types are met depending upon the nerve root involved. Rarely, both types are present together.

Erb's palsy: This is the most common type when the **5th and 6th and rarely the 7th cervical nerve roots are involved.** The resulting paralysis causes the arm to lie on the side (adducted) with extension of the elbow, pronation of the forearm and flexion of the wrist (Waiter's tip). Winging of the scapula is common. Moro reflex is absent. There may be associated ipsilateral phrenic nerve (diaphragmatic) paralysis (C3, 4, 5).

Klumpke's palsy: This type of palsy is due to the affection of the lower cords of the plexus involving **7th and 8th cervical or even the first thoracic nerve roots.** There is paralysis of the muscles of the forearm. The arm is flexed at the elbow and the wrist is extended. The forearm is supinated and a claw-like deformity of the hand is observed. When the first thoracic nerve is involved, there may be homolateral ptosis with small pupil due to sympathetic nerve involvement (Horner's syndrome). **Treatment consists of** immobilization and prevention of contractures. Physical therapy and passive movements are advocated. Full recovery takes weeks or even months. Severe injury may produce permanent disability (Fig. 33.7). Bony injury should be excluded with radiography.

Prognosis is usually good, if it is due to stretching. But if it is due to hemorrhage or avulsion, the deformity may be permanent.

Brachial plexus injury: The incidence is about 0.1–0.2% of shoulder dystocia, even in normal delivery, macrosomia, malpresentation and instrumental deliveries. The entire arm is flaccid. All reflexes are absent.

Phrenic nerve injury (C3, 4 or 5) cause paralysis of the ipsilateral diaphragm. This is due to excessive stretching of the neck at birth. Risk factors are: Breech or difficult forceps delivery. Infants present with respiratory distress, cyanosis and tachypnea. Diagnosis is made by USG showing paradoxical movement of the diaphragm.

Treatment is supportive. Continuous positive airway pressure (CPAP) or mechanical ventilation may be needed. Recovery is usually completed in 1–3 months time.

FRACTURES:

- ***Skull bone***—(See p. 558)
- ***Spines***—Fracture of the odontoid process or fracture dislocation of the fifth-sixth cervical vertebrae may occur due to acute bending of the spine while delivering the aftercoming head or in shoulder dystocia. The result is instantaneous death of the baby due to compression on the medulla.
- ***Long bones***—Bones commonly involved in fractures are—the humerus, the clavicle and the femur. These occur in breech delivery. Fractures are usually of greenstick type but may be complete. Rapid union occurs with callus formation. Deformity is a rarity even where the bone ends are not in good alignment.

Treatment— Fracture femur and humerus are treated by immobilization. X-ray studies are done. Closed reduction and casting are needed when bones are displaced. Limb motion is restricted. Healing with callus formation occurs over 2–4 weeks. Usually there is complete recovery.

DISLOCATIONS: The common sites of dislocations of joints are shoulder, hip, jaw and fifth-sixth cervical vertebrae. Confirmation is done by radiology or ultrasonography and the help of an orthopedic surgeon should be sought.

VISCERAL INJURIES: Liver, kidneys, adrenals or lungs are commonly injured mainly during breech delivery. The most common result of the injury is intraperitoneal hemorrhage. Severe hemorrhage is fatal. In minor hemorrhage, the baby presents features of blood loss in addition to the disturbed function of the organ involved. Treatment is directed: (1) To correct hypovolemia, anemia and coagulation disorders; (2) Specific management—surgical or otherwise, to tackle the injured viscera.

PERINATAL INFECTIONS

Infection is still one of the leading cause of neonatal death in the developing countries. The neonates are more susceptible to infection as they are deficient in natural immunity and acquired immunity. Preterm infants are at high-risk for perinatal infections. Neonates those survive from sepsis often suffer from severe neurological as well as severe parenchymal lung disease. **Early-Onset Sepsis (EOS)** occurs within first 3 days of life.

Risk Factors for Neonatal Infection

- Rupture of membranes > 18 hours
- Maternal intrapartum fever > 100.4°F
- Low birth weight infant (< 2,500 g)
- Prematurity (< 37 weeks)
- Chorioamnionitis
- Male infants
- Mother with Gr B β-hemolytic streptococcal (GBS) infection
- Repeated vaginal examination in labor
- Invasive procedures of monitoring

MODE OF INFECTION

- **Antenatal**
- **Intranatal**
- **Postnatal**

ANTENATAL: ***Transplacental:*** Maternal infection that can affect the fetus through transplacental route are predominantly the viruses. They are rubella, cytomegalovirus, herpes virus, HIV, chickenpox and hepatitis-B virus (see Chapter 19). Other infections are syphilis, toxoplasmosis and tuberculosis (see Chapter 20).

Amnionitis: Amnionitis following premature rupture of the membranes can affect the baby following aspiration or ingestion of infected amniotic fluid.

INTRANATAL:

- Aspiration of infected liquor or meconium following early rupture of the membranes or repeated internal examination.
- While the fetus is passing through the infected birth passage—(a) eyes are infected—ophthalmia neonatorum or (b) oral thrush with *Candida albicans*.
- Improper asepsis while caring the umbilical cord.

POSTNATAL: Nosocomial infections—(i) Transmission due to human contact—infected mother, relatives or staff of the nursery; (ii) Cross-infection from an infected baby in the nursery; (iii) Infection through feeding, bathing, clothing or airborne and (iv) Infection in the environment of neonatal intensive care unit (NICU) or invasive monitoring.

Clinical presentation of early-onset neonatal sepsis: It is abrupt and 90% infants become symptomatic by 24 hours of age. Tachypnea, grunting, lethargy, hypotension, cyanosis, jaundice, vomiting, diarrhea and RDS are the common symptoms. Other less common presentations are: DIC, meningitis and persistent pulmonary hypertension of the newborn (PPHN). There may be hypothermia (preterm), or hyperthermia (in term infants) infants.

Common pathogens are: Group B *Streptococcus* (GBS), *Staphylococcus aureus, E. coli, Klebsiella, Hemophilus, Enterobacter, B. fragilis* and *Citrobacter, Pseudomonas*, fungus (*Candida*) and anaerobes. The infection is acquired during intrapartum period from the genital tract. The infant is colonized with pathogen in the perinatal period. The primary sites of colonization are: skin, nasopharynx, oropharynx, conjunctiva and the umbilical cord.

DIAGNOSIS: Laboratory evaluation includes: Complete blood count (CBC), platelet count, blood and urine culture and acute phase reactants. An elevated WBC (>40,000) count with polymorphonuclear cells or a depressed total WBC (<5,000) and absolute neutropenia (<1,500) are commonly found. C-reactive protein (CRP) remains elevated with inflammation and decline rapidly with resolution. Serial tests are needed.

Imaging studies: Chest X-ray, renal ultrasound are needed depending upon the presentation.

PREVENTION OF NEONATAL INFECTION: This has been mentioned on pages 155 and 156 . GBS prophylaxis can reduce EOS significantly. Penicillin is the drug of choice. Ampicilin, cefazolin or vancomycin may be used.

TREATMENT: Antibiotic therapy (Table 33.3)—Broad spectrum are given to cover the Gram-positive and Gram-negative organisms as well as the anaerobes. Injection Ampicillin 150 mg/kg/every 12 hours, Gentamicin 3–4 mg/kg/every 24 hours, usually are started. In a severely ill patient, cefotaxime or ceftazidime is also added. **Supportive therapy** and **management of complications** are continued as needed, e.g. mechanical ventilation for RDS, dopamine for hypotension, anticonvulsant for seizures and sodium bicarbonate for metabolic acidosis (p. 546). Immunotherapy with IV immunoglobulin (IVIG), monoclonal antibodies, granulocyte colony stimulating factor (GM–CSF) are used as an adjuvant to the antibiotics.

Table 33.3: Antibiotics Used to Treat Neonatal Infection

Drug	Nature	Bacterial Coverage	Daily Dose (per kg body wt)	Route	Side Effects
Ampicillin	Bactericidal	Both Gram-positive and Gram-negative	150 mg/kg/q 12h	IV/IM	Diarrhea, skin rash, nausea, vomiting
Amikacin	Bactericidal	Primarily Gram-negative	15 mg/kg/q 24h	—do—	Nephrotoxicity, ototoxicity
Gentamicin	Bactericidal	Primarily Gram-negative	3–4 mg/kg/q 24h	—do—	Ototoxicity, nephrotoxicity
Cefotaxime	Bactericidal	Mainly Gram-negative	50 mg/kg/q 12h	—do—	Hypersensitivity, thrombophlebitis, diarrhea
Cefuroxime	Bactericidal	Both Gram-positive and Gram-negative	50 mg/kg/q 12h	—do—	Generally free from toxicity, hypersensitivity (rarely)
Ceftriaxone	Bactericidal	—do—	50 mg/kg/q 12h	—do—	Diarrhea, eosinophilia, skin rash, neutropenia
Meropenem	Bactericidal	Aerobic and anaerobic Gram-positive and Gram-negative	20 mg/kg/q 12h	—do—	Diarrhea, leukopenia, thrombocytopenia, anaphylaxis
Aqueous pencillin	Bactericidal	Mainly Gram-positive	2 lac units/kg/ divided 8 hourly	—do—	Allergic reactions, rash, fever, diarrhea
Vancomycin	Bactericidal (MRSA)	Most Gram-positive and enterococci	15 mg/kg/day IV every 12 hours	Allergy	allergy, ototoxicity, nephrotoxicity, thrombophlebitis

IM = Intramuscular; IV = Intravenous; q 12h = every 12 hours; q 24h = every 24 hours

COMMON SITES OF INFECTION :

Trivial but may be serious: (i) Eyes—ophthalmia neonatorum; (ii) Skin; (iii) Umbilicus; (iv) Oral thrush.

Severe systemic: (i) Respiratory tract; (ii) Septicemia; (iii) Meningitis; (iv) Intra-abdominal infection.

OPHTHALMIA NEONATORUM (CONJUNCTIVITIS)

Ophthalmia neonatorum is defined as inflammation of conjunctiva during first month of life.

CAUSES: The common causative agents are: (i) *Chlamydia trachomatis* (oculogenitalis); (ii) Other bacterial causes: (a) *Gonococcus* (rare), *Staphylococcus*, *Pseudomonas;* (b) Chemical—silver nitrate; (c) Viral: herpes simplex (type II).

MODE OF INFECTION: Infection occurs mostly during delivery by contaminated vaginal discharge. It is more likely in face or breech delivery. During neonatal period, there may be direct contamination from other sites of infection or by chemical.

The **clinical picture** varies and the discharge may be watery, mucopurulent to frank purulent in one or both eyes. The eyelids may be sticky or markedly swollen. Cornea may be involved in severe cases.

Prognosis is favorable to most cases except in neglected cases with rare gonococcal infection. Fortunately, effective methods of prophylaxis and treatment have almost eliminated the risk of blindness.

PREVENTION: Any suspicious vaginal discharge during the antenatal period should be treated and the most meticulous obstetric asepsis is maintained at birth. The newborn baby's closed lids should be thoroughly cleansed and dried.

INVESTIGATIONS: The discharge is taken for—(a) gramstain smear; (b) culture and sensitivity; (c) scraping material from lower conjunctiva for Giemsa staining and also culture in suspected chlamydial infection and (d) culture in special viral media for suspected herpes simplex infection.

TREATMENT: Prophylaxis: 1% **silver nitrate** solution (1–2 drops to each eye), 0.5% **erythromycin** ophthalmic ointment, **2.5% povidone iodine** solution (1 drop each eye) is administered within 1 hour of birth and is continued for few days.

Treatment depends on the specific etiology.

(a) **Gonococcal**—Infant is isolated during the first 24 hours of treatment. Eyes are irrigated with sterile isotonic saline every 1–2 hours until clear. In severe and culture positive cases systemic ceftriaxone 25–50 mg/kg/IV/IM or cefotaxime 100 mg/kg is given IM/IV. Single dose in infant without dissemination or for 7 days when there is dissemination, is usually given.

(b) **Chlamydia**—Erythromycin suspension 40 mg/kg daily orally divided into 4 doses for 14 days is given to prevent systemic infection. Topical treatment alone is ineffective.

(c) **Herpes simplex**—The infant is isolated. Systemic therapy with acyclovir 20 mg/kg every 8 hours for 2 weeks is given IV. Topical use of 3% vidarabine or 0.1% iododeoxyuridine ointment 5 times a day for 10 days is used.

Ophthalmologist should be consulted for any severe infection.

SKIN INFECTIONS

SKIN INFECTIONS—Newborn's skin infections may manifest as skin rashes, pustulosis or cellulitis. The causative organisms are: Gram-positive, Gram-negative and anaerobic organisms. *Staphylococcus aureus* is the predominant one. Common sites of infections are: face, axilla, groin, scalp and periumbilical area. Colonization of the newborn skin occurs during birth from vaginal flora as well as from the environment (nosocomial, cross-infection from the carriers).

Localized infections are often due to traumatized skin. The common sites are: venopuncture or scalp electrode.

PUSTULOSIS—is usually caused by *S. aureus*. Rarely it may be epidemic and results in septicemia or pyemia. Some skin lesions may be bullous or scalded.

Treatment of *S. aureus* pustulosis depends on the severity of infections and condition of the infant. Mild infections may be treated with topical mupirocin and oral therapy with amoxycillin/or cephalexin. More extensive lesions require therapy with nafcillin or oxacillin IV, MRSA infection need to be treated by vancomycin.

Cellulitis—usually occurs at a traumatic skin site (see above). It is usually treated with local antibiotic ointment (bacitracin). In severe infections or in a premature infant, complete blood count (CBC), blood culture are to be obtained. Systemic antibiotic (Oxacillin or nafcillin and gentamicin) IV is given.

Epidemic outbreaks due to nosocomial acquisition of *S. aureus* in newborn nurseries or NICU need intensive surveillance of the staff members and the newborns with culture.

UMBILICAL SEPSIS (OMPHALITIS)

It is not uncommon for mild umbilical sepsis to occur. The causative organisms include both Gram-positive and negative organisms. Anaerobic infections with Clostridium tetani may occur rarely. **The infection is manifested by** serous or seropurulent umbilical discharge which may be offensive. The base of the cord stump looks moist and the periumbilical skin becomes red and swollen. There is delay in falling off of the cord. Systemic manifestations include pyrexia and features of toxemia or jaundice in severe infection.

Spread of infection: (1) Periumbilical cellulitis with suppuration; (2) Thrombophlebitis of the umbilical vein with extension of the infection to the liver producing hepatitis or pyemic liver abscess; (3) Peritonitis and (4) Necrotizing fascitis.

Prevention: Antiseptic and aseptic precaution should be taken right from the time of cutting the cord to the time of complete epithelization of the area after falling of the cord. The care of the umbilical cord as mentioned in Chapter 30 should be followed.

Curative: Treatment: Complete septic work up (CBC, blood and umbilical swab culture) is done. Antibiotic therapy with nafcillin and gentamicin or oxacillin or piperacillin/tazobactam may be used depending upon the severity of infection. The wound is dressed like any surgical wound with spirit and antiseptic powder.

TETANUS NEONATORUM: It is rare nowadays but may cause concern in the tropical countries. The infection is caused by Clostridium tetani and the portal of entry is through the umbilical cord. The features are evident within 5–15 days after birth.

The striking features are: **Inability to suck associated with marked trismus followed by rigidity of the body with opisthotonus, pyrexia and convulsions.**

Prevention includes immunization of the mother during pregnancy with tetanus toxoid. Babies born in unhygienic conditions without previous immunization of the mother, should be given 1,500 IU of antitetanus serum intramuscularly soon after birth.

Curative treatment includes: (1) **The baby should be isolated in the infectious disease hospital;** (2) Tetanus immune globulin (human) 6,000 IU is given intramuscularly; (3) Antitetanus serum (ATS) should be started immediately in doses of 50,000–100,000 units intramuscularly or intravenously. The same dose may have to be repeated after 12 hours; (4) Antibiotics, particularly penicillin should be given in heavy doses; (5) Sedation should be ensured by intramuscular administration of either (a) Chlorpromazine 5–10 mg/kg per day or (b) Phenobarbitone 15 mg/kg per day in divided doses. Both may be combined so as to be more effective; (6) Endotracheal intubation and ventilation may be needed and (7) Nutrition is to be maintained by intragastric feeding. **Prognosis:** Mortality is up to 60–80%.

NECROTIZING ENTEROCOLITIS

This is a life-threatening condition associated with ischemic and inflammatory necrosis of the relatively immature intestine. Pathogenesis is multifactorial. The role of any toxin is yet to be established.

Risk factors: (a) Premature infants; (b) Perinatal asphyxia; (c) Hypotension; (d) Polycythemia; (e) Umbilical cord catheter-related thromboembolism; (f) Septicemia due to *E. coli*, *Klebsiella*, pseudomonas; (g) Exchange transfusion and (h) Congenital heart disease.

Pathophysiology: There is ischemic and/or toxic damage to the mucous membrane of the gut commonly in the ileocecal region. It is associated with bacterial proliferation and gas formation. Gradually there is ischemic necrosis of the muscular wall of the gut, gangrene ultimately leading to perforation and peritonitis.

Diagnosis: Systemic signs: Respiratory distress, lethargy, feeding intolerance, hypertension, acidosis, oliguria and bleeding diathesis. **Abdominal signs are:** Abdominal distension, tenderness, bloody stools, vomiting.

Imaging studies: X-ray abdomen reveals abdominal gas pattern with dilated loops. There may be absent bowel gas with pneumoperitoneum. Ultrasonography including Doppler can detect gas bubbles in liver parenchyma, portal venous system, bowel necrosis and perforation. Grossly bloody stool is common in NEC. **Thrombocytopenia, metabolic acidosis and hyponatremia are the triad** of signs to confirm the diagnosis.

Prevention: Human (mother's) milk can prevent NEC. Probiotics and nutrients enhance the growth of beneficial microbes. Prolonged use of antibiotics should be avoided.

TREATMENT: (i) **Respiratory system**: Supplemental O_2 and mechanical ventilation may be needed; (ii) Support to the cardiovascular system: Circulatory volume, blood pressure, arterial blood gas, tissue perfusion is maintained.

Nutrition—(i) Discontinuation of oral feeding and to start nasogastric suction; (ii) Total parenteral nutrition; (iii) Laboratory monitoring for arterial blood gas, serum electrolytes, blood glucose, platelet count, acid-base balance and septic work up are done; (iv) Antibiotics—vancomycin, piperacillin/tazobactam, gentomycin and metronidazole; (v) Bowel resection in the case of perforation. **Prognosis:** Mortality is up to 40% when associated with perforation. **Overall mortality is about 10–12%.**

MUCOCUTANEOUS CANDIDIASIS

ORAL THRUSH: Infection of the buccal mucous membranes and the tongue by the fungus *Candida albicans* is not uncommon especially in bottle fed babies. Contamination by the organisms occurs from the feeding bottle, teats, nurse's hand, mother's nipple and infected vagina. The fungus grows on the mucous membrane and produces milky white elevated patches resembling milk curd, which cannot be easily wiped off with gauze. Rarely, the fungal infection may spread down to involve the gastrointestinal or respiratory tract.

It usually appears in the late first week or during the 2nd week. The infant refuses to take feeds. Constitutional upset is unusual but becomes evident in extra-oral spread to the respiratory tract. The typical patches are visible on the mouth and **an attempt to remove the patch leaves behind a raw oozing surface. Spots on the edges of the tongue are diagnostic**, as suckling would remove the milk curd from that region.

PROGNOSIS: If effectively treated, cure is very prompt but in neglected cases especially with alimentary or respiratory tract involvement, rapid deterioration occurs.

PREVENTION: Maternal fungal infection in the vagina is to be adequately treated before delivery. Utensils including feeding bottles and teats are to be properly cleansed before and after each feed.

TREATMENT: Local applications of 1% aqueous solution of **gentian violet** on the oral mucous membrane twice daily after feeds for 2–3 days is quite effective but not used now due to many side effects. **Nystatin** oral suspension (100,000 U/mL), 1 mL is applied to each side of the mouth 4 times a day for about 2–3 weeks. Systemic fluconazole is highly effective in chronic mucocutaneous candidiasis. Infants with chronic thrush **refractory** to usual treatment should be investigated for immunodeficiency. **Mothers with breast ductal candidiasis**, concurrent treatment of both the mother and the infant is done to eliminate cross infection. **Diaper candidal dermatitis** is treated with topical 2% nystatin ointment, 2% miconazole ointment or 1% clotrimazole cream. Intestinal colonization should be treated with oral nystatin at the same time.

CONGENITAL MALFORMATIONS AND PRENATAL DIAGNOSIS

The incidence of significant congenital malformations is about 2–5% at birth, a lower incidence of 1 in 500, is however, reported from the hospital statistics of India. In the Western countries, however, major fetal abnormalities account for about 20% of perinatal deaths and many survivors are physically and/or mentally handicapped. Defects in the central nervous system account for about 50% of malformations.

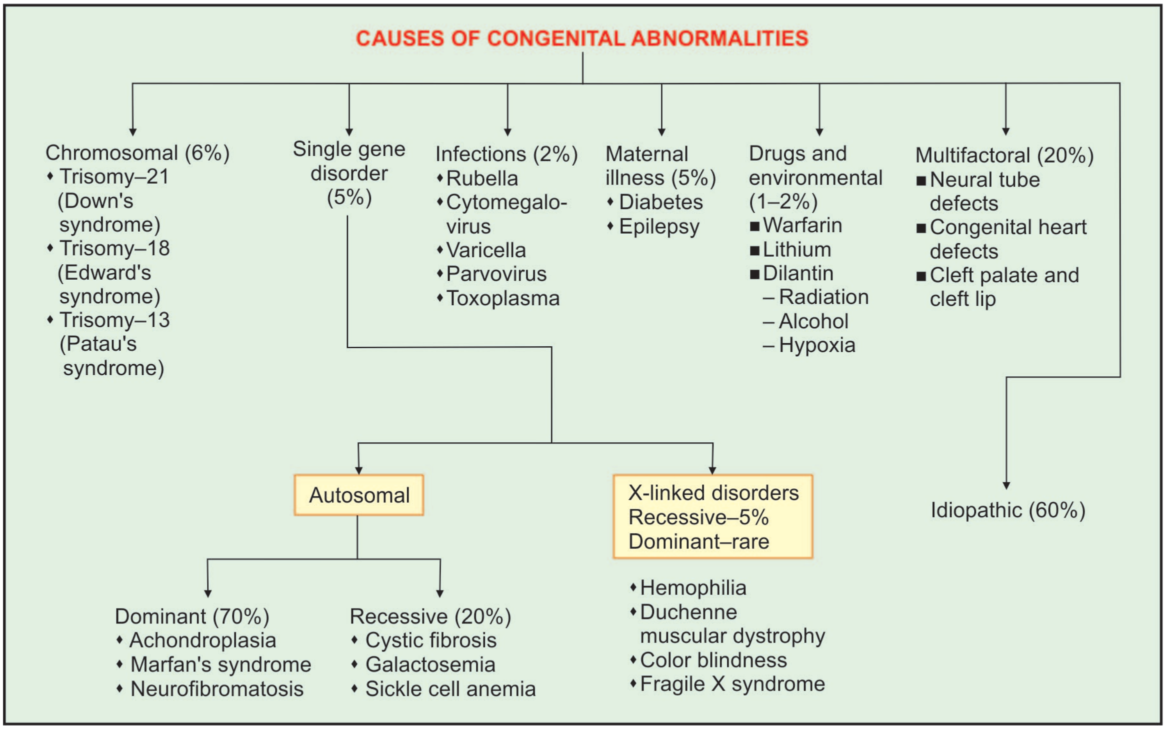
ETIOLOGY: The causes are not fully understood and are grouped as follows:

When a fetus is exposed to a teratogenic agent the resultant effect will depend on the duration of gestation and the genetic susceptibility of the fetus. Calculating from the first day of LMP, D 31 to D 71 is the critical period of organ development.

GENETICS: The defect is inherited through the genes in the ovum or sperm. Single gene disorders either autosomal or X-linked, which may be dominant or recessive may be found.

ENVIRONMENTAL: The fetal affection due to a given teratogen will depend on the dose administered, the gestational age at exposure and the maternal and fetal immune response to the agent. The fetus is, in fact, potentially susceptible to some teratogenic effect even after the completion of morphogenesis. **The net effect may be death, malformation, growth retardation or functional disorder.**

- **Advancing maternal age** increases the incidence of Down's syndrome (Mongolism) to the extent of 1 in 100 births at the age of 40 years. Increasing parity is associated with high incidence of malformations except anencephaly or spina bifida which is comparatively common in first birth.
- **The adverse effects of drugs** (Fig. 33.3) on the preimplantation and postimplantation early ovum remain unpredictable. However, warfarin, lithium, dilantin, antifolic acid group of drugs have got established untoward effects on the growing conceptus. The untoward effects of the various drugs are mentioned in Chapter 34.
- **Infections—**maternal rubella, cytomegalovirus, toxoplasma either latent or overt in the first trimester, produces congenital malformation of the fetus. The correlation with other maternal infections are described in Chapter 20.


CAUSES OF CONGENITAL ABNORMALITIES
Chromosomal (6%)
• Trisomy–21 (Down's syndrome)
• Trisomy–18 (Edward's syndrome)
• Trisomy–13 (Patau's syndrome)
Single gene disorder (5%)
Infections (2%)
• Rubella
• Cytomegalovirus
• Varicella
• Parvovirus
• Toxoplasma
Maternal illness (5%)
• Diabetes
• Epilepsy
Drugs and environmental (1–2%)
■ Warfarin
■ Lithium
■ Dilantin
– Radiation
– Alcohol
– Hypoxia
Multifactoral (20%)
■ Neural tube defects
■ Congenital heart defects
■ Cleft palate and cleft lip
Idiopathic (60%)
Autosomal
X-linked disorders
Recessive–5%
Dominant–rare
Dominant (70%)
• Achondroplasia
• Marfan's syndrome
• Neurofibromatosis
Recessive (20%)
• Cystic fibrosis
• Galactosemia
• Sickle cell anemia
• Hemophilia
• Duchenne muscular dystrophy
• Color blindness
• Fragile X syndrome

- **Irradiation** is a potential danger to the fetus especially in early embryonic phase. Irradiation of gonads of either parent may result in mutation of genes which is recessive in character. Though maximal ionizing radiation currently thought to be relatively safe for the human embryo and fetus at any stage of gestation (as stated by the National Committee on Radiation Protection) is 10 rads, **it is safer to limit its use especially during first trimester.**
- **Maternal malnutrition, metabolic and endocrinal disorders** like uncontrolled diabetes, epilepsy are related with increased incidence of fetal malformations.

MULTIFACTORIAL: Most of the malformations probably result from delicate and complex interactions between genetic predisposition and altered environmental factors, the nature of which remains obscure in majority of cases. The malformation may affect a single organ and a particular sex.

Prenatal diagnosis and fetal therapy (see p. 129)

DOWN'S SYNDROME (TRISOMY 21)

Trisomy 21 is the **most frequent autosomal (chromosomal) syndrome. The defect is due to:**

(1) Inclusion of an **additional chromosome,** trisomy 21 (95%)—47 instead of 46 chromosomes. Triplication may be caused either by the presence of an entire additional chromosome 21 or the addition of only band q 22.

(2) Chromosomal translocation defect (14 : 21) (rare)—especially occurring in young mothers. There is transfer of a segment of one chromosome to a different site of the same chromosome or to a different chromosome. **There is 30% chance of recurrence in translocation defect.**

Incidence: The overall incidence is 1 in 600. The incidence rises with advancing age of the mother, **reaching a peak of about 1 in 25 by the age of 45 years.**

Diagnosis of the affected baby:

- **General appearance:** Craniofacial abnormalities include small ears (100%), brachycephaly, upwards and outwards slanting of the eyes with epicanthic folds; short upper lip with small mouth and macroglossia. The baby's face resembles that of the Mongolian race (Fig. 33.8). The hands are short and broad **with a**

single palmar crease (30%). There is increased (50%) association of congenital heart disease (VSD), omphalocele, cataracts and esophageal atresia, duodenal atresia and imperforate anus. The affected baby is mentally retarded.

Hypotonia may cause breathing difficulties, poor swallowing and aspiration. Joint hyperextensibility is observed.

Expectation of life is reduced. Adult Mongol is likely to develop leukemia. Male infertility is the rule. In female puberty may be delayed and may be fertile.

- Confirmation is established by chromosomal analysis (karyotype) using bone marrow aspiration or leukocyte culture.

Genetic counseling in subsequent pregnancy. The risk of recurrence due to trisomy 21 is 1%. That of translocation is higher. Following amniocentesis, if karyotyping of the exfoliated cells shows the abnormal chromosome, therapeutic termination will have to be seriously considered (see p. 127).

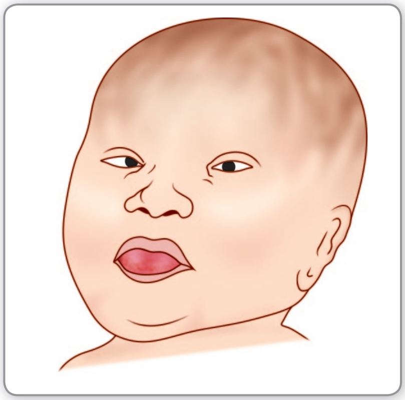


Fig. 33.8: Baby with Down's syndrome

CONGENITAL MALFORMATIONS IN NEWBORN AND THE SURGICAL EMERGENCIES

- **Imperforate anus**
- **Exomphalos**
- **Esophageal atresia**
- **Diaphragmatic hernia**
- **Meconium ileus**
- **Duodenal atresia**

IMPERFORATE ANUS: It is more prevalent in males than females. Two types are met with (A) high imperforate anus, where rectum ends above the puborectalis sling. There may be associated rectourinary fistula in males or rectovaginal fistule in females. (B) Low imperforate anus where rectum has traversed the puborectalis sling. This variant may be associated with or without perineal fistula. **Diagnosis is made by:** (1) Absence of meconium passage, (2) Absence of anal opening, (3) Failure to pass a rectal thermometer rubber catheter or lubricated little finger, (4) Radiology is helpful to detect the extent of atresia. **Radiography is taken with the baby held in inverted position by holding the legs, with a coin placed over the anal pit (invertogram).** The distance between the highest level of the intestinal gas and the shadow of the coin gives the extent of the atresia. (5) **Imaging study** (X-ray, USG) of the lumbosacral spine and urinary tract should be done to exclude any other abnormality in this area.

Management: (1) Cruciate incision (perineal anoplasty) is made on the membrane in case of the simple membranous obstruction which is evidenced by marked bulging over the anal pit when the baby cries. (2) **In high imperforate anus**, colostomy is done and pull through operation is done at a later date.

ESOPHAGEAL ATRESIA: The esophagus ends blindly about 12 cm from the nares. Babies born of mothers having hydramnios should be checked carefully at birth to exclude this abnormality. Simultaneous distal tracheoesophageal fistula (TEF) too often (85%) coexists. Excessive salivation, increasing respiratory distress and even a small amount of fluid by mouth causing cough and cyanosis point strongly towards the entity. Distal TEF causes reflux of gastric contents into the tracheobronchial tree causing chemical pneumonitis and pneumonia. Diagnosis is made by failure to pass a nasogastric tube down through the esophagus. **Confirmation is done by radiography** with prior insertion of a radiopaque catheter into the esophagus.

Management: (1) Withhold fluids by mouth, (2) Frequent suctioning to prevent aspiration, (3) Place the baby in relatively upright position (45°) to prevent reflux, (4) Broad spectrum antibiotic should be administered, (5) Placement of a gastrostomy tube, (6) Ligation of tracheoesophageal fistula and esophageal anastomosis by thoracotomy or thoracoscopy are the principal steps of the operation.

MECONIUM ILEUS: It is a manifestation of fibrocystic disease of the pancreas. Deficiency of the pancreatic enzyme makes the meconium in the intestine inspissated which in turn obstructs the lumen of the lower ileum. Diagnosis is based on clinical manifestations of small gut obstruction. **Sweat test:** A patient with cystic fibrosis is found to lose large quantities of sodium in the sweat. Immunoreactive trypsinogen (IRT) is done as a newborn screening. High value suggests further testing (CF gene mutation). **Confirmation is done by** straight radiographic picture of the abdomen showing the solid nature of the meconium with a granular appearance. Rectal mucosal biopsy may have to be done to demonstrate the absence of ganglion cells in **Hirschsprung disease.** Contrast enema (Meglumine diatrizoate) can be both diagnostic and therapeutic. **Surgery includes** resection and anastomosis of the gut containing the inspissated meconium followed by treatment with pancreatic enzymes and vitamins. **Surgical therapy:** Surgery may be done open, laparoscopic and transanal. Different methods are: Staged repair with colostomy, one stage pull through or delayed I-stage repair when the infant has gained (double) weight. **Prenatal diagnosis** with DNA probes is possible from chorionic villus sampling (see p. 129).

Table 33.4: Age for Elective Surgical Procedures

Malformation	Optimal Age
Cleft lip	6 weeks–6 months
Cleft palate	9–12 months
Umbilical hernia	After 1 year, if required
Inguinal hernia	As early the infant's general condition permits
Hydrocele	After 1 year
Undescended testicle	Between 1 and 2 years of age
Patent ductus arteriosus	At birth
Coarctation of aorta	3–4 years or soon after the infant has been medically stabilized
Hypospadius (other than glandular)	6–18 months

EXOMPHALOS (OMPHALOCELE): It is a congenital herniation of the abdominal contents (usually small gut) through the defect in the abdominal wall at the base of the umbilical cord. The anterior abdominal wall is defective in its entire thickness. Associated congenital anomalies occur in about 30–40% of infants (chromosomal abnormalities, CDH, cardiac defects). **Omphalocele** differs from that of **gastroschisis** by the following anatomic features: (a) A protective membrane encloses the abdominal contents, (b) Contents of umbilical cord course individually over the sac and come out at the apex. **Every effort should be made to protect the membranes from rupture.** Cesarean delivery may prevent rupture of this sac. A moist sterile saline dressing should be applied and arrangement is made for immediate surgical closure, if possible, in one stage (< 5 cm opening) or in two stages. **Prenatal diagnosis** with ultrasound is possible.

CONGENITAL DIAPHRAGMATIC HERNIA (CDH): Congenital diaphragmatic hernia occurs where the abdominal contents herniate through a defect in the diaphragm (patent pleuroperitoneal canal) into the thorax. **It usually occurs on the left side** through foramen of Bochdalek (95%). CDH following delivery, infants may develop (A) Pulmonary parenchymal insufficiency due to hypoplastic lungs, and (B) Pulmonary hypertension of the newborn. **Symptoms include** acute respiratory distress with marked cyanosis which may be relieved by holding the baby in an upright position. **Signs include** unequal movements of the thorax, absent breath sounds on the affected side with scaphoid abdomen. In left-sided CDH, apical impulse is shifted to the right and heart sounds are better heard over the right side of chest. X-ray chest reveals gas shadow of small bowel in the thorax and mediastinal shift away from the affected side (Fig. 33.9). It may be associated with trisomies (13,18) and 45XO. **Prenatal diagnosis** with ultrasound is possible.

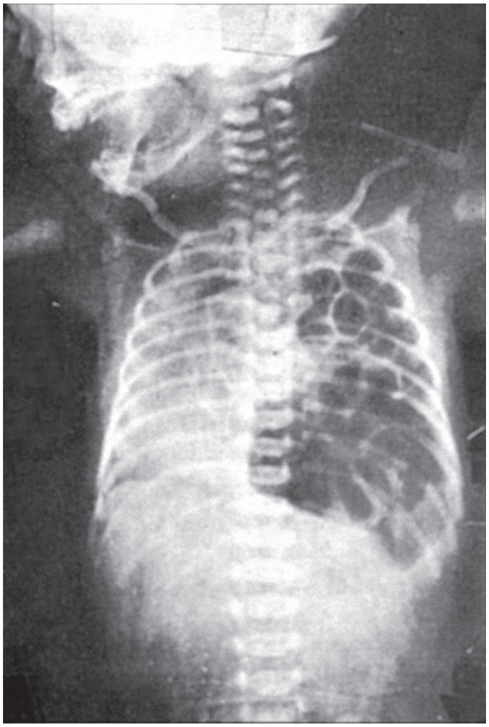


Fig. 33.9: Diaphragmatic hernia

***Management*: Supportive care:** Intubation and PPV is to be initiated immediately. Replacement of surfactant is helpful. (1) To insert a large bore open-ended nasogastric tube into the stomach to lessen gaseous distension of the bowel, (2) Insert an endotracheal tube and give positive pressure oxygen of less than 30 mm Hg, (3) Correct acidosis, (4) Blood gas levels should be monitored by an indwelling arterial catheter, (5) Extracorporeal membrane oxygenation is used for neonates with respiratory failure due to pulmonary hypoplasia, (6) **CDH medication:** Sildenafil has been found to lower PPHN in neonates, (7) Surgical repair is done by reduction of intrathoracic intestines and closure of diaphragmatic defect. Surgery may be delayed following improvement of pulmonary function and (8) Intrauterine fetal surgery has been done in few cases to prevent pulmonary hypoplasia.

***Prognosis*:** Mortality rate in CDH is up to 50%.

DUODENAL ATRESIA: In atresia, the lumen is completely obstructed whereas in stenosis, it is narrowed. **It is usually common in babies born of mothers with hydramnios, with IUGR and in trisomy 21 (Down's syndrome–33%).** The 70% of cases have other malformation (cardiac, GI). Vomiting is a prominent feature, the vomitus being copious and bile stained (atresia is usually below the ampulla of Vater). The upper abdomen may be distended and following the passage of meconium (usually white), no further stools are passed.

Plain X-ray of the abdomen or USG in upright position shows the typical **'double bubble appearance'**—gas in fundus of stomach and in the vault of the proximal half of duodenum with no air in the small or large bowel. Prenatal diagnosis is made with **ultrasound**.

***Management*:** (1) Withhold fluids by mouth, (2) Parenteral replacement of fluids and electrolytes, (3) Prompt corrective surgery of duodenojejunostomy.

NONIMMUNE FETAL HYDROPS (NIFH)

Nonimmune fetal hydrops is defined as the accumulation of extracellular fluid in tissues and serous cavities in conditions other than Rh incompatibility (see p. 388). It is usually associated with increased skin thickness (> 5 mm), due to generalized subcutaneous edema in the fetus, placental enlargement, pericardial effusion, pleural effusion and/or ascites. With complete prevention of Rh problem, more than 75% of the fetal hydrops are related to NIFH.

Causes:

1. *Chromosomal abnormality (10%)*: Trisomies (13, 18, 21), Turner syndrome, triploidy, aneuploidy
2. *Congenital cardiac lesions*: Congenital heart block, supraventricular tachycardia, structural major cardiac abnormality (hypoplastic left heart)
3. *Congenital abnormalities*: Diaphragmatic hernia, renal abnormality, cystic hygroma
4. *Hematological*: Beta-thalassemia, Glucose-6-Phosphate dehydrogenase (G6 PD) deficiency, leukemia
5. *Infections (8%)*: Parvovirus, rubella, toxoplasma, syphilis, cytomegalovirus, hepatitis
6. *Placental and umbilical cord pathology*: Twin-to-twin transfusion, chorioangioma, umbilical vein thrombosis, TRAP (see p. 240)
7. *Maternal diseases (5%)*: Uncontrolled diabetes, severe anemia, thyrotoxicosis
8. *Miscellaneous (10%):* CNS malformations, skeletal abnormalities, lysosomal disorders
9. *Idiopathic*: 20%

Pathology: Pathology depends on the etiological factor. However, ultimate pathology is development of severe anemia, hypoproteinemia (decreased colloid osmotic pressure), asphyxia, increased capillary permeability and heart failure.

Investigations: Prenatal diagnosis is possible nowadays with the advent of high resolution ultrasound scan, Doppler flow study and cordocentesis.

(i) **Maternal blood** for complete blood count, ABO and Rh group, red cell antibody titers, hemoglobin electrophoresis, VDRL, Kleihauer test, Glucose tolerance test, tests for thalassemia, G-6 PD deficiency and serological tests for infections (see Chapter 20). Doppler study for fetal anemia measuring MCA peak velocity.

(ii) **Ultrasound–detailed scan** of the fetus for echocardiography, structural lesions, and Doppler flow studies is most important for diagnosis and follow up.

(iii) **Amniocentesis** for chromosomal, biochemical and enzyme studies (see p. 130).

(iv) **Cordocentesis** for study of chromosomal and single gene disorders, enzymes, plasma proteins, blood gases and antibodies, hemoglobin electrophoresis, PCR, DNA studies (see p. 130).

(v) **Neonatal:** Chromosomal study, placental examination, autopsy study if there is still born (see p. 127).

Management: It is directed according to the cause and severity of the pathology. Termination of pregnancy may be an option when the parents desire, especially in presence of chromosomal or structural abnormality. **Transplacental therapy** for fetal dysrhythmias could be made by administering digoxin orally to the mother.

Direct fetal therapy may be done by intraperitoneal, intramuscular or intravascular (umbilical vein) routes. **Fetal transfusion** may be given through umbilical vein or peritoneal cavity to improve anemia. **Drainage of** pleural fluid, pericardial fluid or ascitic fluid under ultrasound guidance may be needed.

Obstetric management: (i) Intrauterine paracentesis or thoracocentesis prior to delivery is helpful for easy delivery and for neonatal resuscitation, (ii) Place of cesarean section depends on obstetric reasons, (iii) Antenatal corticosteroid therapy is to be given when delivery is planned preterm, (iv) Intensive neonatal care including ventilator support is needed.

Prognosis: Perinatal mortality is high (50–100%) especially in presence of a structural abnormality.

QUESTIONS

1. Mention the important causes of respiratory distress syndrome (RDS)? Outline the management of a newborn with RDS? (p. 547, 549)

Write Short Notes on:

A. Apgar score (p. 544)
B. Causes of jaundice in the newborn (p. 551)
C. Causes of nonimmune fetal hydrops (p. 571)

Chapter 34

Pharmacotherapeutics in Obstetrics

OXYTOCICS IN OBSTETRICS

DEFINITION: Oxytocics are the drugs of varying chemical nature that have the power to excite contractions of the uterine muscles. Among a large number of drugs belonging to this group, the following are the important ones and are extensively used in clinical practice.

- **Oxytocin**
- **Ergot derivatives**
- **Prostaglandins**

OXYTOCIN

PHARMACOLOGY: Oxytocin is a nonapeptide. In 1950, de Vigneaud and coworkers did the Nobel prize winning work on structure of oxytocin. It is synthesized in the supraoptic and paraventricular nuclei of the hypothalamus. By nerve axons it is transported from the hypothalamus to the posterior pituitary where it is stored and eventually released.

Oxytocin has a half-life of 3–4 minutes and a duration of action of approximately 20 minutes. It is rapidly metabolized and degraded by oxytocinase.

MODE OF ACTION: Myometrial oxytocin receptor concentration increases maximum (100-200 fold) during labor. Oxytocin acts through receptor and voltage-mediated calcium channels to initiate myometrial contractions. It stimulates amniotic and decidual prostaglandin production. Bound intracellular calcium is eventually mobilized from the sarcoplasmic reticulum to activate the contractile protein. The uterine contractions are physiological, i.e. causing fundal contraction with relaxation of the cervix.

PREPARATIONS USED: (i) Synthetic oxytocin (Syntocinon-Sandoz or Pitocin-Parke-Davis) is widely used. It has only got oxytocic effect without any vasopressor action. The Syntocinon is available in ampoules containing 5 IU/mL: Pitocin 5 IU/mL. (ii) Syntometrine (Sandoz)—A combination of syntocinon 5 units and ergometrine 0.5 mg. (iii) Desamino-oxytocin—It is not inactivated by oxytocinase and is 50–100 more effective than oxytocin. It is used as buccal tablets containing 50 IU. (iv) Oxytocin nasal solution contains 40 units/mL.

EFFECTIVENESS: In the first trimester, the uterus is almost refractory to oxytocin. In the second trimester, relative refractoriness persists, and, as such, oxytocin can only supplement other abortifacient agents in induction of abortion. In later months of pregnancy and during labor in particular, it is highly sensitive to oxytocin even in small doses. *Oxytocin loses its effectiveness unless preserved at the correct temperature (between 2°C and 8°C).*

INDICATIONS: Oxytocin may be conveniently used in pregnancy, labor or puerperium. The indications are grouped as follows:

♦ **Therapeutic** ♦ **Diagnostic**

THERAPEUTIC: ♦ **Pregnancy** ♦ **Labor** ♦ **Puerperium**

Pregnancy

Early:
- To accelerate abortion—inevitable or missed and to expedite expulsion of hydatidiform mole
- To stop bleeding following evacuation of the uterus
- Used as an adjunct to induction of abortion along with other abortifacient agents (PGE_1 or PGE_2).

Late:
- To induce labor
- To ripen the cervix before induction.

Labor:
- Augmentation of labor
- Uterine inertia
- Inactive management of third stage of labor
- Following expulsion of placenta as an alternative to ergometrine.

***Puerperium*:** To minimize blood loss and to control postpartum hemorrhage.

Table 34.1: Contraindications of Oxytocin

Pregnancy	Labor	Any Time
Grand multipara	All the contra-indication in pregnancy	Hypovolemic state
Contracted pelvis	Obstructed labor	Cardiac disease
History of cesarean section or hysterotomy	Incoordinate uterine contraction	
Malpresentation	Fetal distress	

DIAGNOSTIC:

- Contraction stress test (CST)—see p. 576
- Oxytocin sensitivity test (OST)—see p. 576

DANGERS OF OXYTOCIN

The dangers are particularly noticed when the drug is administered late in pregnancy or during labor.

♦ **Maternal** ♦ **Fetal**

MATERNAL

Uterine hyperstimulation (overactivity)—is a frequently observed side effect. There may be excessive duration of uterine contraction (**hypertonia**) or increased frequency (> 6 in 10 min time) of contractions (**polysystole**). It is often associated with **abnormal FHR pattern (see p. 418, Fig. 25.2)**.

Uterine rupture—may be seen with violent uterine contractions common. **High-risk cases are**: grand multipara, malpresentation, contracted pelvis, prior uterine scar (hysterotomy) and excessive oxytocin use.

Water intoxication is due to its antidiuretic function when used in high dose (30–40 mIU/min). Water intoxication is manifested by hyponatremia, confusion, coma, convulsions, congestive cardiac failure and death. **It is prevented by** strict fluid intake and output record, use of salt solution and by avoiding high dose oxytocin for a long time.

Hypotension: Bolus IV injections of oxytocin cause hypotension especially when patient is hypovolemic or with a heart disease. Occasionally oxytocin may produce **anginal pain**.

Antidiuresis: Antidiuretic effect is observed when oxytocin infusion rate is high (40–50 mIU/min) and continued for a long time.

FETAL: **Fetal distress**, fetal hypoxia or even fetal death may occur due to uterine hyperstimulation. Uterine hypertonia or polysystole causes reduced placental blood flow.

ROUTES OF ADMINISTRATION

- Controlled intravenous infusion is the widely used method
- Bolus IV or IM—5–10 units after the birth of the baby as an alternative to ergometrine
- Intramuscular—the preparation used is syntometrine
- Buccal tablets or nasal spray—Limited use on trial basis.

METHODS OF ADMINISTRATION OF OXYTOCIN

- **Controlled intravenous infusion**
- **Intramuscular**

CONTROLLED INTRAVENOUS INFUSION: Oxytocin infusion should be ideally by infusion pump. Fluid load should be minimum. It is started at low dose rates (1–2 mIU/min) and increased gradually.

- **For induction of labor**
- **For augmentation of labor**

For induction of labor

Principles: (1) Because of safety, **the oxytocin should be started with a low dose** and is escalated at an interval of 20–30 minutes where there is no response. **When the optimal response is achieved** (uterine contraction sustained for about 45 seconds and numbering 3 contractions in 10 minutes), the administration of the particular concentration in mIU/minute is to be continued. This is called oxytocin titration technique. (2) **The objective of oxytocin administration** is not only to initiate effective uterine contractions but also to maintain the normal pattern of uterine activity till delivery and at least 30–60 minutes beyond that.

Calculation of the infused dose: Nowadays the infusion is expressed in terms of **milliunits per minute**. This can give an accurate idea about the exact amount administered per minute irrespective of the concentration of the solution.

Regulation of the drip: **The drip is regulated by**—(1) Manually, counting the drops per minute commonly practiced. (2) Oxytocin infusion pump which automatically controls the amount of fluid to be infused.

Convenient regime: Because of wide variation in response, **it is a sound practice to start with a low dose (1–2 mIU/min) and to escalate by 1–2 mIU/min at every 20 min intervals up to 8 mIU/min.** The patient should preferably lie on one side or in semi-Fowler's position to minimize venacaval compression.

High-dose oxytocin begins with 4 mIU/min and increased 4 mIU/min at every 20–30 min interval. It is mainly used for augmentation of labor and in active management of labor. Risks of uterine hyperstimulation and fetal heart irregularities are more with high-dose regime.

In majority of cases, a dose of less than 16 mIU/min (2 units in 500 mL Ringer solution with drop rate of 60/minute) is enough to achieve the objective. Conditions where fluid overload is to be avoided, infusion with high concentration and reduced drop rate is preferred (Tables 34.2 and 34.3).

Table 34.2: Calculation of the Dose Delivered in Milliunits (mIu) and Its Correlation with Drop Rate Per Minute

Units of oxytocin mixed in 500 mL Ringer solution (1 unit = 1000 milliunits) (mIU)	Drops per minute (15 drops = 1 mL) 15	30	60
	In terms of mIU/minute		
1	2	4	8
2	4	8	16
8	16	32	64

Table 34.3: Showing the Convenient Regime

Dose of oxytocin	Solution used	Escalating drop rate at intervals of 20–30 min.
To start with 1 unit	500 mL	15 - 30 - 60 Ringer solution
If no response – 2 units	-do-	-do-
If still no response – 8 units	-do-	-do-

For augmentation of labor

Oxytocin infusion is used during labor in uterine inertia or for augmentation of labor or in the active management of labor (details see p. 605). The procedure consists of low rupture of the membranes followed by oxytocin infusion when the liquor is clear. Fetopelvic disproportion must be ruled out beforehand.

Observation during oxytocin infusion

- **Rate of flow of infusion** by counting the drops per minute or monitoring the pump.
- **Uterine contractions**—number of contractions per 10 min duration of contraction and period of relaxation are noted. 'Fingertip' palpation for the tonus of the uterus in between contractions (Fig. 34.1) may be done where gadgets are not available.
- **Peak intrauterine pressure** of 50–60 mm Hg with a resting tone 10–15 mm Hg is optimum when intrauterine pressure monitoring is used (see Fig. 25.2).
- **FHR monitoring** is done by **auscultation** at every 15 min interval or by continuous EFM (see p. 692).
- **Assessment of progress of labor** (descent of the head and rate of cervical dilatation—see p. 151)

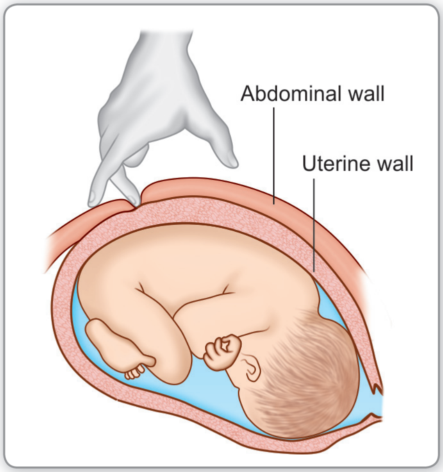


Fig. 34.1: To note the uterine tonus

Indications of stopping the infusion

(1) Nature of uterine contractions—(a) Abnormal uterine contractions occurring frequently (every 2 minutes or less) or lasting more than 60 sec (hyperstimulation) or polysystole. (b) Increased tonus in between contractions.

(2) Evidences of fetal distress (see p. 692). (3) Appearance of untoward maternal symptoms (see p. 573).

DIAGNOSTIC USE OF OXYTOCIN

- ♦ Contraction Stress Test (CST)
- ♦ Oxytocin Sensitivity Test (OST)

CONTRACTION STRESS TEST (CST) (Syn: Oxytocin challenge test)

It is an invasive method to assess the fetal well-being during pregnancy. When there is alteration in FHR in response to uterine contractions, it suggests fetal hypoxia.

Principles: The test is based on determination of the respiratory function of the fetoplacental unit during induced contractions when the blood flow through the unit is curtailed. The objective is to detect the degree of fetal compromise so that a suitable time can be selected to terminate the pregnancy. ***Candidates for CST:*** (1) intrauterine growth restriction, (2) postmaturity, (3) hypertensive disorders of pregnancy, (4) diabetes. ***Contraindications:*** (i) Compromised fetus, (ii) Previous history of cesarean section, (iii) Complications likely to produce preterm labor, (iv) APH, (v) multiple pregnancy.

Procedure: The oxytocin infusion is started in the same manner as mentioned earlier. The initial rate of infusion is 1 mIU/minute which is stepped up at intervals of 20 minutes until the effective uterine contractions are established (vide supra). The alteration of the FHR during contractions is recorded by electronic monitoring (see p. 693). Alternatively, clinical

Interpretation of CST

- *Positive* — **Persistent late deceleration of FHR** with 50% or more of uterine contractions (see p. 695)
- *Negative* — **No late or significant variable deceleration**
- *Suspicious* — **Intermittant late or variable decelerations**
- *Unsatisfactory* — <3 contractions per 10 minutes or an unpredictable tracing
- *Hyperstimulation* — **Decelerations** with contractions lasting >90 seconds or occurring more frequently than every 2 minutes

monitoring can effectively be performed using hand to palpate the hardening of the uterus during contraction and auscultation of FHR during contraction and for 1 minute thereafter. It takes at least 1–2 hours to perform the test.

Importance**:** **A negative test** is associated with good fetal outcome. Whereas a **positive CST** is associated with increased incidence of IUD, fetal distress in labor and low Apagar score. But there **is 50% chance of false-positive results** and as such positive test cases are subjected to other methods of evaluation (Biophysical profile p. 109 and Doppler studies p. 109) for the well-being of the fetus. Suspicious CST should have a repeat test in 24 hours.

ERGOT DERIVATIVES

Out of many ergot derivatives, two are used extensively as oxytocics. **These are:**

- **Ergometrine** (Ergonovine in USA)
- **Methergine** (Methyl ergonovine in USA)

CHEMISTRY: **Ergometrine** is an alkaloid isolated by Dudley and Moir in 1935 from Ergot, a fungus *Claviceps purpurea* that develop commonly in cereals like rye, wheat, etc. The alkaloids are detoxified in the liver and eliminated in the urine. **Methergine** is a semisynthetic product derived from lysergic acid.

MODE OF ACTION: Ergometrine acts directly on the myometrium. It excites uterine contractions which come so frequently one after the other with increasing intensity that the uterus passes into a state of spasm without any relaxation in between.

EFFECTIVENESS: Keeping the physiological functions in mind, **it should not be used in the induction of abortion or labor.** On the contrary, it is highly effective in hemostasis – to stop bleeding from the uterine sinuses, either following delivery or abortion. Methergine is somewhat slower in producing uterine response taking 96 seconds, in contrast to 55 seconds by ergometrine when administered intravenously.

Table 34.4: Composition of Different Ergot Preparations

Preparations	Ampoules	Tablet
Ergometrine (Ergonovine)	0.25–0.5 mg	0.5–1 mg
Methergine (Methyl-ergonovine)	0.125–0.250 mg	0.125–0.5 mg
Syntometrine (Sandoz)	0.5 mg – Ergometrine + 5 units – Syntocinon	

MODE OF ADMINISTRATION: Ergometrine and methergine can be used parenterally or orally. As it produces tetanic uterine contractions, the preparation **should only be used either in the late second stage of labor (after the delivery of the anterior shoulder) or following delivery of the baby**. Syntometrine should always be administered intramuscularly.

Table 34.5: Comparative Study of Onset and Duration of Action of Different Oxytocics

Onset of Action				
Routes	**Ergometrine**	**Methergine**	**Syntometrine**	**Syntocinon on Puerperal Uterus**
IV	45–60 sec	$1^{1}/_{2}$ min	Not to be used	30 sec
IM	6–7 min	7 min	$2^{1}/_{2}$ min	$2^{1}/_{2}$ min
Oral	10 min	10 min	—	—
	Duration of Action			
	3 hrs	3 hrs	3 hrs	8 min

HAZARDS:

(1) Common side effects are **nausea and vomiting**.

(2) Because of its **vasoconstrictive action**, it may **precipitate rise of blood pressure**, myocardial infarction, stroke and bronchospasm.

(3) Prolonged use may lead to **gangrene of the toes** due to its vasoconstrictive effect.

(4) Prolonged use in puerperium may **interfere with lactation** by lowering prolactin level.

USES OF ERGOMETRINE/METHERGINE

Indications	Contraindications
A. Prophylactic **Active management of third stage of labor** (p. 164) as prophylaxis to excess bleeding following delivery	**A. Prophylactic** (1) **Suspected pleural pregnancy**: If given accidentally with the delivery of the first baby, the second baby is compromised by the tetanic contractions of the uterus. (2) **Organic cardiac diseases**: Results in sudden squeezing of blood from the uterine circulation into the systemic circulation causing overloading of the right heart and failure. (3) **Severe preeclampsia and eclampsia**: Sudden rise of blood pressure or development of fits (eclampsia). (4) **Rh-negative mother**: More risk of fetomaternal microtransfusion.
B. Therapeutic To stop the **atonic uterine bleeding,** following delivery, abortion or expulsion of hydatidiform mole	**B. Therapeutic** **Heart disease or severe hypertensive disorders**—because of its vasoconstrictive effect, it may cause transient hypertension or cardiac failure especially when given intravenously. **Oxytocin is a better substitute in such cases.**

CAUTIONS: Ergometrine should not be used during pregnancy, first stage of labor, second stage prior to crowning of the head and in breech delivery prior to crowning.

COMMENTS: As a hemostatic in uterine hemorrhage following expulsion of the fetus irrespective of duration of pregnancy, ergometrine or methergine is the drug of choice (Table 34.6). On the contrary, oxytocin is predominantly used to initiate uterine contractions (induction) and to accelerate uterine contractions in labor.

Table 34.6: Comparative Study of Ergot Derivatives and Oxytocin

	Ergot Derivatives	Oxytocin
Mode of action	Acts directly on the myometrium producing tetanic contraction with complete loss of polarity	Acts on the physiological uterine contractile system. Law of polarity is maintained.
Onset of action	Comparatively slower	Faster in action
Duration	Long sustained	Short lived
Clinical uses	▪ **To stop hemorrhage** following delivery, abortion or expulsion of H mole ▪ Prophylactic use in late second or in third stage to hasten separation of placenta and **to minimize blood loss**	▪ In the **induction of labor** ▪ To **augment uterine contraction** during labor ▪ To **stop postpartum** or **postabortal hemorrhage** along with ergometrine or in isolation
Hazards	▪ Nausea and vomiting ▪ Rise in blood pressure, stroke ▪ Rarely gangrene of the toe	▪ Uterine hyperstimulation, ▪ Antidiuretic effect, uterine rupture ▪ Anginal pain or rarely hypotension
Contraindication	See above	See p. 574

PROSTAGLANDINS (PGS)

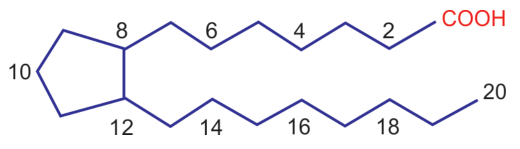
Prostaglandins are the derivatives of prostanoic acid from which they derive their names. They have the property of acting as "local hormones". Prostaglandins were first described and named by Von Euler in 1935.

***Chemistry*:** Prostaglandins are 20-carbon carboxylic acids with a cyclopentane ring which are formed from polyunsaturated fatty acids. **Of the many varieties of prostaglandins, PGE$_2$ and PGF$_{2\alpha}$ are exclusively used in clinical practice. The subscript numeral after the letter indicates the degree of unsaturation.** Inactivation is done in lungs and liver.

***Source*:** Prostaglandins are synthesized from one of the essential fatty acids, arachidonic acid, which is widely distributed throughout the body. **In the female, these are identified in menstrual fluid, endometrium, decidua and amniotic membrane.**

***Prostaglandins (PGs)*:** Increased biosynthesis of PGs of E and F series in the uterus is a prerequisite for labor both term and preterm. PGs are paracrine/autocrine **hormones** as they act on locally at their site of production. Their half-life in the peripheral circulation is about 1–2 minutes. **Decidua is the main source of PGF$_{2\alpha}$, fetal membranes (amnion) produce PGE$_2$ and the myometrium mainly produce PGI$_2$.** In vivo, PGF$_{2\alpha}$ promotes myometrial contractility. PGE$_2$ induces labor with cervical effacement and dilatation. It shortens induction to delivery interval. PGs promote myometrial contraction irrespective of the duration of gestation, whereas oxytocin acts predominantly on the uterus at term or in labor. This has helped the widespread use of PGs to effect first trimester medical termination of pregnancy and also for induction of labor at term. Side effects of PGs are less when used vaginally. Local application of PGE$_2$ gel is the **gold standard** for cervical ripening (Table 34.7).

USE IN OBSTETRICS



Prostanoic acid

- Induction of abortion (MTP and missed abortion see p. 202)
- Termination of molar pregnancy (see p. 227)
- Induction of labor (see p. 598)
- Cervical ripening prior to induction of abortion or labor
- Augmentation (acceleration) of labor
- Management of atonic postpartum hemorrhage (see p. 479)
- Medical management of tubal ectopic pregnancy (see p. 217).

CONTRAINDICATIONS: ♦ Hypersensitivity to the compound ♦ Uterine scar ♦ Active cardiac, pulmonary, renal or hepatic disease; hypotension (PGE$_2$) ♦ Bronchial asthma (PGF$_{2\alpha}$)

MECHANISM OF ACTION: Both PGE$_2$ and PGF$_{2\alpha}$ have got an oxytocic effect on the pregnant uterus when used in appropriate dose. The probable **mechanism of action** is change in myometrial cell membrane permeability and/or alteration of membrane-bound Ca^{++} (see p. 136, 137). PGs also sensitize the myometrium to oxytocin. PGE$_2$ is at least 5 times more potent than PGF$_{2\alpha}$. **PGF$_{2\alpha}$ acts predominantly on the myometrium, while PGE$_2$ acts mainly on the cervix due to its collagenolytic property**. PGE$_2$ causes dissolution collagen bundles and increases submucosal water content of the cervix.

Table 34.7: Prostaglandins in Obstetrics

Advantages	Disadvantages and Side Effects
■ Powerful oxytocic effect, irrespective of duration of gestation ■ Induction of labor (PGE$_2$, PGE$_1$): In cases with (a) low preinduction score; (b) IUFD ■ Used in induction of abortion (PGE$_1$) with success ■ It has got no antidiuretic effect (cf oxytocin) ■ PGE$_1$ (misoprostol) can be used for augmentation of labor	■ It is costly compared to oxytocin ■ Nausea, vomiting, diarrhea, pyrexia, bronchospasm, tachycardia and chills ■ Cervical laceration may occur (PGF$_{2\alpha}$) when used as an abortifacient ■ Tachysystole (hyperstimulation) of the uterus, may occur during induction and may continue for a variable period ■ Risk of uterine rupture in cases with previous scar

Preparations:

Prostaglandin E_2 is widely used because it is less toxic and more effective than **$PGF_{2\alpha}$**. It is however more costly.

Vaginal tablet—Contains 3 mg dinoprostone (Prostin E_2). In the posterior fornix followed by 3 mg after 6–8 hours maximum 6 mg.

Vaginal pessary (with retrieval device) releasing dinoprostone approximately 10 mg over 24 hours. It is removed when cervical ripening is adequate.

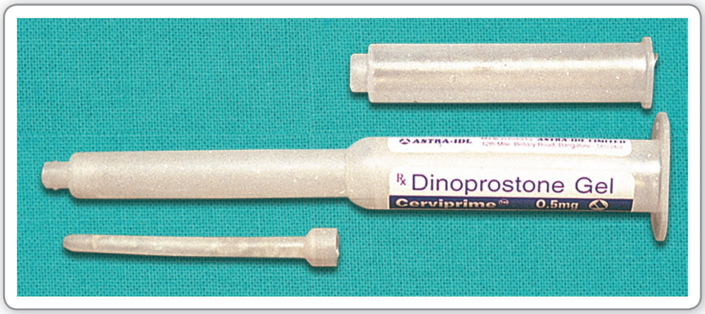


Fig. 34.2: Dinoprostone gel in disposable syringe with catheter

Prostin E_2 (Dinoprostone) gel—500 µg into the cervical canal, below the level of internal os or 1–2 mg in the posterior fornix (Fig. 34.2).

Parenteral: (a) PGE_2 (IV)—Prostin E_2 containing 1 mg/mL, (b) $PGF_{2\alpha}$—Prostin $F_{2\alpha}$ (Dinoprost tromethamine) containing 5 mg/mL, (c) Methyl analogue of $PGF_{2\alpha}$ (Carboprost–containing 250 µg/mL).

PGE_2 is effective for induction of labor causing cervical effacement and dilatation. It reduces the need of oxytocin use and cesarean delivery. PGE_2 preparations are relatively expensive and require refrigeration otherwise it becomes unstable at room temperature.

Methyl ester of PGE_1 (Misoprostol). It is rapidly absorbed and is more effective than oxytocin or dinoprostone for induction of labor.

Misoprostol (PGE_1) has been used for cervical ripening. Primarily it has been used for peptic ulcer disease. Transvaginal misoprostol is used for induction of labor. Oral misoprostol can be used as it is convenient to the patient. It is given 25–50 µg every 3–6 hours by oral or vaginal route. Low doses of oral PGE_1 (dissolving 200 µg tablet in 200 mL tap water) 2–25 µg in solution is safe. It is to be repeated at interval of 2 hours. Buccal or sublingual misoprostol is also used for induction of labor and vaginal delivery. Oxytocin when needed may be added after 4 hours. Misoprostol has been found to be as effective as PGE_2 for cervical ripening and induction of labor. To date no evidence of teratogenic or carcinogenic effects has been observed.

Advantages of PGE_1 over PGE_2: Misoprostol is cheap, stable at room temperature, long self life, easily administered (oral, vaginal or rectal) and has less side effects. Induction delivery interval is short. Need of oxytocin augmentation is less. Failure of induction is less.

Risks: Incidence of tachysystole (hyperstimulation), fetal heart rate changes and meconium passage are high. Rupture of uterus, though rare, has also been observed. It should not be used for cases with previous cesarean birth because the risk of rupture is high. Misoprostol is not yet approved for use in pregnancy by FDA. Use of misoprostol for induction of abortion has been discussed in p. 203-204.

Tachy Systole: Contractions less than 5 in 10 min time averaged over a 30 min window. It may occur in spontaneously or stimulated labor. FHR changes may or may not be present.

REMARKS ABOUT OXYTOCICS: All the oxytocics have got their places in obstetrics

- **To arrest hemorrhage** following delivery (PPH) or abortion, ergot preparation (methergine, ergometrine) is the life saving drug. In **refractory cases of atonic PPH**, $PGF_{2\alpha}$ (IM/intra-myometrial) or PGE_1 (misoprostol) 1,000 µg (rectal) is an effective choice.
- **For induction of labor—**either prostaglandins or oxytocin can be used. **With favorable preinduction cervical score**, there is very little to choose between oxytocin and prostaglandin, **but when the score is unfavorable** as in IUD, shorter period of gestation or in elderly primigravida, **prostaglandins have got a distinct advantage** over oxytocin. **Misoprostol** has certain advantages over PGE_2.
- **In augmentation or acceleration of labor**, oxytocin still enjoys its popularity although prostaglandins are equally effective.
- **For induction of abortion**—(see p. 202). Prostaglandins (misoprostol—PGE_1) has got a distinct advantage over oxytocin. Oxytocin may supplement the effects of PGs in the process.

KEY POINTS

- **Oxytocics** are drugs used to stimulate uterine contractions.
- **Oxytocin** can be used in **pregnancy, labor and puerperium** (p. 574). **Contraindications** (p. 574) and **dangers** (p. 574) of oxytocin use must be carefully assessed.
- **Oxytocin** is given by controlled intravenous infusion (p. 575). During infusion, the woman must be monitored carefully (p. 576). Oxytocin must be preserved between 2°C and 8°C to be effective.
- **Methergine** (ergot derivative) can be used orally or parenterally (p. 577). It differs with oxytocin in its action (Table 34.5). Indications (p. 578), contraindications (p. 578) and hazards (p. 577) of ergot derivatives must be assessed.
- **Prostaglandins** are widely used in obstetrics (p. 579). They have many advantages (p. 579). Of the different preparations, PGE$_2$, PGF$_{2\alpha}$, and PGE$_1$ are commonly used (p. 580). Prostaglandins (PGE$_2$, PGE$_1$) have some advantages over oxytocin in medical induction of labor (p. 579-580).

ANTIHYPERTENSIVE THERAPY

Antihypertensive drugs are essential when the **BP is 160/110 mm Hg to protect the mother from eclampsia, cerebral hemorrhage, cardiac failure and placental abruption**. Aim is to reduce BP to a mean less than 125 mm Hg. Their benefit in mild or moderate hypertension is not yet known. If there is any risk of target organ damage (kidney) antihypertensives are given to maintain BP ≤ 140 mm Hg. **First line therapy** is either methyldopa or labetalol. **Second line drug** is nifedipine. ACE inhibitors are avoided in pregnancy. **The following are the drugs with their pharmacological property and clinical use**:

- **In preeclampsia and eclampsia (see p. 265, 269, 274)**
- **Chronic hypertension (see p. 277, 278)**

***The commonly used drugs are grouped into*:**

I. Sympatholytics	II. Andrenergic receptor blocking agents	III. Vasodilators	IV. Calcium channel blockers	V. ACE inhibitors (see p. 582)
■ Methyldopa ■ Reserpine	■ Labetalol ■ Propranolol	■ Hydralazine ■ Nitroglycerin ■ Sodium nitroprusside	■ Nifedipine ■ Nicardipine	■ Captopiril ■ Lisinopril

Table 34.8: Antihypertensive Drugs

Drugs	Mechanism of Action	Dose	Side Effects	Contraindications and Precautions
METHYLDOPA	Drug of first choice. Central and peripheral antiadrenergic action. Effective and safe for both the mother and the fetus	■ Orally – 250 mg bid – may be increased to 1 g qid depending upon the response ■ IV infusion—250–500 mg	■ Maternal – Postural hypotension, hemolytic anemia, sodium retention, excessive sedation. Coombs' test may be positive ■ Fetal – Intestinal ileus	Hepatic disorders, psychic patients, congestive cardiac failure Postpartum (risk of depression)
HYDRALAZINE	Acts by peripheral vasodilatation as it relaxes the arterial smooth muscle. Orally it is weak and should be combined with methyldopa or β blockers. It increases the cardiac output and renal blood flow	■ Orally – 100 mg/day in four divided doses ■ IV 5–10 mg every 20 min maximum 20 mg	■ Maternal hypotension, tachycardia, arrhythmia, palpitation, lupus like syndrome, fluid retention ■ Fetal – reasonably safe ■ Neonatal — thrombocytopenia	Because of variable sodium retention, diuretics should be used. To control arrhythmias, propranolol may be administered intravenously

Table 34.8: contd. from previous page

LABETALOL	Combined α and β adrenergic blocking agent	■ Orally – 100 mg tid may be increased up to 2,400 mg daily ■ IV infusion (Hypertensive crisis) 20–40 mg IV every 10–15 min until desired effect, maximum up to 220 mg	■ Tremors, headache, asthma, congestive cardiac failure ■ Efficacy and safety with short-term use appear equal to methyldopa	■ Hepatic disorders ■ Asthma, congestive cardiac failure
NIFEDIPINE	Direct arteriolar vasodilatation by inhibition of slow inward calcium channels in vascular smooth muscle	Orally 5–10 mg tid maximum dose 60–120 mg/day	Flushing, hypotension, headache, tachycardia, inhibition of labor	Simultaneous use of magnesium sulfate could be hazardous due to synergistic effect
SODIUM NITROPRUSSIDE	Direct vasodilator (arterial and venous)	IV infusion 0.25–8 µg/kg/min	■ Maternal: Nausea, vomiting, severe hypotension ■ Fetal toxicity due to metabolites — cyanide and thiocynate	**Drug of last resort** for acute hypertension. Should be used in critical care unit for very short time (10 minutes)
NITROGLYCERINE	Relaxes mainly the venous but also arterial smooth muscle	Given as IV infusion 5 µg/min to be increased at every 3–5 min up to 100 µg/min	Tachycardia, headache, Methemoglobinemia	**Used in hypertensive crisis for short time only.** Contraindicated in hypertensive encephalopathy as it increases blood flow and intracranial pressure
ACE inhibitors/ Angiotensin-II receptor blockers (AARB)	ACE inhibitors: Inhibit formation of angiotensin II from angiotensin I. ARB–Blocks Angiotensin-II receptors	■ Captopril Orally 6.25 mg bid ■ Telmisartan orally 20–40 mg a day	■ Maternal: Hypotension, headache, asthenia, arrhythmias ■ Fetal: Oligohydramnios, IUGR, fetal renal tubular dysgenesis, neonatal renal failure, pulmonary hypoplasia	**Should be avoided in pregnancy.** Suitable for chronic hypertension in nonpregnant state or postpartum

DIURETICS

The diuretics are used in the following conditions during pregnancy:

- Pregnancy-induced hypertension with pathological edema
- Eclampsia with pulmonary edema
- Severe anemia in pregnancy with heart failure
- Prior to blood transfusion in severe anemia
- As an adjunct to certain antihypertensive drugs such as hydralazine or diazoxide.

COMMON PREPARATIONS USED: *Frusemide (Loop diuretic)*—Dose – 40 mg tablet daily following breakfast for 5 days a week. In acute conditions, the drug is administered parenterally in doses of 40–120 mg daily. **Mode of action**—It directly prevents reabsorption of sodium and potassium mainly from the loop of Henle.

Hazards—(a) **Maternal complications include**—weakness, fatigue, muscle cramps, hypokalemia and postural hypotension. These can be corrected by potassium supplement during therapy. (b) **Fetal**—In preeclampsia, its routine use should be restricted, as it is likely to cause further reduction of maternal plasma volume, which is already lowered. This may result in diminished placental perfusion leading to fetal compromise. Other hazards include thrombocytopenia and hyponatremia.

Thiazide diuretic is often used in conjunction with other antihypertensives. It is safe in pregnancy. **Dose**: 12.5 mg twice daily maximum up to 50 mg daily may be used. **Side effects are**: Maternal and fetal hyponatremia, acute pancreatitis, rise in uric acid levels, and neonatal thrombocytopenia. In a diabetic patient, it may cause hyperglycemia.

Spironolactone potentiates thiazide or loop diuretics by antagonizing aldosterone. It is a potassium-sparing diuretic. It is contraindicated in hyperkalemia. **Drospirenone** (see p. 622) is an analog of spironolactone. It has antiandrogenic and antimineralocorticoid properties. The dose used in COCs is usually not associated with hyperkalemia (see p. 709).

TOCOLYTIC AGENTS

Preterm labor and delivery can be delayed by drugs in order to improve the perinatal outcome. Short-term delay of 48 hours allows the use of corticosteroids that can reduce the perinatal mortality and serious morbidity significantly. The commonly used drugs are: Betamimetics, Prostaglandin synthetase inhibitors, Magnesium sulfate, Calcium channel blockers, Oxytocin receptor antagonists, Nitric oxide donors and progesterone.

DRUGS: The commonly used drugs are given in the Table 34.9.

Table 34.9: Tocolytic Agents

Drugs	Mechanism of Action	Doses	Side Effects and Precautions
Calcium channel blockers Nifedipine Nicadipine Verapamil	Nifedipine blocks the entry of calcium inside the cell. It is equally effective to $MgSO_4$	**Oral (not sublingual)** 10–20 mg every 3–6 hrs	**Maternal:** Hypotension, headache, flushing and nausea **Combined therapy** with β mimetics or $MgSO_4$ should be avoided
Magnesium sulfate	It acts by **competetive inhibition to calcium ion** either at the motor end plate at the cell membrane reducing calcium influx Decreases acetylcholine release and its sensitivity at the motor end plate. Direct depressant action on the uterine muscle	**Loading dose** 4–6 g IV (10–20% solution) over 20–30 min followed by an infusion of 1–2 g/hr → to continue tocolysis for 12 hrs after the contractions have stopped. Tocolytic effect is poor	**$MgSO_4$ is relatively safe.** Common **maternal side effects** are flushing, perspiration, headache and muscle weakness, rarely pulmonary edema. Neonatal side effects are lethargy, hypotonia, rarely respiratory depression. **Monitoring**: See p. 584. **Contraindication:** Myasthenia gravis and impaired renal function
(Indomethacin) Cyclo-oxygenase inhibitor [**Sulindac** another NSAID is also used as it has less placental transfer]	Reduces synthesis of PGs, thereby **reduces** intracellular free Ca^{++}, activation of MLCK (see p. 136) and uterine contractions. (PGs cause ↑ in free intracellular Ca^{++} and activation of MLCK)	Loading dose 50 mg PO or PR followed by 25 mg every 6 hrs for 48 hrs	**Maternal:** Heartburn, asthma GI bleeding, thombocytopenia, renal injury, platelet dysfunction **Contraindications:** Hepatic disease, active peptic ulcer, coagulation disorders. **Fetal and Neonatal side effects:** (i) Constriction of the ductus arteriosus (due to inhibition of synthesis of PGI_2 and PGE_2) (ii) Oligohydramnios, (iii) Neonatal pulmonary hypertension (iv) IUGR

Table 34.9: contd. from previous page

Drugs	Mechanism of Action	Doses	Side Effects and Precautions
Betamimetics - Terbutaline - Ritodrine - Isoxsuprine (Effective for 48 hrs to allow time for steroids and antibiotics to work)	**Activation** of the intracellular enzymes (adenylate cyclase, cAMP, Protein kinase), **reduces** intracellular free calcium (Ca^{++} ↓↓) and inhibits activation of MLCK (↓↓) → Reduced interaction of actin and myosin → smooth muscle relaxation (see p. 136, 137). β (β_2) receptor stimulation causes smooth muscle relaxation	**Ritodrine** is given by IV infusion, 50 µg/min and is increased by 50 µg every 10 min until contractions cease. Infusion is continued for about 12 hrs after the contractions cease. **Terbutaline** has longer half-life and has fewer side effects. Subcutaneous injection of 0.25 mg every 3 to 4 hrs is given	**Side effects of β mimetics** are more when used parenterally **Maternal:** Headache, palpitation, tachycardia, pulmonary edema, hypotension, cardiac failure, hyperglycemia, ARDS, hyperinsulinemia, lactic acidemia, hypokalemia and even death. **Fetal:** Tachycardia, heart failure, IUFD. **Neonatal:** Hypoglycemia and intraventricular hemorrhage
Oxytocin Antagonists Atosiban	**Oxytocin analogue (antagonist)** that blocks myometrial oxytocin receptors. It inhibits intracellular calcium release (see p. 136), release of PGS and thereby inhibits myometrial contractions	**IV infusion** 300 µg/min Initial bolus may be needed	Nausea, vomiting, chest pain (rarely)
Nitric oxide (NO) Donors Glyceryl trinitrate (GTN)	Smooth muscle relaxant	Patches	May cause cervical ripening - Headache (see p. 625)

ANTICONVULSANTS

Convulsion in pregnancy is largely due to eclampsia. Other causes are—epilepsy, meningitis, cerebral malaria and cerebral tumors. **Eclampsia should be considered first unless proved otherwise by history, examination and investigations.**

The commonly used drugs are given in the tabulated form (Table 34.10).

Table 34.10: Anticonvulsants

Drug	Mode of Action	Dose	Side Effects
MAGNESIUM SULFATE (also see p. 273)	**It decreases the acetylcholine release from the nerve endings and reduces the motor end-plate sensitivity to acetylcholine.** It also **blocks the calcium channels.** It causes vasodilatation, increases cerebral, uterine and renal blood flow. It decreases intracranial edema	**IM**—*Loading dose*: 4 g IV (20% solution) over 3–4 min. To follow 10 g (50% solution) deep IM, 5 g in each buttock. *Maintenance dose:* 5 g (50% solution) deep IM on alternate buttock every 4 hrs **IV**— *Loading dose:* 4–6 g. IV over 15-20 min. *Maintenance dose:* 1–2 g/hr IV infusion **Repeat injections are given only if** knee jerks are present, urine output > 30 mL/hr and respiration rate > 12/min. Therapeutic level of serum Mg is 4–7 mEq/L	**$MgSO_4$ is relatively safe** and is the drug of choice. Muscular paresis (diminished knee jerks), respiratory failure. Renal function is to be monitored. It does not affect the duration of labor **Antidote**: Injection calcium gluconate 10% 10 mL IV **Fetal effects** are usually absent. It is **contraindicated** in patients with myasthenia gravis

Table 34.10: contd. from previous page

Drug	Mode of Action	Dose	Side Effects
DIAZEPAM	Central muscle relaxant and anticonvulsant	Initially 20–40 mg IV to be followed by an infusion containing 500 mL of dextrose with 40 mg of diazepam, the drip rate being 30 drops/min or adjusted as per need **Status epilepticus:** 10–20 mg IV slowly, to be repeated if needed after 1 hr may be followed by IV infusion to a maximum of 3 mg/kg over 24 hrs	■ **Mother**—Hypotension ■ **Fetus**—Respiratory depressant effect which may last for even 3 weeks after delivery. Hypotonia, thermoregulatory problem in newborn
PHENYTOIN	Centrally acting anticonvulsant (for other anticonvulsant drugs see p. 338).	**Eclampsia:** 10 mg/kg IV — at the rate not more than 50 mg/min followed 2 hrs later by 5 mg/kg **Epilepsy:** 300–400 mg daily orally in divided doses **Status epilepticus:** 18 mg/kg slow IV (50 mg/min); maintenance dose of about 100 mg is given at an interval of 6–8 hrs. BP and ECG monitoring should be done	**Maternal** — Hypotension, cardiac arrhythmias, and phlebitis at the injection site. **Fetal hydantoin syndrome** (when used in first trimester) is observed in 5–10% offspring. This may be due to the disease itself with a genetic predisposition, or due to drug metabolism and deficient folate level. For the abnormalities (see p. 338)

ANTICOAGULANTS

Anticoagulants are not commonly used in pregnancy. Cardiac disease, venous thrombosis, antiphospholipid syndrome are some of the indications. The commonly used drugs are discussed here.

Table 34.11: Anticoagulants

Drug	Mode of Action	Dose	Side Effects
Heparin ■ (unfractionated heparin) ■ **Low-molecular-weight heparin (LMWH)**	Inhibits action of thrombin, it also enhances the activity of antithrombin III, increases factor Xa inhibitor **Antidote:** Protamine sulfate	5,000–10,000 IU to be administered parenterally. **DVT and pulmonary embolism:** loading dose 5,000 units IV followed by continuous infusion of 18 units/kg/hr. **Pregnancy:** 5,000-10,000 SC every 12 hrs (with monitoring). **LMWH:** Enoxaparin 1 mg/kg twice daily SC, less antithrombotic effect	**Maternal:** Hemorrhage, urticaria with long-term use thrombocytopenia and osteopenia, hyperkalemia **Fetal:** It does not cross the placenta, not teratogenic **Low-molecular-weight heparins**—as effective and safe as unfractionated heparin. Longer half-life and once daily dose is convenient. Standard dose does not require monitoring
Warfarin	**Interferes with synthesis of vitamin K dependent factors** (II, VII, IX, X)	5–10 mg orally daily for initial 2 days then 3–9 mg daily (taken at the same time each day) depending upon the prothrombin time (INR) (INR – 2.5–3.5)	**Maternal:** Hemorrhage **Fetal:** Warfarin embryopathy (5%), nasal hypoplasia, bone stiplings, optic atrophy, mental retardation, microcephaly, chondrodysplasia punctata. Women with mechanical heart valves, warfarin is preferred. To avoid first trimester.

MATERNAL DRUG INTAKE AND BREASTFEEDING

Maternal drug intake during nursing may have adverse effect not only on lactation but also on the baby through the ingested breast milk. Any drug ingested by a nursing mother may be present in her breast milk, but its concentrations are usually low compared to blood levels in the mother. Usually such low levels are not of any clinical significance to the infant.

However, milk concentrations of some drugs (e.g. iodides) may exceed those in the maternal plasma so that therapeutic doses in the mother may cause toxicity to the infant (Table 34.9). **Common side effects** from maternal medication on breastfed infants are: *diarrhea (antibiotics), irritability (antihistaminics), drowsiness (sedatives, antidepressants, antiepileptics)*.

Benefits of breastfeeding are well known. *The risk of drug exposure to the neonate must be weighed against these benefits.* If the drug amount is 1–2% of the mother, usually no adverse effects are noted. Short-term effects of most drugs on breastfed infants are little. Benefits of breastfeeding must be weighed against the theoretical effects of small amount of drug.

Factors Involved in Drug Transfer into Milk

- Chemical properties of the drug
- Molecular weight
- Degree of protein binding
- Degree of ionic dissociation
- Lipid solubility
- Tissue pH
- Drug concentration in maternal blood
- Duration of exposure time

Guidelines for Medication During Lactation

- Benefits of medication must outweigh the risks
- Select drugs that are most widely tested and with short half-life
- Monitor the infant during the course of therapy

Nonionized, low molecular weight, lipid soluble compounds are usually excreted through breast milk.

Table 34.12: Effects of Various Medications on Lactation and Neonates

Maternal Medication	Effects on Lactation and the Neonate
Oral pill (combined)	Suppression of lactation
Bromocriptine	– do –
Ergot	Vomiting, diarrhea, convulsions in infants
Progesterone (only pill)	It is ideal for breastfeeding mother
Metronidazole (single dose regimen)	No adverse effects have been reported Temporary cessation of lacation (12–24 hrs) is advised.
Antithyroid drugs (PTU)	Can continue with supervision of infant
Warfarin:	Safe in therapeutic doses. Prophylactic vitamin K to the infant (see p. 510)
ACE inhibitors, β blockers	Generally no adverse effects
Cytotoxic agents	Risk of immune suppression. Risks may outweigh the benefits depending on individual drug
Lithium	Lethargy, hypotonia, poor feeding
Narcotics, sedatives and anticonvulsants	Generally no adverse effects
Tetracycline	Tooth staining, delayed bone growth

Drugs Contraindicated During Breastfeeding (AAP 2001)

- Cytotoxic drugs (cyclosporine, doxorubicin, cyclophosphamide): might cause immune suppression.
- Drugs of abuse: Cocaine, heroin, marijuana.
- Radioactive compounds: ^{131}I, Technetium – 99 m
- Drugs, whose effects on nursing infants are unknown but may be of concern: amiodarone (hypothyroidism), sertraline. Benefits of breastfeeding are to be weighed against the negative effects.

Medical complications during pregnancy and lactation are best managed without any drug whenever possible. Risk and benefit ratio of any drug is to be weighted before a drug is to be used. Therapy may be deferred whenever possible till the first trimester is over.

FETAL HAZARDS ON MATERNAL MEDICATION DURING PREGNANCY

TERATOLOGY AND PRESCRIBING IN PREGNANCY

Teratogen causes permanent alteration in the structure and/or function of an organ, acting during the embryonic or fetal life. The *teratogens may be chemical agents (drugs) or physical agents (radiation, heat). The dose (amount) and duration of teratogen exposure may cause variable response from no effect level to lethal level.* **Final results of an abnormal development are: death, malformation, growth restriction and functional disorder**.

The term 'placental barrier' is a contradiction. Virtually drugs cross the barrier with the exception of few with large organic ions such as heparin and insulin. *Approximately 25% of human development defects are genetic in origin, 2–3% are due to drug exposure and about 65% are either unknown or from combination of genetic and environmental factors.*

Mechanism of Teratogenicity: The actual mechanism is unknown. Teratogens may affect through the following ways:

(1) **Folic acid deficiency** leads to deficient methionine production and RNA, DNA synthesis. Folic acid is essential for normal meiosis and mitosis. Periconceptional folate deficiency leads to neural tube defects, cleft lips and palate (see p. 471, 472).

(2) **Epoxides or arena oxides** are the oxidative inter metabolites of many drugs like hydantoin and carbamazepine. These intermediary metabolites have carcinogenic and teratogenic effects unless they are detoxified by fetal epoxide hydrolase.

(3) **Environment and Genes**. Abnormalities that are **multifactorial** depend on the ultimate interaction between the environment and fetal gene mutation. Genotype of the embryo and their susceptibility to teratogens (valproic acid) are the important determinants. Embryonic period (2nd–8th weeks) is most vulnerable. Homozygous gene mutations are associated with more anomalies.

(4) **Maternal disease and drugs** (epilepsy and anticonvulsants) have an increased risk of fetal anomalies. Paternal exposure to drugs or mutagens (polycyclic hydrocarbons) can cause gene mutation and chromosomal abnormality in sperm.

(5) **Homeobox genes** are groups of regulatory genes that control the expression of other genes involved in the normal development of growth and differentiation. Teratogens like retinoic acid can dysregulate these genes to cause abnormal gene expression.

TIMING OF TERATOGEN EXPOSURE AND THE HAZARDS

- **Before D 31**: Teratogen produces an all or none effect. The conceptus either does not survive or survives without anomalies. In early conception only few cells are there. So any damage at that phase is irreparable and is lethal.
- **D 31-D 71** is the critical period for organ formation. Effects of teratogen depend on the following factors: (i) Amount of the drug reaching the fetus, (ii) Gestational age at the time of exposure, (iii) Duration of exposure.
- **After D 71** development of other organs continues. Diethylstilbestrol (DES) related uterine anomalies occur with exposure around 20 weeks (Fig. 34.3).

Brain continues to develop throughout pregnancy and neonatal period. Fetal alcohol syndrome occurs in late pregnancy.

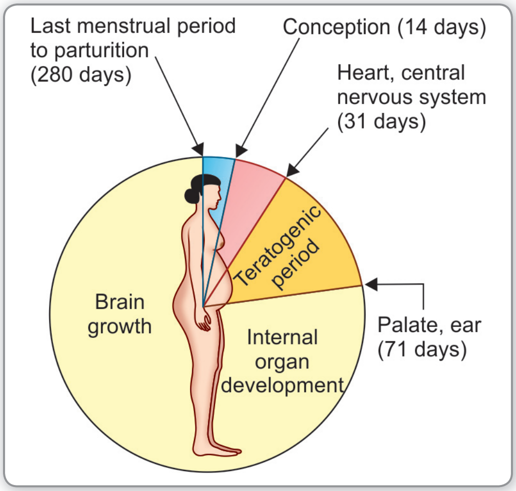

Last menstrual period to parturition (280 days)
Conception (14 days)
Heart, central nervous system (31 days)
Teratogenic period
Palate, ear (71 days)
Brain growth
Internal organ development

Fig. 34.3: Gestational age and teratogenicity

Placental transfer of drugs: Most drugs cross the placental barrier by simple diffusion. The factors responsible for transfer are—(i) Molecular weight (molecular weight > 1,000 Da do not cross the placenta), (ii) Protein binding, (iii) Concentration of free drug, (iv) Lipid solubility, (v) Degree of ionization and tissue pH, (vi) Uteroplacental blood flow and (vii) Placental surface area. The rate of drug transfer across the placenta is increased in late pregnancy.

This is due to: (i) increased unbound drug available for transfer, (ii) increased uteroplacental blood flow, (iii) increased placental surface area, (iv) decreased thickness of the placental membranes.

Table 34.13: FDA Risk Categories for Drugs and Medications: FDA Drug Bulletin (1994)

Category	Definitions
A	Well controlled studies in pregnant women have failed to demonstrate a fetal risk.
B	**No evidence of risk in humans:** well controlled studies in pregnant women have not shown any increased risk of fetal malformation despite adverse findings in animals. The chance of fetal harm is remote but remains a possibility.
C	**Risk cannot be ruled out.** Adequate, well controlled human studies are lacking. Animal studies have shown a risk to the fetus or are lacking as well. Potential benefit may outweigh the risk.
D	**Positive evidence of risk:** Studies in humans have demonstrated fetal risk. Potential benefits from the use of the drug (life-threatening situation) may outweigh the potential risk.
X	**Contraindicated in pregnancy:** Proven fetal risks clearly outweigh any possible benefit. **Drugs in this group are:** Alcohol, ACE inhibitors, Lithium, Methotrexate, Valproic acid, Mifepristone, Danazol, Isotretinoin, Radioactive iodine and others.

Teratogen information databases:
Organization of Teratology Information Services (OTIS): http: ## www. otispregnancy. org.

Keeping these in mind, the following guidelines are formulated:

- If the benefit outweighs the potential risks, only then can the particular drug be used with prior counseling
- Only, well tested and reputed drugs are to be prescribed and that too using the minimum therapeutic dosage for the shortest possible duration.

Table 34.14: Fetal or Neonatal Affections Caused by Various Maternal Medications

Maternal Medication	Fetal or Neonatal Affection and Comment
Cytotoxic drugs	Teratogenic, Abortion, FGR, IUFD, Myelosuppression
Diethylstilbestrol	Vaginal adenosis, cervical hoods, uterine hypoplasia of the female offspring
Androgenic steroids	Masculinization of the female offspring
Lithium	Cardiovascular (Ebstein's) anomalies, fetal diabetes insipidus, polyhydramnios, neonatal goitre, hypotonia and cyanosis, prenatal diagnosis with echocardiography needed
Anticonvulsants	▪ Benefits of treatment outweigh the risks to the fetus. Polytherapy should be avoided
▪ Phenytoin (see p. 399)	▪ Fetal hydantoin syndrome (see p. 338). It includes microcephaly, IUGR, mental retardation, craniofacial abnormalities, hypertelorism, hypoplasia of the nails and distal phalanges
▪ Valproate ▪ Carbamazepine	▪ Increased risk of neural tube defects, ASD cleft palate, polydactyly, hypospadias, craniosynostosis
Aspirin	High doses in the last few weeks cause premature closure of ductus arteriosus, persistent pulmonary hypertension and kernicterus in newborn. Risk of prolonged pregnancy and maternal bleeding due to platelet dysfunction is there

Table 34.14: contd. from previous page

Maternal Medication	Fetal or Neonatal Affection and Comment
Cocaine	Congenital anomalies (cardiac, CNS), abortion, placental abruption, microcephaly
Antimalarials	Chloroquine, Quinine — no evidence of fetal toxicity in therapeutic doses; benefits outweigh the risk
Corticosteroids	High doses (> 10 mg prednisolone daily) may produce fetal and neonatal adrenal suppression. Prolonged systemic use may cause IUGR
Aminoglycosides	Nephrotoxicity, ototoxicity in preterm infants
Chloramphenicol	"Gray baby" Syndrome (peripheral vascular collapse)
Tetracycline	Dental discoloration (yellowish), chelate calcium and deformity Inhibition of bony growth — hypoplasia of the enamel
Quinolones	Arthropathy in animal studies. No malformation was noted
Long-acting sulfonamides	Neonatal hemolysis (G6PD deficiency), Jaundice and kernicterus
Nitrofurantoin	Hemolysis in newborn with G6PD deficiency, if used at term. No congenital defects noted
Metronidazole	No evidence of teratogenic risk, high-dose regimens should not be used
Cimetidine, Omeprazole Ranitidine	No known teratogenic risk
Isotretinoin	Human teratogen
Efavirenz	Known to be teratogenic (see p. 352), risks of NTD
Acyclovir	No increased risk of malformation
ACE inhibitors	Mentioned earlier (see p. 581)
Vitamin A (large dose)	May be teratogenic
All live viral vaccines	Potentially dangerous to the fetus
Narcotics	Maternal: abortion, FGR. Fetal: Depression of CNS—apnea, bradycardia and hypothermia
Smoking (Nicotine, CO, and other polycyclic hydrocarbons)	Placental abruption, placenta previa, prematurity and IUGR increased

Anticoagulants (see p. 585), antihypertensives (see p. 581), diuretics (see p. 582), beta-blockers (see p. 582), and oral contraceptives (see p. 621) are mentioned earlier

ALCOHOL: Heavy drinkers (≥ 3 oz) have major risk to the fetus (6%). **Fetal Alcohol Syndrome (FAS)** is defined as the presence of at least one characteristic from each of the following three categories:

1. **Growth restriction** before and/or after birth.
2. **Facial anomalies**: Small palpebral fissures, indistinct or absent philtrum, epicanthic folds, flattened nasal bridge, short length of nose, thin upper lip, low set and unparallel ears and retarded midfacial development.
3. **CNS dysfunction**: Microcephaly, mental retardation, abnormal neurobehavioral development (attention deficit with hyperactivity).

PATERNALLY MEDICATED DRUGS AFFECTING HUMAN PROGENY: Adverse effects on human progeny has been observed in the form of abortion, congenital malformations, low birth weight and increased perinatal loss when the father has been exposed to lead, anesthetic agents, smoking or caffeine ingestion. These agents probably alter the morphology of the spermatozoa or cause some change in the composition of the semen.

ANALGESIA AND ANESTHESIA IN OBSTETRICS

Relief of pain during labor and delivery is an essential part in good obstetric care. Choice of anesthesia depends upon the patient's conditions and the associate disorders. Anesthetic complications may cause maternal death. Anesthesia following full meal may cause maternal death due to vomiting and aspiration of gastric contents. **Maternal risk factors for anesthesia are:** Short stature, short neck, marked obesity, severe preeclampsia, bleeding disorders, placenta previa, medical disorders, like cardiac, respiratory and neurological disease.

ANATOMICAL AND PHYSIOLOGICAL CONSIDERATIONS

NERVE SUPPLY OF THE GENITAL TRACT: Uterus is under both nervous and hormonal control. Hypothalamus controls the uterine activity through the reticular formation which balances the effects of the two autonomic divisions.

***Motor nerve supply*:** The uterus receives both sympathetic and parasympathetic nerve fibers. The sympathetic nerve fibers arise from lower thoracic and upper lumbar segments of the spinal cord. **The parasympathetic fibers arise from** sacral 2, 3 and 4 segments of the spinal cord (Fig. 34.4).

The *preganglionic fibers of the* sympathetic nerves arising from the spinal cord pass through the ganglia of the sympathetic trunk to aorticorenal plexus where they synapse. The aorticorenal plexus continues as the superior hypogastric plexus or presacral nerve and passes over the bifurcation of aorta and divides into right and left hypogastric nerves. Each hypogastric nerve joins the pelvic parasympathetic nerve of the corresponding side and forms the pelvic plexus (right and left) or inferior hypogastric plexus. The pelvic plexus then continues along the course of the uterine artery as paracervical plexus on each side of the cervix.

Sensory pathway: **Sensory stimuli from the uterine body** are transmitted through the pelvic, superior hypogastric and aorticorenal plexus to the 10th, 11th and 12th dorsal and the first lumbar segments of the spinal cord. Sensory stimuli from cervix pass through the pelvic plexus along the pelvic parasympathetic nerves to sacral segments 2, 3 and 4 of the spinal cord. **Sensory stimuli from upper vagina** pass to 2, 3 and 4 sacral parasympathetic segments and **from lower vagina** pass through the pudendal nerve. The perineum receives both motor and sensory innervation from sacral roots 2, 3 and 4 through the pudendal nerve. The branches of ilioinguinal and genital branch of genitofemoral nerves supply the labia majora and also carry the impulses from the perineum.

NERVOUS CONTROL OF UTERINE ACTIVITY: Regarding motor innervation of the uterus, the sympathetic nerves rather than the parasympathetic have the influences over the uterine activity.

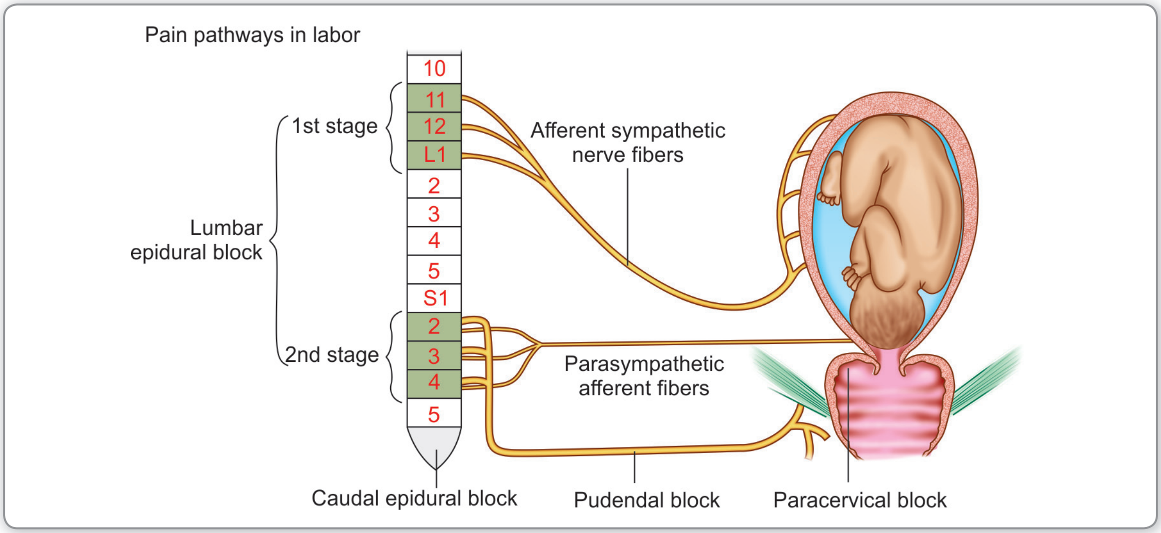
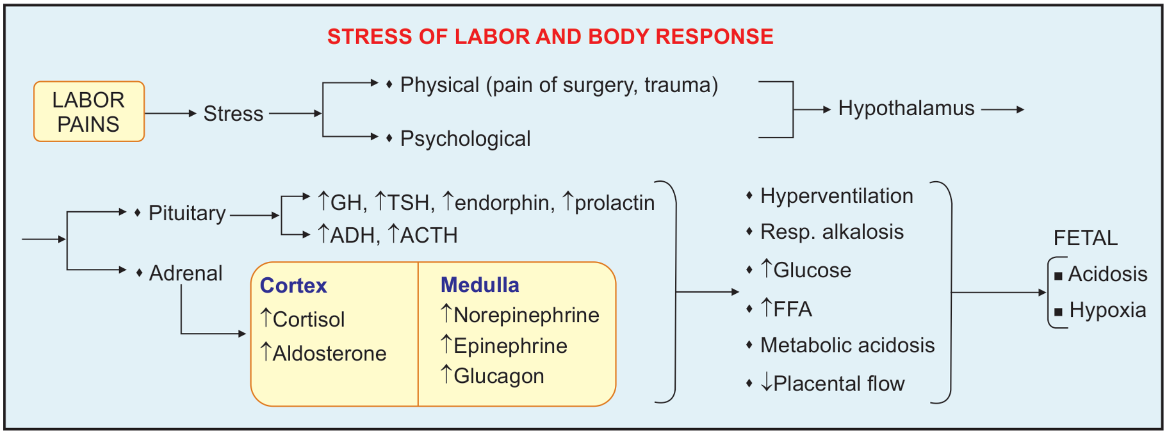


Fig. 34.4: Diagrammatic representation of the pain pathways during labor and the methods of their interruption

HORMONAL CONTROL: It is generally agreed that intact nerve supply is not essential for the initiation and progress of labor. Total spinal block does not inhibit uterine activity, provided blood pressure is not allowed to fall, and normal vaginal delivery can occur in the paraplegic patient. It is believed that some hormones are essential for the control of uterine activity. Oxytocin, a hormone derived from posterior pituitary maintains the uterine activity during labor. Progesterone is the pregnancy-stabilizing hormone. Labor commences when it is withdrawn. **Adrenaline with its beta activity inhibits the contraction of uterus, while its alpha activity excites it**.


STRESS OF LABOR AND BODY RESPONSE
LABOR PAINS
Stress
Physical (pain of surgery, trauma)
Psychological
Hypothalamus
Pituitary
↑GH, ↑TSH, ↑endorphin, ↑prolactin
↑ADH, ↑ACTH
Adrenal
Cortex
↑Cortisol
↑Aldosterone
Medulla
↑Norepinephrine
↑Epinephrine
↑Glucagon
Hyperventilation
Resp. alkalosis
↑Glucose
↑FFA
Metabolic acidosis
↓Placental flow
FETAL
Acidosis
Hypoxia

ANALGESIA DURING LABOR AND DELIVERY

Pain during labor results from a combination of uterine contractions and cervical dilatation. During cesarean delivery incision is usually made around the T12 dermatome anesthesia is required from the level of T4 to block the peritoneal discomfort. Labor pain is experienced by most women with satisfaction at the end of a successful labor. Antenatal (mothercraft) classes, sympathetic care and encouraging environment during labor can reduce the need of analgesia. Drugs have an important part to play in the relief of labor pain but it must not be supposed that they are of greater importance than proper preparation and training for childbirth. The intensity of labor pain depends on the intensity and duration of uterine contractions, degree of dilatation of cervix, distension of perineal tissue, parity and the pain threshold of the subject. **The most distressing time during the whole labor is just prior to full dilatation of the cervix.**

Methods of Pain Relief

- Psychoprophylaxis
- Sedatives and analgesics
- Inhalation agents
- Patient controlled analgesia (PCA)
- Transcutaneous electric nerve stimulation (TENS)
- Regional (neuraxial) analgesia
- General anesthesia

Commonly Used Sedatives and Analgesics in Labor

Drug	Usual Dose	Frequency	Neonatal Half-life (Approx)
Pethidine	50–100 mg IM	4 hrs	13–20 hrs
Fentanyl	50–100 µg IV	1 hr	5 hrs
Nalbuphine	10 mg (IV/IM)	3 hrs	4 hrs
Morphine	10 mg IM	4 hrs	7 hrs
Meperidine	25–50 mg (IV/IM)	4 hrs	

The ideal procedure should produce efficient relief of pain but should neither depress the respiration of the fetus nor depress the uterine activity causing prolonged labor. **The drug must** be nontoxic and safe for both mother and fetus. But it is regretted that no such agent is available at present that fulfills all these conditions. Every case of labor does not require analgesia and only sympathetic explanation may be all that is required.

SEDATIVES AND ANALGESICS

The following factors are important to control the dose of sedative and analgesics:

(1) **Pain threshold**: The threshold of pain varies from patient to patient. Some patients experience severe pain though the uterine contractions are relatively weak. In such cases, it is preferable to control the pain adequately.

(2) **Parity**: The multiparous women need less analgesia due to added relaxation of the birth canal and rapid delivery.

(3) **Maturity of the fetus—**Minimal doses of drugs are indicated while the fetus is thought to be premature to avoid neonatal asphyxia.

For the purpose of selecting a general analgesic drug, labor has been divided arbitrarily into two phases. The first phase corresponds up to 8 cm dilatation of the cervix in primigravidae and 6 cm in case of multipara. The second phase corresponds to dilatation of the cervix beyond the above limits up to delivery. **The first phase is controlled by** sedatives and analgesics, and **the second phase is controlled by** inhalation agents. The idea is to avoid the risk of delivery of a depressed baby.

OPIOID ANALGESICS—Pethidine: For a long time pethidine has been used as an analgesic in labor. It has got strong sedative but less analgesic efficacy. Pethidine is generally used in the early first stage of labor and indicated when the discomfort of labor merges into regular, frequent and painful contractions. The initial dose is 100 mg (1.5 mg/kg body weight) IM and repeated as the effect of the first dose begins to wane, without waiting for the reestablishment of labor pain.

The side effects of pethidine to the mother are nausea, vomiting, delayed gastric emptying. **Ranitidine should be given to inhibit gastric acid production, and emetic effect is counteracted by metoclopramide (10 mg IM)**. Pethidine crosses the placenta and accumulates in fetal tissues. Pethidine reduces baseline variability, depresses respiration and suckling of the newborn when administered before delivery.

Meperidine: Compared to morphine, analgesic effect is one tenth, but respiratory depression effect is less. It is used 25–50 mg (1–3 mg/kg IM) or a PCA pump 15 mg every 10 minutes. Repeated use or PCA in labor, infants may need naloxone at delivery. Maximum placental transfer and neonatal depression occur 2–3 hours of use.

Fentanyl is a short acting synthetic opioid and is equipotent to pethidine. It has less neonatal effects and less maternal nausea and vomiting. It needs frequent dosing. It can be used as PCA.

Phenothiazines: Promethazine (phenergan) is commonly used in labor in combination with an opioid. It does not cause major neonatal depression. Promethazine is a weak antiemetic drug and causes sedation in the mother.

Narcotic antagonists are used to reverse the respiratory depression induced by opioid narcotics. **Naloxone** is given to mother 0.4 mg IV in labor. It may have to be repeated. It is given to the newborn 10 µg/kg IM or IV and is repeated if necessary when the infant is born with narcotic depression. **Naloxone** is given to a newborn born of a narcotic addicted mother, with proper ventilation arrangement only otherwise withdrawal symptoms are precipitated.

Benzodiazepines (Diazepam): It is well tolerated by the patient. It does not produce vomiting and helps in the dilatation of cervix. It is metabolized in the liver. The usual dose is 5–10 mg. It may be used in larger doses in the management of preeclampsia. However, diazepam is avoided in labor. **Major disadvantages are**: Loss of beat to beat variability in labor, neonatal hypotonia and hypothermia. **Flumazenil** is a specific benzodiazepine antagonist. It can reverse the respiratory depression effect of benzodiazepines.

Combination of narcotics and antiemetics: Narcotics may be used in combination with promethazine, metoclopramide or ondansetron. The advantages claimed that the combination potentiates the action of narcotic, produces less respiratory depression and prevents vomiting. But there are also disadvantages like hypotension and delay of second stage of labor.

INHALATION METHODS

Premixed nitrous oxide and oxygen: Cylinders contain 50% nitrous oxide and 50% oxygen mixture. Entonox apparatus has been approved for use by midwives. This agent is used in the second phase (from 8 cm dilatation of cervix to delivery). It can be self administered. Entonox is most commonly used inhalation agent during labor in the UK. Hyperventilation, dizziness, hypocapnia are the side effects. The woman is to take slow and deep breaths before the contractions and to stop when the contractions are over. The woman should be monitored with pulse oximetry.

Table 34.15: Commonly Used Local Anesthetic Agents in Obstetrics

Drug	Usual Dose	Onset	Duration (Min)	Use in Obstetrics
Lignocaine	7 mg/kg	Rapid	60–90 min	Local or pudendal block and also for epidural or spinal for cesarean delivery
Bupivacaine	3 mg/kg	Slow	90–150 min	Epidural or spinal for cesarean delivery

Toxicity—Central Nervous System: Depression, dizziness, tinnitus, metallic taste, numbness of tongue, slurred speech, muscle fasciculation. Rarely generalized convulsions and loss of consciousness. **Cardiovascular toxicity:** Hypotension, cardiac arrythmias and fetal distress due to impaired placental circulation.

REGIONAL (NEURAXIAL) ANESTHESIA

When complete relief of pain is needed throughout labor, epidural analgesia is the safest and simplest method for procuring it. It provides sensory as well as various degrees of motor blockade over a region of the body. But anesthetists/obstetricians have to be trained properly to make use of this very valuable method in normal and abnormal labor.

Continuous lumbar epidural block: A lumbar puncture is made between L_2 and L_3 with the epidural needle (Tuohy needle). With the patient on her left side, the back of the patient is cleansed with antiseptics before injection. When the epidural space is ensured, a plastic catheter is passed through the epidural needle for continuous epidural analgesia. A local anesthetic agent (0.5% bupivacaine) is injected into the epidural space. Full dose is given after a test dose when there is no toxicity. For complete analgesia a block from T10 to the S5 dermatomes is needed. For cesarean delivery a block from T4 to S1 is needed. Repeated doses (top ups) of 4–5 mL of 0.5% bupivacaine or 1% lignocaine are used to maintain analgesia. Epidural analgesia, as a general rule should be given when labor is well established. Maternal hydration should be adequate with normal saline or Hartmann's solution (crystalloid) infusion prior commencing the blockade. The patient's blood pressure, pulse and the fetal heart rate should be recorded at 15 minutes interval following the induction of analgesia and hypotension, if occurs, should be treated immediately. The woman is kept in semilateral position to avoid aortocaval compression.

Advantages of Regional Anesthesia

- The patient is awake and can enjoy the birth time
- CSE allows women to move
- Newborn apgar score generally good
- Lowered risk of maternal aspiration
- Postoperative pain control is better.

Epidural analgesia is especially beneficial in cases like pregnancy-induced hypertension, breech presentation, twin pregnancy and preterm labor. Previous cesarean section is not a contraindication. Epidural analgesia when used there is no change in duration of first stage of labor. But second stage of labor appears to be prolonged by 15–30 minutes. This might lead to frequent need of instrumental delivery like forceps or ventouse.

Contraindications of Epidural Analgesia	Complications of Epidural Analgesia
■ Maternal coagulopathy or anticoagulant therapy	♦ Hypotension due to sympathetic blockade (vasodilatation). Parturient should be well hydrated with (IL) crystalloid solution beforehand, (left lateral change or vasopressure, phenylephrine, may be used)
■ Supine hypotension	♦ Pain at the insertion site. Back pain
■ Hypovolemia	♦ Postspinal headache due to leakage of cerebrospinal fluid through the needle hole in the dura
■ Neurological diseases	♦ Total spinal due to inadvertent administration of the drug in the subarachnoid space
■ Spinal deformity or chronic low back pain	♦ Injury to nerves, convulsions, pyrexia
■ Skin infection at the injection site	♦ Ineffective analgesia

Paracervical nerve block: Is useful for pain relief during the first stage of labor. Following the usual antiseptic safe guards, a long needle (15 cm or more) is passed into the lateral fornix, at the 3 and 9 o'clock positions.

Five to ten milliliter of 1% lignocaine are injected at the site of the cervix and the procedure is repeated on the other side. This dose is quite sufficient to relieve pain for about an hour or two, and injections can be given more than once if necessary. **Bupivacaine is avoided due to its cardiotoxicity**. Paracervical block should not be used where placental insufficiency is present (Fig. 34.5).

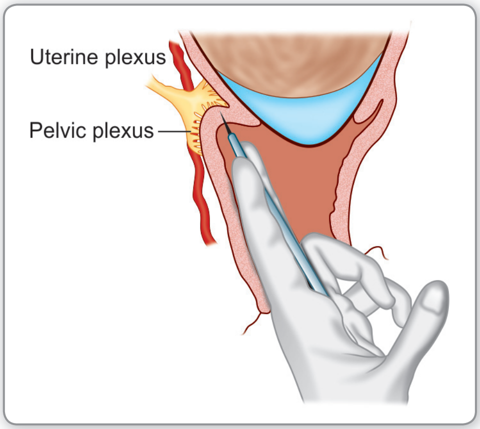


Fig. 34.5: Methods of paracervical block

Although paracervical block may be used from 5 cm dilatation of the cervix, it is most useful toward the end of the first stage of labor to remove the desire to bear down earlier. Paracervical block can only relieve the pain of uterine contraction and the perineal discomfort is removed by pudendal nerve block. **Fetal bradycardia** is a known complication. This is due to decreased placental perfusion resulting from uterine artery vasoconstriction or its direct depressant effect on the fetus following transplacental transfer. This method is not commonly used.

Pudendal nerve block: It is a safe and simple method of analgesia during delivery. Pudendal nerve block does not relieve the pain of labor but affords perineal analgesia and relaxation. **Pudendal nerve block is mostly used for forceps and vaginal breech delivery.** Simultaneous perineal and vulval infiltration is needed to block the perineal branch of the posterior cutaneous nerve of the thigh and the labial branches of the ilioinguinal and genitofemoral nerves (vide supra). This method of analgesia is associated with less danger, both for mother and baby than general anesthesia.

Technique: The pudendal nerve may be blocked by either the transvaginal or the transperineal route.

Transvaginal route: Transvaginal route is commonly preferred. A 20 mL syringe, one 15 cm (6") 22 gauge spinal needle and about 20 mL of 1% lignocaine hydrochloride are required. The index and middle fingers of one hand are introduced into the vagina, the finger tips are placed on the tip of the ischial spine of one side. The needle is passed along the groove of the fingers and guided to pierce the vaginal wall on the apex of ischial spine and thereafter to push a little to pierce the sacrospinous ligament just above the ischial spine tip. After aspirating to exclude blood, about 10 mL of the solution is injected. The similar procedure is adopted to block the nerve of the other side by changing the hands (Fig. 34.7).

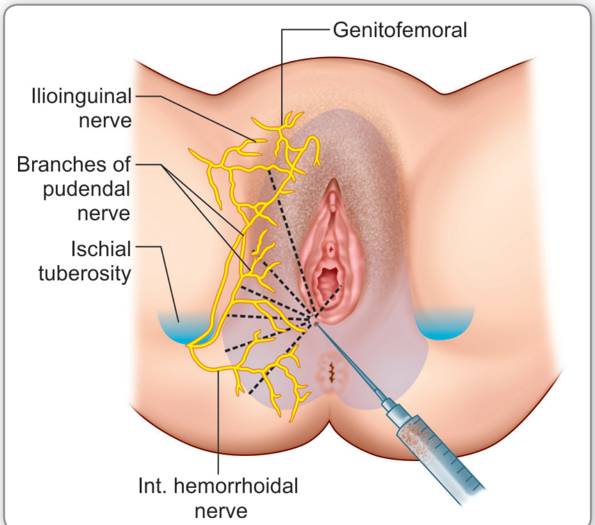


Fig. 34.6: Methods of labial-perineal block for episiotomy during outlet forceps/ventouse operation

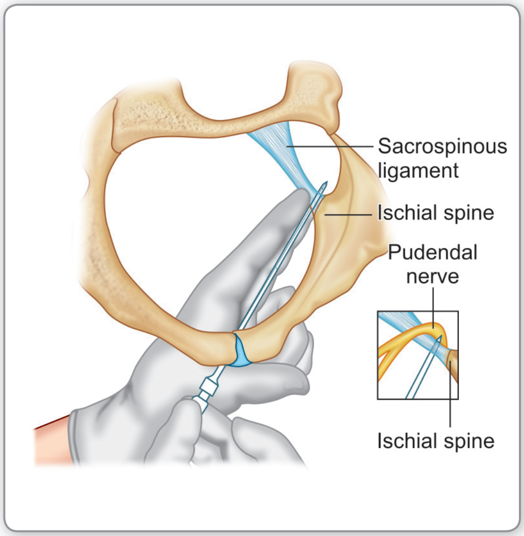


Fig. 34.7: Methods of transvaginal pudendal block anesthesia. Note the relation of the pudendal nerve to the ischial spine in the inset

Complications: Hematoma formation, infection and rarely intravascular injection or allergic reaction. Toxicity may affect: (A) CNS: excitation, ringing in the ears and convulsions. (B) Cardiovascular: tachycardia, hypotension, arrythmias, even cardiac arrest.

Spinal anesthesia: Spinal anesthesia is obtained by injection of local anesthetic agent into the subarachnoid space. It has less procedure time and high success rate. Spinal anesthesia can be employed to alleviate the pain of delivery and during the third stage of labor. For normal delivery or for outlet forceps with episiotomy, ventouse delivery, block should extend from T10 (umbilicus) to S1. For cesarean delivery level of sensory block should be up to T4 dermatome. Hyperbaric bupivacaine (5–10 mg) or lignocaine (25–50 mg) is used. Addition of fentanyl (to enhance the onset of block) or morphine (to improve pain control) may be done. Brief or minimal spinal anesthesia is far safer than prolonged spinal anesthesia. **The advantages of spinal anesthesia are**: (a) less fetal hypoxia unless there is hypotension and (b) minimal blood loss. The technique is not difficult and no inhalation anesthesia is required, but postspinal headache occurs in 5–10% of patients.

Side Effects of Spinal Anesthesia

- Hypotension due to blocking of sympathetic fibers leading to vasodilatation and low cardiac output
- Respiratory depression may occur due to paralysis of respiratory muscles including diaphragm (C_3–C_5)
- Failed block, chemical meningitis, epidural abscess
- Total spinal—due to excessive dose or improper positioning
- Postspinal headache—due to low or high CSF pressure and leakage of CSF
- Meningitis due to faulty asepsis
- Transient or permanent paralysis
- Toxic reaction of local anesthetic drugs
- Paralysis and nerve injury
- Nausea and vomiting are not uncommon
- Urinary retention (bladder dysfunction)

Spinal anesthesia can be obtained by injecting the drug into the subarachnoid space of the third or fourth lumbar interspace with the patient lying on her side with a slight head uptilt. The blood pressure and respiratory rate should be recorded every 3 minutes for the first 10 minutes and every 5 minutes thereafter. Oxygen should be given for respiratory depression and hypotension. Sometimes vasopressor drugs may be required if a marked fall in blood pressure occurs. It is used during vaginal delivery, forceps, ventouse and cesarean delivery.

Combined spinal-epidural analgesia (CSE): An introducer needle is first placed in the epidural space. A small gauge spinal needle is introduced through the epidural needle into the subarachnoid space (needle through needle technique). A single bolus of 1 mL 0.25% bupivacaine with 25 µg fentanyl is injected into the subarachnoid space. The spinal needle is then withdrawn. An epidural catheter is thus sited for repeated doses of anesthetic drug. The method gives rapid and effective analgesia during labor and cesarean delivery. It allows women to move (walking epidural) during labor.

INFILTRATION ANALGESIA

Perineal infiltration: *For episiotomy*—Perineal infiltration anesthesia is extensively used prior to **episiotomy**. A 10 mL syringe, with a fine needle and about 8–10 mL 1% lignocaine hydrochloride (Xylocaine) are required. The perineum on the proposed episiotomy site is infiltrated in a fanwise manner (Fig. 34.6) starting from the middle of the fourchette. **Each time prior to infiltration, aspiration to exclude blood is mandatory.** Episiotomy is to be done about 2–5 minutes following infiltration.

For outlet forceps or ventouse—(Perineal and labial infiltration): The combined perineal and labial infiltration is effective in outlet forceps operation or ventouse traction. A 20 mL syringe, a long fine needle and about 20 mL of 1% lignocaine hydrochloride are required. The needle is inserted just posterior to the introitus. About 10 mL of the solution is infiltrated in a fanwise manner on both sides of the midline (as for episiotomy). The needle is then directed anteriorly along each side of the vulva as far as the anterior-third to block the genital branch of the genitofemoral and ilioinguinal nerve. Five milliliter is required to block each side (Fig. 34.6).

Local abdominal for cesarean delivery: This method is rarely used where regional block is patchy or inadequate. **Technique**: The skin is infiltrated along the line of incision with diluted solution of lignocaine (2%) with normal saline. The subcutaneous fatty layer, muscle, rectus sheath layers are infiltrated as the layers are seen during operation. The operation should be done slowly for the drug to become effective.

PATIENT CONTROLLED ANALGESIA (PCA): Narcotics are administered by mother herself from a pump at continuous or intermittent demand rate through intravenous route. Total dose is limited as there is a lockout interval.

This offers better pain control than high doses given at a long interval by the midwife. Maternal satisfaction is high with this method. Drugs commonly used are fentanyl, meperidine or remifentanil.

PSYCHOPROPHYLAXIS (*Syn: Natural childbirth*): It is psychological method of antenatal preparation designed to prevent or at least to minimize pain and difficulty during labor. For most women, labor is a time of apprehension, fear and agony. As a result of suitable antenatal preparation, majority of women have labor that is easy and painless.

Relaxation and motivation can reduce the fear and apprehension to a great extent. Patient is taught about the physiology of pregnancy and labor in antenatal (mothercraft) classes. Relaxation exercises are practiced. Husband or the partner is also involved in the management. His presence in labor would encourage the bearing down efforts. Need of analgesia would be less.

TRANSCUTANEOUS ELECTRIC NERVE STIMULATION (TENS): It is a noninvasive procedure and is preferred by many women during labor. Electrodes are placed over the level of T10 – L1 and S2 – 4. Current strength can be adjusted according to pain. It works by inhibiting transmitter release through interneuron level. However, no change in pain score was observed when TENS was switched on.

GENERAL ANESTHESIA FOR CESAREAN SECTION

The following are the important considerations of general anesthesia for cesarean section:

- Cesarean section may have to be done either as an elective or emergency procedure
- Ryel's tube aspiration of gastric contents is to be done, especially when the stomach contains food materials
- A large number of drugs pass through the placental barrier and may depress the baby
- Uterine contractility may be diminished by volatile anesthetic agents like ether, halothane
- Halothane, isoflurane cause cardiac depression, hepatic necrosis and hypotension
- Hypoxia and hypercapnia may occur
- Time interval from uterine incision to delivery is related directly to fetal acidosis and hypoxia
- Longer the exposure to general anesthetic before delivery the more depressed is the Apgar score.

Preoperative preparations: These safety measures should be taken to prevent complications of general anesthesia.

- **Preoperative medication with sedatives or narcotics is not required** as they cause respiratory depression of the fetus.
- Fasting of about 6 hours is preferable for an elective surgery.
- High-risk women in labor should preferably not be allowed to eat.
- Ryel's tube aspiration of gastric contents is to be done when the stomach contains food materials.
- H_2-blocker (Ranitidine 150 mg orally) should be given night before (elective procedure). H_2 receptor blocking agent and metoclopramide is to be given IM especially to women with high risks (obesity).
- Non-particulate antacid (0.3 molar sodium citrate 30 mL) is given orally before transferring the patient to theater to neutralize the existing gastric acid.
- While on the theater table, left lateral tilt of the woman is maintained with a wedge on the back. This is to avoid autocaval compression as it is detrimental to both mother and fetus.
- Metoclopramide (10 mg IV) is given after minimum 3 minutes of preoxygenation to decrease gastric volume and to increase the tone of lower esophageal sphincter.
- Intubation with adequate cricoid pressure following induction should be done.
- **Uterine incision — Delivery (U-D) interval** is more predictive of neonatal status (Apgar score). Prolonged U-D interval of more than 3 minutes results in lower Apgar scores and neonatal acidosis.
- Awake extubation should be a routine.

Preoxygenation with 100% oxygen is administered by tight mask fit for more than 3 minutes. **Induction** of anesthesia is done with the injection of thiopentone sodium 200–250 mg (4 mg/kg) as a 2.5% solution intravenously.

Muscle relaxants: Succinylcholine is commonly used immediately after the induction drug to facilitate intubation. It is a short acting muscle relaxant with rapid onset of action.

Intubation: An assistant is asked to apply **cricoid pressure** as soon as the consciousness is lost. **Intubation** is done with a cuffed endotracheal tube and the cuff is inflated. Presence of obesity, severe edema, neck abnormalities, short stature or airway abnormalities make intubation difficult.

Anesthesia is maintained with 50% nitrous oxide, 50% oxygen and a trace (0.5%) of halothane. Relaxation is maintained with nondepolarizing muscle relaxant (vecuronium bromide 4 mg or atracurium 25 mg). After delivery of the baby, the nitrous oxide concentration should be increased to 70% and narcotics are injected intravenously to supplement anesthesia.

Complications of general anesthesia: Aspiration of gastric contents (Mendelson's syndrome) is a serious and life threatening one. Delayed gastric emptying due to high level of serum progesterone, decreased motilin and maternal apprehension during labor is the predisposing factor. The complication is due to aspiration of gastric acid contents ($pH < 2.5$) with the development of chemical pneumonitis, lung damage, atelectasis and bronchopneumonia. Right lower lobe is commonly involved as the aspirated food material reach the lung parenchyma through the right bronchus. **Clinical presentation:** tachycardia, tachypnea, bronchospasm, rhonchi, rales, cyanosis, decreased PaO_2 and hypotension. X-ray chest reveals right lower lobe involvement.

Management: Immediate suctioning of oropharynx and nasopharynx is done to remove the inhaled fluid. Bronchoscopy may be needed if there is any large particulate matter. Continuous positive pressure ventilation to maintain arterial oxygen saturation of 95% is done. Pulse oximeter is a useful guide. Antibiotics are administered when infection is evident. Role of corticosteroid is doubtful.

Other complications of general anesthesia are: (i) Failure in intubation and ventilation, (ii) Nausea, vomiting and sore throat.

KEY POINTS

- **Commonly used antihypertensives** in pregnancy are: Methyldopa, Labetalol, Hydralazine and Nifedipine (p. 582). Hydralazine, Labetalol, nitroglycerine and sodium nitroprusside are used for hypertensive crisis (p. 582). ACE inhibitors should be avoided in pregnancy.
- **Commonly used tocolytics are** — Betamimetics (Terbutaline, Ritodrine, Isoxsuprine), Indomethacin, Calcium channel blockers, Magnesium sulfate, Oxytocin antagonists and Nitric oxide donors (p. 583, 584).
- Tocolytics are used to delay preterm labor for a short-term period (48 hours). Side effects and the precautions of use must be known (see p. 583).
- **Anticonvulsants** used in pregnancy are — Magnesium sulfate, Diazepam and Phenytoin (p. 584). $MgSO_4$ is the drug of choice in eclampsia.
- **While breastfeeding,** the benefits of breastmilk must be weighed against the risk drug exposure to the neonate (p. 586). Information as regard some commonly used drugs are available (Table 34.12).
- **Teratogens** exert their effects through different mechanisms (p. 587). The hazards of drugs depend upon the placental transfer of drugs and the period of gestation (p. 587). Information as regard some commonly used drugs are available (Table 34.14).
- **Opioid analgesics** are commonly used in labor (p. 592). They work primarily as a sedative. Of the inhalation methods, premixed nitrous oxide and oxygen are commonly used (p. 592).
- **Epidural analgesia** is the safe, effective and simple method of regional anesthesia. One must know the contraindications and complications of its use (p. 593). In obstetrics, it is especially beneficial for some cases (p. 593).
- **Pudendal block** is good for perineal analgesia and is used for forceps and vaginal breech delivery.
- **Spinal anesthesia** has some advantages but it should be used carefully to avoid the side effects (p. 595).
- Complications of general anesthesia could be reduced when few preoperative safety measures are taken (p. 596).
- **Mendelson's syndrome** is a serious complication of general anesthesia. This can be prevented when the safety measures are taken beforehand (p. 597).

QUESTIONS

1. What are oxytocics? Mention the commonly used oxytocics in obstetrics? Discuss in brief how oxytocin could be used for augmentation of labor? (p. 573-74)

Write Short Notes on:

A. Contraindications of methergine (ergometrine) (p. 578)

B. Advantages of prostaglandin use in obstetrics over oxytocin (p. 579, 601)

C. Complications of spinal anesthesia (p. 595)

Chapter 35

Induction of Labor

Induction of labor (IOL) means initiation of uterine contractions (after the period of viability) **by any method (medical, surgical or combined) for the purpose of vaginal delivery**. The patient and the family members are informed about the benefits, potential complications and the possibility of cesarean delivery. Overall induction rate is 10%. **Augmentation** of labor is the process of stimulation of uterine contractions (both in frequency and intensity) that are already present but found to be inadequate.

PURPOSE OF INDUCTION OF LABOR: When the risks of continuation of pregnancy either to the mother or to the fetus is more, induction is indicated. Before induction one must ensure the gestational age as well as pulmonary maturity of the fetus. Rarely, preterm induction may have to be done.

Elective induction of labor means initiation of labor at term pregnancy without any acceptable medical or obstetric indication. It is done for the convenience of the patient, obstetrician or the hospital. Unless for a selective patient (e.g. who have history of rapid labors) the **social indications should not be recommended**. The **major risks are** iatrogenic prematurity, increased cesarean delivery for failed induction.

Table 35.1: Indications for Induction of Labor (IOL)

- Pre-eclampsia, eclampsia (see p. 266, 275) (hypertensive disorders in pregnancy)
- Maternal medical complications
  - Diabetes mellitus (see p. 325)
  - Chronic renal disease (see p. 278)
  - Cholestasis of pregnancy (p. 336)
- Postmaturity (see p. 371)
- Abruptio placentae (see p. 294)
- Intrauterine Growth Restriction (IUGR) see p. 533
- Rh-isoimmunization (see p. 386)
- Premature rupture of membranes (see p. 370)
- Fetus with a major congenital anomaly
- Intrauterine death of the fetus (see p. 378)
- Oligohydramnios, polyhydramnios (see p. 251)
- Unstable lie-after correction into longitudinal lie (see p. 459)

Table 35.2: Contraindications of Induction of Labor

- Contracted pelvis and cephalopelvic disproportion
- Malpresentation (breech, transverse or oblique lie)
- Previous classical cesarean section or hysterotomy
- Uteroplacental factors: Unexplained vaginal bleeding, vasaprevia, placenta previa
- Active genital herpes infection
- High-risk pregnancy with fetal compromise
- Heart disease
- Pelvic tumor
- Elderly primigravida with obstetric or medical complications
- Umbilical cord prolapse
- Cervical carcinoma

Common Indications for Induction of Labor (IOL)

- Postmaturity
- Pre-eclampsia/eclampsia
- Intrauterine fetal death
- Premature rupture of the membranes
- Congenital malformation of the fetus
- Antepartum hemorrhage
- Chronic hydramnios

Dangers of Induction of Labor

Maternal	Fetal
♦ Psychological upset when there is induction failure and cesarean section is done ♦ Tendency of prolonged labor due to abnormal uterine action ♦ Increased need of analgesia during labor ♦ Increased operative interference ♦ Increased morbidity	♦ Iatrogenic prematurity ♦ Hypoxia due to uterine dysfunction ♦ Prolonged labor ♦ Operative interference

PARAMETERS TO ASSESS PRIOR TO INDUCTION OF LABOR: When induction is considered for **fetal interest**, one must ensure the gestational age and maturity (pulmonary) of the fetus. However, induction for **maternal interest** may compel to ignore the fetus.

Maternal	Fetal
■ To confirm the indication for IOL ■ Exclude the contraindication of IOL ■ Assess Bishop score (score > 6, favorable) ■ Perform clinical pelvimetry to assess pelvic adequacy ■ Adequate counseling about the risks, benefits and alternatives of IOL with the woman and the family members	♦ To ensure fetal gestational age ♦ To estimate fetal weight (clinical and USG p. 84). ♦ Ensure fetal lung maturation status (p. 124). ♦ Ensure fetal presentation and lie. ♦ Confirm fetal well-being (p. 119).

Table 35.3: Predictive Factors for Successful Induction of Labor

Period of gestation	Pregnancy nearer the term or post-term—more the success
Preinduction score	Bishop score ≥ 6 is favorable. Dilatation of the cervix is most important.
Sensitivity of the uterus	Positive oxytocin sensitivity test (see p. 575) is favorable for IOL
Cervical ripening	Favorable in multiparous and in cases with PROM. Less responsive in elderly primigravidae or cases with prolonged retention of dead fetus.
Presence of fetal fibronectin (fFN) (p. 366) in vaginal swab (> 50 ng/mL)	Favorable for successful IOL
Other positive factors	Maternal height > 5'; Normal BMI, EFW < 3 kg

Cervical Ripening is a series of complex biochemical changes in the cervix which is mediated by the hormones. There is alteration of both cervical collagen and ground substance. Ultimately, the cervix becomes soft and pliable.

Table 35.4: Methods of Cervical Ripening

Pharmacological Methods	Nonpharmacological Methods
Prostaglandins (PGs) ■ Dinoprostone (PGE_2): Gel, tablet, suppository ■ Misoprostol (PGE_1): Tablets.	■ Stripping the membranes
Oxytocin	■ Amniotomy (artificial rupture of the membranes)
Progesterone receptor antagonists ■ Mifepristone (RU 486)	■ Mechanical dilators, osmotic dilators (laminaria)
Relaxin: a protein hormone from corpus luteum, dissolutes cervical connective tissue	■ Transcervical balloon catheter
Hyaluronic acid	■ Extra-amniotic saline infusion
Estrogen	

Use (off-level) of Misoprostol (PGE_1) for cervical ripening is safe and effective (ACOG–2003)

Table 35.5: Bishop's Preinduction Cervical Scoring System (Modified)

Parameters	**Score**			
	0	**1**	**2**	**3**
Cervix				
■ Dilatation (cm)	Closed	1–2	3–4	5 +
■ * Effacement (%)	0-30	40-50	60-70	≥ 80
■ Consistency	Firm	Medium	Soft	–
■ Position	Posterior	Midline	Anterior	–
Head: Station	–3	–2	–1,0	+ 1, + 2
Total score = 13; Favorable score = 6-13; Unfavorable score = 0-5				
* Cervical length (cm)	> 4	2-4	1-2	< 1

*** Modification (1991) replaces effacement (%) with cervical length in cm.**

METHODS OF INDUCTION OF LABOR

♦ **Medical** ♦ **Surgical** ♦ **Combined**

MEDICAL INDUCTION

DRUGS USED: ♦ **Prostaglandins PGE$_2$, PGE$_1$** ♦ **Oxytocin** ♦ **Mifepristone**

Prostaglandins (Table 35.7): Act locally (autocrine and paracrine hormones) on the contiguous cells. PGE$_2$ and PGF$_{2\alpha}$ both cause myometrial contraction. But **PGE$_2$ is primarily important for cervical ripening whereas PGF$_{2\alpha}$ for myometrial contraction. PGE$_2$ has** greater collagenolytic properties and also sensitizes the myometrium to oxytocin. Intracervical application of dinoprostone (PGE$_2$ – 0.5 mg) gel is the **gold standard** for cervical ripening (see p. 580). It may be repeated after 6 hours for 3 or 4 doses if required. The woman should be in bed for 30 minutes following application and is monitored for uterine activity and fetal heart rate. Side effects are few (p. 579).

Misoprostol (PGE$_1$) is currently being used either **transvaginally** or **orally** for induction of labor (ACOG 2003). Oral use of misoprostol is less effective than vaginal administration. A dose of 25 µg vaginally every 4 hours is found either superior or similarly effective to that of PGE$_2$ for cervical ripening and labor induction. With the above dose schedule, the risk of uterine hyperstimulation, meconium stained liquor and fetal heart irregularities are reduced. Total 6–8 doses are used. Buccal and sublingual use of misoprostol can avoid the first pass hepatic circulation and can maintain the serum bioavailability similar to that of vaginal use. **Side effects are** (see p. 580): Tachysystole, meconium passage and possibly uterine rupture. **It is contraindicated in women with previous cesarean birth (see p. 580)**.

Table 35.6: Methods of Induction of Labor and the Common Clinical Conditions

Medical Methods	**Surgical Methods**	**Combined Methods**
■ Intrauterine fetal death ■ Premature rupture of membranes ■ In combination with surgical induction (ARM)	■ Abruptio placentae ■ Chronic hydramnios ■ Severe pre-eclampsia/ eclampsia ■ In combination with medical induction ■ To place scalp electrode for electronic fetal monitoring	■ To shorten the induction — delivery interval (commonly done). Medical methods followed by surgical or surgical methods followed by medical

Table 35.7: Merits and Demerits of Oxytocin and Prostaglandins in Medical Induction of Labor

	Oxytocin	Prostaglandins (PGE_2, PGE_1)
Cost	Cheaper	PGE_2 costly, PGE_1 less costly
Stability	Needs refrigeration (may be kept for 1 month at 30°C).	PGE_2 needs refrigeration; PGE_1 is stable at room temperature
Administration	Intravenous (IV) infusion	Intravaginally or orally
Effectiveness	Less with: ▪ Low Bishop score ▪ IUFD ▪ Lesser weeks of pregnancy	More effective in those cases as it has got more collagenolytic properties and it also sensitizes the myometrium to oxytocin
Side effects	Uterine hyperstimulation mainly with high dose (ceases following stoppage of infusion)	Low dose schedule has got minimal side effects (p. 580) Tachysystole may last longer — (may need Inj. terbutaline 0.2 mg sc) (see p. 584).
Systemic side effects	Less; water intoxication	Systemic side effects may be troublesome specially with oral or intravenous infusion. Vaginal route use has got minimal side effects.
Antidiuretic (ADH) effect	In high dose	No such

Comments: With favorable preinduction cervical score, there is very little to choose but where the score is poor, prostaglandin has got a distinct advantage over oxytocin.

Oxytocin is an endogenous uterotonic that stimulates uterine contractions. Oxytocin receptors present in the myometrium are more in the fundus than in the cervix. Receptor concentrations increase during pregnancy and in labor (cf. prostaglandins). Oxytocin acts by (a) receptor mediation; (b) voltage-mediated calcium channels (see p. 573) and (c) prostaglandin production. Because of short half life (3–4 minutes) plasma levels fall rapidly when intravenous infusion is stopped (see p. 573-74). Oxytocin is effective for induction of labor when the cervix is ripe. It is less effective as a cervical ripening agent.

Mifepristone (progesterone receptor antagonists) blocks both progesterone and glucocorticoid receptors. RU 486, 200 mg vaginally daily for 2 days has been found to ripen the cervix and to induce labor. **Onapristone (ZK 98299)** is a more selective progesterone receptor antagonists.

Mechanical methods (p. 599) are effective. Advantages are: low cost, low risk of tachysystole. Disadvantages: infection.

SURGICAL INDUCTION

METHODS:

- **Artificial rupture of the membranes (ARM) (Fig. 35.1)**
- **Stripping the membranes**

Low rupture of the membranes (LRM)

Mechanism of onset of labor: May be related with (a) stretching of the cervix; (b) separation of the membranes (liberation of prostaglandins) and (c) reduction of amniotic fluid volume.

Effectiveness depends on: (1) State of the cervix; (2) Station of the presenting part. Induction delivery interval is shorter when amniotomy is combined with oxytocin than when either method is used singly.

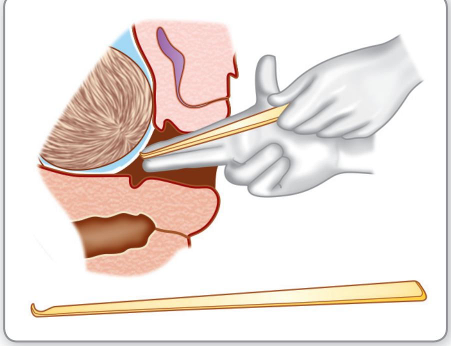


Fig. 35.1: Artificial rupture of the membranes (ARM) using amni-hook

Advantages of amniotomy: (a) High success rate; (b) Chance to observe the amniotic fluid for blood or meconium; (c) Access to use fetal scalp electrode or intrauterine pressure catheter or for fetal scalp blood sampling.

Limitation: It cannot be employed in an unfavorable cervix (long, firm cervix with os closed). The cervix should be at least one finger dilated.

Indications: See Table 35.6.

Contraindications: Intrauterine fetal death, Maternal AIDS, Genital active herpes infection.

Immediate beneficial effects of ARM

- Lowering of the blood pressure in pre-eclampsia-eclampsia.
- Relief of maternal distress in hydramnios.
- Control of bleeding in APH.
- Relief of tension in abruptio placentae and initiation of labor.

These benefits are to be weighed against the risks involved in the indications for which the method is adopted.

HAZARDS OF ARM

- Once the procedure is adopted, **there is no scope of retreating from the decision of delivery**.
- **Chance of umbilical cord prolapse** — The risk is low with engaged head or rupture of membranes with head fixed to the brim.
- **Amnionitis** — Careful selection of cases with favorable preinduction score will shorten the induction-delivery interval. Meticulous asepsis during the procedure reduces the risk.
- **Accidental injury** to the placenta, cervix or uterus, fetal parts or vasa previa (see p. 301). Care taken during rupture of the membranes minimizes the problem.
- **Liquor amnii embolism** (rare).

LOW RUPTURE OF THE MEMBRANES (LRM)

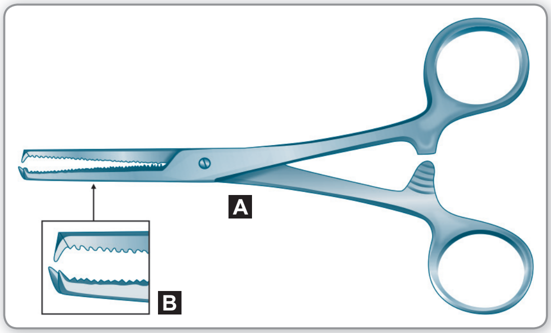
It is widely practised nowadays with high degree of success. The membranes below the presenting part overlying the internal os are ruptured to drain some amount of amniotic fluid.

Contraindication: It is preferably avoided in chronic hydramnios, as there is risk of sudden massive liquor drainage. Sudden uterine decompression may precipitate early placental separation (abruption). In such a case controlled ARM (see p. 249) is done.

Procedures: ***Preliminaries:*** **It is an indoor procedure**. The patient is asked to empty her bladder. The procedure may be conducted in the labor ward or in the operation theater if the risk of cord prolapse is high.

Actual steps (Fig. 35.3):

- FHR status is monitored before and after the procedure.
- The patient is in lithotomy position.
- Full surgical asepsis is to be taken.
- Two fingers are introduced into the vagina smeared with antiseptic ointment. The index finger is passed through the cervical canal beyond the internal os. The membranes are swept free from the lower segment as far as reached by the finger.
- With one or two fingers still in the cervical canal with the palmar surface upwards, a long Kocher's forceps (Fig. 35.2) with the blades closed or an *amnion hook* is introduced along the palmar aspect of the fingers up to the membranes.



Figs 35.2A and B: (A) Kocher's artery forceps. (B) Showing the tip of the Kocher's

- The blades are opened to seize the membranes and are torn by twisting movements. **Amnihook** is used to scratch over the membranes. This is followed by visible escape of amniotic fluid.

If the head is not engaged, an assistant should push the head to fix it to the brim of the pelvis to prevent cord prolapse. If the head is deeply engaged and the drainage of liquor is insignificant, gentle pushing of the head up, facilitates escape of desired amount of amniotic fluid.

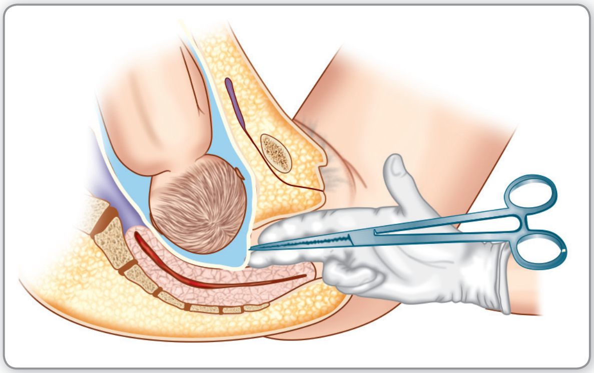


Fig. 35.3: Methods of low rupture of membranes by Kocher's artery forceps

After the membranes rupture, the following are to be assessed:

- ♦ (a) Color of the amniotic fluid; (b) Status of the cervix; (c) Station of the head; (d) Detection of cord prolapse if any; (e) FHR pattern is again checked. In high-risk cases scalp electrode for fetal monitoring is applied.
- ♦ A sterile vulval pad is placed. Prophylactic antibiotic may be prescribed.

Hazards: (1) Cord prolapse; (2) Uncontrolled escape of amniotic fluid and placental abruption; (3) Injury to the cervix or the presenting part; (4) Rupture of vasa previa leading to fetal blood loss; (5) Amnionitis.

STRIPPING THE MEMBRANES

Stripping (sweeping) of the membranes means digital separation of the chorioamniotic membranes from the wall of the cervix and lower uterine segment. It is thought to work by release of endogenous prostaglandins from the membranes and decidua. Manual exploration of the cervix triggers Ferguson reflex (see p. 136) which promotes oxytocin release from maternal pituitary. Sweeping of the membranes is done prior to ARM. It is simple, safe and beneficial for induction of labor.

As an isolated procedure, stripping the membranes off from its attachment from the lower segment is an effective procedure for induction provided cervical score is favorable. It is used as a preliminary step prior to rupture of the membranes. It is also used to make the cervix ripe.

Criteria to be fulfilled for membrane stripping are: (a) The fetal head must be well applied to the cervix; (b) The cervix should be dilated so as to allow the introduction of the examiner's finger.

Table 35.8: Merits and Demerits of Oxytocin and Low Rupture of the Membranes (Amniotomy) as an Isolated Method

	Oxytocin	Amniotomy (LRM)
Exclusive indications	▪ IUD	▪ APH ▪ Hydramnios ▪ Severe pre-eclampsia/eclampsia
Prerequisites	Can be employed irrespective of the state of the cervix and the station of the head	The cervical canal must be at least one finger dilated and the head should preferably be engaged.
Effectiveness	Quite satisfactory and the procedure can be repeated at intervals	If the procedure fails to initiate labor within 4 hours, should be supplemented by oxytocin if required.
Special benefits	Reversibility of the decision	(a) Observation of liquor for blood or meconium stain. (b) Access to uterine cavity for the use of — (i) fetal scalp electrode— electronic monitoring, (ii) fetal scalp blood sampling, (iii) intrauterine pressure catheter
Hazards	See p. 574	See p. 601-602

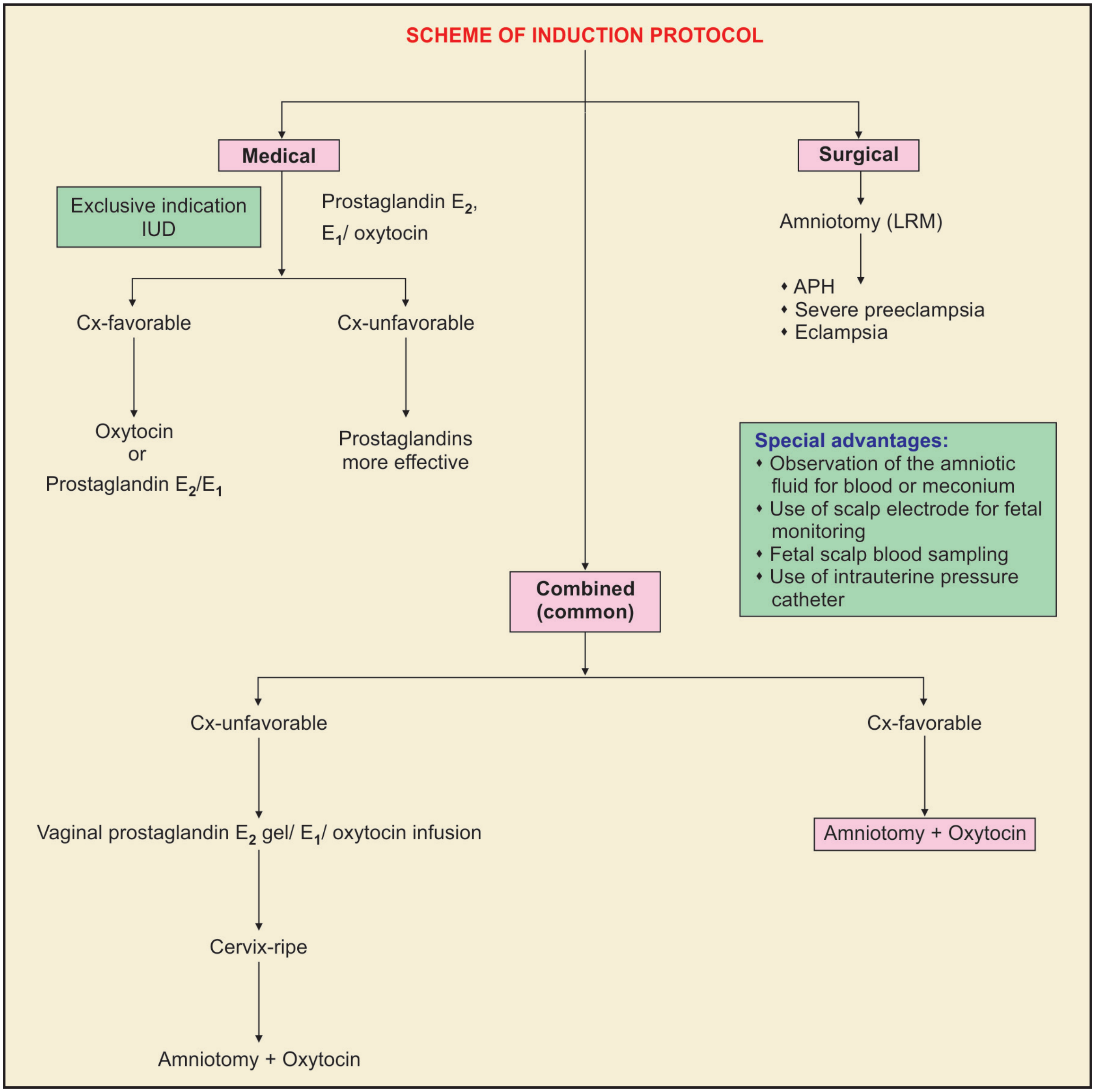
Comments: Each method has got its limitations and hazards. For induction of labor, each case should be judged on individual basis.

Mechanical: ***Dilators*** **—** act by release of endogenous prostaglandins from the membranes and maternal decidua to induce labor and cervical ripening. Hygroscopic dilators, e.g. laminaria (desiccated seaweed), lamicel (magnesium sulfate in polyvinyl alcohol) act by absorption of water. They swell and forcibly dilate the cervix. Mechanical dilators are as safe and effective as PGE_2 in cervical ripening.

Transcervical Balloon Catheter (Foley catheter) and extra-amniotic saline infusion is effective for cervical ripening.

COMBINED METHOD

The combined medical and surgical methods are commonly used to increase the efficacy of induction by reducing the induction-delivery interval. The oxytocin infusion is started either prior


SCHEME OF INDUCTION PROTOCOL
Medical
Surgical
Exclusive indication IUD
Prostaglandin E_2, E_1/ oxytocin
Amniotomy (LRM)
• APH
• Severe preeclampsia
• Eclampsia
Cx-favorable
Cx-unfavorable
Oxytocin or Prostaglandin E_2/E_1
Prostaglandins more effective
Special advantages:
• Observation of the amniotic fluid for blood or meconium
• Use of scalp electrode for fetal monitoring
• Fetal scalp blood sampling
• Use of intrauterine pressure catheter
Combined (common)
Cx-unfavorable
Cx-favorable
Vaginal prostaglandin E_2 gel/ E_1/ oxytocin infusion
Amniotomy + Oxytocin
Cervix-ripe
Amniotomy + Oxytocin

to or following rupture of the membranes depending mainly upon the state of the cervix and head brim relation. With the head nonengaged, it is preferable to induce with prostaglandin gel or to start oxytocin infusion followed by ARM. The advantages of the combined methods are: (1) More effective than any single procedure; (2) Shortens the induction-delivery interval and thereby—(a) minimizes the risk of infection and (b) lessens the period of observation.

ACTIVE MANAGEMENT OF LABOR

(Syn: Augmentation of labor)

Active management of labor was introduced by O'Driscoll and his colleagues in 1968 at National Maternity Hospital, Dublin. The term "Active" refers to the active involvement of the consultant-obstetrician in the management of primigravid labor.

Active management applies exclusively to primigravidas with singleton pregnancy and cephalic presentation who are in spontaneous labor and with clear liquor. Husband or the partner is present during the course of labor (see p. 155). **Partograph** is maintained to record the progress of labor (Fig. 35.5).

The essential components of active management of labor (AMOL):

- Antenatal classes to explain the purpose and the procedure of AMOL **(prenatal education)**
- Woman is admitted in the labor ward only after the diagnosis of labor (regular painful uterine contractions with cervical effacement)
- One to one nursing care with **partographic monitoring of labor** (Fig. 35.5)
- Amniotomy (ARM) with confirmation of labor
- Oxytocin augmentation (escalating dose) if cervical dilatation is <1 cm/hr. (p. 576)
- Delivery is completed within 12 hours of admission
- Epidural analgesia if needed (p. 593)
- Fetal monitoring by intermittent auscultation or by continuous electronic monitoring (Fig. 35.4)
- Active involvement of the consultant obstetrician.

The key to active management involves strict vigilance (one to one care), active and informed intervention in time. The incidence of operative delivery is not increased and less analgesia is required.

Aim: To expedite delivery within 12 hours without increasing maternal morbidity and perinatal hazards.

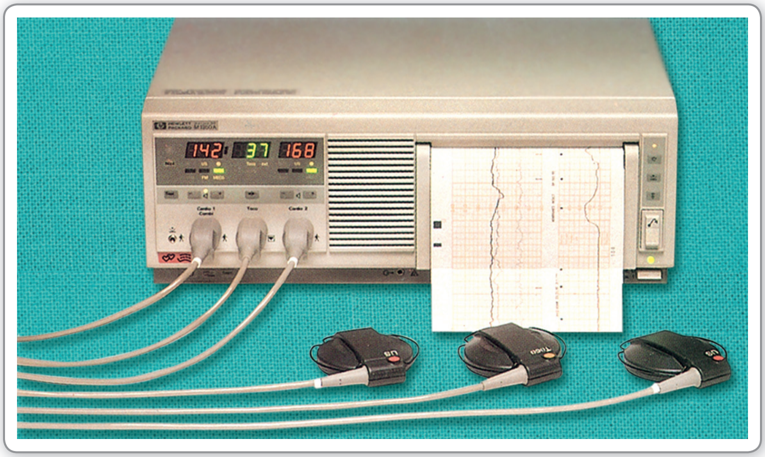
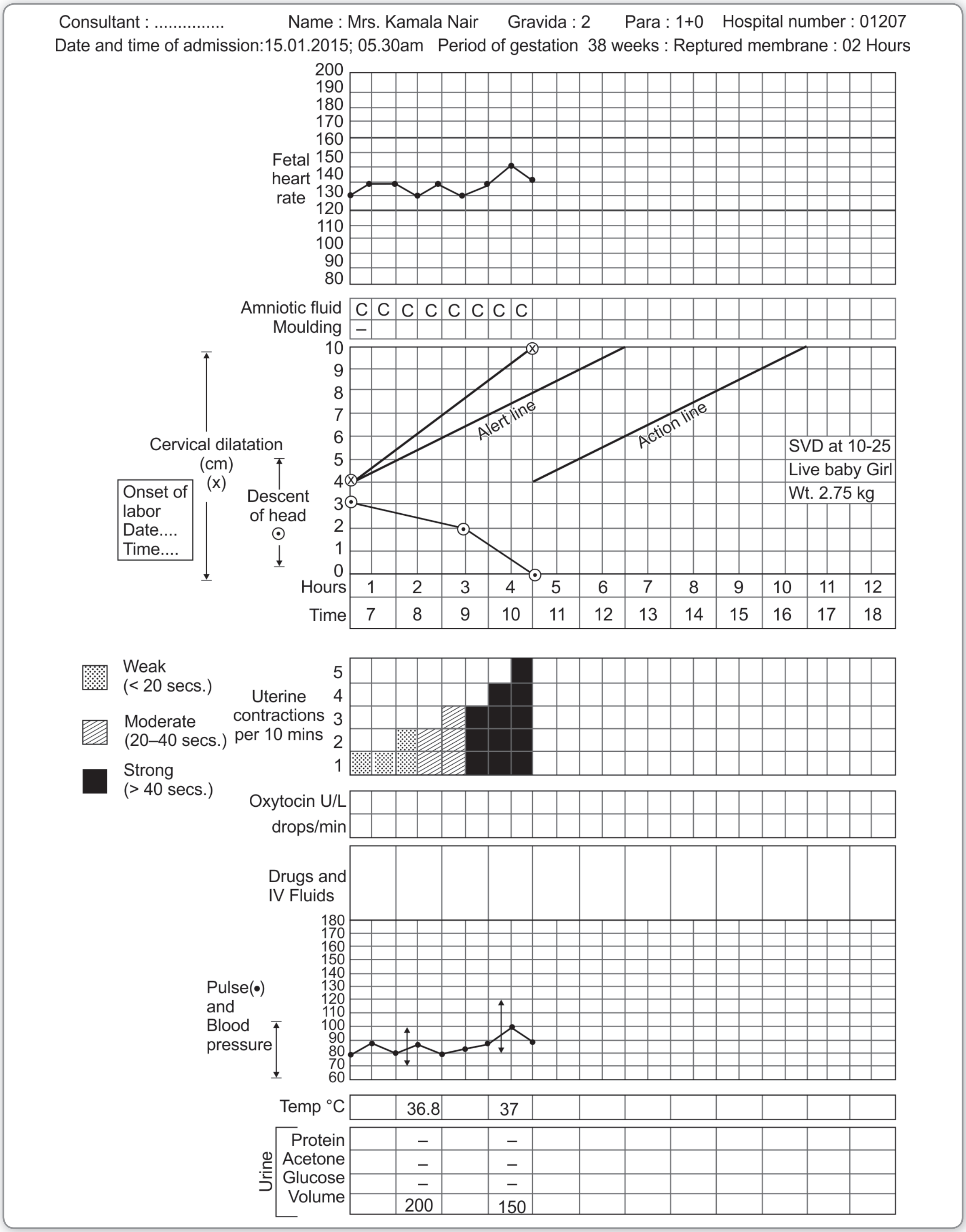


Fig. 35.4: Electronic fetal monitor with abdominal transducers



Consultant : .... Name : Mrs. Kamala Nair Gravida : 2 Para : 1+0 Hospital number : 01207

Date and time of admission:15.01.2015; 05.30am Period of gestation 38 weeks : Reptured membrane : 02 Hours

Onset of labor Date.... Time....

SVD at 10-25

Live baby Girl

Wt. 2.75 kg

Fig. 35.5: Partograph (modified WHO) representing graphically the important observations in labor. The cervical dilatation and descent of head are shown in relation to alert and action lines (See also p. 465, Fig. 27.2). Intensity and duration of uterine contraction are shown with shades

Active management of labor: Objective is — (a) early detection of any delay in labor; (b) diagnose its cause and (c) initiate management.

Emotional support in labor: Stress and anxiety during labor can make labor prolonged. Presence of a supportive companion during labor (husband/female relative of choice) reduces the duration of labor, need of analgesics and oxytocin augmentation. Such social support is a low cost useful intervention. Stress-induced high levels of endogenous adrenalin is thought to inhibit uterine contractions via stimulation of uterine muscle beta receptors.

Limitations of active management of labor: It is employed only in selected cases and in selected centers where intensive intrapartum monitoring by trained personnel is possible. It requires more staff involvement in the antenatal clinic and labor ward.

Advantages are:

- Less chance of dysfunctional labor
- Shortens the duration of labor (< 12 hours)
- Fetal hypoxia can be detected early
- Low incidence of cesarean birth
- Less analgesia
- Less maternal anxiety due to support of the caregiver and prenatal education.

Contraindications are:

- Presence of obstetric complication
- Presence of fetal compromise
- Multigravida (not a routine).

PARTOGRAPH

Partograph is a composite graphical record of key data (maternal and fetal) during labor, entered against time on a single sheet of paper (Fig. 35.5).

In **cervicograph** (Philpott & Caste — 1972), the **alert line** starts at 4 cm (WHO) of cervical dilatation and ends at 10 cm dilatation (at the rate of 1 cm/hr). The **action line** is drawn 4 hours to the right and parallel to the alert line. **In a normal labor, the cervicograph (cervical dilatation) should be either on the alert line or to the left of it.** When it falls on Zone 2 (see p. 464-65 and Fig. 27.2) it is abnormal and need to be critically assessed. When it falls in Zone 3 case should be reassessed by a senior person. Decision is to be made either for termination of labor (cesarean section) or for augmentation of labor (amniotomy and or oxytocin).

The components of a partograph are: (a) Patient identification; **(b)** Time — recorded at hourly interval. Zero time for spontaneous labor is the time of admission in the labor ward and for induced labor is the time of induction; **(c)** Fetal heart rate — recorded at every 30 minutes; **(d)** State of membranes and color of liquor **:** to mark 'I' for intact membranes, 'C' for clear and 'M' for meconium stained liquor; **(e)** Cervical dilatation and descent of the head (see p. 151); **(f)** Uterine contractions — the squares in the vertical columns are shaded according to duration and intensity (see p. 158); **(g)** Drugs and fluids; **(h)** Blood pressure (recorded in vertical line) at every 2 hours and pulse at every 30 minutes; **(i)** Oxytocin — concentration in the upper box and dose (m IU/min) in the lower box; **(j)** Urine analysis; **(k)** Temperature record.

Advantages of a partograph: (i) A single sheet of paper can provide details of necessary information at a glance; (ii) No need to record labor events repeatedly; (iii) It can predict deviation from normal progress of labor early. So, appropriate steps could be taken in time (see Fig. 27.2, p. 465); (iv) It facilitates handover procedure; (v) Introduction of partograph in the management of labor (WHO 1994) has reduced the incidence of prolonged labor and cesarean section rate. There is improvement in maternal morbidity, perinatal morbidity and mortality.

KEY POINTS

- **Induction of labor** means initiation of uterine contractions (after fetal viability) for the purpose of vaginal delivery. **Augmentation** is the process of stimulation of uterine contraction that are already present but found to be inadequate (p. 598).
- Induction of labor should be done when benefits of delivery to either the mother or the baby outweigh the risks of pregnancy continuation.
- **Indications and contraindications** (p. 598) must be carefully judged to avoid the dangers of induction of labor (p. 598).
- **Methods of cervical ripening** are many (p. 599). Bishop's preinduction cervical score (Table 35.5) can predict the success of induction (p. 600). Score ≥ 6 is favorable.
- **Methods of induction** may be medical, surgical or combined (p. 600), depending upon the individual case. Each method has got its merits and demerits (Table 35.6, 35.7, 35.8).
- Induction of labor with sweeping of the membranes is effective. Combined use of amniotomy (ARM) and IV oxytocin is more effective than ARM alone.
- **Active management of labor** needs some criteria to be fulfilled (p. 605). It has many advantages (p. 607).
- **Partograph** is a composite graphical record of labor events (maternal and fetal) entered against time on a single sheet of paper (Fig. 35.5). It has many advantages (p. 607). It can predict deviation from normal progress of labor early so that early steps could be taken.

QUESTIONS

1. What is induction of labor? What are the different methods of induction of labor? Discuss the advantages of active management of labor? (p. 598, 600, 605)

Write Short Notes on:

A. Common indications of induction of labor (p. 598)

B. Merits and demerits of prostaglandins in medical induction of labor (p. 600)

Chapter 36

Population Dynamics and Control of Conception

POPULATION DYNAMICS

Demography (demos = population, graphy = to study) is the branch of science which deals with the study of human population. An accurate idea of the vital events like crude death rate, birth rate and the changing pattern of population is of paramount importance for a nation. From these informations, the national plan outlay is formulated and the impact of the health care delivery system in a given period of time is evaluated.

POPULATION DYNAMICS: The population is not static but is always in a dynamic state. **The factors involved in population dynamics are**—(1) deaths, (2) births and (3) migration. Ever since the second half of the 20 century, there has been a substantial fall in crude death rate throughout the developing world. Eradication of epidemics such as smallpox and to some extent of malaria and kala-azar, and improvement of health care delivery system are some of the factors responsible for reduction of crude death rate. On the contrary, there has been only a marginal fall in the birthrate throughout the years. It is this disparity between the death rate and birthrate which results in the rapid rise of population. This is especially pronounced in the developing countries comprising about 70% of the total world population. The problem becomes intensified by the dynamic migration of population from one state to the other or amongst different parts of the same state, e.g. rural to urban.

MAGNITUDE OF THE PROBLEM: The total **world population** was estimated to be about 6 billion in the last century. Earlier in the last century, the rate of increase of population was about 10 million per year. It is now increasing at a much faster rate of 100 million per year. If the rate of increase continues at the same pace, the projected population would be 8,000 million in 2025.

India, with 2.5% of the world's land surface area has to accommodate about 16% of the world population and is the second most populous country in the world, next to China. Moreover, it is not uniformly distributed. The density of the population is 300 per sq. kilometer. The death rate stood at the level of 8 per 1,000 and the birthrate at 25 per 1,000 in mid 2003. Thus, there is a wide gap between the births and deaths resulting in rapid rise of population. One can have an idea of the magnitude of population explosion from the following. In India, during the period 1991–2001, the population increase was to the extent of 160 million. This means an increase in population to the extent of 16 million per year. This increase in population per year is almost equal to the total population of Australia with a land area 2.5 times more than that of India. As the rate of population growth continues unabated, **India's population has reached 1,027 million in 2001 and the projected population in 2025 is 1,363 million. In 2050, the projected population is 1,628 million when India would be the world's most populous country exceeding China**.

IMPACT OF INCREASED POPULATION: The rapid increase of population has got an adverse effect on the national economy. The fruits of improvements in the different sectors are being eroded by the growing population. Moreover, increasing number of births have got a deleterious effect on the health of the mother and the child and hinders social and economic upliftment of the family. High parity is also related to increased maternal, perinatal and infant deaths and is associated with various obstetric and gynecological complications and nutritional problems. For these reasons, population control by appropriate family welfare program is considered to be a **branch of preventive and community medicine.** Considering the magnitude of the problem, many developing countries, India in particular, have taken this as a national program of vital importance.

CONTROL OF CONCEPTION

FAMILY PLANNING

AIMS: The aims of family welfare planning are: (1) **To bring down** population growth, so as to ensure a better standard of living; (2) **From economic and social point of view** — the already existing population of nearly 1,027 million are deficient in their basic needs of food, clean water, clothing, housing, education and proper health care. Spacing of birth and small family norm will improve the health of the mothers and their children so that a healthier society can emerge; (3) **To reduce the maternal and infant mortality rates**—there are about 600,000 maternal deaths each year throughout the globe of which 99% occur in the developing world. Maternal mortality and morbidity could be reduced significantly by effective use of contraception. Nearly 6 million infant deaths might be avoided if all pregnancies occurred to women between the ages of 18–35 years; if the intervals between pregnancies were at least 2 years and if no woman had more than four children; (4) **To prevent** *pregnancies* that are **too early**, **too frequent and too many** and the number of unsafe abortions.

OBJECTIVES

Conception control:

- **To bring down the birthrate** to a realistic minimum during a given period of time. There are about 168 million eligible couples in India according to census 2001. **The term "eligible couple" is applied** to couples with wives in the reproductive age group of 15–45 years and who require the use of some sort of family planning method. Latest report shows that about 44% of the eligible couples in India are practicing effective methods of contraception.
- **To bring about certain social changes like — (a) To educate and motivate** the sexually active and fertile couple to accept the small family norm; **(b) To increase the literacy rate**, especially amongst women in rural areas; **(c) To raise the marriageable age** of both boys and girls. Low age of marriage not only contributes to the increased birthrate but adversely affects the health of the woman. In 1978, the Indian Parliament approved the bill fixing the **minimum age of marriage at 21 years for men and 18 years for women; (d) To maximize the access** of good quality, wide variety, client-oriented family planning services and to fulfill the unmet need of contraception.

Maternity and child health services:

- Maternity services are to be extended through antenatal, intranatal and postnatal care with immunization against tetanus and prevention and correction of anemia.
- Children are to be protected through an immunization schedule and vitamin supplementation program.

Other services:

These include: (i) Sex education and marriage guidance; (ii) Research and evaluation of the program, research about normal reproduction, investigation and treatment of infertility and recurrent abortion. It also includes evaluation of pregnancy termination (safe abortion) as a method of family limitation.

CONTRACEPTION

Contraception and fertility control are not synonymous. Fertility control includes both fertility inhibition (contraception) and fertility stimulation. While the fertility stimulation is related to the problem of the infertile couples, **the term contraception includes all measures, temporary or permanent, designed to prevent pregnancy due to the coital act**.

Ideal contraceptive methods should be highly (100%) effective, acceptive, safe, reversible, cheap, having non-contraceptive benefits, simple to use and requiring minimal motivation, maintenance and supervision.

CONTRACEPTIVE EFFECTIVENESS: The failure rate of any contraceptive is calculated in terms of pregnancy rate per hundred women years (HWY) of use. It is calculated according to the following formula (Pearl index):

$$\textbf{Pregnancy failure rate/HWY} = \frac{\text{Number of accidental pregnancies} \times 1{,}200^*}{\text{Number of patients observed} \times \text{months of use}}$$

* 1,200 = number of months in 100 years

Example: If 100 couples have used a method for a period of 2 years and have resulted in 20 pregnancies, the pregnancy rate is calculated to be: $\frac{20 \times 1200}{100 \times 24} = 10$

When the pregnancy rate is below 10, the effectiveness of the particular method is considered to be high. If it is more than 20, it is said to be below. The following are the effectiveness of the commonly used contraceptive methods.

METHODS OF CONTRACEPTION

The various methods of contraception are schematically depicted below:

TEMPORARY

Temporary methods are commonly used to postpone or to space births. However, the methods are also frequently being used by the couples even though they have got strong desire for no more children (Table 36.1).

BARRIER METHODS

These methods prevent sperm deposition in the vagina or prevent sperm penetration through the cervical canal. The objective is achieved by mechanical devices or by chemical means which produce sperm immobilization, or by combined means. The following are used (Box 36.1).

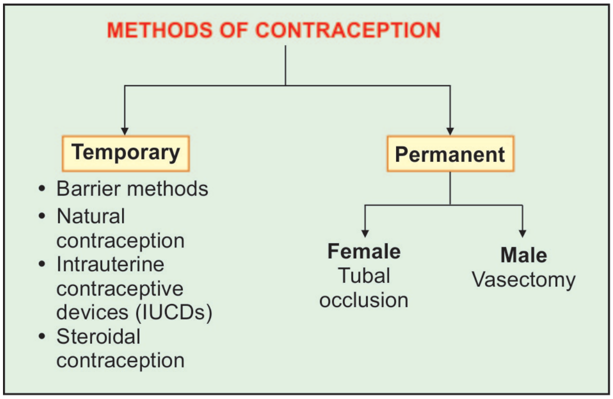


Table 36.1: Failure Rate of Contraceptive Methods in First 12 Months of Use

Methods	Pregnancy Rate per 100 Women Years (approx.)
No method	85
Natural (calendar, temperature, mucus)	25
Withdrawal	27
Lactational amenorrhea	2
Condom (male)	15
Condom (female)	21
Diaphragm	16
IUCD :	
CuT 380A	0.8
LNg 20	0.1
Combined oral pill	0.1
Progestin only pill	1
Patch	0.38
DMPA and NET injectables	0.3
Norplant	0.05
Implanon	0.01
Vasectomy	0.15
Tubectomy	0.15

Failure rate is further less when methods are used correctly and consistently

CONDOM (MALE)

Condoms are made of polyurethane or latex. Polyurethane condoms are thinner and suitable for those who are sensitive to latex rubber. It is the most widely practiced method used by the male. In India, one particular brand (latex) is widely marketed as "Nirodh". The efficacy of condoms can be augmented by improving the quality of the products and by adding spermicidal agents during its use. **Protection against sexually transmitted disease is an additional advantage**. Occasionally, the partner may be allergic to latex.

The method is suitable for couples who want to space their families and who have contraindications to the use of oral contraceptive or IUD. These are also suitable to those who have infrequent sexual intercourse.

BOX 36.1

- **Mechanical**

Male — Condom
Female — Condom, diaphragm, cervical cap

- **Chemical**

(Vaginal contraceptives)
Creams — Delfen (nonoxynol-9, 12.5%)
Jelly — Koromex, Volpar paste
Foam tablets — Aerosol foams, Chloramin T or Contab, Sponge (Today)

- **Combination**

Combined use of mechanical and chemical

FEMALE CONDOM (FEMIDOM) (Fig. 36.1A): It is a pouch made of polyurethane which lines the vagina and also the external genitalia. It is 17 cm in length with one flexible polyurethane ring at each end. Inner ring at the closed end is smaller compared to the outer ring. Inner ring is inserted at the apex of the vagina and the outer ring remains outside. It gives protection against sexually transmitted disease and pelvic inflammatory disease. It is expensive. Multiple uses can be made with washing, drying and with lubrication. Failure rate is about 5–21/HWY.

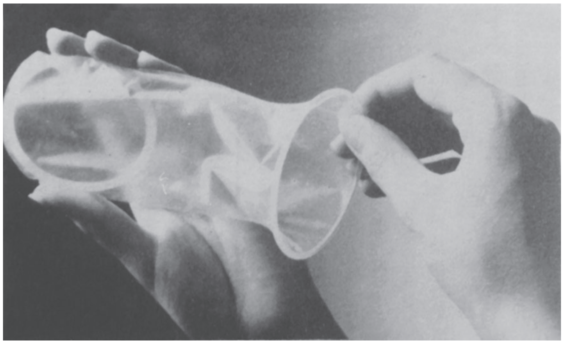


Fig. 36.1A: Female condom

USE OF CONDOM: (1) As an elective contraceptive method; (2) As an interim form of contraception during pill use, following vasectomy operation (see later) and if an IUD is thought lost until a new IUD can be fitted; (3) During the treatment of trichomonal vaginitis of the wife, the husband should use it during the course of treatment irrespective of contraceptive practice; (4) Immunological infertility — male partner to use for 3 months. For other **non-contraceptive benefits**, see Table 36.2.

Table 36.2: Condom

Advantages	
■ Cheaper with no contraindications ■ No side effects ■ Easy to carry, simple to use and disposable ■ Protection against sexually transmitted diseases, e.g. gonorrhea, chlamydia, HPV and HIV ■ Protection against pelvic inflammatory diseases ■ Reduces the incidence of tubal infertility and ectopic pregnancy ■ Protection against cervical cell abnormalities ■ Useful where the coital act is infrequent and irregular	EACH CONDOM TO BE USED ONCE ONLY हर बार नया कण्डोम इस्तेमाल कीजिए निरोध • NIRODH चिकनाई सहित LUBRICATED **Disadvantages** ■ May accidentally break or slip off during coitus ■ Inadequate sexual pleasure ■ Allergic reaction (Latex) ■ To discard after one coital act ■ Failure rate — 15/HWY

Precautions: (i) To use a fresh condom for every act of coitus; (ii) To cover the penis with condom prior to genital contact; (iii) Create a reservoir at the tip; (iv) To withdraw while the penis is still erect; (v) To grasp the base of the condom during withdrawal.

Table 36.3: Diaphragm

Advantages	Disadvantages
■ Cheap	■ Requires help of a doctor or paramedical person to measure the size required
■ Can be used repeatedly for a long time	■ Risk of vaginal irritation, ablation and urinary tract infection are there
■ Reduces PID/STIs to some extent	
■ Protects against cervical precancer and cancer	■ Not suitable for women with uterine prolapse *Failure rate—16/HWY*

DIAPHRAGM (Table 36.3 and Fig. 36.1B)

It is an intravaginal device made of latex with flexible metal or spring ring at the margin. Its diameter varies from 5 cm to 10 cm. It requires a medical or paramedical personnel to measure the size of the device. The distance between the tip of the middle finger placed in the posterior fornix and the point over the finger below the symphysis pubis gives the approximate diameter of the diaphragm. Diaphragm should completely cover the cervix. As it cannot effectively prevent ascent of the sperms alongside the margin of the device, additional chemical spermicidal agent should be placed on the superior surface of the device during insertion, so that it remains in contact with the cervix. **The device is introduced up to 3 hours before intercourse and is to be kept for at least 6 hours after the last coital act**. Ill-fitting and accidental displacement during intercourse increase the failure rate. Overall failure rate is 6–16%.

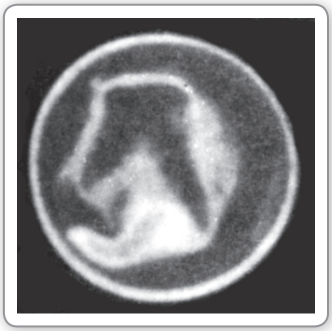


Fig. 36.1B: Commonly used conventional contraceptives (Diaphragm)

VAGINAL CONTRACEPTIVES (FIG. 36.1C)

SPERMICIDES: Spermicides are available as vaginal foams, gels, creams, tablets and suppositories. Usually, they contain **surfactants like nonoxynol–9, octoxynol or benzalkonium chloride**. These agents mostly cause sperm immobilization. **The cream or jelly** is introduced high in the vagina with the help of the applicator soon before coitus. **Foam tablets** (1–2) are to be introduced high in the vagina at least 5 minutes prior to intercourse. In isolation, it is not effective (18–29/HWY), but enhances the efficacy of condom or diaphragm when used along with it. There may be occasional local allergic manifestations either in the vagina or vulva.

DELEEN
ORTHO

Fig. 36.1C: Vaginal contraceptive (nonoxynol–9, 12.5%)

***Vaginal contraceptive sponge (Today)*:** It is made of polyurethane impregnated with 1 g of nonoxynol-9 as a spermicide. Nonoxynol-9 acts as a surfactant which either immobilizes or kills sperm. **It releases spermicide during coitus, absorbs ejaculate and blocks the entrance to the cervical canal**. **The sponge should not be removed for 6 hours after intercourse. Its failure rate (HWY) is about — Parous women: 32-20, Nulliparous 16-9**. Currently it is observed that nonoxynol–9 is not effective in preventing cervical gonorrhea, chlamydia or HIV infection. Moreover, it produces lesions in the genital tract when used frequently. Those lesions are associated with increased risk of HIV transmission. Sponge may cause allergic reactions.

FERTILITY AWARENESS METHOD (TABLE 36.4)

Fertility awareness method requires partner's cooperation. The woman should know the fertile time of her menstrual cycle.

Table 36.4: Fertility Awareness Methods (Rhythm Method)

Advantages	Disadvantages
■ No cost ■ No side effects *Failure rate*—20–30/HWY	■ Difficult to calculate the safe period reliably ■ Needs several months training to use these methods ■ Compulsory abstinence from sexual act during certain periods ■ Not applicable during lactational amenorrhea or when the periods are irregular

RHYTHM METHOD

This is the only method approved by the Roman Catholic Church. **The method is based on identification of the fertile period of a cycle and to abstain from sexual intercourse during that period**. This requires partner's cooperation. The methods to determine the approximate time of ovulation and the fertile period include — (a) recording of previous menstrual cycles (**calendar rhythm**), (b) noting the basal body temperature chart (**temperature rhythm**), and (c) noting excessive mucoid vaginal discharge (**mucus rhythm**). **The users of the calendar method** obtain the period of abstinence from calculations based on the previous twelve menstrual cycle records. **The first unsafe day is obtained** by subtracting 20 days from the length of the shortest cycle and **last unsafe day** by deducting 10 days from the longest cycle. **Users of temperature rhythm** require abstinence until the 3rd day of the rise of temperature. **Users of mucus rhythm** require abstinence on all days of noticeable mucus and for 3 days thereafter.

COITUS INTERRUPTUS (WITHDRAWAL) (TABLE 36.5)

It is the oldest and probably the most widely accepted contraceptive method used by man. **It necessitates withdrawal of penis shortly before ejaculation**. It requires sufficient self-control by the man so that withdrawal of penis precedes ejaculation.

Table 36.5: Coitus Interruptus

Advantages	Disadvantages
■ No appliance is required ■ No cost. *Failure rate*—27/HWY	■ Requires sufficient self-control by the man ■ The woman may develop anxiety neurosis, vaginismus or pelvic congestion ■ Chance of pregnancy is more: • Precoital secretion may contain sperm • Accidental chance of sperm deposition into the vagina

BREASTFEEDING, LACTATIONAL AMENORRHEA (LAM)

Prolonged and sustained breastfeeding offers a natural protection of pregnancy. This is more effective in women who are **amenorrheic than those who are menstruating**. The risk of pregnancy to a woman who is fully breastfeeding and amenorrheic is **less than 2% in the first 6 months**. Otherwise, the **failure rate** is high (1–10%). Thus, during breastfeeding, additional contraceptive support should be given by condom, IUCD or injectable steroids where available to provide complete contraception.

When the women is **fully breastfeeding**, a contraceptive method should be used in the **3rd postpartum month and with partial or no breastfeeding, she should use it in the 3rd postpartum week**.

FERTILITY AWARENESS BASED METHODS

(1) **Natural contraception** (rhythm method, coitus interruptus and lactational amenorrhea method)
(2) **Barrier method** (condoms, diaphragm and spermicides).

INTRAUTERINE CONTRACEPTIVE DEVICES (IUCDs)

The intrauterine device has been used throughout the world. During the last couple of decades, however, there has been a significant improvement in its design and content. The idea is to obtain maximum efficacy without increasing the adverse effects. **The device is classified as open**, when it has got no circumscribed aperture of more than 5 mm so that a loop of intestine or omentum cannot enter and become strangulated, if accidentally, the device perforates through the uterus into the peritoneal cavity. Lippes loop, Cu T, Cu 7, Multiload and Progestasert are examples of open devices. **If closed devices**, like Grafenberg ring and Birnberg bow, accidentally enter the abdominal cavity, they have the potential of causing strangulation of the gut and hence are obsolete. **The device may be non-medicated** as Lippes loop or **medicated (bioactive)** by incorporating a metal copper, in devices like Cu T-200, Cu T-380A, Multiload-250, Multiload-375 (Fig. 36.2).

Hormone containing IUD either releasing progesterone (Progestasert) or levonorgestrel (LNG-IUS) has also been introduced. Nowadays the following medicated intrauterine contraceptive devices are in use:

• **Multiload 250**	• **Multiload 375**	• **Cu T 380A**	• **LNG – IUS**	• **GyneFix®**

DESCRIPTION OF THE DEVICES (FIG. 36.2)

Copper T 200 — The widely used medicated device is Copper T 200 B. It carries 215 mm^2 surface area of fine copper wire wounded round the vertical stem of the device. Stem of the T-shaped device is made of a polyethylene frame. It has a polyethylene monofilament tied at the end of the vertical stem. These two threads are used for detection and removal. In spite of the copper being radiopaque, additional barium sulfate is incorporated in the device. The device contains 124 mg of copper. The copper is lost at the rate of about 50 µg per 24 hours during a period of 1 year. It is supplied inside a sterilized sealed packet. **The device is to be removed after 4 years**. **Cu T 200** carries 200 mm^2 surface area of wire containing 120 mg of copper and **is removed after 3 years**. **Apart from the use of Cu T as a contraceptive, it is used following synechiolysis**:

Cu T 380A — Cu T 380A carries total 380 mm^2 surface area of copper wire wound around the stem (314 mm^2) and each copper sleeve on the horizontal arms (33 mm^2). The frame contains barium sulfate and is radiopaque. Replacement is every 10 years.

Multiload Cu 250 — The device emits 60–100 µg of copper per day during a period of 1 year. **The device is to be replaced every 3 years**.

Multiload–375 — The device is available in a sterilized sealed packet with an applicator. There is no introducer and no plunger. It has 375 mm^2 surface area of copper wire wound around its vertical stem. Replacement is every 5 years (Fig. 36.2).

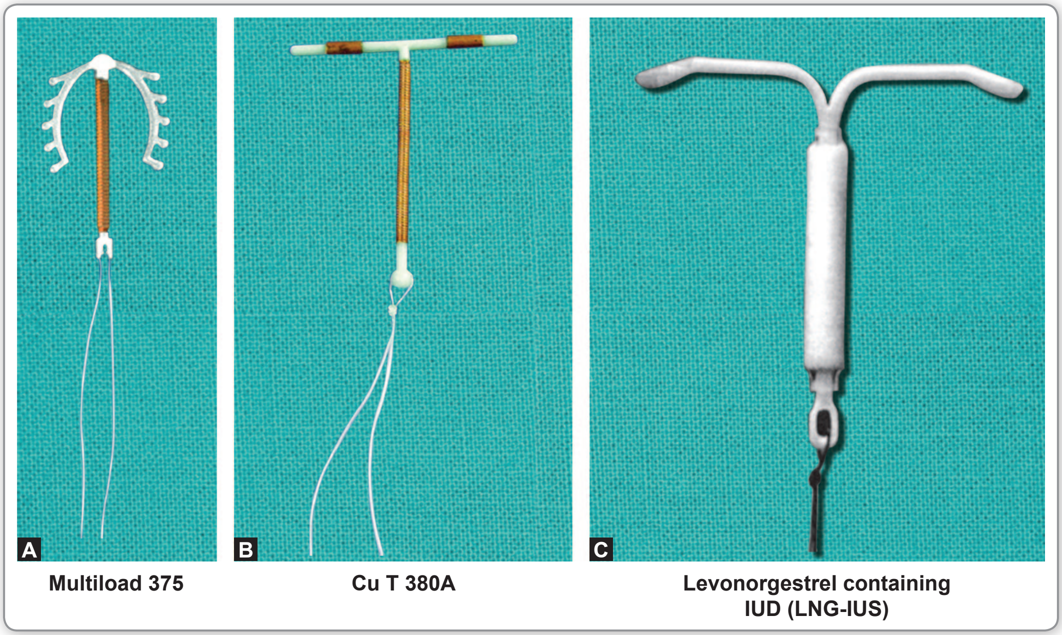
Levonorgestrel intrauterine system (LNG-IUS) (Fig. 36.2C) — This is a T-shaped device, with polydimethylsiloxane membrane around the stem which acts as a steroid reservoir. Total amount of levonorgestrel is 52 mg and is released at the rate of 20 µg/day. This device is to be replaced every 7 years. Its efficacy is comparable to sterilization. It has many non-contraceptive benefits also (p. 620).

Progestasert — It is a progesterone (38 mg) containing IUD. Progestasert is no longer manufactured.

Lippes loop — It is a non-medicated open intrauterine device. It is no longer used in India.

Mode of action: Mechanism of antifertility effect of all the IUDs is not yet clear. They act predominantly in the uterine cavity and do not inhibit ovulation. Probable factors are:

- **Biochemical and histological changes in the endometrium** — There is a nonspecific inflammatory reaction along with biochemical changes in the endometrium which have got gametotoxic and spermicidal property. Lysosomal disintegration from the macrophages attached to the device liberates prostaglandins, which are toxic to spermatozoa. Macrophages cause phagocytosis of spermatozoa.
- **There may be increased tubal motility** which prevent fertilization of the ovum.
- **Endometrial inflammatory response** decreases sperm transport and impedes the ability of sperm to fertilize the ovum.



Figs 36.2A to C: Commonly used IUD

- **Copper devices** — Ionized copper has got an additional local antifertility effect by **preventing blastocyst implantation through enzymatic interference**. Copper initiates the release of cytokines which are cytotoxic. Serum copper level is not increased. It seems that the progressive calcium deposition in the device prevents copper diffusion, if kept for longer period.
- **Levonorgestrel-IUS (Mirena)**— It induces strong and uniform suppression of endometrium. Cervical mucus becomes very scanty. Anovulation and insufficient luteal phase activity has also been mentioned. Serum progesterone level is not increased.

Contraindications for Insertion of IUCD: (1) Presence of pelvic infection, current or within 3 months; (2) Undiagnosed genital tract bleeding; (3) Suspected pregnancy; (4) Distortion of the shape of the uterine cavity as in fibroid or congenital uterine malformation; (5) Severe dysmenorrhea; (6) Past history of ectopic pregnancy; (7) Within 6 weeks following cesarean section; (8) STIs — Current or recurrent; (9) Trophoblastic disease; (10) Significant immunosuppression. **Additionally for CuT** are: (11) Wilson disease and (12) Copper allergy. **For LNG-IUS are:** (13) Hepatic tumor or hepatocellular disease (active); (14) Current breast cancer and (15) Severe arterial disease.

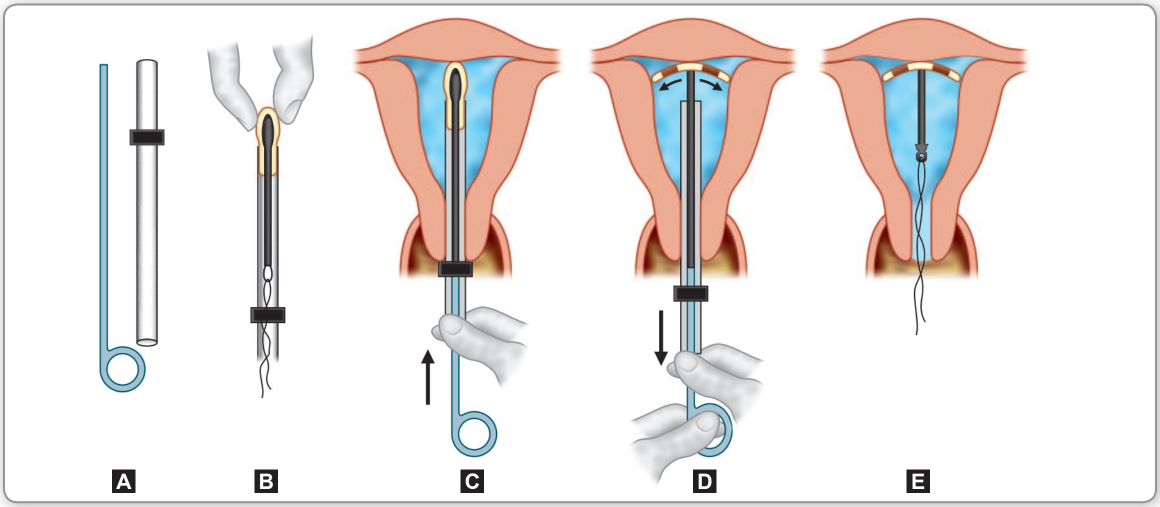
Time of Insertion

(a) ***Interval*** (When the insertion is made in the interconceptional period beyond 6 weeks following childbirth or abortion) — **It is preferable to insert 2–3 days after the period is over**. But it can be inserted any time during the cycle even during menstrual phase which has certain advantages (open cervical canal, distended uterine cavity, less cramp). However, during **lactational amenorrhea**, it can be inserted at any time.

(b) ***Postabortal*** — Immediately following termination of pregnancy by suction evacuation or D&E, or following spontaneous abortion, the device may be inserted. The additional advantage of preventing uterine synechiae can help in motivation for insertion.

(c) ***Postpartum*** — Insertion of the device can be done before the patients are discharged from the hospital. Because of high rate of expulsion, it is preferable to withhold insertion for 6 weeks when the uterus will be involuted to near normal size.

(d) ***Postplacental delivery*** — Insertion immediately following delivery of the placenta could be done, but the expulsion rate is high.



Figs 36.3A to E: Withdrawal technique of insertion of Cu T

Methods of Insertion (Figs 36.3 and 36.4)

- **Cu T 200**
- **Cu T 380A**

(A) Preliminaries

(1) History taking and examinations (general and pelvic) to exclude any contraindication of insertion; (2) Patient is informed about the various problems, the device is shown to her and consent is obtained; (3) **The insertion is done in the outpatient department**, taking aseptic precautions without sedation or anesthesia. To reduce cramping pain, ibuprofen (NSAID) may be given (200–400 mg) 30 minutes before insertion; (4) **Placement of the device inside the inserter** — the device is taken out from the sealed packet. The thread, the vertical stem and then the horizontal stem folded to the vertical stem are introduced through the distal end of the inserter. The device is now ready for introduction. **"No-touch" insertion method is preferred** (see below).

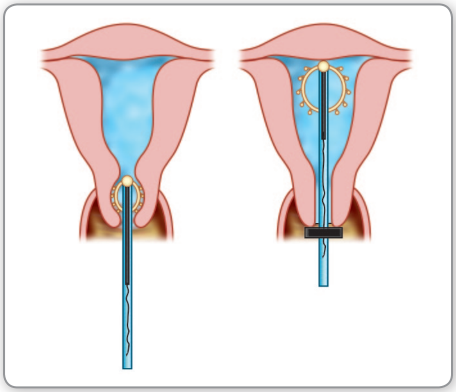


Fig. 36.4: Technique of insertion of multiload IUCDs

(B) Actual steps

(1) The patient empties her bladder and is placed in lithotomy position. Uterine size and position are ascertained by pelvic examination; (2) Posterior vaginal speculum is introduced and the vagina and cervix are cleansed by antiseptic lotion; (3) The anterior lip of the cervix is grasped by Allis forceps. A sound is passed through the cervical canal to note the position of the uterus and the length of the uterine cavity. The appropriate length of the inserter is adjusted depending on the length of the uterine cavity; (4) The inserter with the device placed inside is then introduced through the cervical canal right up to the fundus and after positioning it by the guard, the inserter is withdrawn keeping the plunger in position. **Thus, the device is not pushed out of the tube but held in place by the plunger while the inserter is withdrawn (withdrawal technique in Fig. 36.3);** (5) The excess of the nylon thread beyond 2–3 cm from the external os is cut. Then the Allis forceps and the posterior vaginal speculum are taken off.

"No-touch" insertion technique includes: (i) Loading the IUD in the inserter without opening the sterile package. The loaded inserter is now taken out of the package without touching the distal end; (ii) Not to touch the vaginal wall and the speculum while introducing the loaded IUD inserter through the cervical canal.

Multiload 375—The applicator with the device is just to be taken out of the sealed packet in a "no-touch" method and **the same is pushed through the cervical canal up to the fundus of the uterus**. The applicator is then withdrawn (Fig. 36.4).

LNG-IUS: The arms of the device are released in the uterine cavity about 2 cm below the fundus. The details of insertion are to be followed as in the instruction package (Fig. 36.2D).

Instructions to the Patient

The possible symptoms of pain and slight vaginal bleeding should be explained. The patient should be advised to feel the thread periodically by the finger. The patient is checked after 1 month and then annually.

Complications

Immediate:

- **Cramp-like pain** — It is transient but at times, severe and usually lasts for 0.5–1 hour. It is relieved by analgesic or antispasmodic drugs.
- **Syncopal attack**—Pain and syncopal attack are more often found in nulliparous or when the device is large enough to distend the uterine cavity.
- **Partial or complete perforation** — It is due to faulty technique of insertion but liable to be met within lactational period when the uterus remains small and soft.

Remote:

- **Pain** — The pain is more or less proportionate to the degree of myometrial distension. A proper size of the device may minimize the pain.
- **Abnormal menstrual bleeding** — The excessive bleeding involves increased menstrual blood loss, prolongation of duration of period and intermenstrual bleeding. The patient may become anemic and is of concern in one who is already anemic. Iron supplement is advocated. Tranexamic acid may be given for short-term relief. **Menstrual loss is much less with the use of third-generation IUDs** (see p. 620).
- **Pelvic infection (PID)** — The risk of developing PID is 2–10 times greater amongst IUD users. The risk is more in the first 3 weeks. Infection with chlamydia and rarely with actinomyces are seen. Newer IUDs reduce the risk (see p. 639).

Pain, abnormal uterine bleeding and PID are the principal factors related to its discontinuation (10–15%).

- **Spontaneous expulsion**—Usually occurs within a few months following insertion, more commonly during the period, at times unnoticed by the patient. Failure to palpate the thread which could be felt before, is an urgent ground to report to the physician. **The expulsion rate is about 5%**. The rate is, however, more following postabortal or puerperal insertions. The expulsion rate is markedly reduced in the successive years. Another device of appropriate size may be reintroduced and this is likely to be retained. **The newer IUDs have got less expulsion rate (see p. 639)**.
- **Perforation of the uterus**—The incidence of uterine perforation is about 1 in 1,000 insertions. Most perforations occur at the time of insertion, but the migration may also occur following initial partial perforation with subsequent myometrial contraction. **It is, however, less common when the device is introduced by the withdrawal technique**.

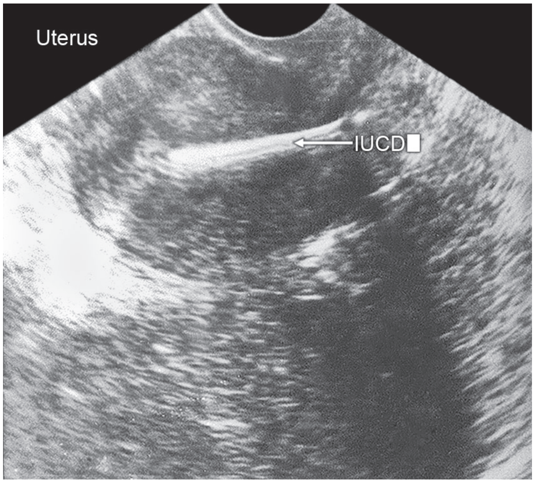


Fig. 36.5: Ultrasonogram showing the Cu T inside the uterine cavity. Thread was missing in this case

Diagnosis: Non-visibility of the thread through the external os and the appearance of pelvic symptoms after a long asymptomatic period are suspicious. **Negative findings on exploration** of the uterine cavity by a probe is suggestive. **Ultrasonography** can detect the IUD in abdominal cavity and is better than radiography. **Straight X-ray**, anteroposterior and lateral views, following introduction of a radiopaque probe (uterine sound) into the uterine cavity is conclusive. The device is found away from the opaque shadow placed in the uterine cavity, if it has perforated the uterine wall (Fig. 36.7).

Management: **Lippes loop** — As it is an open device made of inert material, it will cause no harm if left in the peritoneal cavity. Adhesions and intestinal injury are unlikely. But for psychological reason or otherwise, **it is better to remove it by laparoscopy or laparotomy**.

Copper device — A copper bearing device induces an intense local inflammatory reaction with adhesions with the surrounding structures. Thus, as soon as the diagnosis is made, it is to be removed by laparoscopy or laparotomy.

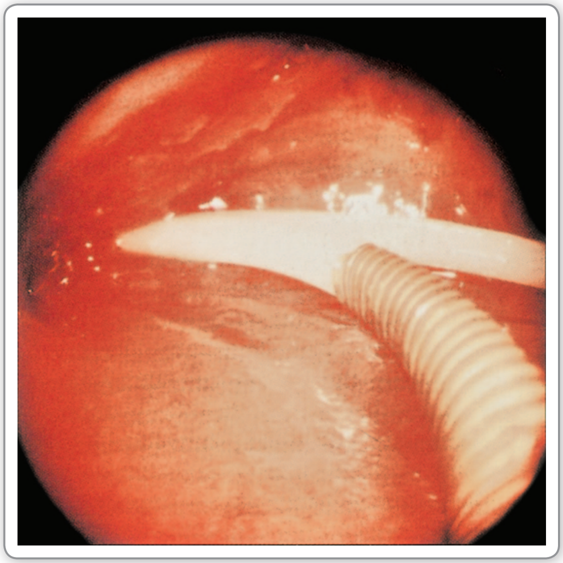


Fig. 36.6: Hysteroscopic view of IUCD in uterine cavity. The threads are coiled inside

- **Pregnancy** — The pregnancy rate with the device *in situ* is about 2 per 100 women years of use. Devices containing less than 300 mm^2 copper have higher failure rate and should not be used routinely. Lowest pregnancy rates are observed with Cu T 380A (0.8/HWY) and LNG-IUS (0.2/HWY). Should pregnancy occur with a device *in situ*, there is risk of ectopic pregnancy (0.02%). IUD can thus prevent an uterine but not an ectopic pregnancy. Third generation of IUDs like Cu T 380 A and LNG-IUS **give some amount of protection against an ectopic pregnancy**.

Management: **If the thread is visible** through the cervix, it is best to remove the device. This will minimize such complications as abortion, preterm labor, sepsis and low birth weight baby. **However, if the thread is not visible** it is better to leave it alone after counseling with the patient about the risks involved in continuing pregnancy. The device is expected to be expelled spontaneously with the delivery of the afterbirths. There is no conclusive evidence of increased risk of fetal anomalies with copper devices.

Indications for removal: (1) Persistent excessive regular or irregular uterine bleeding; (2) Flaring up of salpingitis; (3) Perforation of the uterus; (4) IUD has come out of place (partial expulsion); (5) Pregnancy occurring with the device *in situ*; (6) Woman desirous of a baby; (7) Missing thread; (8) 1 year after menopause; (9) When effective lifespan of the device is over.

IUD removal is simple and can be done at any time. It is done by pulling the strings gently and slowly with a forceps.

Missing Thread: The thread may not be visible through the cervical os due to — (a) Thread coiled inside; (b) Thread torn through; (c) Device expelled

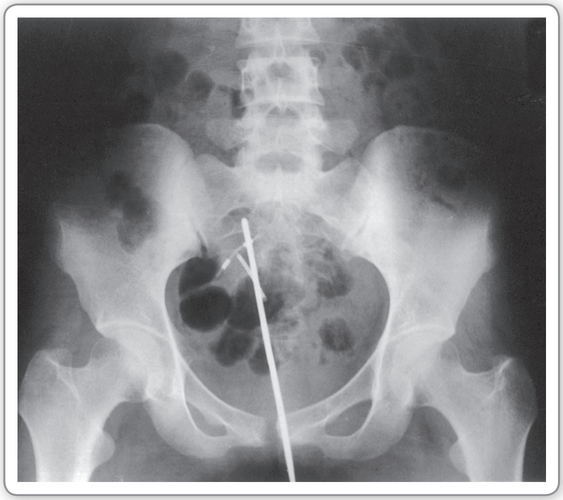


Fig. 36.7: Straight X-ray of the pelvis with an uterine sound placed inside the uterine cavity — (a) the displaced IUCD seems inside the uterine cavity with AP view. Lateral view is needed for confirmation

Courtesy: Dr Swati Shirodkar, HOD, Dept of Obst Gyn, MGM Medical College, Aurangabad outside unnoticed by the patient; (d) Device perforated the uterine wall and is lying in the peritoneal cavity; (e) Device pulled up by the growing uterus in pregnancy.

Methods of identification: **Pregnancy is to be excluded first** —

1. **Ultrasonography** can detect the IUD either within the uterine cavity or in the peritoneal cavity (if perforated). It is preferred to radiography (Fig. 36.5).
2. **Hysteroscopy** can be used for direct visualization of the uterine cavity and it could be removed simultaneously (Fig. 36.6).
3. **Sounding** the uterine cavity by a probe (Fig. 36.7).
4. If negative, **straight X-ray** after introducing radiopaque probe (uterine sound) into the uterine cavity. This will not only reveal the presence or absence of the device but also its existence outside the uterine cavity (Fig. 36.7).

Table 36.6: IUD (Cu Devices and Hormone Releasing IUDs)

Advantages	**Disadvantages**
▪ Inexpensive : Cu T distributed free of cost through Government channel	▪ Require motivation
▪ Simplicity in techniques of insertion and most cost effective of all methods	▪ Limitation in its use
▪ Prolonged contraceptive protection after insertion (5–10 years) and suitable for the rural population of developing countries	▪ Adverse local reactions manifested by menstrual abnormalities, PID, pelvic pain and heavy periods. Beside effects are less with third generation of IUDs
▪ Systemic side effects are nil. Suitable for hypertensives, breastfeeding women and epileptics	▪ Risk of ectopic pregnancy.
▪ Reversibility to fertility is prompt after removal	*Failure rate—0.1–2/HWY*

Removal

A. Device inside the uterine cavity: It can be removed by any of the following methods mentioned below:

(i) Specially designed blunt hook
(ii) Artery forceps (Fig. 42.8)
(iii) Uterine curette (Fig. 42.17)
(iv) Hysteroscopically under direct vision (Fig. 36.6).

B. Outside the uterus but inside the abdominal cavity: (i) Laparoscopy; (ii) Laparotomy (rarely).

Advantages of third generation of IUDs (Cu T 380A, Multiload 375 and Levonorgestrel IUS) *over the others.*

(1) Higher efficacy with lowest pregnancy rate (<1 per 100 women years); (2) Longer duration of action (5–10 years); (3) Low expulsion rate and fewer indications for medical removal; (4) Risk of ectopic pregnancy is significantly reduced (Cu T 380A and LNG-IUS: 0.02/HWY); (5) Risk of PID is reduced, anemia is improved; (6) **Non-contraceptive benefits especially with LNG-IUS:** (i) **Significant reduction** in menstrual blood loss, menorrhagia, dysmenorrhea and premenstrual tension syndrome (PMS), (ii) It can be used **in the treatment of** endometrial hyperplasia, adenomyosis, endometriosis, uterine leiomyomas and endometrial cancer, (iii) It can be used **as an alternative to** hysterectomy for menorrhagia and DUB. (iv) It provides excellent benefits of **hormone replacement therapy** (HRT) when used over the transition years of reproduction to perimenopause. (Fibroplant p. 640).

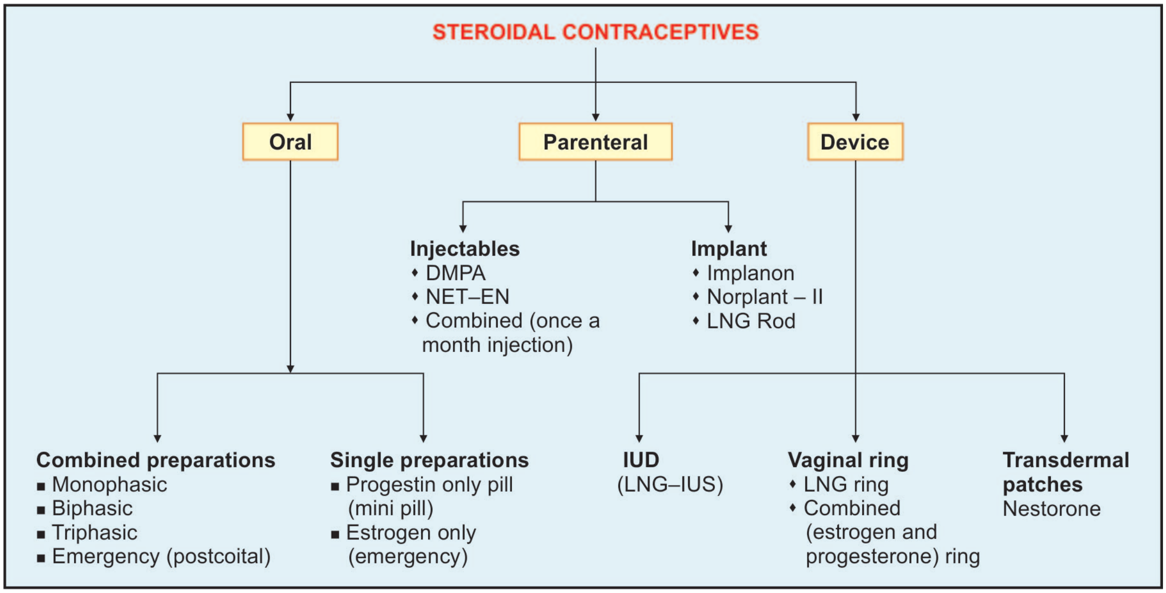
Disadvantages of third generation of IUDs: ♦ Expensive ♦ LNG-IUS is not available through government channel in India currently ♦ Amenorrhea (5%) is a cause of its discontinuation ♦ Malpositioning with long duration of use may cause pregnancy (failure) or expulsion.

SUMMARY OF IUD

Intrauterine contraceptive device is a widely acceptable reversible method of contraception for spacing of births. Amongst many, either a copper impregnated device like Cu T, multiload or a hormone releasing device like LNG-IUS is commonly used. **Its mode of action** is not clear. Probably, it produces nonspecific biochemical and histological changes in the endometrium and ionized copper has got spermolytic and gametotoxic effects. LNG-IUS induces uniform suppression of endometrium and produces very scanty cervical mucus. **It should not be used in newly married women or when any pelvic pathology is present.** The device can be introduced in the interval period or following abortion or following childbirth. The introduction is an outdoor procedure and can be done even by a trained paramedical personnel without anesthesia. The technique employed is either “push-out” in Lippes loop or “withdrawal” in Cu T. **The immediate complications** include cramp-like pains or even syncopal attacks. **The delayed complications** include pelvic pain, menstrual irregularities, expulsion of the IUD or even perforation of the uterus. Complications are much less with third generation of IUDs. **The indications of its removal are** missing threads, persistent pelvic pain, menorrhagia, pregnancy, displacement of the device and flaring up of pelvic infection. While Cu T 200 should be removed after 3–4 years, Multiload 375 is replaced after 5 years, Cu T 380A after 10 years and LNG-IUS after 5 years. **The failure rate is** about 0.5–2/HWY. **Devices with less than 300 mm^2 of copper have higher failure rate.** Copper device can also be used as postcoital contraception and following synaecolysis.

STEROIDAL CONTRACEPTIONS

Enovid (norethynodrel 10 mg and mestranol 0.15 mg) was used in the first contraceptive field trial in Puerto Rico in 1956 by Pincus and his colleagues. Intensive pharmacological research and clinical trials were conducted during the following years to minimize the adverse effects of estrogen without reducing the contraceptive efficacy, resulted in lowering the dose of estrogen to a minimum of 20 µg or even 15 µg in the tablet.


STEROIDAL CONTRACEPTIVES
Oral
Parenteral
Device
Injectables
♦ DMPA
♦ NET–EN
♦ Combined (once a month injection)
Implant
♦ Implanon
♦ Norplant – II
♦ LNG Rod
Combined preparations
▪ Monophasic
▪ Biphasic
▪ Triphasic
▪ Emergency (postcoital)
Single preparations
▪ Progestin only pill (mini pill)
▪ Estrogen only (emergency)
IUD
(LNG–IUS)
Vaginal ring
♦ LNG ring
♦ Combined (estrogen and progesterone) ring
Transdermal patches
Nestorone

COMBINED ORAL CONTRACEPTIVES (PILLS)

The combined oral steroidal contraceptives are the most effective reversible method of contraception. In the combination pill, **the commonly used progestins are either levonorgestrel or norethisterone or desogestrel and the estrogens are principally confined to either ethinyl estradiol or mestranol** (3-methyl ether of ethinyl estradiol). Currently "lipid friendly", **third-generation progestins**, namely desogestrel, gestodene and norgestimate are available. Some of the preparations available in the market are mentioned in the Table 36.7 and 36.8. Only Mala-N is distributed through government channel free of cost (Fig. 36.8).

Table 36.7: Some of the Oral Contraceptives and Their Composition

Commercial Names	Composition		No. of Tablets
	Progestins (mg)	Estrogen (μg)	
1. Mala N (Govt of India)	Levonorgestrel (0.15)	Ethinyl estradiol (30)	21 + 7 iron tablets
2. Mala-D	Levonorgestrel (0.15)	—do—	21 + 7 iron tablets
3. Loette (Wyeth)	Desogestrel (0.15)	Ethinyl estradiol (20)	21
4. Yasmin (Schering)	Drospirenone 3 mg (p. 507)	Ethinyl estradiol (30)	21

Depending on the amount of ethinyl estradiol (E) and the types of progestin (P) used, pills are defined as: **1st generation** — with E 50 μg or more; **2nd generation** — with E 20–35 μg and P as levonorgestrel or norgestimate; **3rd generation** — with E 20–30 μg and P as desogestrel or gestodene; **4th generation** — E as third generation, with P as drospirenone, dienogest or nomegestrol. Low dose pills have E less than 50 μg.

4th generation: Drospirenone which is an analog of spironolactone is used as progestin. It has antiandrogenic and antimineralocorticoid action. It causes retention of K^+.

Mode of action: The probable mechanisms of contraception are:

- **Inhibition of ovulation** — Both the hormones synergistically act on the hypothalamo-pituitary axis. The release of gonadotropin releasing hormone (GnRH) from the hypothalamus is prevented through a negative feedback mechanism. There is thus no peak release of FSH and LH from the anterior pituitary. So follicular growth is either not initiated or if initiated, recruitment does not occur.
- **Producing static endometrial hypoplasia**— There is stromal edema, decidual reaction and regression of the glands making endometrium nonreceptive to the embryo.
- **Alteration of the character of the cervical mucus** (thick, viscid and scanty) so as to prevent sperm penetration.
- **Probably interferes** with tubal motility and alters tubal transport. Thus, even though accidental breakthrough ovulation occurs, the other mechanisms prevent conception.

Estrogen inhibits FSH rise and prevents follicular growth. It is also useful for better cycle control and to prevent breakthrough bleeding.

Progestin: Anovulatory effect is primarily by inhibiting LH surge. **It is also helpful to counteract the adverse effects of estrogen on the endometrium** (endometrial hyperplasia and heavy withdrawal bleeding). It is also responsible for changes in the cervical mucus (vide supra).

Selection of the patient: History and general examination should be thorough, taking special care to screen cases for contraindications (headache, migraine). Examination of the breasts for any nodules, weight and blood pressure are to be noted. **Pelvic examination** to exclude cervical pathology is mandatory. Pregnancy must be excluded. **Cervical cytology** to exclude abnormal cells is to be done. Thus, any woman of reproductive age group without any systemic disease and contraindications listed is a suitable candidate for combined pill therapy. Growth and development of the pubertal and sexually active girls are not affected by the use of "pill".

How to prescribe a pill: INSTRUCTION: New users should normally start their pill packet **on day 1 of their cycle**. One tablet is to be taken daily preferably at bed time for consecutive 21 days. It is continued for 21 days and then have a 7 days break; with this routine there is contraceptive protection from the first pill. Next pack should be started on the 8th day, irrespective of bleeding (same day of the week, the pill finished). **Thus, a simple regime of "3 weeks on and 1 week off" is to be followed**. Packing of 28 tablets, there should be no break between packs. Seven of the pills are dummies and contain either iron or vitamin preparations. However, a woman can start the pill up to day 5 of the bleeding. In that case she is advised to use a condom for the next 7 days. The pill should be started on the day after abortion. Following childbirth in non-lactating woman, it is started after 3 weeks and in lactating woman it is to be withheld for 6 months (see later in the chapter).

FOLLOW-UP: The patient should be examined after 3 months, then after 6 months and then yearly. **The patient above the age of 35 years should be checked more frequently**. At each visit, any adverse symptoms are to be noted. Examination of the breasts, weight and blood pressure recording and pelvic examination including cervical cytology, are to be done and compared with the previous records.

MISSED PILLS: Normally there is return of pituitary and ovarian follicular activity during the pill-free interval (PFI) of 7 days. Breakthrough ovulation may occur in about 20% cases during the time. Lengthening of PFI due to omissions, malabsorption or vomiting, either at the start or at the end of a packet, increases the risk of breakthrough ovulation and therefore pregnancy.

Contraindications of Combined Oral Contraceptives (WHO/FRM/FPP—2001)

Absolute			Relative
(A) *Circulatory diseases (past or present) Thromboembolic disorder (current or past)* ♦ Arterial or venous thrombosis ♦ Severe hypertension, stroke ♦ Valvular heart disease, ischemic heart disease, angina ♦ Diabetes with vascular complications ♦ Focal migraine ♦ Severe hypercholesterolemia ♦ Smokers over age 35 years	**(B) *Diseases of the liver*** ♦ Active liver disease ♦ Liver adenoma, carcinoma ♦ Liver tumors	**(C) *Others*** ♦ Pregnancy ♦ Undiagnosed genital tract bleeding ♦ Estrogen-dependent neoplasm, e.g. breast cancer ♦ Breastfeeding (within 6 weeks postpartum) ♦ Major surgery or prolonged immobilization	♦ Age > 40 years ♦ Smoker or < 35 years ♦ History of jaundice ♦ Diabetes ♦ Gall bladder disease ♦ Hyperlipidemia ♦ Post breast cancer ♦ Breastfeeding (postpartum 6 weeks to 6 months) ♦ Sickle cell disease ♦ CIN

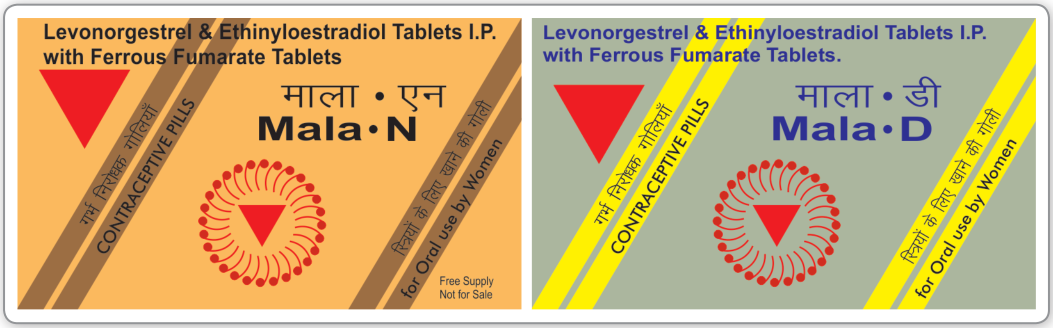

Levonorgestrel & Ethinyloestradiol Tablets I.P. with Ferrous Fumarate Tablets
माला • एन
Mala•N
गर्भ निरोधक गोलियाँ
CONTRACEPTIVE PILLS
स्त्रियों के लिए खाने की गोली
for Oral use by Women
Free Supply
Not for Sale
Levonorgestrel & Ethinyloestradiol Tablets I.P. with Ferrous Fumarate Tablets.
माला • डी
Mala•D
गर्भ निरोधक गोलियाँ
CONTRACEPTIVE PILLS
स्त्रियों के लिए खाने की गोली
for Oral use by Women

Fig. 36.8: Some commonly used oral contraceptives

Management: When a woman forgets to take **1 pill** (late up to 24 hours), she should take the missed pill at once and continue the rest as schedule. There is nothing to worry.

When she **misses 2 pills** in the 1st week (days 1–7), she should take 2 pills on each of the next 2 days and then continue the rest as schedule. Extra precaution has to be taken for next 7 days either by using a condom or by avoiding sex.

If 2 pills are missed in the 3rd week (days 15–21) or if more than 2 active pills are missed at any time, another form of contraception should be used as backup for next 7 days as mentioned above. She should start the next pack without a break.

If she misses any of the **7 inactive pills** (in a 28-day pack only) she should throw away the missed pills. She should take the remaining pills once a day and start the new pack as usual.

Drug interactions: Effectiveness of some drugs (aspirin, oral anticoagulants, oral hypoglycemics) are decreased and that for some other drugs (beta-blockers, corticosteroids, diazepam, aminophylline) are increased by oral contraceptives.

ADDITIONAL CONTRACEPTION: To ensure cent percent efficacy, additional mechanical contraceptives (usually condom) are to be used in the following circumstances:

(1) **When broad-spectrum antibiotics like** ampicillin, amoxicillin, tetracycline, doxycycline are used — as they impair the absorption of ethinyl estradiol.

(2) When enzyme-inducing drugs are used, e.g., (a) barbiturates, (b) all antiepileptic drugs except sodium valproate and clonazepam, (c) rifampicin, (d) ketoconazole, (e) griseofulvin, (f) lansoprazole, (g) ritonavir, (h) nevirapine and (i) efavirenz—under such circumstances high-dose preparations (ethinyl estradiol of 50 µg or more) are to be used to counterbalance the increased liver metabolism.

INDICATIONS FOR WITHDRAWAL: While the majority tolerates the combined pill, in some susceptible individuals, gross adverse symptoms develop which necessitate its withdrawal. **The indications for withdrawal of the pill are**—(1) Severe migraine; (2) Visual or speech disturbances; (3) Sudden chest pain; (4) Unexplained fainting attack or acute vertigo; (5) Severe cramps and pains in legs; (6) Excessive weight gain; (7) Severe depression; (8) Prior to surgery (it should be withheld for at least 6 weeks to minimize postoperative vascular complications) and (9) Patient desires pregnancy.

Continuous or extended use of COCs: It can be used by women who prefer to bleed at an interval of 60–80 days (3–4 times a year). For extended use of pills, the woman should take the active pills from pill pack and immediately start the next pack of active pills. The pills should be continued for 60–80 days, then a withdrawal bleed is allowed. Formulations with 84 active pills followed by 7 placebo pills are being made. This results in a "seasonal" withdrawal bleed at an interval of 4 months. Any monophasic pill may be used in this manner.

Pill regimen with 24 active pills followed by 4 placebo pills results in menses at a 28 days interval with lesser bleeding both in amount and days. Failure rate is also less.

How long can the pill be continued? Potential benefits of pills are greater when compared to risks in a well-selected individual. A woman who does not smoke and has no other risk factor for cardiovascular disease, may continue the pill (with careful monitoring) until the age of 50 years. This offers the dual advantages of effective contraception and hormone replacement therapy. However, for spacing of births, use of 3–5 years is considered enough and safe.

General and Metabolic Effects of Combined Oral Contraceptives: The combined preparations containing estrogen and progestin have got a wide range of metabolic activities which affect almost all the systems of the body. The changes are almost similar to those of pregnancy and almost completely revert back to normal after the drug is withdrawn. **The effects are related either to the estrogen (OGN) or to the progestin (PGN) or to both (OGN + PGN) of the compounds**.

BENEFITS OF COMBINED ORAL CONTRACEPTIVES (COCs)

(a) **Contraceptive benefits:** (i) Protection against unwanted pregnancy (failure rate – 0.1/HWY); (ii) Convenient to use; (iii) Not intercourse related; (iv) Reversibility; (v) Improving maternal and child health care.

(b) **Non-contraceptive benefits:** *Improvement of menstrual abnormalities*—(1) Regulation of menstrual cycle; (2) Reduction of dysmenorrhea; (40%) (3) Reduction of menorrhagia; (50%) (4) Reduction of premenstrual tension syndrome (PMS); (5) Reduction of Mittelschmerz's syndrome; (6) Protection against iron-deficiency anemia. **Protection against health disorders** — (7) Pelvic inflammatory disease (thick cervical mucus); (8) Ectopic pregnancy; (9) Endometriosis; (10) Fibroid uterus; (11) Hirsutism and acne; (12) Functional ovarian cysts; (13) Benign breast disease; (14) Osteopenia and postmenopausal osteoporotic fractures; (15) Autoimmune disorders of thyroid; (16) Rheumatoid arthritis. **Prevention of malignancies**—(17) Endometrial cancer (50%); (18) Epithelial ovarian cancer (50%); (19) Colorectal cancer (40%).

Table 36.8: Combined Preparations

Advantages	Disadvantages
■ Highly effective	■ Requires education and motivation
■ Good cycle control	■ Limitation in its use (see above)
■ Well-tolerated in majority	■ Requires initial checkup and periodic supervision
■ Additional non-contraceptive— benefits are many (see above)	■ Inconvenience caused in its use due to daily schedule
■ Low-dose pill with "lipid-friendly" progestins further reduces the risk	■ Risk of drug interactions (p. 624)
■ Reversibility rate is prompt	■ Costly but free supply through government channel (Mala-N)
	Failure rate—0.1/HWY

ADVERSE EFFECTS OF COMBINED ORAL CONTRACEPTIVES

The minor complications or ailments are —

- **Nausea, vomiting, headache (OGN) and leg cramps (PGN)** — These are transient and often subside following continuous use for 2–3 cycles.
- **Mastalgia (OGN + PGN)** — Heaviness or even tenderness in the breast is often transient.
- **Weight gain (PGN)** — Though progestins have got an anabolic effect due to its chemical relation to testosterone, use of low-dose COCs does not cause any increase in weight.
- **Chloasma (OGN) and acne (PGN)** are annoying for cosmetic reasons. Low-dose oral contraceptives improve acne as levonorgestrel preparations are less androgenic.
- **Menstrual abnormalities**—(a) ***Breakthrough bleeding (BTB)*** is commonly due to subthreshold blood level of hormones. Other causes of break-through bleeding in pill takers are (i) disturbance of drug absorption — diarrhea, vomiting; (ii) use of enzyme-inducing drugs (mentioned earlier), missing pills, use of low-dose pills; (iii) pregnancy complications (miscarriage); (iv) diseases—cervical ectopy or carcinoma. Usually, it settles after 3–4 cycles when there is no other specific cause for BTB. Exogenous estrogen (conjugated estrogen 1.25 mg or estradiol 2 mg) given daily for 7 days can control the bleeding. Doubling up the active pills for 2–3 days, or until bleeding stops, is helpful. A pill containing higher dose of estrogen, with different progestin could be helpful. BTB is not associated with any increased failure rate.

(b) **Hypomenorrhea (PGN)** — It is of little significance, although disturbing to the patient. It is due to the local endometrial changes. (c) **Menorrhagia (OGN)** — It is usually pre-existing and use of compounds with progestin preponderance is helpful. (d) **Amenorrhea (OGN or PGN)** — Postpill amenorrhea of more than 6 months duration occurs in less than 1% cases. The association is casual, not causal. It is usually more in women with pre-existing functional menstrual disorders. Spontaneous resumption of menstruation occurs in majority of cases. A refractory case (≥ 12 months) should be investigated as a case of secondary amenorrhea.

- **Libido:** Libido may be diminished **(PGN)** probably due to dryness of the vagina. More often, it may either remain static or at times, may even increase due to loss of fear of pregnancy.
- **Leukorrhea:** It may be due to excessive cervical mucus secretion **(OGN)** or due to increased preponderance of monilial infection **(OGN + PGN)**.

The major complications are:

- **Depression:** Low-dose estrogen preparations are not associated with depression.
- **Hypertension (OGN):** Current low-dose COCs rarely cause significant hypertension. Pre-existing hypertension is likely to be aggravated. Changes are seen only in systolic but not in diastolic blood pressure. The effect on blood pressure is thought to involve the renin-angiotensin system. There is marked increase in plasma angiotensinogen. The changes, however, reverse back to normal in 3–6 months after stoppage of pill.
- **Vascular complications (OGN):** (a) *Venous thromboembolism (VTE)* — The overall risk is to the extent of 3–4 times more than the non-users. Pre-existing hypertension, diabetes, obesity, thrombophilias (inherited or acquired) and elderly patient (over 35 years especially with smoking habits) are some of the important risk factors. Ethinyl estradiol used with a dose of 20 μg in the pill markedly reduces the incidence.

Current studies estimate the annual number of nonfatal VTE per 100,000 users as: no COC use = 5, second generation COC = 15, COC containing desogestrel and gestodene = 30, pregnancy = 60. The absolute risk is very small compared to pregnancy. The risk of death from VTE due to COCs is extremely low at 1–5 per million per year. The most important risk factor is genetic thrombophilia (factor V Leiden mutation). This is rare in Asians (0.4%) compared to Caucasian (5%). (b) Arterial thrombosis — **The high risk factors for myocardial infarction and stroke (ischemic and hemorrhagic) are hypertension, smoking habit, age over 35 years and diabetes.** Women with multiple risk factors for cardiovascular disease generally should not use COCs. However, low-dose COCs do not increase the risk of myocardial infarction or stroke in a healthy, nonsmoking woman, irrespective of age.

- **Cholestatic jaundice**—Susceptibility is increased in women with previous history of idiopathic recurrent jaundice in pregnancy or hepatitis.
- **Neoplasia (OGN)** — Combined oral contraceptives (COCs) **reduce the risk of epithelial ovarian (50% ↓) and endometrial (50% ↓) carcinoma**. This protective effect persists for 10–15 years even after stopping the method following a use of 6 months to 1 year. No major association has been established between breast carcinoma and low-dose COC use. Conclusions regarding association of COC and cervical carcinoma are not definite. However, pill users should have regular HPV DNA and **cervical cytology screening**. **No increased risk of hepatocellular adenomas** has been found with low-dose preparations. It gives protection against benign cystic breast disease and cystic ovaries.

GENERAL METABOLIC EFFECTS: ♦ **Carbohydrate (PGN):** Progestins impair glucose tolerance promoting insulin resistance and hyperglycemia. This was observed in preparations containing 150 μg or more levonorgestrel. Low-dose COCs have no effect on insulin, HbA1C, and fasting glucose levels. ♦ **Protein (OGN):** Estrogen has got some stimulatory effect on the hepatic secretion of many proteins. The level of sex hormone binding globulin (SHBG) is increased. ♦ **Lipid (OGN):** Plasma lipids and lipoproteins are increased. Total cholesterol and triglycerides are increased. Low dose estrogen increases HDL cholesterol and decreases LDL cholesterol thereby exerts its protective effect against atherosclerosis. Progestins, however, decrease HDL cholesterol and increase LDL cholesterol thereby promote heart disease. Preparations with more selective, lipid friendly and third-generation progestins namely desogestrel, gestodene or norgestimate, HDL level is somewhat elevated. However, most changes are within the normal range and not clinically relevant.

- **Vitamins and minerals:** Vitamin B_6, vitamin B_{12}, folic acid, calcium, manganese, zinc and ascorbic acid levels are decreased while vitamins A and K levels are increased.

EFFECTS ON ORGANS: ♦ **Hypothalamopituitary axis:** Both FSH and LH levels remain low as found in early proliferative phase and remain throughout the cycle at such static low level. ♦ **Ovary:** Ovarian function remains quiescent with occasional evidence of breakthrough ovulation. There is evidence of fibrosis, progressive wastage of unripe ova with advancing age without evidence of corpus luteum. The endogenous hormones remain static at a low level. ♦ **Endometrium (PGN)** — Stromal edema, decidual reaction and glandular exhaustion out of depletion of glycogen are more or less constant findings.

- **Cervix (PGN + OGN)**—Increased glandular hyperplasia and downgrowth of the endocervical epithelium beyond the squamocolumnar junction gives the appearance of an ectopy. Relative risk of cervical cancer with COC use is 1.1. It may be due to the persistent exposure of the pill users to HPV infection or due to their more sexual activity. ♦ **Uterus (OGN)**—Uterus may be slightly enlarged. Low dose COCs do not usually increase the size of a pre-existing fibroid. COCs can reduce the amount of menstrual bleeding. ♦ **Vagina (PGN)**—Cytohormonal study reflects the picture of early luteal phase. ♦ **Other organs:** (i) *Liver* — The liver functions are depressed; (ii) *Gastrointestinal tract* — There is increased incidence of mesenteric vein thrombosis, (iii) *Urinary* — There is increased incidence of urinary tract infection but is probably related to increase in sexual activity.

EFFECTS ON REPRODUCTION: ♦ **Ovulation returns within 3 months of withdrawal of the drug in 90% cases.** ♦ **Risk to fetus:** When COC is taken during early pregnancy inadvertently, there is no greater risk of significant congenital anomaly. Risk of congenital abnormality in general is 2–3%. ♦ **Lactation (OGN + PGN):** Lactation is probably affected by a reduction in the milk production and also by alteration of the quality of the milk (reduction of protein and fat content). Moreover, significant amount of the steroids are ingested by the infant, the effects are as yet unknown. Mini-pill is a better alternative for the breastfeeders.

TRIPHASIC FORMULATIONS OF COMBINED ORAL PILLS: In these preparations, the hormonal doses of each compound vary over the course of the cycle. Minimum doses are provided for contraceptive effect in the early part of the cycle and slightly higher doses later in the cycle to prevent breakthrough bleeding. **It is an attempt to minimize undesirable side effects on lipid metabolism.** This is due to low total amount of steroids and the balanced estrogen-progestogen relationship.

Triquilar tablets (Schering-AG) — First 6 tablets contain 0.05 mg levonorgestrel and 30 mg of ethinyl estradiol; next 5 tablets contain 0.075 mg levonorgestrel and 40 mg ethinyl estradiol; the last 10 tablets contain 0.125 mg levonorgestrel and 30 mg ethinyl estradiol. It has to be taken like conventional "pills".

PROGESTIN ONLY CONTRACEPTION (POP/MINI PILL)

POP is devoid of any estrogen compound. It contains very low dose of a progestin in any one of the following form — levonorgestrel 75 µg, norethisterone 350 µg, desogestrel 75 µg, lynestrenol 500 µg or norgestrel 30 µg. **It has to be taken daily from the 1st day of the cycle**.

Mechanism of action: It works mainly by making cervical mucus thick and viscous, thereby prevents sperm penetration. Endometrium becomes atrophic, so blastocyst implantation is also hindered. In about 2% of cases ovulation is inhibited and 50% women ovulate normally.

How to prescribe mini pill: The first pill has to be taken on the 1st day of the cycle and then continuously. It has to be taken **regularly and at the same time of the day**. There must be no break between the packs. **Delay in intake** for more than 3 hours, the woman should have missed pill immediately and the next one as schedule. Extra precaution has to be taken for next 2 days (see above).

Advantages: (1) Side effects attributed to estrogen in the combined pill are totally eliminated; (2) No adverse effect on lactation and hence can be suitably prescribed in lactating women and as such it is often called "Lactation Pill"; (3) Easy to take as there is no "on and off" regime; (4) It may be prescribed in patient having (medical disorders) hypertension, fibroid, diabetes, epilepsy, smoking and history of thromboembolism; (5) Reduces the risk of PID and endometrial cancer.

Disadvantages: (1) There may be acne, mastalgia, headache, breakthrough bleeding, or at times amenorrhea in about 20–30% cases; (2) All the side effects, attributed to progestins may be evident;

(3) Simple cysts of the ovary may be seen, but they do not require any surgery; (4) Failure rate is about 0.5–2 per 100 women years of use. Failure is more in young compared to women over 40 years. Women using drugs that induce liver microsomal enzymes (mentioned above) should avoid this method of contraception.

Contraindications: (i) Pregnancy; (ii) Unexplained vaginal bleeding; (iii) Recent breast cancer; (iv) Arterial disease; (v) Thromboembolic disease.

INJECTABLE PROGESTINS

The preparations commonly used are depot medroxyprogesterone acetate (DMPA) and norethisterone enanthate (NET-EN). Both are administered intramuscularly (deltoid or gluteus muscle) within 5 days of the cycle. The injection (IM) should be deep, Z-tract technique and the site not to be massaged. DMPA in a dose of 150 mg every 3 months (WHO 4 months) or 300 mg every 6 months; NET-EN in a dose of 200 mg given at 2-monthly intervals.

Depo-Sub Q provera 104, contains 104 mg of micronized preparation of DMPA. It is given subcutaneously over the anterior thigh or abdomen. It suppresses ovulation for 3 months as it is absorbed more slowly.

Mechanism of action: (1) Inhibition of ovulation — by suppressing the mid cycle LH peak; (2) Cervical mucus becomes thick and viscid, thereby prevents sperm penetration; (3) Endometrium is atrophic preventing blastocyst implantation.

Advantages: (1) It eliminates regular medication as imposed by oral pill; (2) It can be used safely during lactation. It probably increases the milk secretion without altering its composition; (3) No estrogen related side effects; (4) Menstrual symptoms, e.g. menorrhagia, dysmenorrhea are reduced; (5) Protective against endometrial cancer; (6) Can be used as an interim contraception before vasectomy becomes effective; (7) Reduction in PID, endometriosis, ectopic pregnancy and ovarian cancer. **The non-contraceptive benefits**: DMPA reduces the risk of—salpingitis, endometrial cancer, iron-deficiency anemia, sickle cell problems and endometriosis.

Disadvantages: Failure rate for DMPA – (0–0.3/HWY). There is chance of irregular bleeding and occasional phase of amenorrhea. Return of fertility after their discontinuation is usually delayed for several months (4–8 months). However, with NET-EN the return of fertility is quicker. Loss of bone mineral density (reversible) has been observed with long-term use of depo-provera. Overweight, insulin-resistant women may develop diabetes. **Other side effects are**: weight gain and headache.

Contraindications: Women with high-risk factors for osteoporosis and the others are same as in POP (see above).

IMPLANT

Implanon is a progestin only delivery system containing 3-ketodesogestrel (etonogestrel). It is a long-term (up to 3 years) reversible contraception. It consists of a single closed capsule (made of polydimethylsiloxane 40 mm × 2 mm) and contains 68 mg of etonogestrel (ENG). It releases the hormone about 60 mcg, gradually reduced to 30 mcg per day over 3 years. Implanon does not cause decrease in bone mineral density (Fig. 36.9).

Mechanism of action: It inhibits ovulation in 90% of the cycles for the 1st year. It has got its supplementary effect on endometrium (atrophy) and cervical mucus (thick) as well.

Insertion: The capsule is inserted subdermally, in the inner aspect of the nondominant arm, 6–8 cm above the elbow fold. It is inserted between biceps and triceps muscles. Preloaded sterile applicator is available. No incision is required. Removal is done by making a 2 mm incision at the tip of the implant and pushing the rod until it pops out. It is done under local anesthetic. It is ideally inserted within D 5 of a menstrual cycle, immediately after abortion and 3 weeks after postpartum.

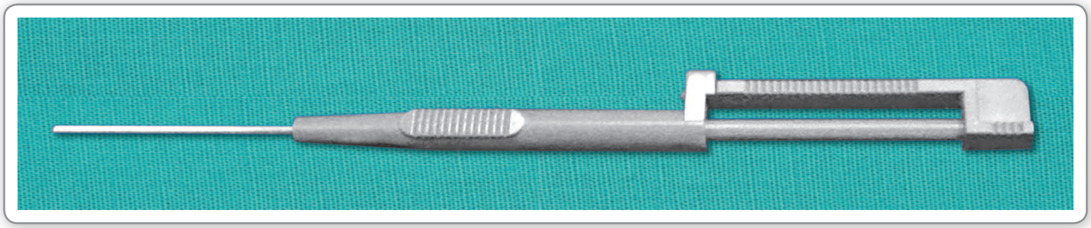


Fig. 36.9: Single implant rod — Implanon (Applicator)

Removal: Implanon should be removed within 3 years of insertion. Loss of contraceptive action is immediate.

Advantages are the same as with DMPA. Others are (i) Highly effective for long-term use and rapidly reversible; (ii) Suited for women who have completed their family but do not desire permanent sterilization. **Efficacy of Implanon** is extremely high with Pearl indices of 0.01. This safe and effective method is considered as "reversible sterilization". **Drawbacks:** Frequent irregular menstrual bleeding, spotting and amenorrhea are common. Difficulty in removal is felt occasionally. **Contraindications** are similar to POP (see above).

NORPLANT–II (Jadelle): Two rods of 4 cm length with diameter of 2.5 mm are used. Each rod contains 75 mg of levonorgestrel. It releases 50 mcg of levonorgestrel per day. Contraceptive efficacy is similar to combined pills. Failure rate is 0.06 per 100 women years. It is used for 3 years. The rods are easier to insert and remove.

EMERGENCY CONTRACEPTION (EC) *(Syn: Postcoital Contraception)*

- **Hormones**
- **IUD**
- **Antiprogesterone**
- **Others**

Indications of emergency contraception: Unprotected intercourse, condom rupture, missed pill, delay in taking POP for more than 3 hours, sexual assault or rape and first time intercourse, as known to be always unplanned. Risk of pregnancy following a single act of unprotected coitus around the time of ovulation is 8%.

HORMONES (Table 36.9)

Levonorgestrel (E. Pills) 0.75 mg, two doses given at 12 hours interval, is very successful and without any side effects. The two tablets (1.50 mg) can be taken as a single dose also. The first dose should be taken within 72 hours (see Fig. 36.10).

Ulipristal acetate is a progesterone receptor modulator. A single dose 30 mg, to be taken orally as soon as possible or within 120 hours of coitus. It acts by suppressing follicular and endometrial growth. It delays ovulation and inhibits implantation. It should not be prescribed in women with severe hepatic dysfunction or with severe asthma. It is more effective compared to levonorgestrel.

Table 36.9: Emergency Contraceptives

Drug	Dose	Pregnancy Rate (%)
Levonorgestrel	0.75 mg stat and after 12 hours	0–1
Ulipristal acetate	30 mg PO	0–1
Copper IUDs (Gold standard)	Insertion within 5 days	0–0.1
Mifepristone	100 mg single dose	0–0.6
Ethinyl estradiol	2.5 mg BD × 5 days	0–0.6
Ethinyl estradiol 50 μg + norgestrel 0.25 mg	2 tab stat and 2 after 12 hours	0–2

Rx
2 Tablets
LEVONORGESTREL TABLETS
आकस्मिक गर्भ निरोधक गोलियाँ
EMERGENCY CONTRACEPTIVE PILLS
ई. पिल्स
E.Pills
स्त्रियों के लिए खाने की गोली
FOR ORAL USE BY WOMEN
Free Supply,
Not for Sale

Fig. 36.10: Levonorgestrel pill

Copper IUD: Introduction of a copper IUD within a maximum period of 5 days can prevent conception following accidental unprotected exposure. This prevents implantation. Failure rate is about 0–1%. It is the **gold standard** method to be offered to all women for EC.

Progesterone receptor modulators: Antiprogesterone (RU 486- Mifepristone) binds competitively to progesterone receptors and nullifies the effect of endogenous progesterone.

Dose: A single dose of 100 mg is to be taken within 17 days of intercourse. Implantation is prevented due to its antiprogesterone effect. Pregnancy rate is 0–0.6%.

Morning-after pill: This is not true contraception, but has rightly been called interception, preventing conception in case of accidental unprotected exposure around the time of ovulation. **Drugs commonly used**, levonorgestrel (see above), ethinyl estradiol 2.5 mg. The drug is taken orally twice daily for 5 days, beginning soon after the exposure but not later than 72 hours.

No fetal adverse effects have been observed when there is failure of emergency contraception. However, induced abortion should be offered to the patient, if the method fails.

Mode of action: The exact mechanism of action remains unclear. The following are the possibilities:

- Ovulation is either prevented or delayed when the drug is taken in the beginning of the cycle.
- Fertilization is interfered.
- Implantation is prevented (except E. Pills) as the endometrium is rendered unfavorable.
- Interferes with the function of corpus luteum or may cause luteolysis.

Drawbacks: Nausea and vomiting are much more intense with estrogen use. Antiemetic (meclizine) should be prescribed.

Combined hormonal regimen (Yuzpe method) is equally effective. Two tablets of Ovral (0.25 mg levonorgestrel and 50 μg ethinyl estradiol) should be taken as early as possible after coitus (< 72 hours) and two more tablets are to be taken 12 hours later.

Oral antiemetic (10 mg metoclopramide) may be taken 1 hour before each dose to reduce the problem of nausea and vomiting.

Postcoital contraception is only employed as an emergency measure and is not effective if used as a regular method of contraception.

SUMMARY OF ORAL CONTRACEPTIVES

- **Combined pills**
- **Triphasic pill**
- **Emergency (postcoital) contraception**
- **Mini pill**

Conventional combined preparations—The widely used oral contraceptives consist of tablets containing estrogen and progestin compounds. It is the most effective and reversible method of contraception. Each tablet usually contains 30 mg of ethinyl estradiol and 1 mg of norethisterone or 0.3 mg norgestrel. **It has got trigger action**—(a) inhibition of ovulation, (b) production of static endometrial hypoplasia and (c) alteration of the character of the cervical mucus. **Its use is absolutely contraindicated in cases** with circulatory diseases, liver diseases, severe migraine and estrogen-dependent tumor. The pill should be started from the day 1 of a cycle and continued as “3 weeks on and 1 week off” regime. Periodic checkup is essential especially when prescribed in women above the age of 35 years. **The pill should be withdrawn** if complications arise such as severe migraine, chest pain, visual disturbances, etc.

The beneficial effects are relief from dysmenorrhea, premenstrual tension, endometriosis, acne, hirsutism and lesser chance of ectopic and PID. It gives protection against ovarian and endometrial carcinomas.

The minor side effects are—nausea, vomiting, break-through bleeding, mastalgia, leg cramp, weight gain, hypomenorrhea or amenorrhea. The major complications are rare and include depression, hypertension and thromboembolic manifestations. The failure rate is about 0.1/HWY.

- **Triphasic pill**—It has got lesser amount of steroids than the conventional monophasic tablets. There is lesser effect on lipid metabolism.
- **Emergency**—Following rape or accidental exposure, either levonorgestrel, 0.75 mg two doses at 12 hours interval or two tablets of the COC preparations are to be taken soon after coitus and two more tablets after 12 hours are quite effective in preventing conception. The first dose should be taken within 72 hours.
- **Mini pill**—The pill contains low doses of progestin— norgestrel 30 mg, levonorgestrel 75 µg or desogestrel 75 µg. It should be taken daily and can be safely prescribed during lactation. It is best suited where estrogen is contraindicated.

STERILIZATION

Permanent surgical contraception, also called voluntary sterilization, is a surgical method whereby the reproductive function of an individual male or female is purposefully and permanently destroyed. **The operation done on male is vasectomy and that on the female is tubal occlusion**, **or tubectomy**.

Couple Counseling

Couple must be counseled adequately before any permanent procedure is undertaken. Individual procedure must be discussed in terms of benefits, risks, side effects, failure rate and reversibility.

VASECTOMY (FIGS 36.11)

It is a permanent sterilization operation done in the male where a segment of vas deferens of both the sides are resected and the cut ends are ligated.

Advantages: (1) The operative technique is simple and can be performed by one with minimal training; (2) The operation can be done as an outdoor procedure or in a mass camp even in remote villages; (3) Complications—immediate or late are few; (4) Failure rate is minimal—1 in 2,000 after 10 years and there is a fair chance of success of reversal anastomosis operation (70–80%); (5) The overall expenditure is minimal in terms of equipment, hospital stay and doctor's training.

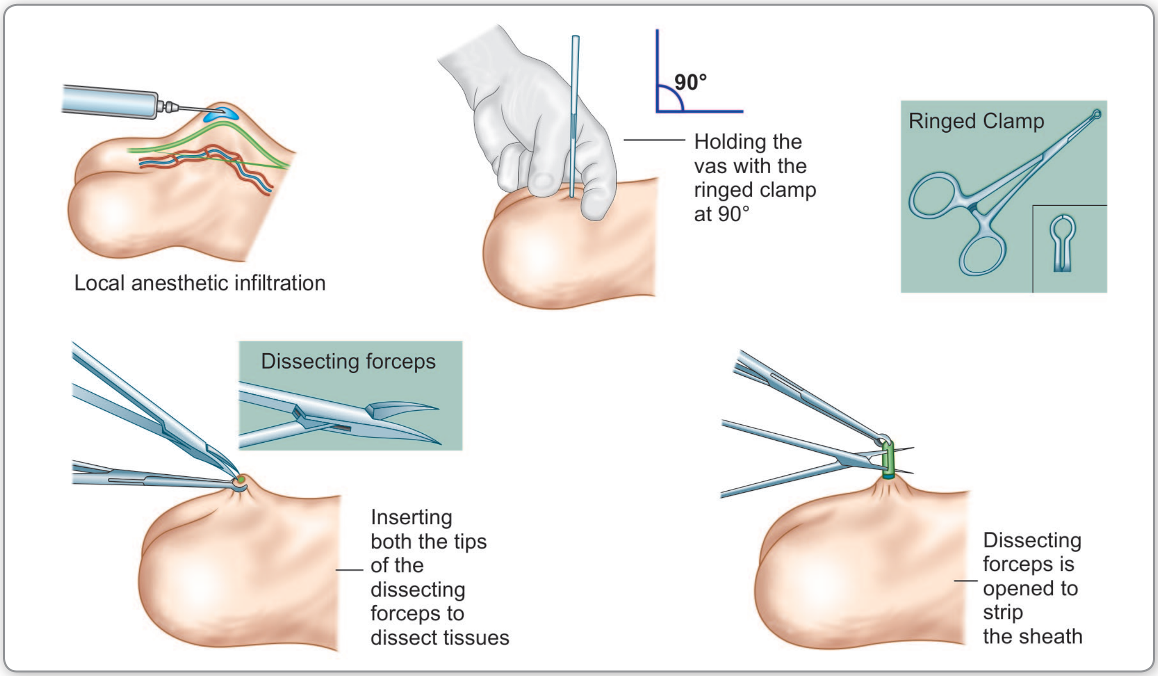

Local anesthetic infiltration
90°
Holding the vas with the ringed clamp at 90°
Ringed Clamp
Dissecting forceps
Inserting both the tips of the dissecting forceps to dissect tissues
Dissecting forceps is opened to strip the sheath

Fig. 36.11: Method of no-scalpel vasectomy (NSV) operation. The dissecting forceps and the ringed clamp are seen in the inset

Drawbacks: (1) Additional contraceptive protection is needed for about 2–3 months following operations, i.e. till the semen becomes free of sperm; (2) Frigidity or impotency when occurs is mostly psychological.

Selection of candidates: Sexually active and psychologically adjusted husband having the desired number of children is an ideal one.

No-Scalpel Vasectomy (NSV): It is commonly done at present in India. It was popularized by Dr Li Shun Qiang of China in 1991.

Technique: Written consent of the person is taken following counseling. The operation is done as an outdoor procedure or in the camp. The local area is shaved and cleaned with povidone-iodine lotion. Full surgical asepsis has to be maintained during operation. Procedure is done under local anesthetic.

The vas is palpated with three fingers of the left hand; index and thumb in front and the middle behind. This is done at the level midway between the top of the testis and the base of the penis. The vas is grasped with a ringed clamp applied perpendicularly on the skin overlying the vas. The skin is punctured with the sharp pointed end of the medial blade of dissecting forceps. The puncture point is enlarged by spreading the tissues (dartos muscle and spermatic fascia) inserting both the tips of the dissecting forceps. The vas is elevated with the dissecting forceps and in hold with the ringed clamp. At least 1 cm of length of vas is made free and mobilized. The vas is ligated at two places 1 cm apart by No. “00” chromic catgut and the segment of the vas in between the ligatures is resected out. Division of the vas should be accompanied by fascial interposition or diathermy. This reduces the failure rate. Hemostasis is secured. No skin suturing is needed. Wound dressing is done and a small pressure bandage is applied. The same procedure is repeated on the other side. A scrotal suspensory bandage is worn. The patient is allowed to go home after half an hour. Histological examination of the excised segment of the vas should be done for confirmation if the surgeon is in any doubt.

Advices: Antibiotic (Injection Penidure LA6 IM) is administered as a routine and an analgesic is prescribed. Heavy work or cycling is restricted for about 2 weeks, while usual activities can be resumed forthwith. For checkup, the patient should report back after 1 week, or earlier, if complication arises. **Additional contraceptive should be used for 3–4 months.**

NVS takes less time and helps in faster recovery due to less tissue injury. Complications are significantly less. However, it needs training on the part of the surgeon.

Precaution: The man does not become sterile soon after the operation as the semen is stored in the distal part of the vas channels for a varying period of about 3 months. **It requires about 20 ejaculations to empty the stored semen**. Semen should be examined either by one test after 16 weeks or by two test at 12 weeks and 16 weeks after vasectomy and **if the two consecutive semen analyses show absence of spermatozoa, the man is declared as sterile. Till then, additional contraceptive (condom or DMPA to wife) should be advised**.

Complications: Complications of NSV are significantly less.

- ***Immediate*** — (1) Wound sepsis which may lead to scrotal cellulitis or abscess; (2) Scrotal hematoma.
- ***Remote***—(1) Frigidity or impotency—It is mostly psychological in origin; (2) Sperm granuloma is due to inflammatory reaction to sperm leakage. This can be prevented by cauterization or fulguration of the cut ends; (3) Chronic intrascrotal pain and discomfort (post-vasectomy syndrome) may be due to scar tissue formation, or tubular distension of the epididymis; (4) There is no increase in prostate or testicular cancer or heart disease; (5) Spontaneous recanalization (1 in 2,000) is rare.

Other Methods to Block the Vas

A. **Electrocoagulation** may be used to encourage scar tissue formation.

B. **Fascial interposition** following ligation, excision and cautery. This is done to prevent recanalization.

FEMALE STERILIZATION

Occlusion of the Fallopian tubes in some form is the underlying principle to achieve female sterilization. It is the most popular method of terminal contraception all over the world.

Indications: (1) **Family planning purposes:** This is the principal indication in most of the developing countries. (2) **Socioeconomic:** An individual is adopted to accept the method after having the desired number of children. (3) **Medicosurgical indications (therapeutic):** Medical diseases such as heart disease, diabetes, chronic renal disease, hypertension are likely to worsen, if repeated pregnancies occur and hence sterilization is advisable. During third time repeat cesarean section or repair of prolapse operation, to avoid the risks involved in the future childbirth process, sterilization operation should be seriously considered.

Time of Operation: (1) ***During puerperium (puerperal):*** If the patient is otherwise healthy, the operation can be done 24–48 hours following delivery. Its chief advantage is technical simplicity. Hospital stay and rest at home following delivery are enough to help the patient to recover simultaneously from the two events, i.e. delivery and operation. (2) ***Interval:*** The operation is done beyond 3 months following delivery or abortion. **The ideal time of operation is following the menstrual period in the proliferative phase**. (3) ***Concurrent with MTP:*** Sterilization is performed along with termination of pregnancy. This is mostly done in the urban centers.

Methods of female sterilization: Occlusion by resection of a segment of both the Fallopian tubes (commonly called tubectomy) is the widely accepted procedure. Currently, occlusion of the tubes with rings or clips or electrocoagulation using a laparoscope is gaining popularity. Hysterectomy during the childbearing period has got an incidental sterilization effect but should not be done for sterilization purpose.

Tubectomy (Figs 36.12 and 36.14): It is an operation where resection of a segment of both the Fallopian tubes is done to achieve permanent sterilization. The approach may be: **(1) Abdominal** **(2) Vaginal**

(1) Abdominal: (A) **Conventional** (B) **Minilaparotomy**

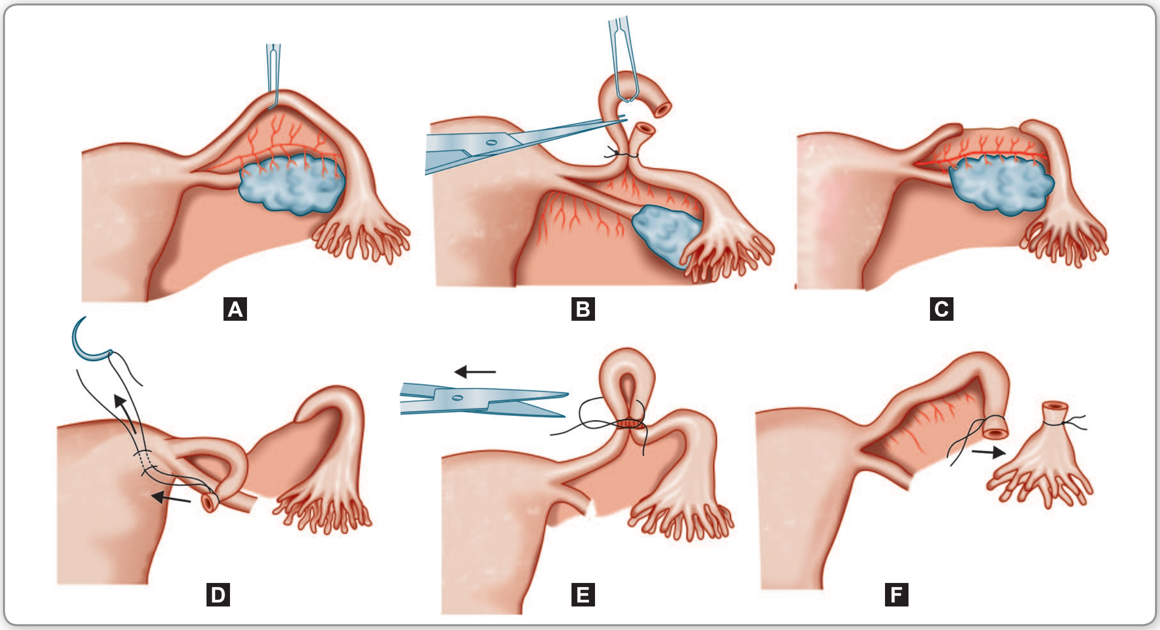
***Conventional (Laparotomy)*—Steps:**

- **Anesthesia:** The operation can be done under general or spinal or local anesthesia. **In mass camp, local anesthesia is preferable.** In case of local anesthesia, premedication with inj. morphine 15 mg or inj. pethidine 100 mg with phenergan 50 mg IM is to be administered at least 30–45 minutes prior to surgery. The incisional area is infiltrated with 1% lignocaine.
- **Incision: In puerperal cases**, where the uterus is felt per abdomen, the incision is made two fingers breadth (1") below the fundal height and in interval cases, the incision is made two fingers breadth above the symphysis pubis. The incision may be either midline or paramedian or transverse. The abdomen is opened by the usual procedure.
- **Delivery of the tube:** The index finger is introduced through the incision. The finger is passed across the posterior surface of the uterus and then to the posterior leaf of the broad ligament from where the tube is hooked out. **The tube is identified by** the fimbrial end and mesosalpinx containing utero-ovarian anastomotic vessels.
- **Techniques — (Fig. 36.12):** (a) **Pomeroy's:** A loop is made by holding the tube by an Allis forceps in such a way that the major part of the loop consists mainly of isthmus and part of the ampullary part of the tube (at the junction of proximal and middle third). Through an avascular area in the mesosalpinx, a needle threaded with No. "0" chromic catgut is passed and both the limbs of the loop are firmly tied together. About 1–1.5 cm of the segment of the loop distal to the ligature is excised. The tube is so excised as to leave behind about 1.5 cm of intact tube adjacent to uterus. **Segment of the loop removed is to be inspected to be sure that the wall has not been partially resected and to send it for histology.** The same procedure is repeated on the other side. Because of the absorption of the absorbable ligature, the cut ends become independently sealed off and are separated after a few weeks.

Advantages: It is easy, safe and very effective in spite of the simplicity of the technique. The failure rate is 0.1–0.5%. The cut ends become independently sealed off and retract widely from each other (Fig. 36.12C).

(b) **Uchida technique**—A saline solution is injected subserosally in the mid portion of the tube to create a bleb. The serous coat is incised along the antimesenteric border to expose the muscular tube. The tube is ligated with No. 0 chromic catgut on either side and about 3–5 cm of the tube is resected off. The ligated proximal stump is allowed to retract beneath the serous coat. The serous coat is closed with a fine suture in such a way that the proximal stump is buried but the distal stump is open to the peritoneal cavity. No failure in this method has been observed so far.

(c) **Irving method —** The tube is ligated on either side and mid portion of the tube (between the ties) is excised. The free medial end of the tube is then turned back and buried into the posterior uterine wall creating a myometrial tunnel (Fig. 36.12D).



Figs 36.12A to F: Steps of tubectomy by **Pomeroy's method**: (A) A segment of the Fallopian tube is lifted up; (B) The loop is ligated with chromic catgut and is cut (about 1.5 cm); (C) End result of the operation—note wide separation; (D) **Irving procedure**: the medial cut end is buried in the myometrium posteriorly and the distal cut end is buried in the mesosalpinx; (E) **Madlener procedure**; (F) **Kroener procedure**: the ampullary end of the tube is ligated and resected

(d) **Madlener technique** (Fig. 36.12E)—It is the easiest method. The loop of the tube is crushed with an artery forceps. The crushed area is tied with black silk. **The loop is not excised**. The failure rate is very high to the extent of 7% and hence, it is abandoned in preference to the Pomeroy's technique.

(e) **Kroener** method of fimbriectomy is not a common procedure (Fig. 36.12F).

The abdomen is closed in layers. Antibiotics are given routinely in the postoperative period. The abdominal stitches are removed on the 5th day and the patient is discharged. However, if the patient has satisfactory postoperative progress, she may be discharged after 48 hours. The stitches may be removed in the outpatient department.

MINILAPAROTOMY (MINI-LAP): When the tubectomy is done through a small abdominal incision along with some device, the procedure is called mini-lap. It has been popularized by Uchida of Japan ever since 1961.

Steps: (1) Anesthesia — Always under local anesthesia; (2) Plan of incision — As described in conventional method but the incision should be ½"–¾"; (3) Specially designed retractor may be introduced after the abdomen is opened; (4) Uterus is elevated or pushed to one side or the other by the elevator that has already been introduced transvaginally into the uterine cavity. This helps manipulation of the tube in bringing it close to the incisional area, when it is seized by artery forceps; (5) The appropriate technique of tubectomy is performed on one side and then repeated on the other side; (6) The peritoneum is closed by purse string suture.

Once conversant with the technique, it can be performed with satisfaction to the patient. It also benefits the organization (turnover of the patient per bed is more than that in the conventional method). The patient is usually discharged within 24–48 hours.

Vaginal Ligation: Tubectomy through the vaginal route may be done along with vaginal plastic operation or in isolation. When done in isolation, **the approach to the tube is through posterior colpotomy.** Surgeon needs additional skill of vaginal surgery. Interval cases (uterus < 12 weeks) are most suited. It is done under general or spinal anesthesia. It takes longer time. Laparotomy may sometimes be needed due to difficulties. **Complications are**: hemorrhage, broad ligament hematoma and rarely rectal injury. Dyspareunia may be a late complication. **Advantage**: Short hospital stay, convenient in obese women. Its limitation and relative merits and demerits are given in Table 36.11.

LAPAROSCOPIC STERILIZATION

Laparoscopy is the commonly employed method of endoscopic sterilization (Fig. 36.13). It is gradually becoming more popular—especially in the camps. The procedure is mostly done under local anesthesia. The operation is done in the interval period, concurrent with vaginal termination of pregnancy or 6 weeks following delivery. It should not be done within 6 weeks following delivery (Fig. 36.13).

The procedure can be done either with single puncture or double puncture technique. The tubes are occluded either by a **silastic ring** (silicone rubber with 5% barium sulfate) devised by **Fallope** or by **Filshie clip** is made of titanium lined with silicone rubber. Only 4 mm of the tube is destroyed. Failure rate is 0.1%. **Hulka-Clemens spring clip** is also used. **Electrosurgical methods**—Dessicates the tissue by heating. Unipolar or bipolar method of tubal coagulation is used. Bipolar cautery is safer than unipolar one but it has higher failure rates (2.1%). Laser photocoagulation is not popular because of high recanalization rate.

Principal steps (Single puncture technique)

Premedication — Pethidine hydrochloride 75–100 mg with phenergan 25 mg and atropine sulfate 0.65 mg are given intramuscularly about half an hour prior to operation.

Local anesthesia—Taking usual aseptic precautions about 10 mL of 1% lignocaine hydrochloride is to be infiltrated at the puncture site (just below the umbilicus) down up to the peritoneum.

Position of the patient—The patient is placed in lithotomy position. The operating table is tilted to approximately 15 degrees of Trendelenburg position. Usual aseptic precaution is taken as in abdominal and vaginal operations. The bladder should be fully emptied by a metal catheter. Pelvic examination is done methodically. A uterine manipulator is introduced through the cervical canal for manipulation for visualization of tubes and uterus at a later step.

Producing pneumoperitoneum—A small skin incision (1.25 cm) is made just below the umbilicus. The Veress needle is introduced through the incision with 45° angulation into the peritoneal cavity. The abdomen is inflated with about 2 L of gas (carbon dioxide or nitrous oxide or room air or oxygen). Choice of gas depends upon the method of sterilization.

Introduction of the trocar and laparoscope with ring loaded applicator—Two silastic rings are loaded one after the other on the applicator with the help of a loader and pusher. The trocar with cannula is introduced through the incision previously made with a twisting movement. The trocar is removed and the laparoscope together with ring applicator is inserted through the cannula (Fig. 36.14).

The ring loaded applicator approaches one side of the tube and grasps **at the junction of the proximal and middle third of the tube.** A loop of the tube (2.5 cm) is lifted up, drawn into the cylinder of the applicator and the ring is slipped into the base of the loop under direct vision. The procedure is to be repeated on the other side (Fig. 36.14).

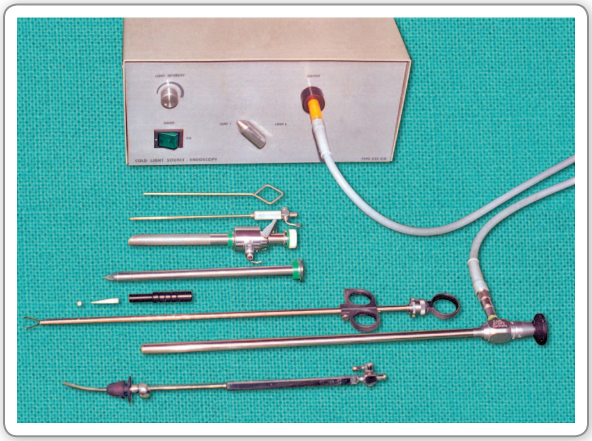


Fig. 36.13: Laparoscopic instruments for tubal sterilization

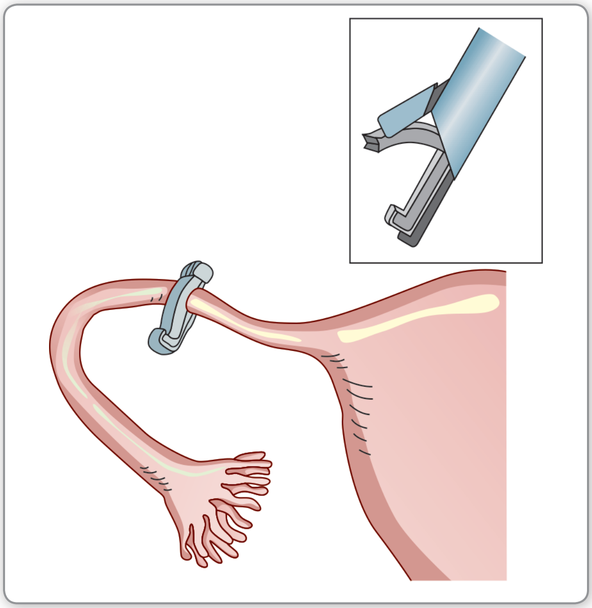


Fig. 36.14: Laparoscopic tubal sterilization by Filshie clip. Filshie (GM Filshie) clip is made of titanium and the inner surfaces are lined with silicon. It is easier to apply and damage to the tube is less (see p. 558)

Removal of the laparoscope: After viewing that the rings are properly placed in position, the tubal loops looking white and there is no intraperitoneal bleeding, the laparoscope is removed. The gas or air is deflated from the abdominal cavity. The abdominal wound is sutured by a single chromic catgut suture.

COMMENTS ON METHODS OF FEMALE STERILIZATION

In the third world countries, mini-lap remains the mainstay in the National Family Planning Program as a method of permanent sterilization. It is safe, has wider applicability, is less expensive and has got a less failure rate compared to laparoscopic sterilization. However, for a quick turnover in an organized mass camp, laparoscopic sterilization offers a promising success (Table 36.11).

HAZARDS OF TUBAL STERILIZATION

Immediate: These are related to general anesthesia and to the particular method used in sterilization. The related complications have already been discussed (Tables 36.10 and 36.11).

Remote: (1) Specific for the approach and (2) related to the sterilization.

(A) The remote complications specific for the approach of the operation, abdominal or vaginal have already been described. **(B)** The complications related to sterilization can be grouped into: (a) **General complications:** These include occasional obesity, psychological upset, and (b) **Gynecological:** (1) Chronic pelvic pain; (2) Congestive dysmenorrhea; (3) Menstrual abnormalities in the form of menorrhagia, hypomenorrhea or irregular periods. Pelvic pain, menorrhagia along with cystic ovaries **constitute a post-ligation syndrome**. It may be vascular in origin. However, the incidence can be minimized, if the blood vessels adjacent to the mesosalpinx are not unduly disturbed; (4) Alteration in libido.

Failure rate: The overall failure rate in tubal sterilization is about 0.7%, **the Pomeroy's technique being the lowest 0.1–0.5%**, in contrast to the Madlener's being 1.5–7%. The failure rate is increased when it is done during hysterotomy or during cesarean section. Failure rates of laparoscopic sterilization depend upon the individual method (electrocoagulation: unipolar 0.75%, bipolar 2.1%, Falope ring 1.7%, Filshie clip 0.1%). Failure may be due to **fistula formation or due to spontaneous reanastomosis**.

Table 36.10: Female Sterilization

	Abdominal Approach	Vaginal Approach
Surgeon	Can be performed by any one conversant with surgery	Can be done only by a surgeon conversant with vaginal plastic operation
Time of operation	Can be done at any time, puerperal or interval	Interval period is most suited May be done in other times, provided the uterus is smaller than 12 weeks
Contraindication	Practically — nil than 12 weeks size	Associated TO mass, uterus—more
Anesthesia	Can be done under local anesthesia	General or spinal anesthesia is usually needed
Complication during operation	Easy to tackle	Difficult at times and laparotomy may be necessary
Duration of operation	Shorter time	Longer time
Complications: *Immediate*	Few Wound infection, peritonitis—rare	Few Hemorrhage, revealed or broad ligament hematoma, injury to the rectum
Late	Incisional hernia, failure rate—less	Dyspareunia, failure rate—more
Hospital stay	Longer—5–6 days Shorter with mini-lap (24–48 hours)	Shorter—24–48 hours

Table 36.11: Mini-lap vis-a-vis Laparoscopic Sterilization

	Mini-lap	Laparoscopic Sterilization
Cost	Minimal maintenance	Expensive, requires adequate maintenance
Personnel	Any medical personnel with surgical skill	Should only be performed by persons with special training
Selection of time	Any time — Puerperium, interval, with MTP	Should not be done within 6 weeks of delivery or with enlarged uterus
Contraindication	Practically none. Can be done in conditions contraindicated for laparoscopy	Lung lesions, organic heart diseases, intra-abdominal adhesions, extreme obesity
Complication life-threatening	Minimal but usually absent	Minimal but at times fatal
Hospital stay	3–5 days	3–4 hours
Failure rate	0.1–0.3%	0.2–0.6%
Reversibility	Difficult due to adhesions and reduced remnant tubal length	Easier and effective. Only 4 mm of the tube is destroyed with the Filshie clip (see p. 635, and Fig. 36.14)

Mortality following tubal sterilization is estimated to be 72 per 100,000 for all methods. Laparoscopic procedures carried the mortality rate of 5–10 per 100,000 compared to 7 per 100,000 for puerperal ligations.

Reversibility: Informed consent must be obtained after **adequate counseling**. Couple must understand the permanency of the procedure, its occasional failure rate, the risks and side effects and its alternatives. Unfortunately, regret is not uncommon. Microsurgical techniques give excellent result for tubal reanastomosis. Pregnancy rates after reversal are high (80%) following use of clips and rings. Reversal of vasectomy with restoration of vas patency is possible in up to 90% of cases. But pregnancy rate is low (50%).

CONTRACEPTIVE COUNSELING AND PRESCRIPTION

Pregnancy carries an overall maternal mortality around 400 per 100,000 total births in the developing countries and the same in the developed countries is less than 10, whereas annual number of deaths per 100,000 exposed to pill is 1.3 and with that of IUDs is 1. The risks of death from automobile driving are 1 in 6,000 per year. Contraception usually carries less risk compared to pregnancy. **Importantly benefits of contraceptive use outweigh the risks of pregnancy**.

No one single universally acceptable method has yet been discovered. The individual should have the liberty to choose any of the currently available well-tested method, which may even vary at each phase in her reproductive life. If one compares the risks and benefits of any contraceptive, it is observed that more deaths occur as a result of unplanned pregnancies than from the hazards of any modern contraceptive method [excluding “Pill” users over 35 years who smoke (see p. 623)].

Important factors for the selection of any contraceptive method for an individual are: relative safety, effectiveness, side effects and willingness to use the method correctly and consistently. The other factors to consider are the frequency of coitus, the need of lactation and prevention of STIs. **Acceptability is probably the most critical factor in the effectiveness of a contraceptive method. Couple (client) should be helped to make an informed choice.** A clear account of the risks and the benefits for an individual method is given. Regular follow-up and compliance with the instructions are to be ensured. It is also essential that an informed (verbal) consent is obtained and recorded.

Sterilization counseling includes a discussion of the following issues: (1) Desire of the individual partner (male/female); (2) Procedure selection; (3) Failure rate; (4) Risks and side effects; (5) Issue of reversibility. Reversal is more likely to be successful after laparoscopic clips compared to laparotomy procedures. However, the risks of ectopic pregnancy are there; (6) Options for alternative long active (equally effective) reversible methods (Implants, CuT 380A) should be given.

Prescription: Conventional contraceptives can be safely prescribed during the entire reproductive period as elective choice or as an alternative to "Pill" or IUD if they are contraindicated or unacceptable to the couple. As such only the advice regarding the use of "Pill" or IUD during different phases of reproductive life is discussed.

Adolescent girls: Low-dose combined pills are most effective for the sexually active adolescents. It is the contraceptive of choice. However, DMPA or Norplant may be an alternative when accepted.

Newly married couple: Provided there is not enough justification to prove early fertility, a highly effective and acceptable contraceptive should be prescribed. IUD may not be prescribed. As such "Pill' is recommended provided there is no contraindication. Apart from effective contraception, "Pill" has got many non-contraceptive benefits as well (see p. 625).

Spacing of Births: ♦ **Postabortal** ♦ **Postpartum** ♦ **Interval**

Postabortal: The contraceptive practice should be started soon following the abortion process is completed. "Pill" is the ideal; IUD is an alternative.

Postpartum: ♦ **Nonlactating** ♦ **Lactating**

Nonlactating—Contraceptive practice should be started after 3 weeks. "Pill" is good; IUD is an equally effective alternative. Injectable depot medroxyprogesterone acetate could be used as it is devoid of any estrogen related side effects. Implanon (etonogestrel) may be prescribed.

Lactating—In fully lactating women (5–6 feeds and spending about 60 minutes in 24 hours), the contraceptive practice may be safely withheld for 10 weeks postpartum. For doubtful adverse effects of steroids on lactation and on the babies through the ingested milk, **"Pill" is better withheld. Minipill or injectable steroids** is ideal. Alternatively, IUD can be inserted.

Interval: Below the age of 35 years, she can have her choice to either "Pill" or IUD following adequate counseling. In women above the age of 35 years, especially who are smokers, IUD should be inserted in preference to "Pill". Injectables (DMPA) or implant (Implanon) where available is the other alternative.

To stop future pregnancies: The decision to advise permanent sterilization should be judiciously given especially to the underprivileged women in the face of high perinatal and infant mortality rate. The cases are to be individualized. However, a two-child formula is usually recommended and as such, a couple having two children who have been fully immunized can have permanent sterilization (husband or wife). If the couple is not motivated to undergo the sterilization operation, any of the temporary methods is to be prescribed till the end of the reproductive period of the wife. Women who have completed their family but do not desire for permanent sterilization, may use IUD (Cu T 380A) or implant if accepted.

Older women: Contraception should be prescribed to avoid unplanned pregnancy. Low-dose pills can be continued till menopause (with monitoring) in the low-risk group. Progestin only pill, injectable progestin and LNG-IUS are the other alternatives. Barrier methods and vaginal spermicides can be used either as a primary or backup method. Usually, fertility is reduced after 40 years of age.

Women at risk of STIs need dual protection against pregnancy and STIs. They should use condom with spermicides or use another contraceptive method in conjunction with condom.

Women using enzyme inducers are advised to take COCs having more than usual dosage (see p. 624) or other method of contraception (injectables, IUDs). **Emergency contraception (postcoital contraception)** when required as emergency, POP, IUD or other methods can be used (see p. 629).

ONGOING TRIALS AND SELECTIVE AVAILABILITY

The following are used on trial basis or are available in selected countries:

Female: ▪ **Centchroman (SAHELI)**—*Ormeloxifene* is a research product of Central Drug Research Institute, Lucknow, India. It is a non-steroidal compound with potent antiestrogenic and weak estrogenic properties. It is taken orally (30 mg) twice a week for first 3 months, then once a week. It works primarily by preventing implantation of fertilized ovum. It does not inhibit ovulation.

Side effects are few. It is avoided in polycystic ovarian disease, with liver and kidney diseases and in tuberculosis. There may be a tendency of oligomenorrhea. The failure rate is about 1–4 per 100 women years of use. Failure rate is less with increased doses. It is devoid of any significant adverse metabolic effect. This may also be used as an emergency contraceptive. It is sold in the market against prescription only and not over the counter.

- **Non-contraceptive use:** Because of its potent antiestrogenic activity, centchroman is being currently tried in the management of dysfunctional uterine bleeding, endometrial hyperplasia, endometriosis and breast cancer. It is used as hormone replacement therapy (HRT), because of its weak estrogenic property.

- **Combined injectable contraceptives (CICs)** — Both estrogen and progestin are combined in these monthly injectables. Preparations available are: DMPA 25 mg with estradiol cypionate 5 mg (Cyclofem) and NET-EN 50 mg with estradiol valerate 5 mg (Mesigyna). Estrogen has been incorporated to improve the menstrual cycle. Use of "natural" estrogens in CICs has got favorable impact on lipid metabolism and cardiovascular effects compared to synthetic estrogen used injection COCs. It is given within first 5 days of menstruation. Next injection should be on the same date of each month (4 week schedule). Fertility return is quick.

Drawbacks—(i) Irregular or prolonged menstrual bleeding; (ii) Not suitable for nursing mothers.

- **Transdermal patch:** ***Nestorone (newer progestin)***—When used as a cream to the skin provides effective contraception. **Patch** delivers 150 µg of norelgestromin (progestin) and 20 µg ethinyl estradiol daily. It has an area of 20 cm^2 (4.5 cm × 4.5 cm). The patch is used weekly for 3 weeks and 1 week off for withdrawal bleeding. It is well-tolerated, safe and effective. **Drawbacks:** Patch detachment, skin reaction and high failure in overweight women (> 90 kg). It is applied over the buttocks, upper and outer arm, or lower abdomen but not over the breasts. Failure rate is 1.2 per 100 women years. Patch failure rate is high in women weighing ≥ 90 kg.

- **Vaginal rings**—Containing levonorgestrel covered by silastic tubing have been introduced. They are 5 and 6 cm in diameter. The vaginal ring delivers levonorgestrel (20 µg/day) to maintain a constant blood level like norplant. The rings are replaced by 90 days. Pregnancy rate is 3 per 100 women years. This method is under woman's control.

- **Combined ring**—Soft, transparent ethylene vinyl ring (NuvaRing) releases ethinyl estradiol (15 µg) and etonogestrel (metabolite of desogestrel) 120 µg daily over a period of 21 days. The ring is inserted on the 1st day of menses and is worn for 3 weeks. The ring must be reinserted within the next 3 hours, if removed for any reason, vaginal route use avoids GI absorption, first pass liver metabolism and has lowest systemic estrogenic side effects. It is then removed and after 1 week (after the withdrawal bleed) a new ring is inserted. It acts by inhibiting ovulation. Pearl index is 0.65 and cycle control is good. The ring (54 mm diameter and 4 mm thick) is inserted within 5 days of menses. Side effects are headache, leukorrhea, vaginitis and expulsion.

- **Uniplant** is a single rod implant, containing 55 mg of nomegestrol (newer progestin) with a release rate of 100 µg/day. It provides contraception for 1 year.

- **Biodegradable implants** are under study. **Capronor** (single capsule) releases levonorgestrel from the polymer E-caprolactone at a rate 10 times faster than from silastic. The longer capsule contains 26 mg of levonorgestrel and inhibits ovulation in about 50% of cycles. Contraceptive efficacy is comparable to norplant. The capsule begins to disappear after 12 months.

- **Injectable contraceptive (biodegradable)** in the form of microspheres using copolymer (lactide-glycolide) have been studied. Hormone currently used in the microsphere (0.06 – 0.1 mm diameter) is either norethindrone or norethindrone combined with ethinyl estradiol. Injection is given over the gluteal muscle. Unlike implant, microspheres cannot be removed once injected.

- **LHRH** agonist (buserelin) and LHRH antagonist (cetrorelix) acts by preventing the pituitary response to the endogenous GnRH. They have the potential to arrest follicular growth and endometrial development. Unwanted side effects (loss of libido and hot flushes) are avoided using add-back therapy. Long term effects are not known as yet.

- **Newer IUDs**—**A frameless IUD (GyneFix)** is made of six copper beads (330 mm^2 of Cu) on a monofilament polypropylene thread. The upper and lower beads are crimped onto the thread. The thread is knotted at one end which is embedded into the fundal myometrium to a depth of 1 cm. This anchors the device at the fundus. **The advantages** of the device (Fig. 36.15) over the framed ones are significantly reduced risk of expulsion, dysmenorrhea,

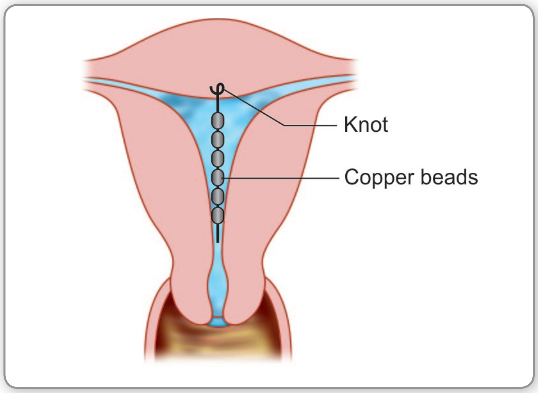


Fig. 36.15: Frameless IUD (GyneFix)

bleeding and infection. Threadless (Butterfly) IUD is also found promising to reduce the risk of side effects (infection). It can be removed with a hook when required. This device is especially suited for nulligravid women.

- **Fibroplant (LNG):** Similar to Mirena (see p. 616), a smaller version of levonorgestrel system is currently being tested. Its small size is suitable for the perimenopausal women in whom the uterus shrinks. It releases LNG at the rate of 14 mcg/day. It is also used as a HRT for postmenopausal women.

TRANSCERVICAL STERILIZATION: ♦ **Quinacrine pellet,** 252 mg is inserted on two occasions 1 month apart into the uterine cavity transcervically through a hysteroscope during the proliferative phase. It is repeated in the next cycle. It acts as a sclerosing agent. Pregnancy rate is 2–3 per 100 women years. Long-term results are awaited.

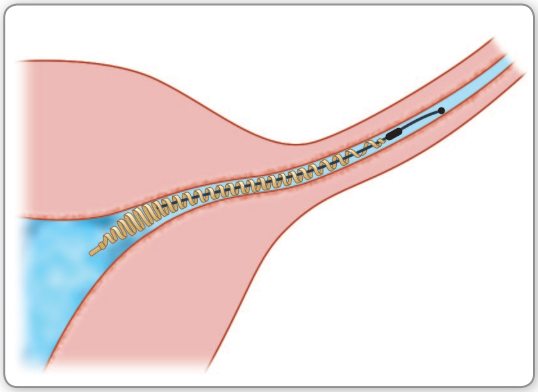


Fig. 36.16: Essure device within the intramural portion of the Fallopian tube. It is inserted using a hysteroscope

♦ **Essure** is a 4 cm long, 2 mm diameter, microcoil (spring like device) made of nickel-titanium steel alloy coil within which lie polyethylene terephthalate fibers (Fig. 36.16). It is inserted into each Fallopian tube transcervically using a hysteroscope. The tube is blocked permanently when scar tissue grows into the device. To ensure proper placement and total occlusion of Essure a hysterosalpingogram is done 3 months after. Its success rate is similar to surgical sterilization (99.74%). For the first 3 months the woman needs to use a temporary contraceptive method in addition, till the scar tissue is formed.

Adiana is a combined procedure. Controlled thermal damage to the proximal tubal epithelium is done by radiofrequency energy. The procedure is done through a hysteroscope. A soft silicone palette (smaller than the grain of a rice) is implanted at the site to stimulate tissue growth for permanent blockage. Hysterosalpingography is done after 3 months for confirmation. Failure rate is about 1.1%.

Male contraception methods

Testosterone or a combination of testosterone and progestin (monthly injection or implant) is found to suppress sperm production. Testosterone undecanoate is used and found successful.

- **GnRH analogs** produce a decline in sperm density, sperm mobility and a decrease in testosterone level. The marked loss of libido makes it unacceptable. Add-back therapy (testosterone) is used to overcome the side effects.
- **Gossypol:** It has been discovered in China; an extract from cotton seed. It acts directly on the seminiferous tubules inhibiting spermatogenesis. The side effects are: fatigue, decreased libido and delayed recovery of sperm count. The serious side effects are hypokalemic paralysis and cardiac arrhythmias.

Intra Vas Device (IVD): Two plugs are implanted in each vas to block sperm transport through the vas deferens. Plugs could be removed to make it a reversible procedure. Its contraceptive effectiveness is being studied.

KEY POINTS

- **Barrier methods** of contraception include condom, diaphragm and vaginal contraceptives (chemicals and sponge today).
- **Natural contraception includes**—rhythm method, coitus interruptus and breastfeeding (p. 614).
- **Conventional contraceptive methods** include use of condom, vaginal diaphragm, spermicidals and rhythm method.
- **The introduction of IUD** is an OPD procedure without anesthesia taking full aseptic precautions. **"No touch" insertion** technique is preferred (p. 617).
- **The contraindications** of insertion of IUD are nulliparity, PID, suspected pregnancy, DUB or suspicious cervix. The pregnancy rate is about 0.5–2/HWY. There is risk of ectopic pregnancy 1–2%. Third generation IUDs have minimal side effects and lowest pregnancy rate (p. 620).

  Immediate complications (IUD) include pain, syncopal attack and uterine perforation. **Remote complications** include—pain, abnormal uterine bleeding, pelvic infection, spontaneous expulsion or even perforation of the uterus (p. 618).
- Indications of **removal of IUD are**—excessive uterine bleeding, flaring up of pelvic infection, uterine perforation, pregnancy, missing thread and patient desirous of a baby (p. 619).

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- Apart from contraception, the IUD is used as an interception and following synechiolysis.
- The **replacement time** for CuT 200 B is 4 years, Multiload 250—3 years, CuT 380A—10 years, Multiload 375—5 years and LNG–IUS is 5 years. Devices containing less than 300 mm^2 copper have higher failure rates.
- LNG-IUS has got many **health benefits** (see p. 620). It is a very safe and effective method for prolonged use.
- **Combined oral contraceptives** are very reliable apart from their many other health benefits (see p. 625).
- **Absolute contraindications** of oral pills (p. 623), major side effects (p. 625, 626) and indications of withdrawal of pills (p. 624) have been discussed.
- The **newer low dose pills** with more specific and "lipid friendly" progestins reduce the health risk further.
- A woman who does not smoke and has no other risk factor for cardiovascular disease, may continue the pill (with careful monitoring) until the age of 50 years.
- **Low-dose progestin pill (mini pill)** is advantageous in lactating women, as it has got no adverse effect on breast milk. It can be used as a suitable alternative where estrogen is contraindicated (p. 627).
- **Overall safety of DMPA** is clearly greater than COC. Norplant and Implanon are safe and effective for long-term use. Both are considered as "reversible sterilization" (p. 628).
- **Emergency contraception** includes hormones, IUD and antiprogesterone (RU-486). Within 72 hours, hormonal preparations are effective; within 5 days, IUD is effective and RU-486 should be taken within day 27 of cycle irrespective of the day and number of intercourse (p. 629).
- **Centchroman** in a nonsteroidal antiestrogenic compound used as once a week contraceptive pill. It acts by preventing the implantation of the fertilized ovum (p. 638).
- A man is not sterilized immediately after vasectomy. As such, additional condom should be advised for at least 3 months (p. 632).
- **No-scalpel vasectomy (NSV)** is done under local anesthetic making a tiny puncture over the stretched skin of the vas (p. 632). It has fewer complications. Both the NSV and scalpel vasectomy (SV) are safe.
- Globally, tubal sterilization is the most common method (20%) of contraception followed by IUDs (15%), oral contraceptives (8%) and condoms (5%).
- **Female sterilization** operation can be done during puerperium, in interval period or concurrent with MTP (p. 633). Hysteroscopic methods of sterilization include insertion of quinacrine pellet and Essure (microcoil), p. 640.
- Apart from conventional or mini-lap abdominal method, **laparoscopic sterilization** is very popular and effective (p. 635).
- Contraceptive prescription should be on an **individual basis**. In an individual, method may vary according to her phase of reproductive life. **Teenage girls, older women** and **sex workers** should also be protected (p. 637).
- It is hard to predict contraceptive trends in the immediate future as the results of contraceptive research are still unclear about the risks and benefits.

QUESTIONS

Write Short Notes on:

A. Lactational amenorrhea (p. 172, 614)
B. Advantages of barrier methods of contraception (p. 612-13)
C. Contraindications of IUD insertion (p. 618)
D. Non-contraceptive benefits of COCs (p. 625)
E. Different methods of emergency contraception (p. 629)
F. No-scalpel vasectomy (p. 632)
G. Merits and demerits of laparoscopic method of sterilization over minilap procedure (p. 634)

Related theory questions (Long & Short), Obstetric Case Discussions, Viva table discussions, Postoperative word round discussions, and MCQs are discussed in author's books:

1. **Bedside Clinic and Viva Voce:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

For further reading:

2. **Master pass in Obstetrics and Gynaecology:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

Chapter 37

Operative Obstetrics

Obstetric operations are surgical procedures and as such irrespective of the nature of operation (major or minor), asepsis and antiseptic precautions are to be taken as outlined in p. 157. Even an internal examination during late pregnancy and labor requires utmost asepsis. The proximity of the bladder and rectum to the operative field should deserve attention prior to any operative procedure.

Before proceeding to vaginal operative or manipulative obstetrics, some protocols are to be maintained. While a uniform guideline is difficult to formulate, **the following preliminaries are to be followed** with a few additions or alterations as and when required. **These are**:

1. **Anesthesia—**either general or local is used. In some cases, the operation may be performed with intravenous diazepam sedation.
2. **The patient is to be placed** in lithotomy position.
3. **Full surgical asepsis** is to be taken:
   (a) Surgical team is to wear sterile cap, mask, thorough hand wash and to wear gown and gloves
   (b) Vulva and vagina are to be swabbed with antiseptic solution
   (c) Cervix is cleaned with povidone-iodine solution
   (d) The perineum is to be draped by sterile towel and the legs with leggings.
4. **To empty the bladder—**If the patient is ambulant, she is asked to empty the bladder before she is placed on the table; otherwise catheterization is to be done.
5. **Vaginal examination** is done.

DILATATION AND EVACUATION (D&E)

The operation consists of dilatation of the cervix and evacuation of the products of conception from the uterine cavity. The operation may be performed:

- **One stage:** Dilatation of the cervix and evacuation of the uterus are done in the same sitting.
- **Two stages:** (a) First phase includes slow dilatation of the cervix
  (b) Second phase includes rapid dilatation of the cervix and evacuation.

ONE STAGE OPERATION

INDICATIONS: (1) Incomplete abortion (most common) (2) inevitable abortion (3) medical termination of pregnancy (6–8 weeks) and (4) hydatidiform mole in the process of expulsion.

PROCEDURES:

Preliminaries: The steps to be followed are those mentioned earlier. The patient is put under general anesthesia. Internal examination is done to note the size and position of the uterus and state of dilatation of the cervix.

Steps: **(Incomplete abortion – recent)**

(1) If the cervix is not sufficiently dilated to admit the index finger (usually it does), it should be dilated.

(2) Sim's posterior vaginal speculum is introduced and an assistant is asked to hold it. The anterior lip of the cervix is grasped by an Allis forceps to steady the cervix. Uterine sound is not to be introduced. Sounding provides no information but risks perforation and bleeding.

(3) The cervical canal is gradually dilated up to the desired extent by the graduated metal dilators (Fig. 42.12).

(4) The products are removed by ovum forceps. The uterine cavity is finally curetted gently by a flushing (blunt) curette. Injection methergine 0.2 mg is to be administered intravenously during the procedure.

(5) The speculum and the Allis forceps are to be removed. The uterus is to be massaged bimanually with the help of the external hand and the internal fingers, placed inside the vagina.

(6) After being satisfied that the uterus is firm and the bleeding is minimal, the vagina and perineum are toileted; a sterile vulval pad is placed and the patient is sent back to her bed.

Postabortion care includes: (a) emergency treatment of complications of any abortion spontaneous or induced (b) family planning counseling and referral services and (c) linkages to other reproductive health services (comprehensive services). Male partner should be involved.

TWO STAGE OPERATION

INDICATIONS: (1) Induction of first trimester abortion (most common) (2) missed abortion (uterus 8–10 weeks) and (3) hydatidiform mole with unfavorable cervix (long, firm and closed os). To prevent damage to the cervix during rapid dilatation, a two-stage operation is, however, preferred in such cases.

PROCEDURES

(A) First phase: It consists of introduction of laminaria tents or lamicel ($MgSO_4$ sponge) into the cervical canal to effect its slow dilatation. The same may be effective by intravaginal insertion of misoprostol (PGE_1), 400 µg 3 hours before surgery. It has less side effects.

Steps of introduction of tents: The preliminaries to be followed are those mentioned earlier (p. 617). (a) The patient should empty her bladder beforehand (b) no anesthesia is required and (c) the appropriate size and number of the tent required are selected. The threads attached to one end are tied to the roller gauze.

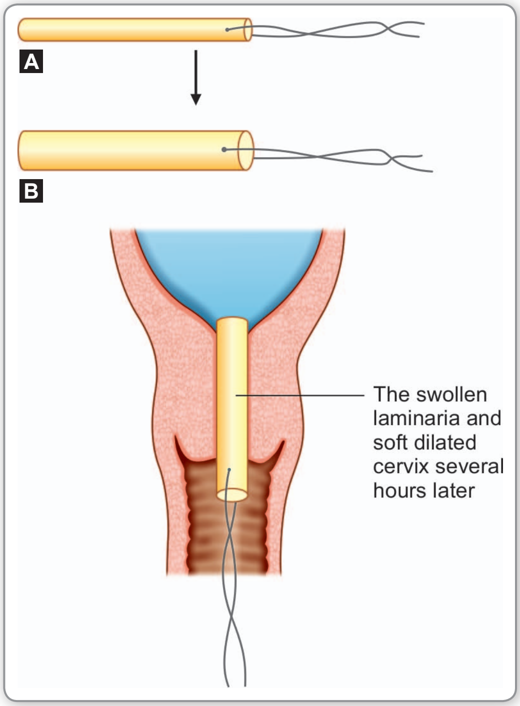
Steps:

(1) Internal examination is done to note the size and position of the uterus and state of the cervix.

(2) Sim's posterior vaginal speculum is introduced and an assistant is asked to hold it. The anterior lip of the cervix is grasped by an Allis forceps to steady the cervix.

(3) The cervical canal may have to be dilated, especially in primigravidae by one or two smaller metal dilators (Hawkin Ambler: size 3/6 or 4/7) to facilitate the introduction of the tents.

(4) The tents are introduced one after the other, holding it by tent introducing forceps (Figs 37.1A and B). The


A
B
The swollen laminaria and soft dilated cervix several hours later

Figs 37.1A and B: Laminaria tent (A) Prior to introduction and (B) Marked swelling due to hygroscopic action while kept in cervical canal tents should be introduced for at least 4 cm (1.5"), so that the tips are placed beyond the internal os. The tents can also be introduced manually.

(5) The roller gauze is used to pack the upper vagina so as to prevent the displacement of the tents.

(6) The patient is returned to her bed.

(7) Prophylactic antibiotic (doxycycline 100 mg PO BID for 3 days and metronidazole PO 400 mg BID for 5 days) is usually administered.

(B) Second phase: It consists of further dilatation of the cervix by graduated metal dilators followed by evacuation of the uterus.

Procedures

- The patient is brought back to the operation theater usually after 12 hours. The patient should empty her bladder beforehand.
- **Preliminaries:** The steps to be followed are those previously mentioned. The operation may be conducted under intravenous diazepam sedation, local paracervical block or under general anesthesia.

Steps: (MTP – 8 weeks)

(1) The posterior vaginal speculum is introduced after removing the roller gauze. The tents are removed with the help of sponge forceps. The vagina and the cervix are swabbed with antiseptic (povidone-iodine) solution. The posterior vaginal speculum is removed.

(2) Vaginal examination is done to note the size of the uterus, position of the uterus and state of dilatation of the cervix.

(3) Posterior vaginal speculum is reintroduced and is to be held by an assistant. The anterior lip of the cervix is to be grasped by the Allis forceps to steady the cervix.

(4) The cervix is dilated with the graduated metal dilators up to the desired extent (10/13 to 12/15) to facilitate introduction of the ovum forceps.

(5) The products are removed by introducing the ovum forceps. Intravenous methergine 0.2 mg is to be given during this stage to minimize blood loss. Firm and well contracted uterus facilitates curettage (Fig. 37.2).

(6) The uterine cavity is thoroughly curetted by a flushing curette.

(7) The posterior vaginal speculum and the Allis forceps are removed. The uterus is massaged bimanually and after being satisfied **that the uterus is empty (evidenced by a well contracted uterus with minimal bleeding),** the patient is sent to her bed after placing a sterile vulval pad.

(8) **Oxytocic agents:** Injection methergine 0.2 mg IM is given. Alternatively oxytocin 20 units in 500 mL of normal saline IV is given intraoperatively and continued after the operation for 30 minutes.

(9) Prophylactic antibiotics (doxycycline and metronidazole) are prescribed.

DANGERS OF D&E OPERATION

Immediate: (1) **Excessive hemorrhage**—may be due to (a) incomplete evacuation or (b) atonic uterus. (2) **injury—**(a) cervical lacerations of varying degree which may lead to formation of a broad ligament hematoma and (b) uterine perforation. (3) **Shock due to:** (a) local anesthesia—convulsions, cardiorespiratory arrest, death due to intravascular injection or over dose. (b) Excessive blood loss. (c) Cervical shock—vasovagal syncope due to cervical stimulation. (4) **Perforation**—injury to major blood vessels, bowel or bladder. Risk is more with advanced gestation. (5) **Sepsis**—endometritis, myometritis and pelvic peritonitis. (6) **Hematometra** may cause pain. (7) **Increased morbidity** and (8) Continuation of pregnancy (failure) – 1%.

Late: (1) Pelvic inflammation (2) infertility (3) cervical incompetence (4) uterine synechiae and in subsequent pregnancy risks are: (5) preterm labor and (6) ectopic pregnancy.

MANAGEMENT PROTOCOL OF UTERINE PERFORATION

The management depends on the location, size and nature of the instrument causing the perforation.

- **The procedure is stopped**.
- **Perforation made by small instruments** such as sound or smaller size dilator – Expectant treatment with observation of pulse and blood pressure. Antibiotic is to be given.
- **Perforation caused by bigger size dilator or ovum or ring forceps or suction cannula:** Diagnostic laparoscopy is helpful to assess the size and site of perforation and the amount of hemorrhage. Operative laparoscopy or laparotomy may be needed to tackle the situation. **One should not forget to inspect the intestine or omentum for evidence of injury.**
- **Lateral cervical tear with broad ligament hematoma or laceration of uterine artery:** Laparotomy followed by repair (conservative surgery) or hysterectomy.
- **Perforation prior to complete evacuation:** Any of the following may be followed—(a) *to stop evacuation,* vaginal evacuation can be done under laparoscopic visualization; (b) *if laparotomy is decided*: (i) complete the evacuation either through the rent or anterior hysterotomy, if preservation of the uterus is necessary and (ii) hysterectomy, if family is completed.

Along with the definitive surgery, simultaneous resuscitative procedure and administration of antibiotics are mandatory.

SUCTION EVACUATION

It is a procedure in which the products of conception are sucked out from the uterus with the help of a cannula fitted to a suction apparatus.

INDICATIONS: (1) Medical termination of pregnancy during first trimester (most common) (2) inevitable abortion (3) recent incomplete abortion and (4) hydatidiform mole.

PROCEDURES: Preliminaries: the steps to be followed are those mentioned in p. 617. General anesthesia is usually not needed. If the patient is apprehensive, intravenous diazepam 5–10 mg (conscious sedation) supplemented by paracervical block is quite effective. The patient is put on the table after she empties her bladder.

Steps (Figure 37.2)

(1) Vaginal examination is done to note the size and position of the uterus and also the state of cervix. *USG (TAS/TVS) should be performed when there is any doubt about the gestational age.*

(2) Posterior vaginal speculum (Fig. 42.4) is introduced and an assistant is asked to hold it.

(3) The anterior lip of the cervix is to be grasped by an Allis forceps. A uterine sound is to be introduced to note the length of the uterine cavity and position of the uterus.

(4) The cervix may have to be dilated with smaller size graduated metal dilators up to one size less than that of the suction cannula. Feeling of “snap” of the endocervix around the dilator is characteristic. Instead laminaria tent 12 hours before (osmotic dilator) or misoprostol (PGE_1) 400 µg given vaginally 3 hours prior to surgery produces effective dilatation.

(5) Intravenous methergine 0.2 mg is administered.

(6) The appropriate suction cannula (Fig. 42.21) is fitted to the suction apparatus by a thick rubber or plastic tubing. The cannula is then introduced into the uterus, the tip is to be placed in the middle of the uterine cavity.

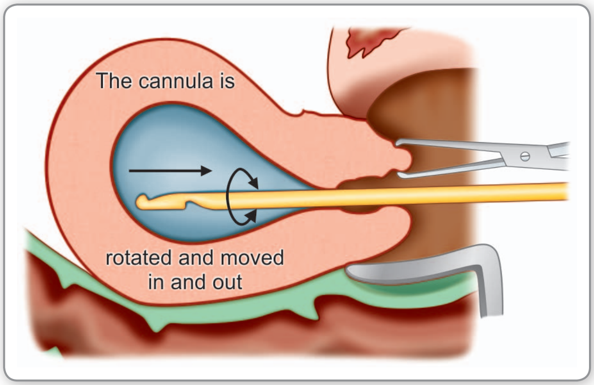


Fig. 37.2: Suction evacuation

(7) **The pressure of the suction is raised to 400–600 mm Hg**. The cannula is moved up and down and rotated within the uterine cavity (360°) with the pressure on. The suction bottle is inspected for the products of conception and blood loss. The suction is regulated by a finger placed over a hole at the base of the cannula.

The endpoint of suction is denoted by: (a) No more material is being sucked out (b) gripping of the cannula by the contracting smaller size uterus (c) grating sensation and (d) appearance of bubbles in the cannula or in the transparent tubing.

(8) **The vacuum should be broken before withdrawing the cannula** down through the cervical canal to prevent injury to the internal os.

(9) It is better to curette the uterine cavity by a small flushing curette at the end of suction and the cannula is reintroduced to suck out any remnants.

(10) After being satisfied that the uterus is remaining firm, and there is minimal vaginal bleeding, the patient is brought down from the table after placing a sterile vulval pad.

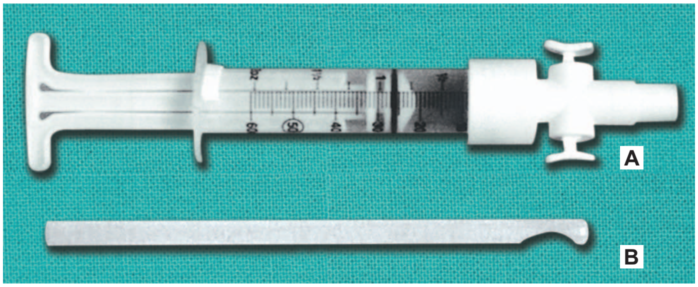
Use of USG during the procedure shortens the operative time and reduces complications.

COMPLICATIONS: Similar complications as mentioned in D&E operation may occur. Use of a plastic cannula can minimize uterine perforation. Blood loss and incomplete evacuation are less likely with pregnancy of 8 weeks or less.

MENSTRUAL REGULATION *(Syn: Induction, Aspiration)*

It is the aspiration of the endometrial cavity within 14 days of missed period in a woman with previous normal cycle (Figs 37.3A and B).

The operation is done as an outpatient or an office procedure (p. 204). It is done with aseptic precautions (p. 642) and in apprehensive patients, sedation or paracervical block anesthesia may be employed. After introducing the posterior vaginal speculum, the cervix is steadied with an Allis forceps. Cervix may be gently dilated using 4 mm or 5 mm size dilators. 5–6 mm suction cannula (Karman's) is then inserted and attached to the 50 mL syringe for suction. **The cannula is rotated, pushed in and out with gentle strokes**.



Figs 37.3A and B: Menstrual regulation equipment — (A) Syringe (B) Plastic cannula with whistle tip used in suction evacuation

The operator should examine the aspirated tissue by floating it in a clear plastic dish over a light source. Placental tissue appears fluffy and feathery when floats in normal saline. **This will help to detect failed abortion, molar pregnancy or ectopic pregnancy. The procedure is contraindicated** in advanced pregnancy and in the presence of local pelvic inflammation. There is risk of continuation of pregnancy (0.5–2%) and ectopic pregnancy. When no chorionic villi are found on tissue examination, ectopic pregnancy should be excluded by estimation of hCG levels and vaginal ultrasonography.

VACUUM ASPIRATION

This procedure is similar to menstrual regulation and is done as outpatient basis (p. 204). The procedure may be **manual vacuum aspiration (MVA)** or **electric vacuum aspiration (EVA)** and is highly effective (98–100%). **Termination of pregnancy is done up to 12 weeks** with minimal cervical dilatation (Fig. 42.20). A hand operated double valve plastic syringe (60 mL) is attached to a Karman's cannula (up to 12 mm size). The cannula is inserted transcervically into the uterus and the vacuum is activated. A negative pressure of 660 mm Hg is created. Aspiration of the products of conception is done. This procedure takes less time (5–15 mins) and is less traumatic. Complications are similar to other surgical methods (p. 644) but are less severe.

HYSTEROTOMY

Hysterotomy is an operative procedure of extracting the products of conception out of the womb before viability (28th week) by cutting through the anterior wall of the uterus. The operation is usually done through the abdominal route. The operation is rarely done these days for the purpose of MTP.

INDICATIONS: (i) Midtrimester MTP where other methods have failed or are contraindicated (ii) fibroids in the lower uterine segment obstructing evacuation (iii) completely low lying placenta (placenta previa) (iv) uterine anomalies (uterine didelphys, septate uterus) (v) cervical cancer with pregnancy and (vi) women with multiple previous cesarean delivery (due to the risk of placenta accreta).

STEPS (ABDOMINAL HYSTEROTOMY): The preparation is similar to that of any other major surgical operation. The anesthesia is either general or epidural. The abdomen is opened either through a low transverse or infraumbilical vertical incision above the symphysis pubis sufficiently large enough to take the uterus out of the abdomen.

Step I: The uterus is drawn out of the incision. The abdominal cavity **and the abdominal wall are to be well packed to prevent contamination by the products of conception (to minimize scar endometriosis)**. If there is difficulty in delivering the uterus out of the abdomen, it can be done with a finger hooked through the uterine incision.

Step II: Methergine 0.2 mg is given intravenously. The loose peritoneum of the uterovesical pouch is cut transversely and pushed up and down (Fig. 37.4). The myometrium is cut vertically for about 5 cm (2") deep enough to make the membranes visible. Alternatively, the uterine incision may be vertical in the middle of the body of the uterus as low down as possible.

Step III: The products of conception are gently coaxed out; the cavity is cleaned with a gauze covered finger.

Step IV: The uterine incision is closed in three layers: (a) Deeper myometrium excluding the decidua (difficult to exclude decidua) is apposed by continuous sutures using No. “0” catgut and round bodied needle; (b) similar second layer of continuous suture is employed taking the entire thickness of the muscle down to the first layer of suture and (c) the peritoneum is apposed transversely using continuous suture.

Step V: Packs are removed; peritoneal toileting is done; another dose of methergine 0.2 mg is administered intramuscularly and the abdominal wall is closed in layers.

COMPLICATIONS: ♦ **Immediate** ♦ **Remote**

- ***Immediate:*** (1) Uterine bleeding (2) peritonitis (3) intestinal obstruction and (4) anesthetic hazards. All these lead to increased morbidity and an occasional death.
- ***Remote:*** (1) Menstrual abnormality—menorrhagia or irregular periods (2) scar endometriosis (1%) (3) scar rupture in subsequent pregnancy. While concurrent sterilization eliminates the hazards, but those left exposed to future pregnancy become a growing concern.

EPISIOTOMY

DEFINITION: A surgically planned incision on the perineum and the posterior vaginal wall during the second stage of labor is called episiotomy (perineotomy). It is in fact an inflicted second-degree perineal injury. It is the most common obstetric operation performed.

OBJECTIVES

- **To enlarge** the vaginal introitus so as to facilitate easy and safe delivery of the fetus: spontaneous or manipulative.
- **To minimize** overstretching and rupture of the perineal muscles and fascia; to reduce the stress and strain on the fetal head.

INDICATIONS: Episiotomy is recommended **in selective cases rather than as a routine**. A constant care during the second stage reduces the incidence of episiotomy and perineal trauma.

- **In elastic (rigid) perineum:** Causing arrest or delay in descent of the presenting part as in elderly primigravidae.

- **Anticipating perineal tear:** (a) Big baby (b) face to pubis delivery (c) breech delivery and (d) shoulder dystocia.
- **Operative delivery:** Forceps delivery, ventouse delivery.
- **Previous perineal surgery:** Pelvic floor repair, perineal reconstructive surgery.

Common indications are: (1) Threatened perineal injury in primigravidae (2) rigid perineum and (3) forceps, breech, occipitoposterior or face delivery.

Timing of the episiotomy: The timing of performing the episiotomy requires judgment. If done early, the blood loss will be more. If done late, it fails to prevent the invisible lacerations of the perineal body and thereby fails to protect the pelvic floor – the very purpose of the episiotomy is thus defeated. **Bulging thinned perineum during contraction just prior to crowning (when 3–4 cm of head is visible) is the ideal time**. During forceps delivery, it is made after the application of blades.

ADVANTAGES

- **Maternal:** It is controversial whether routine episiotomy has got any major benefits. The suggested benefits are: (a) a clear and controlled incision is easy to repair and heals better than a lacerated wound that might occur otherwise (b) reduction in the duration of second stage and (c) reduction of trauma to the pelvic floor muscles—that reduces the incidence of prolapse and perhaps urinary incontinence.
- **Fetal:** It minimizes intracranial injuries, especially in premature babies or after-coming head of breech.

TYPES

The following are the various types of episiotomy (Fig. 37.4):

- **Mediolateral**
- **Median**
- **Lateral**
- **'J' shaped**

Mediolateral: The incision is made downwards and outwards from the midpoint of the fourchette either to the right or to the left. It is directed diagonally in a straight line which runs about 2.5 cm away from the anus (midpoint between anus and ischial tuberosity).

Median: The incision commences from the center of the fourchette and extends posteriorly along the midline for about 2.5 cm (Table 37.1).

Lateral: The incision starts from about 1 cm away from the center of the fourchette and extends laterally. It has got many drawbacks including chance of injury to the Bartholin's duct. It is totally condemned.

'J' shaped: The incision begins in the center of the fourchette and is directed posteriorly along the midline for about 1.5 cm and then directed downwards and outwards along 5 or 7 O'clock position to avoid the anal sphincter. Apposition is not perfect and the repaired wound tends to be puckered. This is also not done widely.

Thus, only mediolateral or median episiotomy is done commonly and as such their relative merits and demerits are given in the tabulated form (Table 37.1).

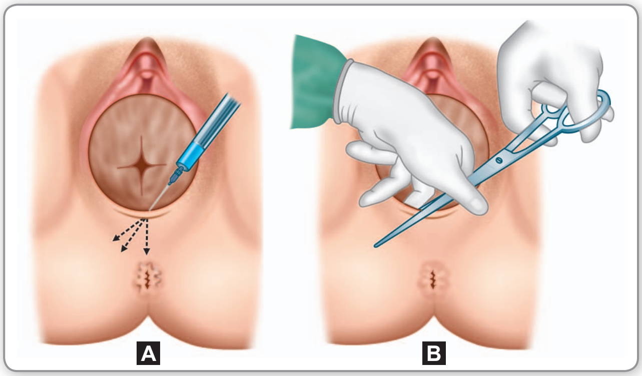
Table 37.1: Relative Merits and Demerits of Median and Mediolateral Episiotomy

	Median	Mediolateral
Merits	▪ The muscles are not cut ▪ Blood loss is least ▪ Repair is easy ▪ Postoperative comfort is maximum ▪ Healing is superior ▪ Wound disruption is rare ▪ Dyspareunia is rare	▪ Relative safety from rectal involvement from extension ▪ If necessary, the incision can be extended
Demerits	▪ Extension, if occurs, may involve the rectum ▪ Not suitable for manipulative delivery or in abnormal presentation or position. As such, its use is selective	▪ Apposition of the tissues is not so good ▪ Blood loss is little more ▪ Postoperative discomfort is more ▪ Relative increased incidence of wound disruption ▪ Dyspareunia is comparatively more

STEPS OF MEDIOLATERAL EPISIOTOMY

STEP I: ***Preliminaries***—The perineum is thoroughly swabbed with antiseptic (povidone-iodine) lotion and draped properly. Local anesthesia: The perineum, in the line of proposed incision is infiltrated with 10 mL of 1% solution of lignocaine.

STEP II: ***Incision***—Two fingers are placed in the vagina between the presenting part and the posterior vaginal wall. The incision is made by a curved or straight blunt pointed sharp scissors (scalpel may also be used), one blade of which is placed inside, in between the fingers and the posterior vaginal wall and the other on the skin. **The incision should be made at the height of an uterine contraction** when an accurate idea of the extent of incision can be better judged from the stretched perineum. Deliberate cut should be made starting from the center of the fourchette extending laterally either to the right or to the left. It is directed diagonally in a straight line which runs about 2.5 cm away from the anus. **The incision ought to be adequate to serve the purpose for which it is needed,** i.e. according to the need of the individual case. The bleeding is usually not sufficient to use artery forceps unless the operation is done too early or the perineum is thick (Figs 37.4A and B).



Figs 37.4A and B: Steps of mediolateral episiotomy—(A) Perineal infiltration; (B) Cutting the perineum

Structures cut are (Fig. 37.5): (1) Posterior vaginal wall (2) superficial and deep transverse perineal muscles, bulbospongiosus and part of levator ani (3) fascia covering those muscles (4) transverse perineal branches of pudendal vessels and nerves (5) subcutaneous tissue and skin.

STEP III: ***Repair***

Timing of repair: the repair is done soon after expulsion of placenta. If repair is done prior to that, disruption of the wound is inevitable, if subsequent manual removal or exploration of the genital tract is needed. Oozing during this period should be controlled by pressure with a sterile gauze swab and bleeding by the artery forceps. Early repair prevents sepsis and eliminates the patient's prolonged apprehension of "stitches".

Preliminaries: The patient is placed in lithotomy position. A good light source from behind is needed. The perineum including the wound area is cleansed with antiseptic solution. Blood clots are removed from the vagina

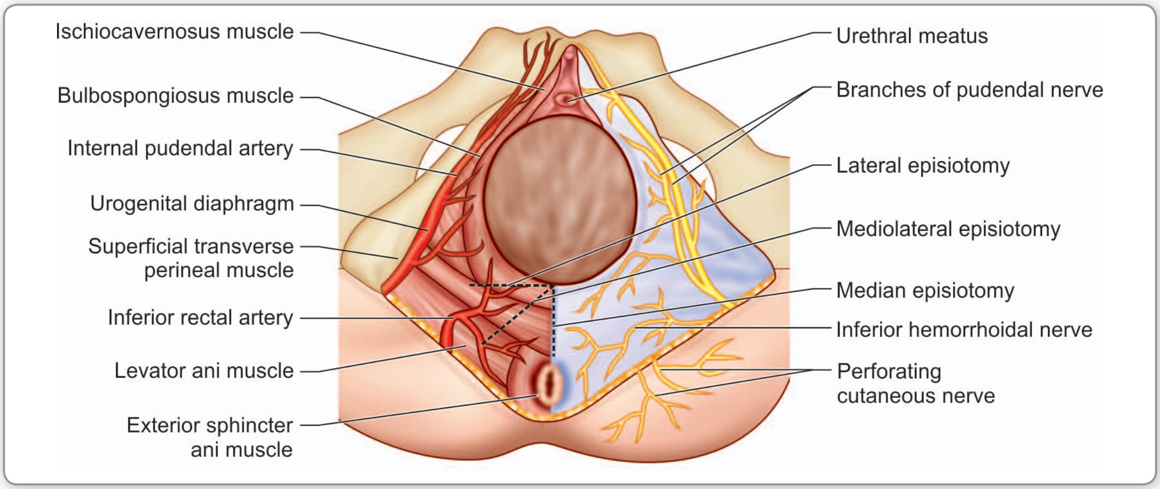
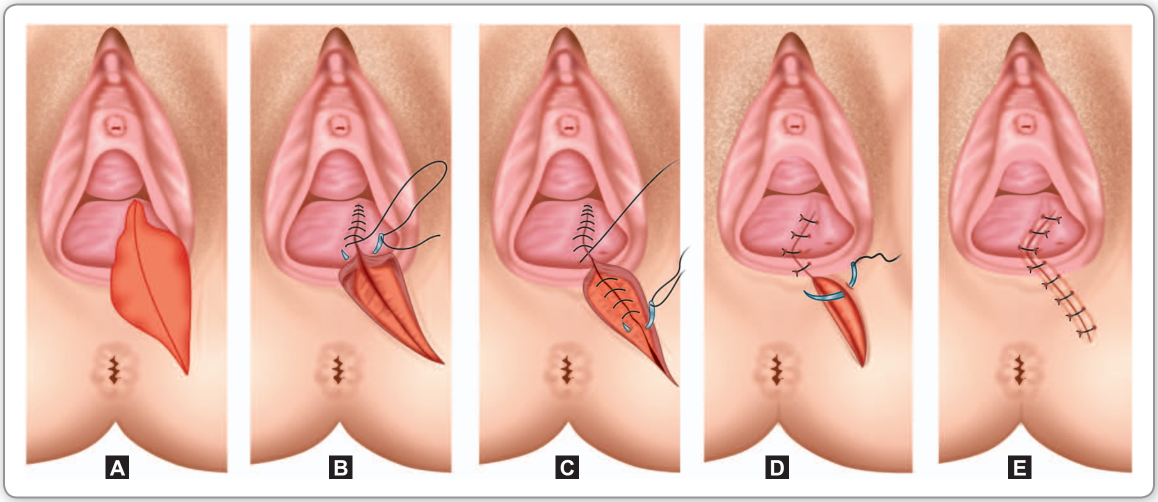

Ischiocavernosus muscle
Bulbospongiosus muscle
Internal pudendal artery
Urogenital diaphragm
Superficial transverse perineal muscle
Inferior rectal artery
Levator ani muscle
Exterior sphincter ani muscle
Urethral meatus
Branches of pudendal nerve
Lateral episiotomy
Mediolateral episiotomy
Median episiotomy
Inferior hemorrhoidal nerve
Perforating cutaneous nerve

Fig. 37.5: Diagrammatic representation of the structures to be cut in different types of episiotomy



Figs 37.6A to E: Steps of repair of episiotomy—(A) Wound on inspection; (B) Repair of vaginal mucosa; (C) Reapir of perineal muscles by interrupted sutures; (D) Apposition of the skin margins; (E) Repaired wound on inspection

and the wound area. The patient is draped properly and repair should be done under strict aseptic precautions. If the repair field is obscured by oozing of blood from above, a vaginal pack may be inserted and is placed high up. **Do not forget to remove the pack after the repair is completed**.

Repair (Figs 37.6A to D): **The repair is done in three layers**. **The principles to be followed are:** (1) perfect hemostasis (2) to obliterate the dead space and (3) suture without tension.

The repair is to be done in the following order:

(1) Vaginal mucosa and submucosal tissues (2) perineal muscles and (3) skin and subcutaneous tissues.

Preliminaries had been discussed in p. 642.

The vaginal mucosa is sutured first. The first suture is placed **at or just above the apex of the tear**. Thereafter, the vaginal walls are apposed by interrupted sutures with polyglycolic acid suture (Dexon) or No. “0” chromic catgut, from above downwards till the fourchette is reached. The suture should include the deep tissues to obliterate the dead space. A continuous suture may cause puckering and shortening of the posterior vaginal wall. Care should be taken not to injure the rectum. Rest of the procedure is discussed before (see “repair of incomplete perineal tear” in p. 490).

POSTOPERATIVE CARE

Dressing: The wound is to be dressed each time following urination and defecation to keep the area clean and dry. The dressing is done by swabbing with cotton swabs soaked in antiseptic solution (povidone-iodine) followed by application of antiseptic powder or ointment (furacin or neosporin).

Comfort: To relieve pain in the area, $MgSO_4$ compression or application of infrared heat may be used. Ice packs reduce swelling and pain also. Analgesic drugs (ibuprofen) may be given when required.

Ambulance: The patient is allowed to move out of the bed after 24 hours. Prior to that, she is allowed to roll over on to her side or even to sit but only with thighs apposed.

Removal of stitches: When the wound is sutured by catgut or Dexon which will be absorbed, the sutures need not be removed. But if nonabsorbable material like silk or nylon is used, **the stitches are to be cut on 6th day**. The number of stitches removed should be checked with the record of the stitches given.

COMPLICATIONS OF EPISIOTOMY

- **Immediate**
- **Remote**

Immediate: (1) **Extension of the incision** to involve the rectum. This is likely in median episiotomy or during delivery of undiagnosed occipitoposterior even with small mediolateral episiotomy (2) **vulval hematoma** (3) **infection:** the clinical features are—(a) throbbing pain on the perineum (b) rise in temperature (c) the wound area looks moist, red and swollen and (d) offensive discharge comes out through the wound margins.

Treatment: (a) To facilitate drainage of pus by cutting one or two stitches (b) local dressing with antiseptic powder or ointment (c) $MgSO_4$ compression or application of infrared heat to the area to reduce edema and pain (d) systemic antibiotic (IV).

(4) **Wound dehiscence** is often due to infection, hematoma formation or faulty repair. The wound should be dressed daily until the local infection subsides and healthy granulation tissue forms in the margins. Secondary, sutures are given under local anesthesia using cutting needle and nylon. The margins are to be saucerized and debridement of all necrotic tissues should be done. This is followed by through-and-through sutures taking tissues right at the bottom of the wound. Usual postoperative dressing is to be given. Systemic (IV) antibiotic is prescribed.

(5) Injury to anal sphincter causing incontinence of flatus or feces. (6) Rectovaginal fistula and rarely. (7) Necrotizing fasciitis (rare) in a woman who is diabetic or immunocompromised.

Remote: (1) **Dyspareunia—**This is due to a narrow vaginal introitus which may result from faulty technique of repair or due to painful perineal scar, (2) **chance of perineal lacerations** in subsequent labor, if not managed properly and (3) **scar endometriosis (rare)**.

OPERATIVE VAGINAL DELIVERY

Operative vaginal delivery refers to any delivery process which is assisted by vaginal operations. Delivery by forceps, ventouse and destructive operations are generally included. Obstetric maneuvers (shoulder dystocia—p. 469) are described under **assisted vaginal delivery**.

FORCEPS

Obstetric forceps is a pair of instruments, especially designed to assist extraction of the fetal head and thereby accomplishing delivery of the fetus.

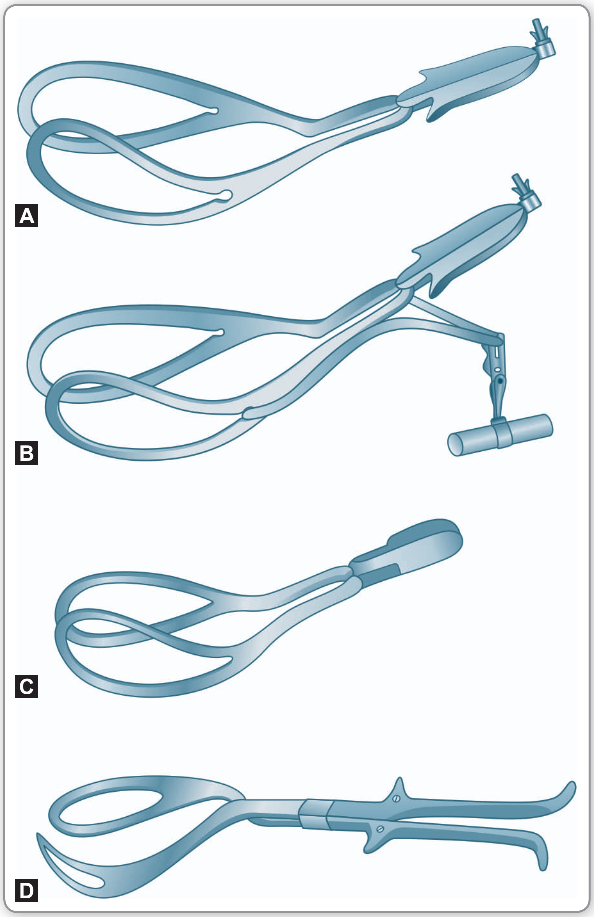
VARIETIES OF OBSTETRIC FORCEPS: Ever since either Peter I or Peter II of the Chamberlen family invented the forceps around AD 1600, more than 700 varieties were invented or modified. Most of them are of historical interest only. But only three varieties are commonly used in present day obstetric practice (Figs 37.7A to D). These are:

- **Long-curved forceps with or without axis-traction device**
- **Short-curved forceps**
- **Kielland's forceps**

The basic construction of these forceps is the same in that each consists of two halves (blades) articulated by a lock.

LONG-CURVED OBSTETRIC FORCEPS

Long-curved obstetric forceps is relatively heavy and is about 37 cm (15") long. **In India, Das's variety (named after Sir Kedar Nath Das) is commonly used with advantages**. It is comparatively lighter and slightly shorter than its Western counterpart but is quite suited for the comparatively small pelvis and small baby of Indian women.



Figs 37.7A to D: Different types of obstetric forceps currently used—(A) Long-curved with axis traction device; (B) The same with attached axis traction device; (C) Wrigley's; (D) Kielland's

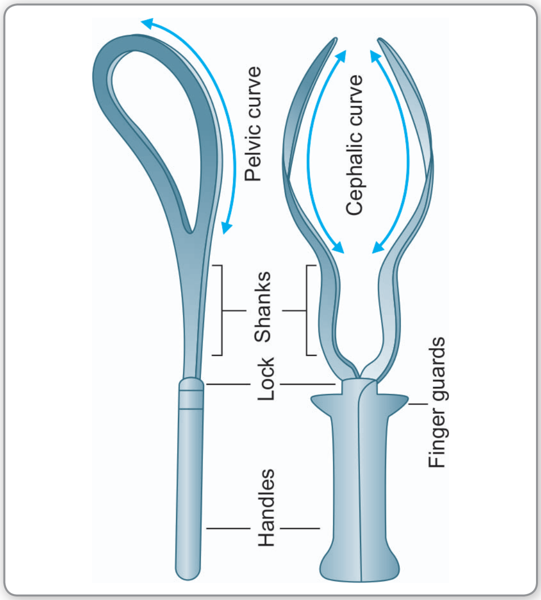


Fig. 37.8: Different parts of a long-curved obstetric forceps

Measurements: Length is 37 cm; distance in between the tips is 2.5 cm and widest diameter between the blades is 9 cm.

BLADES: There are two blades and **are named right or left in relation to maternal pelvis in which they lie when applied. Each blade consists of the following parts:** (1) Blade (2) shank (3) lock and (4) handle with or without screw.

Blade: The blade is fenestrated to facilitate a good grip of the fetal head. There is usually a slot in the lower part of the fenestrum of the blades to allow the upper end of the axis-traction rod to be fitted.

The **toe of the blade** refers to the tip and the **heel** to the end of the blade that is attached to the shank.

The blade has got two curves (Fig. 37.8):

- **Pelvic curve:** The curve on the edge is to fit more or less the curve on the axis of the birth canal **(curve of Carus)**. It forms a part of a circle whose radius is 17.5 cm (7"). **The front of the forceps is the concave side of the pelvic curve**. Pelvic curve permits ease of application along the maternal pelvic axis.
- **Cephalic curve:** It is the curve on the flat surface which when articulated grasps the fetal head without compression. The radius of the curve is 11.5 cm (4.5").

Shank: It is the part between the blade and the lock and usually measures 6.25 cm (2.5"). It increases the length of the instrument and thereby, facilitates locking of the blades outside the vulva. When the blades are articulated, the shanks are not apposed together.

Lock: The common method of articulation consists of a socket system located on the shank at its junction with the handle (English lock). Such type of lock requires introduction of the left blade first.

Handle: The handles are apposed when the blades are articulated. It measures 12.5 cm (5"). There is a finger guard on which a finger can be placed during traction.

A screw is attached usually at the end (or at the base) of one blade (commonly left). It helps to keep the blades in position.

AXIS-TRACTION DEVICE: It can be applied with advantage in midforceps operation, especially following manual rotation of the head. It provides traction in the correct axis of the pelvic curve and as such, less force is necessary to deliver the head. It consists of: (1) Traction rods (two—right and left) (2) Traction handle (Fig. 42.25).

Identification of the traction rods—right or left? Hold the knob pointing inwards and let the rod hang. The small transverse bar at the bottom is to be directed forward. The groove attached to the bar is pointing to the side (in relation to the maternal pelvis) to which the traction rod belongs and accordingly the same is attached to the corresponding blade of the forceps.

HOW TO IDENTIFY THE BLADES?

When articulated: Place the instrument in front of the pelvis with the tip of the blades pointing upwards and the concave side of the pelvic curve forward. **The blade which corresponds to the left of the maternal pelvis is the left blade and that to the right side is the right blade**.

When isolated: (1) The tip should point upwards (2) the cephalic curve is to be directed inwards and the pelvic curve forwards.

Short-Curved Obstetric Forceps (Wrigley)

The instrument is lighter, about a third of the weight of an ordinary long-curved forceps. **The instrument is short which is due to reduction in the length of the shanks and handles (Fig. 37.7C)**. It has a marked cephalic curve with a slight pelvic curve.

Kielland's Forceps

It is a long almost straight (very slight pelvic curve) obstetric forceps without any axis-traction device. It has got a sliding lock which facilitates correction of asynclitism of the head. One small knob on each blade is directed towards the occiput.

CHOICE OF FORCEPS OPERATION (Table 37.2)

Outlet forceps: It is a variety of low forceps where the head is on the perineum (Table 37.2). Thus, all outlet forceps are low forceps but not all low forceps are outlet forceps operations.

Low forceps (90%): The head is near the pelvic floor or even visible at the introitus. It is commonly used nowadays with advantages.

Midforceps (10%): Prerequisites are: (i) Must be associated with less maternal morbidity than Cesarean section (ii) should not cause any fetal damage. Unless the prospect of successful vaginal delivery is high midforceps delivery is best avoided. Manual rotation may be needed before traction. In a selective case, delivery by rotational forceps by an expert is safe. Otherwise, it is better to wait for the head descent and complete rotation. An oxytocin drip may be helpful if not contraindicated. Ventouse may be an alternative.

Table 37.2: Classification for Operative Vaginal (Forceps/ Ventouse) Delivery (ACOG–2000)

Types of Procedure	Criteria
Outlet	■ Scalp is visible at the introitus without separating the labia ■ Fetal skull has reached the level of the pelvic floor ■ Sagittal suture is in direct anteroposterior diameter or in the right or left occiput anterior or posterior position ■ Fetal head is at or on the perineum (Fig. 37.9) ■ Rotation is < 45°
Low	Leading point of the fetal skull (station) is at +2 cm or more but has not yet reached the pelvic floor. (a) Rotation is ≤ 45° (b) Rotation is > 45°
Mid	Fetal head is engaged. Head is 1/5 palpable per abdomen but station is above +2 cm but not above the ischial spines (Figs 12.21 and 37.10A to C)
High	Head is not engaged. This type is not included in classification

TYPES OF APPLICATION OF FORCEPS BLADES

Cephalic application: The blades are applied along the sides of the head grasping the biparietal diameter in between the widest part of the blades. The long axis of the blades corresponds more or less to the occipitomental plane of the fetal head. It is the ideal method of application as it has got a negligible compression effect on the cranium.

Pelvic application: When the blades of the forceps are applied on the lateral pelvic walls ignoring the position of the head, it is called pelvic application. **If the head remains unrotated, this type of application puts serious compression effect on the cranium and thus must be avoided**.

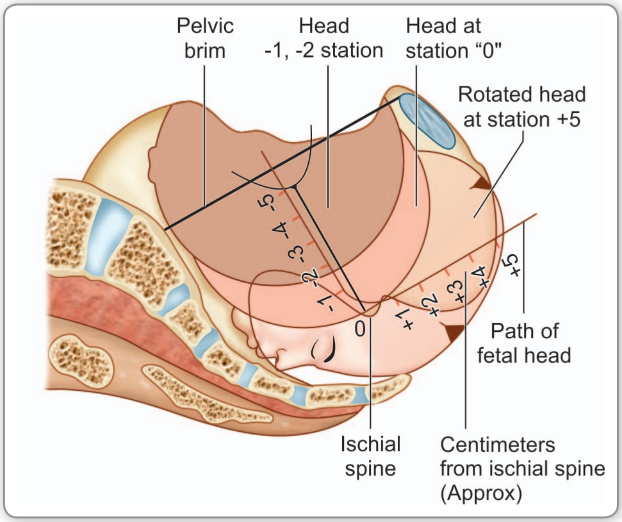


Fig. 37.9: Different types of forceps operations (ACOG—2000)

Functions of Forceps

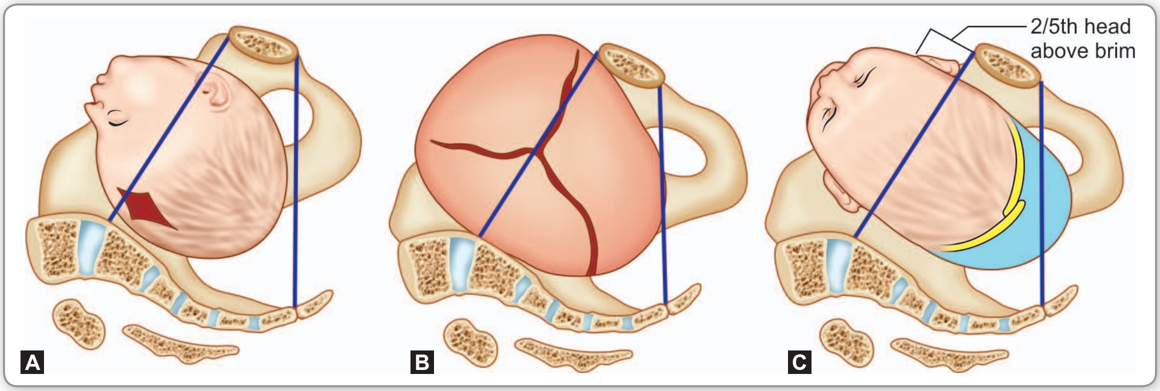
- **Traction force:** In primigravidae, the traction force required is about 20 kg and that in multiparae about 13 kg.
- **Rotation of the head:** Can be achieved by Kielland's forceps.
- **Acts as a protective cage** for the head from the pressure of the birth canal as in a premature baby.
- **Controlled delivery of the after-coming head in** breech to lessen the dangers of sudden decompression.
- **One forceps blade may be used as a vectis** to deliver head in cesarean section.
- **The compression effect** of forceps, on the cranium should be minimal when correctly applied over the biparietal, bimalar placement, and should not be more than required to grasp the fetal head.

Indications for Operative Vaginal Delivery (Forceps/Ventouse)

Maternal	Fetal	Others
▪ Inadequate expulsive efforts ▪ Maternal exhaustion (distress) ▪ Where expulsive efforts (Valsalva) are to be avoided (e.g. cardiac disease, hypertensive crises, cerebrovascular diseases, spinal cord injury)	▪ Nonreassuring fetal heart rate—fetal distress (e.g. low-birth-weight baby, postmaturity) ▪ After-coming head of breech ▪ Suspicion of fetal compromise	▪ Prolonged second stage of labor (nullipara >2 hour; multipara >1 hour) ▪ To cut short the second stage of labor as in severe preeclampsia, cardiac disease, postcesarean pregnancy

Prerequisites for Operative Vaginal Delivery (Forceps or Vacuum Application) (SOGC 2004, RANCZOG 2002)

Fetal and Maternal Criteria		Others
▪ Fetal head engaged (head ≤ 1/5 palpable per abdomen) ▪ The cervix must be fully dilated ▪ The membranes must be ruptured ▪ Fetal head position is exactly known ▪ Pelvis deemed adequate (Table 37.2)	▪ Bladder must be emptied ▪ Adequate maternal analgesia (regional block for mid cavity or pudendal block) ▪ Informed consent (verbal or written) with prior clear explanation	▪ Experienced operator ▪ Aseptic techniques ▪ Back up plan and facilities in case of failure ▪ Presence of a neonatologist ▪ Willingness to abandon the procedure when difficulties faced (see p. 658)



Figs 37.10A to C: (A) Engaged head with lower pole below the level of ischial spine, appropriate for instrumental vaginal delivery; (B) Supermoulding of the head due to brim contraction with the lower pole at the ischial spines, yet not engaged, misleading the station of head; (C) Excessive moulding and caput formation often mislead the actual level of head in the pelvis. Abdominal palpation (see p. 153) is essential before instrumental vaginal delivery

LOW FORCEPS OPERATION

Preliminaries: Same as those mentioned earlier (p. 642). The following are specially emphasized:

- **Anesthesia:** Pudendal block supplemented by perineal and labial infiltration with 1% lignocaine hydrochloride is quite effective in producing local anesthesia
- **Catheterization**
- **Internal examination to assess:** (a) State of the cervix (b) membranes status (c) presentation and position of the head (d) assessment of the pelvic outlet (sacro-coccygeal plateau, TDO and subpubic arch).
- **Episiotomy:** It is usually done during traction when the perineum becomes bulged and thinned out by the advancing head.

STEPS: The operation consists of the following steps:

- Identification of the blades and their application
- Locking of the blades
- Traction
- Removal of the blades

Step I: Identification and application of the blades

The identification of the blades is to be made after articulation as mentioned earlier (Fig. 37.11). **The left or lower blade is to be introduced first**.

The four fingers of the semi-supinated right hand are inserted along the left lateral vaginal wall, the palmar surface of the fingers rest against the side of the head

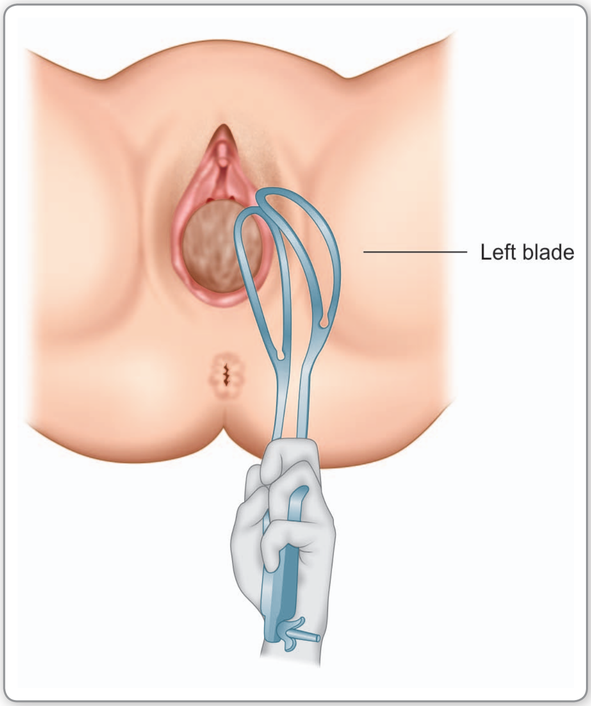


Fig. 37.11: Identification of forceps blades. Articulated forceps are held in front of the perineum in a position that they are expected to assume after application. This practice is known as *"ghosting"*

(Figs 37.12A to F). The fingers are used to guide the blade during application and to protect the vaginal wall. The handle of the left blade is taken lightly by three fingers of the left hand – index, middle and thumb in a pen holding manner and is held vertically almost parallel to the right inguinal ligament. The fenestrated portion of the blade is placed on the right palm with the tip (toe) pointing upwards. The right thumb is placed at the junction of the blade and the shank (heel).

The blade is introduced between the guiding internal fingers and the fetal head, manipulated by the thumb. As the blade is pushed up and up, the handle is carried downwards and backwards, traversing wide arc of a circle towards the left until the shank is to lie straight on the perineum. Utmost gentleness is required while introducing the blade. No assistant is usually required to hold the handle in low forceps operation. When correctly applied, the blade should be over the parietal eminence, the shank should be in contact with the perineum and the superior surface of the handle should be directed upwards.

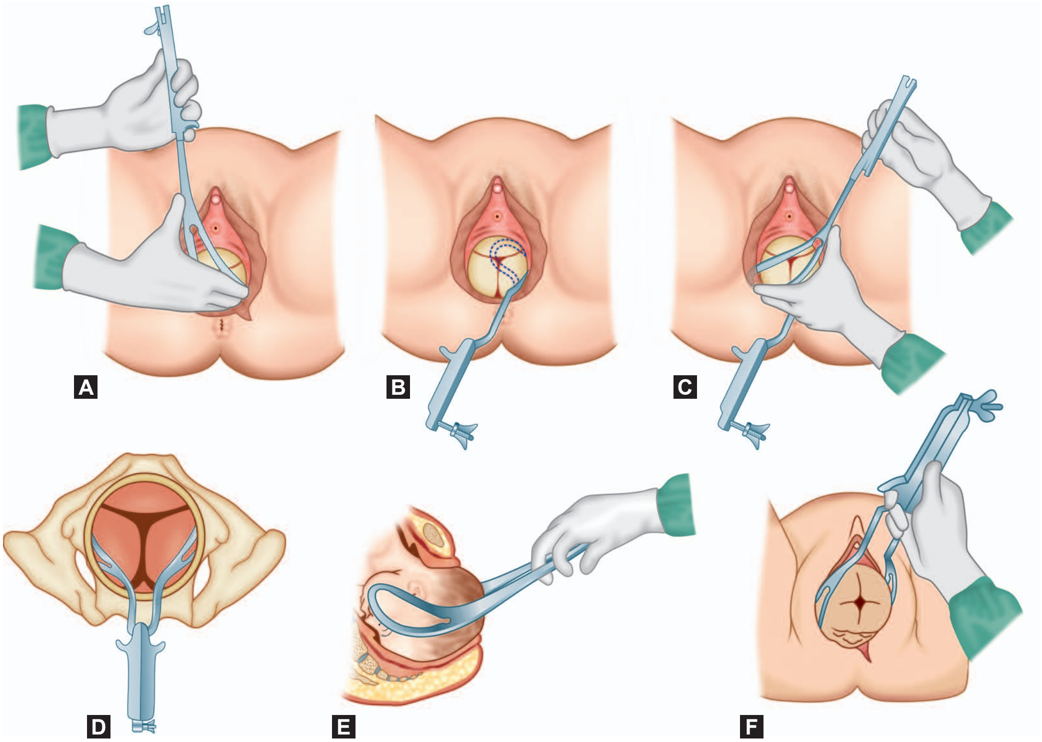
Introduction of the right blade: The two fingers of the left hand are now introduced into the right lateral wall of the vagina alongside the baby's head. The right blade is introduced in the same manner as with left one but holding it with the right hand.

Step II: Locking of the blades

When correctly applied (bimalar, biparietal placement), the blades should be articulated with ease. Minor difficulty in locking can be corrected by depressing the handles on the perineum. In case of major difficulty, the blades are to be removed, the causes are to be sought for (vide infra) and the blades are to be reinserted. The handles should never be forced to lock them.

Step III and IV: Traction and removal of blades

Before traction is applied, correct application of the blades is to be ensured. Correct application is evidenced by: (a) easy locking (b) the blades are equidistant from the lambdoid suture (Fig. 37.13) (c) firm gripping of the head on the biparietal diameter – as judged by a few tentative pulls.



Figs 37.12A to F: Steps of low forceps operation — (A) Introduction of the left blade; (B) The handle lying flat on the perineum after introduction; (C) Introduction of the right blade; (D) Showing perfect apposition and locking of the blades; (E) Bimalar, biparietal placement of blades and position of the fingers during traction; (F) Change in the grip in the final stage of delivery

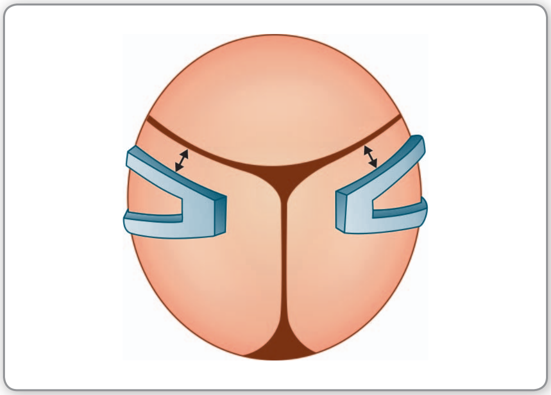


Fig. 37.13: The blades are equidistant from the lambdoid suture indicating correct application

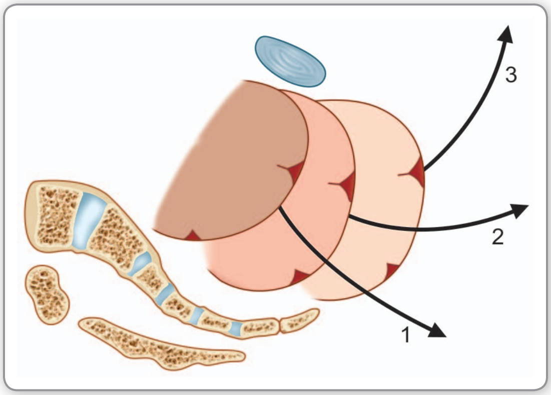


Fig. 37.14: Showing direction of traction along the axis of the birth canal — (1) Downwards and backwards; (2) Straight horizontal pull; (3) Upwards and forwards

Principles: Steady but intermittent traction should be given if possible during contraction. However, in outlet forceps the pull may be continuous. Strong traction is not needed as the only resistance to overcome is the perineum and the coccyx.

Gripping of the articulated forceps during traction: The traction is given by gripping the handle, placing the middle finger in between the shanks with the ring and index fingers on either side on the finger guard. During the final stage of traction, the four fingers are placed in between the shanks and the thumb which is placed on the under surface of the handles and exerts the necessary force.

Direction of the pull: The direction of the pull corresponds to the axis of the birth canal (Fig. 37.14). In low forceps operation depending upon the station of the head, the direction of the pull is downwards and backwards until the head comes to the perineum. The pull is then directed horizontally straight towards the operator till the head is almost crowned. The direction of pull is **gradually changed to upwards and forwards, towards** the mother's abdomen to deliver the head by extension. **The blades are removed one after the other, the right one first**.

Following the birth of the head, usual procedures are to be taken as in normal delivery. Routine injection oxytocin 10 IU, IM or intravenous methergine 0.2 mg is to be administered with the delivery of the baby. Episiotomy is repaired in the usual method (see p. 649). Lacerations on the vaginal walls or perineum are to be excluded.

OUTLET FORCEPS OPERATION

Wrigley's forceps are used exclusively in outlet forceps operation. Perineal and vulval infiltration with 1% lignocaine is enough for local anesthesia. The blades are introduced as in the low forceps operation with long-curved forceps except that two fingers are to be introduced into the vagina for the application of the left blade. Traction is given holding the articulated forceps with the fingers placed in between the shanks and the thumb on the under surface of the handles. **The direction of the pull is straight horizontal and then upwards and forwards**.

MIDFORCEPS OPERATION

The most common indication of midforceps operation is following manual rotation of the head in malrotated occipitoposterior position. The commonly used forceps is long curved one with or without axis-traction device. Kielland is useful in the hands of an expert.

Procedures

- General anesthesia is preferable.
- **Introduction of the blades:** The introduction of the blades is to be done after prior correction of the malrotation.

(a) *Without axis-traction device:* The blades are introduced as in the low forceps operation. **An assistant is required to hold the left handle after its introduction** **(b)** *With axis-traction device:* While applying the left blade, the traction-rod already attached to the blade is held backwards. During introduction of the right blade, the traction-rod must be held forwards otherwise it will prevent locking of the blades.

- **Traction:** **(a)** *Without axis-traction device:* The direction of pull is first downwards and backwards, then (horizontal or straight pull) and finally upwards and forwards **(b)** *With axis-traction device:* The traction handle is to be attached to the traction rods. **During traction, the traction rods should remain parallel with the shanks**. When the base of the occiput comes under the symphysis pubis, the traction-rods are to be removed.

DIFFICULTIES IN FORCEPS OPERATION

The difficulties are encountered mainly due to faulty assessment of the case before the operative delivery is undertaken. However, there is hardly any difficulty in low forceps operation.

During application of the blades: **The causes are:** (1) Incompletely dilated cervix (2) unrotated or nonengaged head.

Difficulty in locking: **The causes are:** (1) Application in unrotated head (2) improper insertion of the blade (not far enough in) (3) failure to depress the handle against the perineum and (4) entanglement of the cord or fetal parts inside the blades.

Difficulty in traction: **The causes of failure to deliver with traction are:** (1) Undiagnosed occipitoposterior position (2) faulty cephalic application (3) wrong direction of traction (4) mild pelvic contraction and (5) Constriction ring.

Slipping of the blades: **The causes are:** (1) The blades are not introduced far enough in (2) faulty application in occipitoposterior position. **The blades should be equidistant from the sinciput and occiput.**

FORCEPS IN OCCIPITO-SACRAL POSITION: Usual application of the blades as like that of occipitoanterior position is made. **The blades should lie equidistant from the sinciput and occiput**, otherwise the blades may slip during traction. Horizontal traction is given until the root of the nose is under the symphysis pubis. The direction is changed to upwards and forwards to deliver the occiput. By a downward movement of the instrument, the nose and chin are delivered.

FORCEPS IN FACE PRESENTATION: Forceps delivery is only reserved for mentoanterior position. The blades are applied as in occipitoanterior position. **But the handles should be kept well forward to avoid** grasping of the neck by the tips of the blade. Traction is made like that of occipitoanterior to bring the chin well below and then round the symphysis pubis.

APPLICATION OF FORCEPS TO THE AFTER-COMING HEAD: The method has been described in p. 444.

KIELLAND'S FORCEPS

The forceps was designed and named after Kielland (Kjelland) of Norway (Rotational forceps, 1916). In the hands of an expert, it is an useful and preferred instrument. **Its advantages over the widely used long-curved forceps are**: (1) It can be used with advantages in unrotated vertex or face presentation (2) facilitates grasping and correction of asynclitic head because of its sliding lock (Fig. 37.15).

IDENTIFICATION OF THE BLADES: The articulated blades are to be held in front of the vulva in a position to be taken up when applied to the head. **The concavity of the slight pelvic curve should correspond to the side towards which the occiput lies**. The blades are named anterior and posterior. **The anterior blade is to be introduced first.**

METHODS OF APPLICATION: There are three methods: (1) Classical (obsolete) (2) wandering and (3) direct.

Indications of rotational forceps are few. It is commonly used in deep transverse arrest with asynclitism of the fetal head. **Wandering method is popular**. The anterior (superior) blade is applied first. The blade is inserted

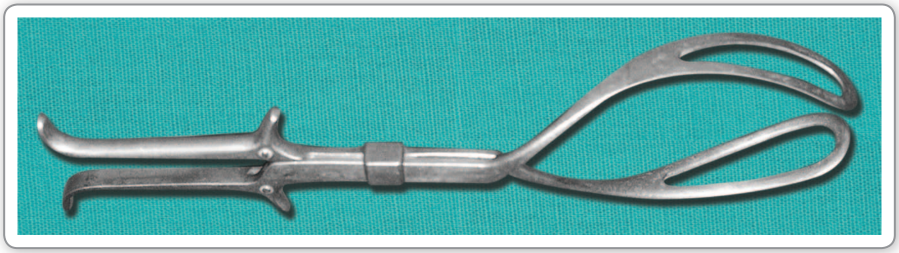


Fig. 37.15: Kielland's forceps

along the side wall of the pelvis and then wandered by swinging it round the fetal face to its anterior position. The posterior blade is inserted directly under guidance of the right hand placed between the head and the hollow of the sacrum. The forceps handles are depressed down and the handle tips are brought into alignment to correct the asynclitism. The occiput is rotated anteriorly. Slight upward dislodgement of the head may facilitate rotation. The position is rechecked and traction is applied. Sitting on a low foot stool or kneeling is convenient for the operator.

LIMITATIONS: Because of complexity in the technique of its application, one should be sufficiently trained before independent use.

HAZARDS: Fetal: Facial bruising, laceration, facial nerve palsy, skull fractures, intracranial hemorrhage. **Maternal:** Perineal sulcus tear, complete perineal tear. **Deep mediolateral episiotomy is mandatory.**

Piper forceps is a specialized forceps, used to assist the delivery of the after-coming head of breech. It has a cephalic curve, reverse pelvic curve, long parallel shanks that permit the baby's body to rest against it during head delivery.

COMPLICATIONS OF FORCEPS OPERATION

The complications of the forceps operation are mostly related to the faulty technique and to the indication for which the forceps are applied rather than the instrument. The complications are grouped into:

- **Maternal**
- **Fetal**

Maternal	Fetal
IMMEDIATE:	**IMMEDIATE:**
■ ***Injury:*** Vaginal laceration or sulcus tear, cervical tear, extension of episiotomy to involve the vaginal vault, complete perineal tear ■ ***Nerve injury:*** Femoral (L2, 3, 4), lumbosacral trunk (L4, 5) with midforceps delivery ■ ***Postpartum hemorrhage*** may be—(i) traumatic or (ii) atonic, requiring blood transfusion or (iii) both, may cause shock ■ ***Anesthetic complications*** (following local or general anesthesia see p. 593, 596) ■ ***Puerperal sepsis and maternal morbidity***	Asphyxia, facial bruising, intracranial hemorrhage (rupture of the great vein of Galen). Cephalohematoma, facial palsy, skull fractures, cervical spine injury (rotational forceps)
REMOTE:	**REMOTE:**
■ Painful perineal scars, dyspareunia, low backache, genital prolapse, stress urinary incontinence and anal sphincter dysfunction	Cerebral or spastic palsy due to residual cerebral injury (rare)

PROPHYLACTIC FORCEPS (ELECTIVE): This type of forceps operation was named after De Lee. **It refers to forceps delivery only to shorten the second stage of labor when maternal and/or fetal complications are anticipated. The indications are:** (1) Eclampsia (2) heart disease (3) previous history of cesarean section (4) postmaturity (5) low-birth-weight baby (6) to curtail the painful second stage and (7) patients under epidural analgesia.

It prevents possible fetal cerebral injury due to pressure on the perineum and spares the mother from the strain of bearing down efforts. **Prophylactic forceps should not be applied until the criteria of low forceps are fulfilled**.

TRIAL FORCEPS: It is a tentative attempt of forceps delivery in a case of suspected midpelvic contraction with a preamble declaration of abandoning it in favor of cesarean section if moderate traction fails to overcome the resistance. The procedure should be conducted in an operation theater keeping everything ready for cesarean section. The conduct of trial forceps requires great deal of skill and judgment. If moderate traction leads to progressive descent of the fetal head, the delivery is completed vaginally, if not cesarean section is done immediately. Many unnecessary cesarean sections or difficult vaginal deliveries can thus be avoided.

CONDITIONS FOR TRIAL OF FORCEPS: (1) Maternal obesity (BMI ≥ 30); (2) clinically big baby (wt ≥ 3 kg); (3) occipitoposterior position; (4) mid-cavity delivery; (5) when 1/5th fetal head palpable per abdomen.

FAILED FORCEPS: When a deliberate attempt in vaginal delivery with forceps has failed to expedite the process, it is called failed forceps. It is often due to poor clinical judgment and skill. Failure in the operative delivery may be due to improper application.

Causes: **The common causes are:** (1) Incompletely dilated cervix (2) unrotated occipitoposterior position (3) cephalopelvic disproportion (4) unrecognized malpresentation (brow) or hydrocephalus (5) constriction ring (6) clinically big baby (≥ 4 kg) (7) maternal BMI ≥ 30 and (8) in a case with mid-cavity delivery.

Prevention: It is a preventable condition. Only through skill and judgment, proper selection of the case ideal for forceps can be identified. **Even if applied in wrong cases, one should resist the temptation to give forcible traction in an attempt to hide the mistake**.

Management: (1) To assess the effect on the mother and the fetus (2) to start a Ringer's solution drip and to arrange for blood transfusion, if required (3) to administer parenteral antibiotic (4) to exclude rupture of the uterus (5) the procedure is abandoned and delivery is done by cesarean section and (6) laparotomy should be done in a case with rupture of uterus.

VENTOUSE

Ventouse is an instrumental device designed to assist delivery by creating a vacuum between it and the fetal scalp. The pulling force is dragging the cranium while in forceps, the pulling force is directly transmitted to the base of the skull.

INSTRUMENTS: Ever since Malmstrom, in 1956 reintroduced and popularized its use, various modifications of the instruments are now available. Each, however, consists of the following basic components (Fig. 37.16):

Metal cups were initially used. *Soft cups, silc cup [silicone rubber or disposable plastic (Mityvac)]* cups have better adherence to the fetal scalp. These cups could be folded and introduced into the vagina without much discomfort. Silastic cup causes less scalp trauma and there is no chignon formation. Rigid plastic cup (Kiwi Omnicup) is safe, effective and is useful for rotational delivery.

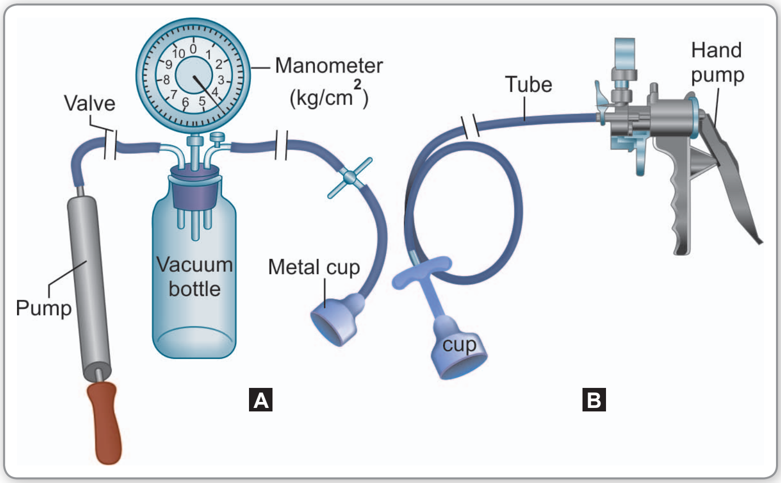
The cup is connected to a pump through a thick-walled rubber tube by which air is evacuated. Vacuum is created by a hand pump or by electric pump. The parts of the device are:

(1) Suction cups with four sizes (30 mm, 40 mm, 50 mm and 60 mm) (2) a vacuum generator and (3) traction tubings (Figs 37.16A and B).

INDICATIONS of ventouse delivery are the same as those of forceps (p. 654).

CONTRAINDICATIONS OF VENTOUSE: (i) Any presentation other than vertex (face, brow, breech) (ii) preterm fetus (< 34 weeks). Chance of scalp avulsion or subaponeurotic hemorrhage (iii) suspected fetal coagulation disorder and (iv) suspected fetal macrosomia (≥ 4 kg).

CONTRAINDICATIONS for operative vaginal delivery (both for ventouse or forceps): (i) Unengaged fetal head (ii) obvious CPD (iii) patient's refusal (iv) fetus having unacute bleeding diathesis (hemophilia).



Figs 37.16A and B: (A) Malmstrom device; (B) Mityvac pump with tube and soft cup

PREREQUISITES FOR OPERATIVE VAGINAL (FORCEPS OR VENTOUSE) DELIVERY (p. 654)

Advantages of Ventouse Over Forceps	Advantages of Forceps Over Ventouse
■ It can be used in unrotated or malrotated head (OP, OT position). It helps in autorotation ■ It is not a space-occupying device like the forceps blades ■ Traction force is less (10 kg) compared to forceps ■ It is comfortable and has lower rates of maternal trauma and genital tract lacerations ■ Analgesia need is less. Pudendal block with perineal infiltration is adequate but for forceps regional or general anesthesia is often needed ■ Reduced maternal pelvic floor injuries and is advocated as the **instrument of first choice**. ■ Perineal injury (3rd and 4th degree tears) are less compared to forceps ■ Postpartum maternal discomfort (pain) are less compared to forceps ■ Easier to learn comparing to forceps ■ Simplicity of use in delivery makes it convenient to the operator (suitable for trained midwives)	♦ In cases, where moderate traction is required, forceps will be more effective compared to ventouse ♦ Forceps operation can quickly expedite the delivery in case of fetal distress where ventouse will be unsuitable as it takes longer time ♦ It is safer at any gestational age baby (even < 36 weeks). The fetal head remains inside the protective cage ♦ It can be employed in anterior face or in after-coming head of breech presentation, where ventouse is contraindicated ♦ Lesser neonatal scalp trauma, retinal hemorrhage, jaundice or cephalhematoma compared to ventouse ♦ Higher rate of successful vaginal delivery as ventouse has got higher failure rates than forceps ♦ Cup detachment (Pop-off) occurs when the vacuum is not maintained in ventouse. No such problems once forceps blades are correctly applied ♦ Number of types of forceps (p. 651) are available for outlet, mid-cavity or rotational delivery. Traction force is more (about 20 kg for a primary and about 13 kg in a multi)

PROCEDURE:

Preliminaries: The procedures to be taken are mentioned in p. 642. Pudendal block or perineal infiltration with 1% lignocaine is sufficient. It may be applied even without anesthesia, especially in parous women. **The instrument should be assembled and the vacuum is tested prior to its application.**

Step I: Application of the cup: The largest possible cup is to be selected. The cup is introduced after retraction of the perineum with two fingers of the other hand. The cup is placed against the fetal head **nearer the occiput (flexion point) with the "knob" of the cup pointing towards the occiput. Flexion or pivot point** is an imaginary site located midsagittally about 6 cm from the center of the anterior fontanel or about 3 cm in front of the posterior fontanel. Traction over this **flexion point** either by ventouse or forceps facilitates flexion and presents the smaller diameter to the pelvis (Fig. 37.16C). The knob indicates the degree of rotation. Betadine (antiseptic) solution is applied to the rim of the malstrom metal cup.

A vacuum of 0.2 kg/cm^2 is induced by the pump slowly, taking at least 2 minutes. A check is made using the fingers round the cup to ensure that no cervical or vaginal tissue is trapped inside the cup. **The pressure is gradually raised at the rate of 0.1 kg/cm^2 per minute until the effective vacuum of 0.8 kg/cm^2 is achieved in about 10 minutes time**. The scalp is sucked into the cup and an artificial **caput succedaneum (chignon)** is produced. The chignon usually disappears within few hours.

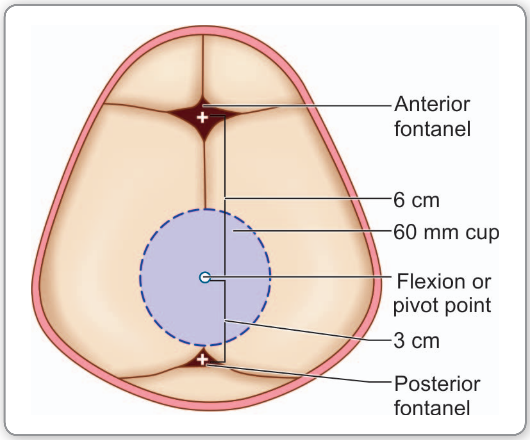
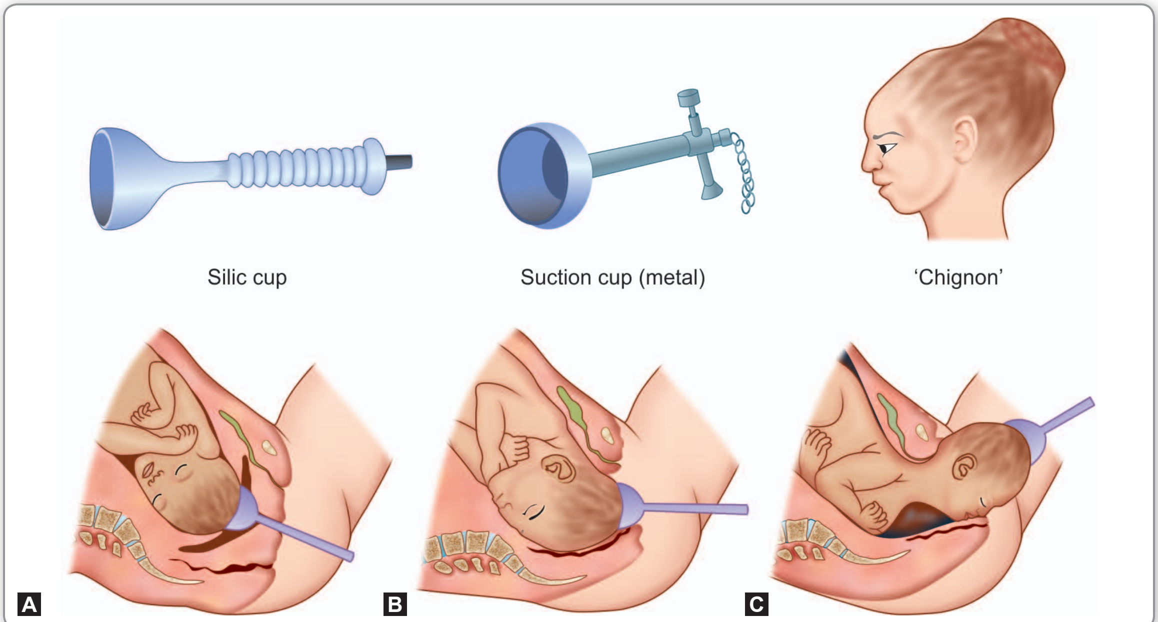


Fig. 37.16C: Ventouse cup should be placed on the flexion or pivot point (see p. 662, Fig. 37.17)

Step II: Traction — *Practical guides are* (Fig. 37.17):

- Traction must be at right angle to the cup
- Traction should be synchronous with the uterine contractions
- Traction is released in between uterine contractions
- Traction should be made using one hand along the axis of the birth canal. The fingers of the other hand are to be placed against the cup to note the correct angle of traction, rotation and advancement of the head
- **Operative vaginal delivery (forceps/ventouse) should be abandoned, where there is no descent of the presenting part with each pull or when delivery is not imminent after three pulls with correctly applied instruments by an experienced operator. On no account, traction should exceed 30 minutes**
- As soon as the head is delivered, the vacuum is reduced by opening the screw-release valve and the cup is then detached. The delivery is then completed in the normal way.



Figs 37.17A to C: Application of vacuum extractor; (A to C) indicating the directions of traction at different stations of the fetal head. Traction over this flexion or pivot point either by ventouse or forceps promotes flexion and presents smaller diameter to the pelvis (Fig. 37.16C)

COMPLICATIONS: ***Neonate:*** (1) Superficial scalp abrasion (2) sloughing of the scalp and (3) cephalhematoma—due to rupture of emissary veins beneath the periosteum. Usually it resolves by one or two weeks (4) subaponeurotic (subgaleal) hemorrhage (not limited by suture line as it is not subperiosteal) (5) intracranial hemorrhage (rare) (6) retinal hemorrhage (no long-term effect) and (7) jaundice.

Maternal: The injuries are uncommon but may be due to inclusion of the soft tissues such as the cervix or vaginal wall inside the cup. However, failure rate is high. **The sequential use of ventouse and forceps increases the risk of trauma both to the mother and the neonate**. Outlet forceps may be used following failure of ventouse.

SUMMARY: Ventouse is an instrument, designed to assist delivery by creating vacuum between it and fetal scalp. The instrument, as deviced by Malmstrom, consists of: (1) suction cup (2) vacuum generator and (3) traction tubing device. **Silc cups** are found more convenient. **The indications are same as those of forceps except that it cannot be employed in face or after coming head of breech**. **Vacuum**: Causes lower rates of maternal trauma and genital tract lacerations, but causes more neonatal scalp trauma and cephalhematoma compared to forceps. Both the instruments (ventouse and forceps) are not inherently dangerous. The operator must have knowledge, experience and skill to use and also the willingness to abandon the procedure when felt difficult. **All operative vaginal delivery procedures should be considered as a trial**.

The rate of anal sphincter injury may be higher in forceps delivery. The risk of fetal injury associated with instrumental vaginal delivery is instrument specific. The sequential use of ventouse and forceps increases the risk of trauma both to the mother and the neonate.

Benefits of operative vaginal delivery: Most women desire a vaginal delivery. Safe and effective use of instrumental delivery fulfills women's desire and satisfaction. Many women (79%) desire subsequent vaginal delivery compared with women delivered by cesarean section (39%).

VERSION

DEFINITION: It is a manipulative procedure designed to change the lie or to bring the comparatively favorable pole to the lower pole of the uterus.

TYPES: According to the methods employed: ♦ **Spontaneous** ♦ **External** ♦ **Internal** ♦ **Bipolar**

Spontaneous: Version process occurs spontaneously. The incidence of spontaneous version in breech presentation is nearly 55% after 32 weeks and about 25% after 36 weeks. It is more common in multiparous women.

External: The maneuver is done solely by external manipulation.

Internal: The conversion is done principally by one hand introducing into the uterus and by the other hand on the abdomen.

***Bipolar* (Braxton-Hicks):** The conversion is done introducing one or two fingers through the cervix and by the other hand on the abdomen.

When the cephalic pole is brought down to the lower pole of the uterus, it is called cephalic version and when the podalic pole is brought down, it is called podalic version.

EXTERNAL CEPHALIC VERSION

External cephalic version is done to bring the favorable cephalic pole in the lower pole of the uterus.

INDICATIONS: ♦ **Breech presentation (see p. 434)** ♦ **Transverse lie (see p. 454)**

Selection of time, contraindication, difficulties and complications have already been described (see p. 440).

The advantages of ECV at term are: (i) By this time spontaneous version will occur in many cases (see above) (ii) If any complications occur during ECV prompt delivery could be done by cesarean section as the baby is at term. Success rate of ECV in general is 60%. Use of tocolytics (ritodrine) increases the success rate of ECV.

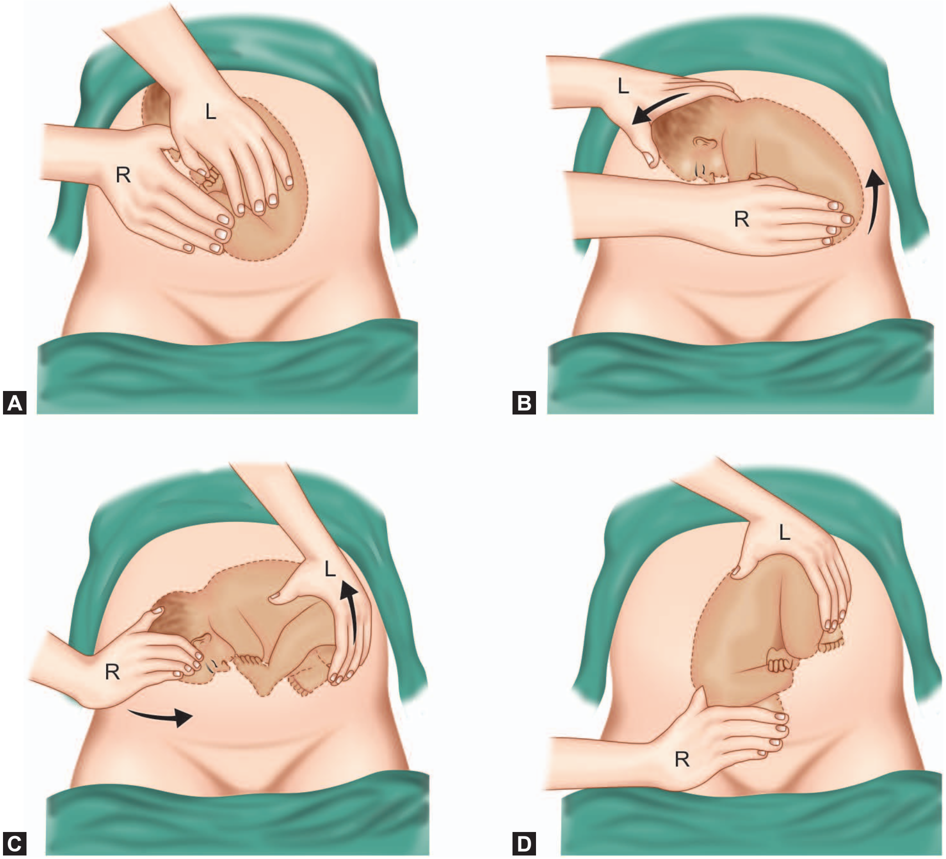
Benefits of ECV: (i) Reduces the incidence of breech presentation at term and of breech delivery (ii) reduces the number of Cesarean delivery (iii) reduces maternal morbidity due to Cesarean or vaginal breech delivery and (iv) reduces the fetal hazards of vaginal breech delivery (p. 439).

PROCEDURES: In breech presentation—The maneuver is carried out after 36 weeks in the labor-delivery complex. **Tocolytic drug** (terbutaline – 0.25 mg SC), **if required, can be administered**. Real time ultrasound examination is done to confirm the diagnosis and adequacy of amniotic flood volume. **A reactive NST should precede the maneuver (p. 122)**.

Preliminaries: The patient is asked to empty her bladder. She is to lie on her back with the shoulders slightly raised and the thighs slightly flexed. Abdomen is fully exposed. The presentation, position of the back and limbs are checked and FHR is auscultated.

Actual steps: **"Forward roll" movement.**

Step I (Figs 37.18A to D): The breech is mobilized using both hands to one iliac fossa towards which the back of the fetus lies. The podalic pole is grasped by the right hand in a manner like that of Pawlik's grip while the head is grasped by the left hand.



Figs 37.18A to D: Steps of external cephalic version — (Breech — LSA): (A) Mobilization of the buttocks to the iliac fossa towards the back using both hands; (B) Rotation of the trunk holding the poles and maintaining flexion of the trunk; (C) Change of hands to prevent crossing after the lie becomes transverse; (D) The lie becomes longitudinal with the cephalic pole being brought to the lower pole of the uterus

Step II : The pressure (firm but not forcible) is now exerted to the head and the breech in the opposite directions to keep the trunk well flexed which facilitates version. The pressure should be intermittent to push the head down towards the pelvis and the breech towards the fundus until the lie becomes transverse. The FHR is once more to be checked.

Step III: The hand is now changed one after the other to hold the fetal poles to prevent crossing of the hand. The intermittent pressure is exerted till the head is brought to the lower pole of the uterus.

A reactive NST should be obtained after completing the procedure (see p. 122). There may be undue bradycardia due to head compression which is expected to settle down by 10 minutes. If however fetal bradycardia persists, the possibility of cord entanglement should be kept in mind and in such cases reversion may have to be considered. **The patient is to be observed for about 30 minutes:** (1) To allow the FHR to settle down to normal and (2) to note for any vaginal bleeding or evidence of premature rupture of the membranes.

INSTRUCTIONS: (1) The patient is advised for follow up to check the corrected position (2) to report to the physician if there is vaginal bleeding or escape of liquor amnii or labor starts and (3) **Rh-negative nonimmunized women must be protected** by intramuscular administration of 100 µg anti-D gamma globulin (see p. 390).

EXTERNAL VERSION IN TRANSVERSE LIE: The version is much easier than in breech. The association of placenta previa or congenital malformation of the uterus should be excluded.

EXTERNAL PODALIC VERSION: The external podalic version may be done in cases when the external cephalic version fails in transverse lie in case of the second baby of twins.

INTERNAL VERSION

Internal version is always a podalic version and is almost always completed with the extraction of the fetus.

INDICATIONS: Internal version is hardly indicated in a singleton pregnancy in present day obstetric practice. **Its only indication being the transverse lie in case of the second baby of twins**.

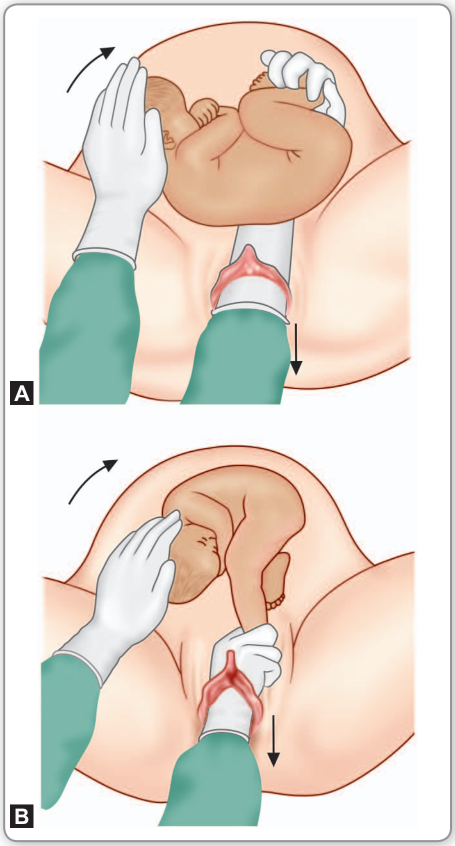
However, it may be employed in singleton pregnancy to expedite delivery in adverse conditions where the cesarean section facilities are lacking. Such conditions are: (1) transverse lie with cervix fully dilated and (2) cord prolapse with cervix fully dilated with transverse lie or head high up and the baby is alive.

CONDITIONS TO BE FULFILLED: (1) The cervix must be fully dilated (2) liquor amnii must be adequate for intrauterine fetal manipulation and (3) fetus must be living.

CONTRAINDICATIONS: It must not be attempted in neglected obstructed labor even if the baby is living.

PROCEDURES: *Assessment* of the lie, presentation and FHR is made by an experienced obstetrician by abdominal palpation, vaginal examination and/or transabdominal ultrasound examination. Close (continuous) FHR monitoring is essential. The steps are to be followed as mentioned earlier (p. 643). Internal version should be done under general or epidural anesthesia. **Actual steps (Figs 37.19A and B):**

Step I: Patient is placed in dorsal lithotomy position. Antiseptic cleaning drapings and catheterization are done. Introduction of the



Figs 37.19A and B: Principal steps of internal podalic version—(A) To introduce the right hand to grasp the upper leg in dorsoanterior position with the head lying on the right iliac fossa; (B) To give traction on the leg gripping in a cigarette holding fashion, the other hand pushes up the head externally

hand—**if the podalic pole of the fetus is on the left side of the mother, the right hand is to be introduced and vice versa**. The hand is to be introduced in a cone-shaped manner. It is then pushed up into the uterine cavity keeping the back of the hand against the uterine wall until the hand reaches the podalic pole.

Step II: The hand is to pass up to the breech and then along the thigh until a foot is grasped. **The identification of the foot is done by palpation of the heel**. It is advantageous to grasp the first foot which one encounters.

Step III: While the leg is brought down by a steady traction, the cephalic pole is pushed up using the external hand.

Step IV: After one leg is brought down, there is no difficulty to deliver the other leg. The delivery is usually completed with breech extraction during uterine contractions.

Step V: Routine exploration of the uterovaginal canal to exclude rupture of the uterus or any other injury.

COMPLICATIONS: Maternal risk includes placental abruption, rupture of the uterus and increased morbidity. **The fetal risk includes** asphyxia, cord prolapse and intracranial hemorrhage apart from all hazards of breech delivery (see p. 439) leading to a high perinatal mortality of about 50%.

BIPOLAR VERSION: The bipolar version named after Braxton-Hicks is an obsolete maneuver in present day obstetric practice. However, it may be a lifesaving procedure at places, specially in the rural areas of the developing countries, where it is not possible to transport the patient with placenta previa to an equipped medical center. Its chief indication is lesser degree of placenta previa when the fetus is dead, deformed or previable. The cervix must be at least two fingers dilated to facilitate manipulation by pushing up of the head to one iliac fossa and to grasp one leg at the ankle. Simultaneous manipulation by the external hand facilitates the procedure. **Bringing down of one leg facilitates compression over the placenta and thereby stops the bleeding**.

Fundal pressure to assist the process of vaginal delivery should not be used. It results in pelvic hematoma formation, orthopedic and neurological complications.

DESTRUCTIVE OPERATIONS

The destructive operations are designed to diminish the bulk of the fetus so as to facilitate easy delivery through the birth canal. In modern obstetric practice, virtually there is hardly any place for destructive operations. **Neglected obstetrics requiring destructive operations are completely preventable**. **These procedures are difficult and may be dangerous too unless the operator is sufficiently skilled**. Unfortunately, one may have to perform such operations while working in the unorganized sector. Some commonly performed operations are discussed here. There are four types of operations**:**

- **Craniotomy**
- **Evisceration**
- **Decapitation**
- **Cleidotomy**

CRANIOTOMY

DEFINITION: It is an operation to make a perforation on the fetal head, to evacuate the contents followed by extraction of the fetus.

INDICATIONS:

- **Cephalic presentation producing obstructed labor with dead fetus:** This is the most common indication of craniotomy in the referral hospitals of the developing countries.
- **Hydrocephalus even in a living fetus:** This is applicable both for the forecoming and the aftercoming head (see p. 470).
- Interlocking head of twins.

CONDITIONS TO BE FULFILLED: (1) The cervix must be fully dilated and (2) baby must be dead (hydrocephalus being excluded).

CONTRAINDICATION: (i) The operation should not be done when the pelvis is severely contracted so as to shorten the true conjugate to less than 7.5 cm (3"). In such condition, the baby cannot be delivered, as the bimastoid diameter (base of the skull) of 7.5 cm cannot be compressed. (ii) Rupture of the uterus where laparotomy is essential.

PROCEDURES: ***Preliminaries:*** The preliminary preparations are the same as mentioned in p. 642. The operation is to be done under general anesthesia.

Actual steps

Step I: The two fingers (index and middle) are introduced into the vagina and the finger tips are to be placed on proposed site of perforation. However, when the suture line cannot be defined because of big caput, the perforation should be done through the dependent part.

Sites of perforation: Vertex: On the parietal bone either side of the sagittal suture. Suture is avoided to prevent collapse of the bone thereby preventing escape of the brain matter. *Face:* Through the orbit or hard palate. *Brow:* Through the frontal bone.

Step II: The Oldham's perforator (Fig. 42.32) with the blades closed is introduced under the palmar aspect of the fingers protecting the anterior vaginal wall and the adjacent bladder (as shown in Figs 37.20A and B) until the tip reaches the proposed site of perforation.

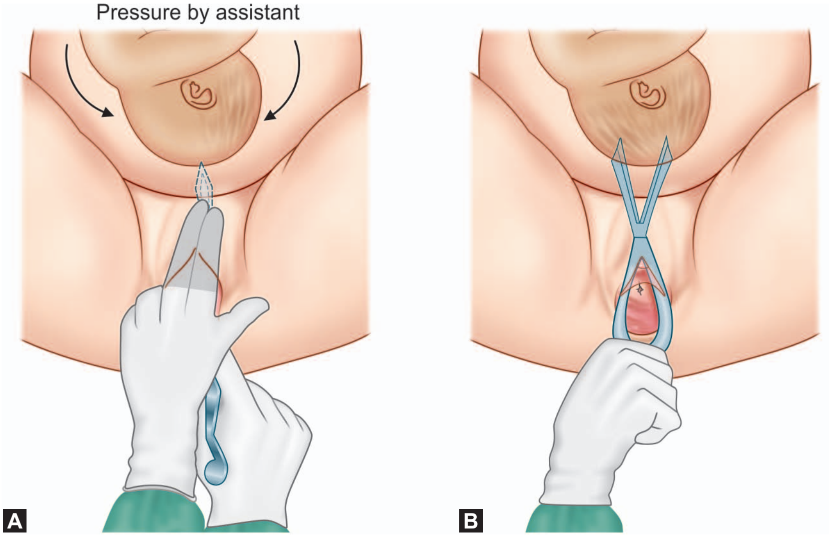
Step III: By rotating movements the skull is perforated. **During this step, an assistant is asked to steady the head per abdomen in a manner of first pelvic grip**. After the skull is perforated, the instrument is thrust up to the shoulders and the handles are approximated so as to allow separation of the sharp blades for about 2.5 cm.

The blades are again apposed by separating the handles. The instrument is brought out keeping the tip of the blades still inside the cranium. The instrument is rotated at right angle and then again thrust in up to the shoulders. The handles are once more to be compressed so as to separate the blades for about 2.5 cm. The perforated area now looks like a cross. The instrument with the blades closed is then thrust in beyond the guard to churn the brain matter. The instrument, with the blades closed, is brought out under the guidance of the two fingers still placed inside the vagina.

Alternative to Oldham's perforator, similar procedure could be performed using a sharp-pointed Mayo's scissors.

Step IV: With the fingers brain matter is evacuated. The idea is to make the skull collapse as much as possible.

Step V: When the skull is found sufficiently compressed, the extraction of the fetus is achieved either by using a cranioclast or by two giant volsella (Fig. 42.23). Giant volsella are used to hold the incised skull and scalp margins.



Figs 37.20A and B: (A) Perforation of the head while an assistant fixes the head suprapubically; (B) Separation of the blades by compression of the handle (for better display, the fingers of the left hand are removed)

Step VI: The traction is now exerted in the same direction as like that mentioned in forceps operation.

Step VII: After the delivery of the placenta, the uterovaginal canal must be explored as a routine for evidence of rupture uterus or any tear.

Injection methergine 0.2 mg is to be given intravenously with the delivery of the anterior shoulder. The rest of the delivery is completed as in normal delivery.

Forceps versus craniotomy in a dead fetus: If the delivery of the uncompressed head can be accomplished without much force with consequent injuries to the mother, forceps delivery is preferred. But if it is found difficult and damaging to the mother, craniotomy is safer.

DECAPITATION

DEFINITION: It is a destructive operation whereby the fetal head is severed from the trunk and the delivery is completed with the extraction of the trunk and that of the decapitated head per vaginum.

INDICATION: (1) Neglected shoulder presentation with dead fetus where neck is easily accessible. (2) Interlocking head of twins.

PROCEDURES: ***Preliminaries***—The preliminaries to be followed are the same as outlined earlier. The operation is done under general anesthesia.

Actual Steps

Step I: If the fetal hand is not prolapsed, bring down a hand. A roller gauze is tied on the fetal wrist and an assistant is asked to give traction towards the side away from the fetal head to make the neck more accessible and fixed.

Step II: Two fingers of the left hand (middle and index) are introduced with the palmar surface downwards and the finger tips are to be placed on the superior surface of the neck—the proposed site of decapitation.

Step III: The decapitation hook with knife is to be introduced flushed under the guidance of the fingers placed into the vagina, **the knob pointing towards the fetal head**. The hook is pushed above the neck and rotated to 90° so as to place the knife firmly against the neck. The internal fingers, in the meantime, are placed on the under surface of the neck to guard the tip of the hook.

Step IV: By upward and downward movements of the hook with knife, the vertebral column is severed (evident by sudden loss of resistance). The rest of the soft tissue left behind may be severed by the same instrument or by embryotomy scissors. While removing the decapitation hook—it is to be pushed up; rotated to 90° and then to take out under the guidance of the internal fingers. The decapitated head is pushed up and the trunk is delivered by traction on the prolapsed arm.

***Step V:* Delivery of the decapitated head**—Any of the following methods may be usually effective:

- By hooking the index finger into the mouth • By holding the severed neck with giant vulsellum (Fig. 42.33) and delivery of the head as that of aftercoming head in breech • Using forceps.

***Step VI:* Routine exploration of the uterovaginal canal to exclude rupture of the uterus or any other injury**.

EVISCERATION

The operation consists of removal of thoracic and abdominal contents piecemeal through an opening on the thoracic or abdominal cavity at the most accessible site. The object is to diminish the bulk of the fetus which facilitates its extraction. If difficulty arises, the spine may have to be divided (spondylectomy) with embryotomy scissors.

The indications are: (1) Neglected shoulder presentation with dead fetus; the neck is not easily accessible and (2) fetal malformations, such as fetal ascites or hugely distended bladder or monsters.

CLEIDOTOMY

The operation consists of reduction in the bulk of the shoulder girdle by division of one or both the clavicles.

The operation is done only in dead fetus (anencephaly excluded) with shoulder dystocia. The clavicles are divided by the embryotomy scissors or long straight scissors introduced under the guidance of left two fingers placed inside the vagina.

POSTOPERATIVE CARE FOLLOWING DESTRUCTIVE OPERATIONS

- **Exploration of the uterovaginal canal must be done** to exclude rupture of the uterus or lacerations on the vagina or any genital injury.
- **A self-retaining (Foley's) catheter is put inside,** especially following craniotomy for a period of 3–5 days or until the bladder tone is regained.
- **Dextrose saline drip is to be continued** till dehydration is corrected. Blood transfusion may be given, if required.
- **Ceftriaxone** 1 g IV infusion is given twice daily.

COMPLICATIONS: (1) Injury to the uterovaginal canal (2) rupture of uterus (3) postpartum hemorrhage—atonic or traumatic (4) shock—due to blood loss and/or dehydration (5) puerperal sepsis (6) subinvolution (7) injury to the adjacent viscera— bladder—vesicovaginal fistula or rarely to rectal wall leading to rectovaginal fistula and (8) prolonged ill health.

CESAREAN SECTION (CS)

DEFINITION: It is an operative procedure whereby the fetuses after the end of 28th weeks are delivered through an incision on the abdominal and uterine walls. This excludes delivery through an abdominal incision where the fetus, lying free in the abdominal cavity following uterine rupture or in secondary abdominal pregnancy. The first operation performed on a patient is referred to as a **primary cesarean section**. When the operation is performed in subsequent pregnancies, it is called **repeat cesarean section**.

Nomenclature and history: Amidst controversy, it appears that the operation derives its name from the notification "lex Cesarea" – a Roman law promulgated in 715 BC which was continued even during Caesar's reign. The law provided either an abdominal delivery in a dying woman with a hope to get a live baby or to perform postmortem abdominal delivery for separate burial. The operation does not derive its name from the birth of Caesar, as his mother lived long time after his birth. The other explanation is that the word cesarean is derived from the Latin Verb "Cedere" which means "to cut". French obstetrician, Francois Mauriceau first reported cesarean section in 1668. In 1876, Porro performed subtotal hysterectomy. It was Max Sanger in 1882, who first sutured the uterine walls. In 1907, Frank described the extraperitoneal operation. Kronig in 1912, introduced lower segment vertical incision and it was popularized by De Lee (1922). Although Kehrer in 1881 did the transverse lower segment operation for the first time, Munro Kerr in 1926 not only reintroduced the present technique of lower segment operation but also popularized it.

INCIDENCE: The incidence of cesarean section is **steadily rising**. During the last decade there has been two-to-threefold rise in the incidence from the initial rate of about 10%. Apart from increased safety of the operation due to improved anesthesia, availability of blood transfusion and antibiotics, the other **responsible factors are**:

Factors for Rising Cesarean Section Rate

- Rising incidence of primary cesarean delivery
- Identification of at risk fetuses before term (FGR)
- Identification of high-risk pregnancy
- Wider use of repeat CS
- Rising rates of induction of labor and failure of induction
- Decline in operative vaginal (midforceps, vacuum) delivery and manipulative vaginal delivery (rotational forceps)
- Decline in vaginal breech delivery
- Increased number of women with age >30 years and associated medical complications. Most are nulliparous
- Adoption of small family norm—neither the obstetricians, nor the patients are ready to accept any risk of abnormal labor
- Wider use of electronic fetal monitoring and increased diagnosis of fetal distress
- Fear of litigation in obstetric practice
- Cesarean delivery on maternal request

INDICATIONS

Indications for cesarean delivery: Cesarean delivery is done when labor is contraindicated (central placenta previa) and/or vaginal delivery is found unsafe for the fetus and/or mother.

The indications are broadly divided into two categories: ♦ **Absolute** ♦ **Relative (common)**

Absolute Indications	Relative Indications
Vaginal delivery is not possible. Cesarean is needed even with a dead fetus Indications are few:	**Vaginal delivery may be possible but risks to the mother and/or baby are high** (More often multiple factors may be responsible)
■ **Central placenta previa**	♦ **Cephalopelvic disproportion** (relative) see p. 410
■ **Contracted pelvis or cephalopelvic** disproportion (absolute)	♦ **Previous cesarean delivery** (p. 385)—(a) when primary CS was due to recurrent indication (contracted pelvis). (b) Previous two CS (c) Features of scar dehiscence. (d) Previous classical CS
■ **Pelvic mass** causing obstruction (cervical or broad ligament fibroid)	♦ **Non-reassuring FHR** (fetal distress)
■ **Advanced carcinoma cervix**	♦ **Dystocia** may be due to **(three Ps) relatively** large fetus **(passenger)**, small pelvis **(passage)** or inefficient uterine contractions **(power)**
■ **Vaginal obstruction** (atresia, stenosis)	♦ **Antepartum hemorrhage:** (a) Placenta previa and (b) abruptio placenta (p. 287, 292, 300)
Common Indications of Cesarean Section	♦ **Malpresentation:** Breech, shoulder (transverse lie), brow (p. 434)
Primigravidae: (1) Failed indication (2) fetal distress (non-reassuring fetal FHR) (3) Cephalopelvic disproportion (CPD) (4) dystocia (dysfunctional labor p. 359) nonprogress of labor (5) malposition and malpresentation (occipito-posterior, breech).	♦ **Failed surgical induction** of labor, **failure to progress** in labor (p. 598) ♦ **Bad obstetric history**—with recurrent fetal loss ♦ **Hypertensive disorders:** (a) Severe preeclampsia, (b) eclampsia—uncontrolled fits even with antiseizure therapy (p. 255, 268)
Mutigravidae: (1) Previous cesarean delivery (2) antepartum hemorrhage (placenta previa, placental abruption) (3) malpresentation (breech, transverse lie).	♦ **Medical-gynecological disorders:** (a) Diabetes (uncontrolled), heart disease (coarctation of aorta, Marfan's syndrome; (b) mechanical obstruction (due to benign or malignant pelvic tumors (carcinoma cervix), or following repair of vesicovaginal fistula

TIME OF OPERATION: ♦ **Elective** ♦ **Emergency (Category 1, 2 and 3)**

Elective: When the operation is done at a prearranged time during pregnancy to ensure the best quality of obstetrics, anesthesia, neonatal resuscitation and nursing services.

Time:

(a) **Maturity is certain**: The operation is done about 1 week prior to the expected date of confinement.

(b) **Maturity is uncertain**: Ultrasound assessment in first or second trimesters (ch. 7) if available is corroborated. Amniocentesis for l:S ratio (p. 124) is used to ensure fetal maturity. Otherwise spontaneous onset of labor is awaited and then CS is done.

Benefits and risks of elective operation: Reduction in perinatal morbidity and mortality as there is no hazard from labor and delivery process. Maternal benefits: no pelvic floor dysfunction.

Maternal risks are: Longer recovery time and hospital stay. Risks of placenta previa and hysterectomy are more in subsequent delivery (p. 486).

Emergency: **Category of C.S (NICE): Emergency:** When the operation is to be done due to an acute obstetric emergency (fetal distress). A time interval of 30 minutes between the decision and delivery is taken as reasonable. **Category 1:** When there is immediate threat to the life of the woman or the fetus. Decision delivery interval should be 30 minutes. **Category 2:** When there is maternal or fetal compromise which is not immediately life threatening. CS should be done within 75 minutes of making decision. **Category 3:** There is no maternal or fetal compromise but needs early delivery. **Category 4:** Delivery is planned to suit the woman, family members and the hospital staff.

TYPES OF OPERATIONS: ♦ **Lower segment** ♦ **Classical or upper segment**

Lower segment cesarean section (LSCS): In this operation, the extraction of the baby is done through an incision made in the lower segment through a transperitoneal approach. It is the only method practiced in present day obstetrics and unless specified, cesarean section means lower segment operation. The operation done through an extraperitoneal approach to the lower segment in infected cases is obsolete.

Classical: **In this operation, the baby is extracted through an incision made in the upper segment of the uterus**. Its indications in present day obstetrics are very much limited and the operation is only done under forced circumstances such as:

- **Lower segment approach is difficult:** (1) Dense adhesions due to previous abdominal operation (2) severe contracted pelvis (osteomalacic or rachitic) with pendulous abdomen.
- **Lower segment approach is risky:** (1) Big fibroid on the lower segment—blood loss is more and contemplating myomectomy may end in hysterectomy (2) carcinoma cervix—to prevent dissemination of the growth and postoperative sepsis (3) repair of high VVF (4) complete anterior placenta previa with engorged vessels in the lower segment—risk of hemorrhage.
- **Perimortem cesarean section:** It is done to have a live baby (rare). Perimortem section is an extreme emergency procedure. Classical section is done in a woman who has suffered a cardiac arrest. The infant may survive if delivery is done within 10 minutes of maternal death.

LOWER SEGMENT CESAREAN SECTION (LSCS)

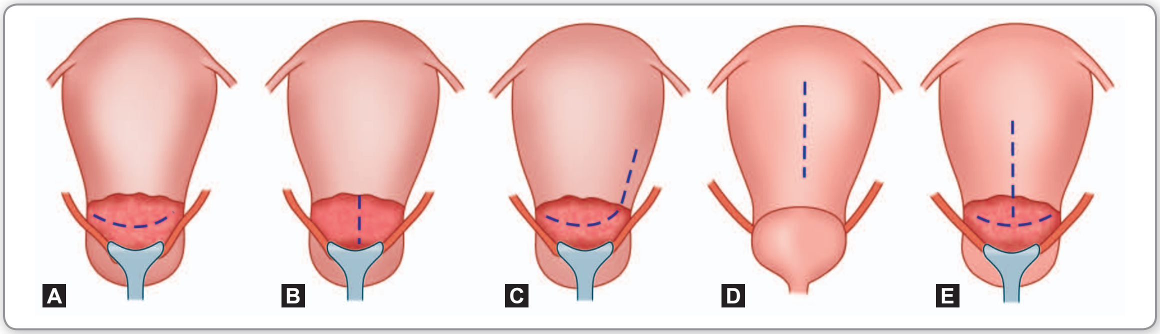
PREOPERATIVE PREPARATION

Informed written permission for the procedure, anesthesia and blood transfusion is obtained.

- Abdomen is scrubbed with soap and nonorganic iodide lotion. Hair may be clipped.
- Premedicative sedative must not be given.
- Nonparticulate antacid (0.3 molar sodium citrate, 30 mL) is given orally before transferring the patient to theater. It is given to neutralize the existing gastric acid.
- Ranitidine (H_2 blocker) 150 mg is given orally night before (elective procedure) and it is repeated (50 mg IM or IV) 1 hour before the surgery to raise the gastric pH.
- The stomach should be emptied, if necessary by a stomach tube (emergency procedure).
- Metoclopramide (10 mg IV) is given to increase the tone of the lower esophageal sphincter as well as to reduce the stomach contents. It is administered after about 3 minutes of preoxygenation in the theater.

Table 37.3: Transverse Abdominal Incision (Modified Pfannenstiel Incision)

Advantages
Postoperative comfort is more Fundus of the uterus can be better palpated during immediate postoperative period Less chance of wound dehiscence Less chance of incisional hernia Cosmetic value
Disadvantages
Takes a little longer time and as such unsuitable in acute emergency situation Blood loss is slightly more Requires competency during repeat section Unsuitable for classical operation



Figs 37.21A to E: Uterine incisions for cesarean section; (A) Lower segments transverse; (B) Lower segment vertical; (C) "J" incision; (D) Classical incision; (E) Inverted T incision

- Bladder should be emptied by a Foley catheter which is kept in place in the perioperative period.
- FHS should be checked once more at this stage.
- Neonatologist should be made available.
- Cross match blood when above average blood loss (placenta previa, prior multiple cesarean delivery) is anticipated.
- Prophylactic antibiotics should be given (IV) before making the skin incision.

♦ ***IV cannula:*** Sited to administer fluids (Ringer's solution, 5% dextrose).

♦ ***Position of the patient***: The patient is placed in the dorsal position. In susceptible cases, to minimize any adverse effects of venacaval compression, a 15° tilt to her left using a wedge till delivery of the baby should be done.

♦ ***Anesthesia***—may be spinal, epidural or general (see p. 593). However, choice of the patient and urgency of delivery are also considered.

♦ ***Antiseptic painting:*** The abdomen is painted with 7.5% povidone-iodine solution or savlon lotion and to be properly draped with sterile towels.

♦ ***Incision on the abdomen:*** The surgeon may choose either a vertical or a transverse skin incision. Vertical incision may be infraumbilical midline or paramedian. Transverse incision, modified Pfannenstiel is made 3 cm above the symphysis pubis. Transverse incision has certain benefits (Table 37.3).

***Packing*: The Doyen's retractor (Fig. 42.14) is introduced.** The peritoneal cavity is now packed off using two taped large swabs. The tape ends are attached to artery forceps. This will minimize spilling of the uterine contents into the general peritoneal cavity.

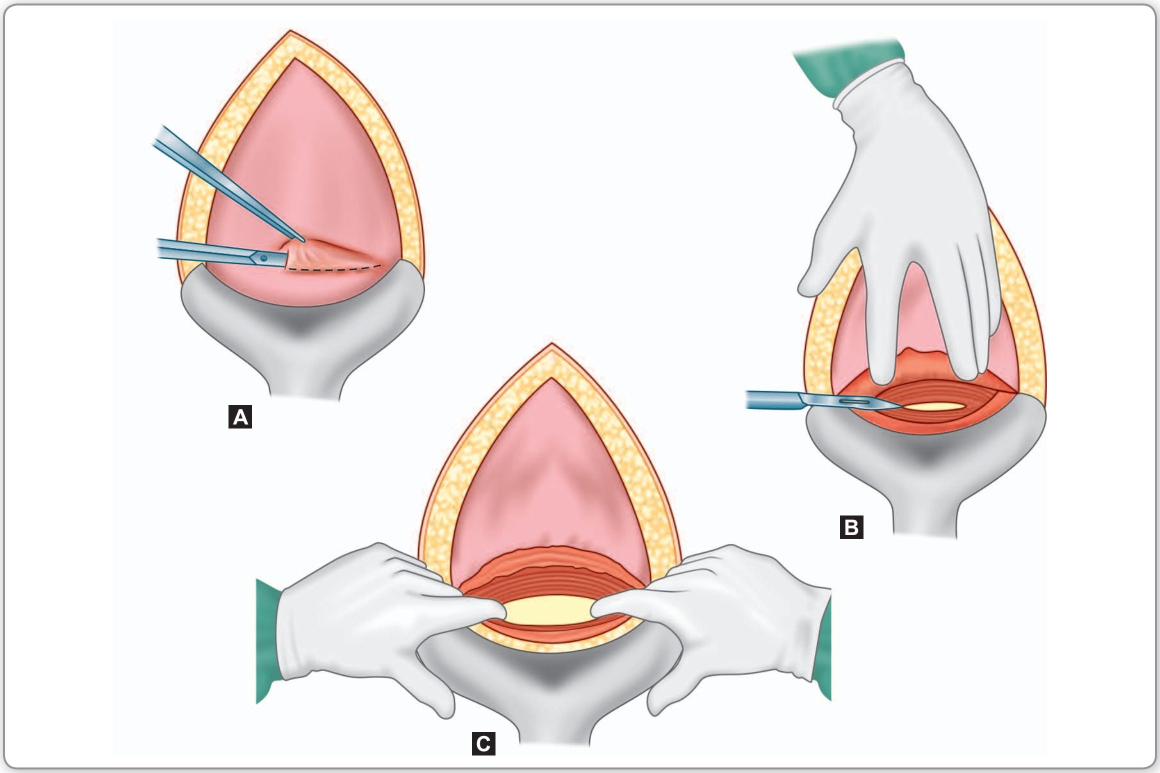
Uterine incision (Figs 37.21A to E)

(a) **Peritoneal incision:** The loose peritoneum of the uterovesical pouch is cut transversely across the lower segment with convexity downwards at about 1.25 cm (0.5") below its firm attachment to the uterus. The lower flap of the peritoneum is pushed down a little.

(b) **Muscle incision (Figs 37.22A to C): The most commonly used incision (90%) is low transverse. Advantages are**: Ease of operation; less bladder dissection, less blood loss, easy to repair, complete reperitonization, less adhesion formation, less risk of scar rupture when trial (VBAC) of labor (p. 384) is given for subsequent delivery.

Other types of uterine incisions are (Figs 37.21A to E): (a) **Lower vertical**—may be extended upwards when needed. (b) classical incision (upper segment). (c) "J" incision—upward vertical extension of the initial transverse incision. (d) inverted "T" incision—upward extension from the mid-transverse incision. **Vertical uterine incision** is made when the lower segment is poorly developed or there is complete anterior placenta previa (p. 292) or any myona occupying the lower segment.

(b) **Low transverse incision:** A small transverse incision is made in the midline by a scalpel at a level slightly below the peritoneal incision until the membranes of the gestation sac are exposed. Two index fingers are then inserted



Figs 37.22A to C: Steps of LSCS: (A) The loose peritoneum on the lower segment is cut transversely; (B) A short incision is made in the midline down to the membranes; (C) The incision of the lower segment is being enlarged using index finger of both hands

Steps contd...

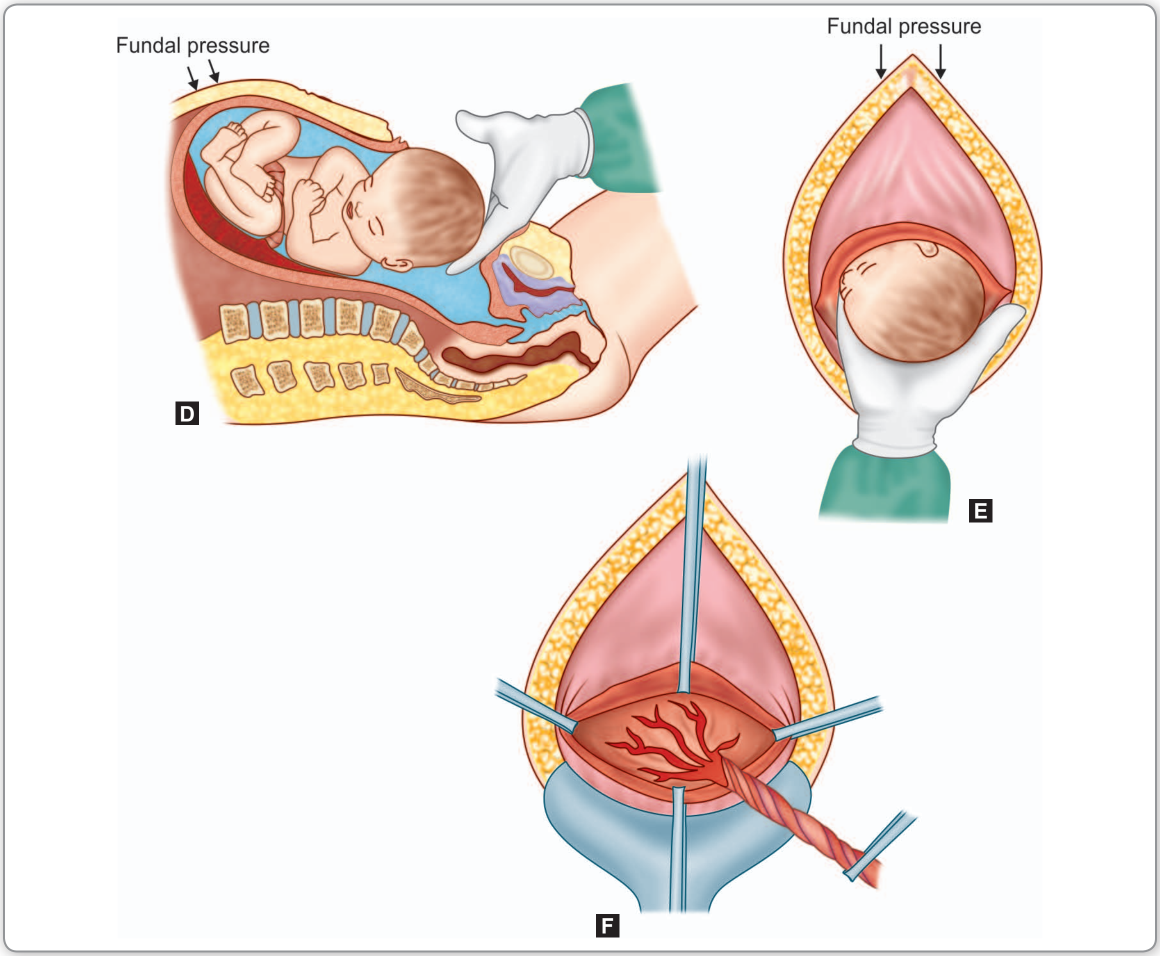
through the small incision down to the membranes and the **muscles of the lower segment are split transversely across the fibers**. **This method minimizes the blood loss but requires experience.** Alternatively, the incision may be extended on either side using a pair of a curved scissors to make it a curved one of about 10 cm (4") in length, the concavity directed upwards.

Delivery of the head (Fig. 37.23): The membranes are ruptured if still intact. The blood mixed amniotic fluid is sucked out by continuous suction. **The Doyen's retractor is removed.** The head is delivered by hooking the head with the fingers which are carefully insinuated between the lower uterine flap and the head until the palm is placed below the head. The head is delivered by elevation and flexion using the palm to act as a fulcrum. As the head is drawn to the incision line, the assistant is to apply pressure on the fundus. **If the head is jammed**, an assistant may push up the head by sterile gloved fingers introduced into the vagina. The head can also be delivered using either Wrigley's or Barton's forceps.

Delivery of the trunk: As soon as the head is delivered, the mucus from the mouth, pharynx and nostrils is sucked out using rubber catheter attached to an electric sucker. After the delivery of the shoulders, intravenous oxytocin 20 units or methergine 0.2 mg is to be administered. The rest of the body is delivered slowly and the baby is placed in a tray placed in between the mother's thighs with the head tilted down for gravitational drainage. The cord is cut in between two clamps and the baby is handed over to the pediatrician. **The Doyen's retractor is reintroduced.**

The **optimum interval between uterine incision and delivery should be less than 90 seconds**. Interval > 90 seconds are associated with poor Apgar scores. There is reflex uterine vasoconstriction following uterine incision and manipulation.

Removal of the placenta and membranes: By this time, the placenta is separated spontaneously. The placenta is extracted by traction on the cord with simultaneous pushing of the uterus towards the umbilicus per abdomen



Figs 37.23D to F: Steps of LSCS contd.: (D) Sagittal section showing insinuation of the fingers between the lower uterine flap and the fetal head until the posterior surface is reached; (E) Methods of delivery of the head; (F) Placenta is being delivered

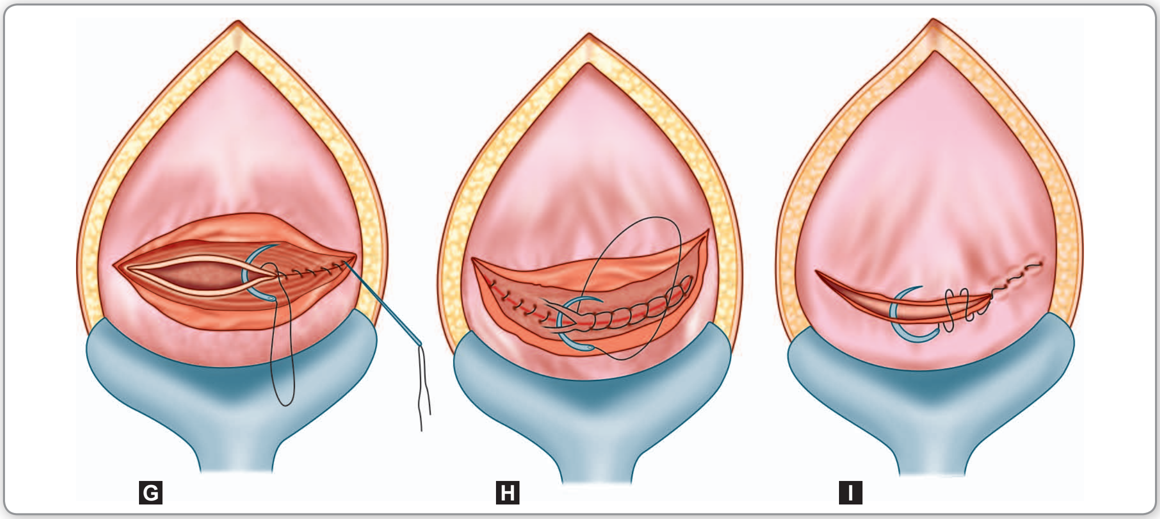
Steps contd...

using the left hand (controlled cord traction). **Routine manual removal should not be done**. Advantages of spontaneous placental separation are: less blood loss and less risk of endometritis. The membranes are carefully removed preferably intact and even a small piece, if attached to the decidua should be removed using a dry gauze. **Dilatation of the internal os is not required. Exploration of the uterine cavity is desirable**.

Suture of the uterine wound (Fig. 37.24): The suture of the uterine wound is done with the uterus keeping in the abdomen. Some, however, prefer to eventrate the uterus prior to suture. The margins of the wound are picked up by Allis tissue forceps or Green Armytage hemostatic clamps (four are required, one each for angle and one for each margin). **The uterine incision is sutured in three layers**.

First layer: The first stitch is placed on the far side in the lateral angle of the uterine incision and is tied. **The suture material** is No "0" chromic catgut or vicryl and the needle is round bodied. **A continuous running suture taking deeper muscles excluding or including the decidua** (very difficult to exclude) ensures effective apposition of the tissues; the stitch is ultimately tied after the suture includes the near end of the angle.

Second layer: A similar continuous suture is placed taking the superficial muscles and adjacent fascia overlapping the first layer of suture. Uterine muscles may be closed using a continuous single layer stitch taking full thickness muscle and decidua. There is controversy as regard the place of single layer or double layer closure in relation to



Figs 37.24G to I: Steps of LSCS contd.: (G) Inserting the continuous catgut (No. '0') suture taking deeper muscles excluding the decidua; (H) Similar method of continuous suture taking superficial muscles and fascia down to the first layer of suture; (I) Continuous peritoneal catgut suture

the risk of subsequent scar rupture. The peritoneal flaps may be apposed by continuous inverting suture (to prevent any raw surface).

Nonclosure of visceral and parietal peritoneum is preferred.

Concluding part: **The mops placed inside are removed and the number verified.** Peritoneal toileting is done and the blood clots are removed meticulously. The tubes and ovaries are examined. **Doyen's retractor is removed.** After being satisfied that the uterus is well contracted, the abdomen is closed in layers. The vagina is cleansed of blood clots and a sterile vulval pad is placed.

POSTOPERATIVE CARE

First 24 hours: **(Day 0)**

- **Observation** for the first 6–8 hours is important. Periodic checkup of pulse, BP, amount of vaginal bleeding and behavior of the uterus (in low transverse incision) is done and recorded.
- **Fluid:** Sodium chloride (0.9%) or Ringer's lactate drip is continued until at least 2.0–2.5 L of the solutions are infused. Blood transfusion is helpful in anemic mothers for a speedy post-operative recovery. Blood transfusion is required if the blood loss is more than average during the operation (average blood loss in cesarean section is approximately 0.5–1.0 L).
- **Oxytocics:** Injection oxytocin 5 units IM or IV (slow) or methergine 0.2 mg IM is given and may be repeated.
- **Prophylactic antibiotics** (cephalosporins, metronidazole) for all cesarean delivery (see p. 726) is given for 2–4 doses. Therapeutic antibiotic is given when indicated.
- **Analgesics** in the form of pethidine hydrochloride 75–100 mg is administered and may have to be repeated.
- **Ambulation:** The patient can sit on the bed or even get out of bed to evacuate the bladder, provided the general condition permits. She is encouraged to move her legs and ankles and to breathe deeply to minimize leg vein thrombosis and pulmonary embolism.
- **Baby** is put to the breast for feeding after 3–4 hours when mother is stable and relieved of pain.

Day 1: • **Oral feeding** in the form of plain or electrolyte water or raw tea may be given. Active bowel sounds are observed by the end of the day.

Day 2: • **Light solid diet** of the patient's choice is given. • **Bowel care:** 3–4 teaspoons of lactulose is given at bed time, if the bowels do not move spontaneously.

Day 5 or day 6: The abdominal skin stitches are to be removed on the D-5 (in transverse) or D-6 (in longitudinal).

Discharge: The patient is discharged on the day following removal of the stitches, if otherwise fit. Usual advices like those following vaginal delivery are given. Depending on postoperative recovery and availability of care at home, patient may be discharged as early as third to as late as seventh postoperative days.

CLASSICAL CESAREAN SECTION

This is relatively easy to perform (Table 37.4). Abdominal incision is always longitudinal (paramedian) and about 15 cm (6") in length, 1/3rd of which extends above the umbilicus. A longitudinal incision of about 12.5 cm (5") is made on the midline of the anterior wall of the uterus starting from below the fundus. The incision is deepened along its entire length until the membranes are exposed which are punctured. In about 40% cases, the placenta is encountered. In such cases, fingers are slipped between the placenta and the uterine wall until the membranes are reached. **The baby is delivered commonly as breech extraction**. Intravenous oxytocin 5 IU IV (slow) or methergine 0.2 mg is administered following delivery of the baby. The uterus is eventrated. The placenta is extracted by traction on the cord or removed manually.

Table 37.4: Merits and Demerits of Lower Segment Operation Over Classical

	Lower Segment	**Classical**
Techniques	Technically slight difficult	Technically easy
	Blood loss is less	Blood loss is more
	The wall is thin and as such apposition is perfect	The wall is thick and apposition of the margins is imperfect
	Perfect peritonization is possible	Not possible
	Technical difficulty in placenta previa or transverse lie	Comparatively safer in such circumstances
Post-operative	Hemorrhage and shock—less	More
	Peritonitis is less even in infected uterus because of perfect peritonization and if occurs, localized to pelvis	Chance of peritonitis is more in presence of uterine sepsis
	Peritoneal adhesions and intestinal obstructions are less	More because of imperfect peritonization
	Convalescence is better	Relatively delayed
	Morbidity and mortality are much lower	Morbidity and mortality are high
	The scar is better healed because of:	**The scar is weak because of:**
Wound healing	Perfect muscle apposition due to thin margins	Imperfect muscle apposition because of thick margins
	Minimal wound hematoma	More wound hematoma formation
	The wound remains quiescent during healing process	The wound is in a state of tension due to contraction and relaxation of the upper segment. As a result, the knots may slip or the sutures may become loose
	Chance of gutter formation is unlikely	Chance of gutter formation on the inner aspect is more.
During future pregnancy	Scar rupture is less (see chapter 22) 0.5–1.5%	More risk of scar rupture (see chapter 22). 4–9%

Suture of the uterine incision: The uterus is sutured in three layers.

- **A continuous suture** is placed with chromic catgut No "0" or vicryl taking deep muscles excluding the decidua.
- **A second layer of interrupted sutures** (1 cm apart) using chromic catgut No. "1" or vicryl taking the entire depth of superficial muscles down to the first layer of suture.
- **The third layer of continuous suture** taking the peritoneum with the adjacent muscles using chromic catgut No "0" and round-bodied needle.

The uterus is returned back into the abdominal cavity. Packings are removed; peritoneal toileting is done and the abdomen is closed in layers.

Merits and Demerits of Lower Segment Transverse over Vertical Incision

Parameters	Lower Segment Transverse	Lower Segment Vertical
Extension of incision	■ May occur to involve the uterine vessels	■ May occur to involve the upper segment or downward the bladder
Bladder dissection	■ Minimal	■ More when extends inferiorly
Uterine closure	■ Easy	■ Difficult
Muscle apposition	■ Good	■ Often imperfect
Reperitonization	■ Complete	■ Often imperfect
Intraoperative bleeding	■ Less	■ More
Subsequent adhesions	■ Less	■ More

Lower segment transverse incision wound healing is perfect and the scar is sound. This is due to: (a) perfect muscle apposition; (b) less wound hematoma; (c) less gutter formation and (d) wound remains quiescent during healing. The risk of scar rupture is less (0.2–1.5%) compared to lower segment vertical incision (1–7%).

COMPLICATIONS OF CESAREAN SECTION

The complications are related either due to the ■ **operations** (inherent hazards), or due to ■ **anesthesia** (see p. 593). The complications are grouped into: ♦ Maternal ♦ Fetal

The maternal complications may be:

■ **Intraoperative** ■ **Postoperative**

INTRAOPERATIVE COMPLICATIONS

- **Extension of uterine incision** to one or both the sides. This may involve the uterine vessels to cause severe hemorrhage, may lead to broad ligament hematoma formation.
- **Uterine lacerations** at the lower uterine incision—may extend laterally or inferiorly into the vagina.
- **Bladder injury**—is rare in a primary CS but may occur in a repeat procedure. Should a bladder injury occur, repair is done with a two layer closure with 2–0 chromic catgut. Continuous bladder drainage is then maintained for 7–10 days.
- **Ureteral injury** is rare (1 in 1,000 procedures). Injury occurs during control of bleeding from lateral extensions.
- **Gastrointestinal tract injury** is rare unless there is prior pelvic/abdominal adhesions.
- **Hemorrhage** may be due to uterine atony or uterine lacerations. Medical management should be started (see p. 481). Surgical management is done (see p. 482) where there are wound lacerations. Blood transfusion is needed.
- **Morbid adherent placenta (placenta accreta)** is commonly seen in cases with placenta previa who had prior cesarean delivery. Total hysterectomy is often needed for such a case to control hemorrhage (see p. 483).

POSTOPERATIVE COMPLICATIONS

MATERNAL: ♦ **Immediate** ♦ **Remote**

IMMEDIATE

- **Postpartum hemorrhage:** The blood loss in cesarean section is more often underestimated. It is mostly related to uterine atony but blood coagulation disorders may rarely occur.
- **Shock:** While most often it is related to the blood loss (see p. 703), it may occur when the operation is done following prolonged labor without correcting preexisting dehydration and ketoacidosis.
- **Anesthetic hazards:** These are mostly associated in emergency operations. The hazards are related to aspiration of the gastric contents. The result may be aspiration atelectasis or aspiration pneumonitis (Mendelson's syndrome, see p. 596). Others are: hypotension and spinal headache (p. 595).
- **Infections:** The common sites are uterus (endomyometritis), urinary tract, abdominal wound, peritoneal cavity (peritonitis) and lungs. Septic pelvic thrombophlebitis may be associated with endometritis (see p. 502). Risk factors for infection are: prolonged duration of labor and that of rupture of membranes, repeated number of vaginal examinations. Prophylactic antibiotics reduce the risk significantly.
- **Intestinal obstruction:** The obstruction may be mechanical due to adhesions or bands, or paralytic ileus following peritonitis.
- **Deep vein thrombosis and thromboembolic disorders** are more likely to occur following cesarean section than vaginal delivery. Septic thrombophlebitis is also a known complication (see p. 509).
- **Wound complications:** Abdominal wound sepsis is quite common. The complications, which are detected on removal of the skin stitches, are: (1) sanguineous or frank pus (2) hematoma (3) dehiscence (peritoneal coat intact) (4) burst abdomen (involving the peritoneal coat) and (5) rarely necrotizing fasciitis.
- **Secondary postpartum hemorrhage**.

REMOTE: ♦ **Gynecological** ♦ **General surgical** ♦ **Future pregnancy**

Gynecological: Menstrual excess or irregularities, chronic pelvic pain or backache.

General surgical: Incisional hernia, intestinal obstruction due to adhesions and bands.

Future pregnancy: There is risk of scar rupture (see ch. 22).

FETAL: **Iatrogenic prematurity and development of RDS** is not uncommon following cesarean delivery. This is seen when fetal maturity is uncertain. Accidental scalpel injury to the baby may occur.

MATERNAL AND PERINATAL MORTALITY: ***Maternal:*** Overall maternal mortality ranges between 6 to 22 per 100,000 procedure. But with adverse patient profile and suboptimal circumstances, which are often interrelated, the maternal mortality ranges from 0.1% to 1%. **The causes of death are:** (1) hemorrhage and shock (2) anesthetic hazards (3) infection and (4) thromboembolic disorders.

Fetal: The perinatal mortality ranges from 5% to 10% and the deaths are mostly related to emergency operations and the complicating factors for which the operations are done. **The causes of death are**: (1) asphyxia may be preexisting (2) RDS (3) prematurity (4) infection and (5) intracranial hemorrhage—attempting breech delivery through a small incision.

Extraperitoneal cesarean section was practised in the past in cases with severe infection. Lower segment is approached extraperitoneally by dissecting through the space of Retzius. Currently, with the availability of potent antimicrobial agents, this is rarely performed.

Cesarean hysterectomy: Cesarean hysterectomy refers to an operation where cesarean section is followed by removal of the uterus. The common conditions are: (1) morbid adherent placenta (2) atonic uterus and uncontrolled postpartum hemorrhage (3) big fibroid (parous women) (4) extensive lacerations due to extension of tears with broad ligament hematoma (5) grossly infected uterus and (6) rupture uterus.

Measures to Reduce Cesarean Births

A Breech presentation	B Dystocia	C Fetal distress, Vaginal birth after cesarean section	D Amnioinfusion	E Symphysiotomy	F Destructive operations
♦ External cephalic version in selected cases (p. 440) ♦ Assisted vaginal delivery of breech in selected cases (see p. 441)	♦ Partographic monitoring in labor management (see p. 607) ♦ Active management of labor (p. 605)	♦ To confirm fetal acidosis by fetal blood sampling (see p. 696) ♦ In selected cases VBAC is successful (see p. 384)	♦ Management of cases with variable or early FHR deceleration due to oligohydramnios, meconium stained liquor (see p. 698)	♦ Selected cases of obstructed labor with live fetus (see p. 468)	♦ Delivery by craniotomy in selected cases of obstructed labor with dead or moribund fetus (see p. 666)

Peripartum hysterectomy is the surgical removal of the uterus either at the time of cesarean delivery or in the immediate postpartum period (even following vaginal delivery). **Subtotal hysterectomy** is commonly done as an emergency (unplanned) procedure. **Benefits of subtotal hysterectomy are**: Less operating time, less blood loss, less risk of injury to other organs (bladder, ureter) and less postoperative morbidity.

Perimortem cesarean delivery refers to the caesarean delivery of a woman who is expected to die within next few moments or has just died. It is done within 4–5 minutes of start of cardiopulmonary resuscitation (CPR) when the fetus is alive.

SYMPHYSIOTOMY

Symphysiotomy is the operation designed to enlarge the pelvic capacity by dividing the symphysis pubis. In the tropical countries, its place has to be duly considered in the perspective of wide prevalence of obstructed labor cases which are rushed to the referral hospitals in a bad shape.

The cases are judiciously selected and symphysiotomy may be done as an alternative to risky cesarean section when there is a likelihood of scar rupture in subsequent labors. Moreover, symphysiotomy produces permanent enlargement of the pelvis, as such future dystocia will be unlikely. **The operation should be done in established obstruction and not when it is only anticipated. The conditions to be fulfilled are:** (1) the pelvis should not be severely contracted; isolated outlet contraction is ideal (2) vertex must be presenting and (3) the FHS must be present.

The operation consists of dividing the symphysis pubis strictly in the midline from above downwards until the arcuate ligament is cut. The fingers of the left hand in the vagina displace the urethra, while catheter is in, to one side. The baby is delivered spontaneously with liberal episiotomy or by traction—ventouse (preferable) or forceps.

Complications: Retropubic pain, osteitis pubis, stress urinary incontinence and rarely vesicovaginal fistula.

QUESTIONS

Related theory questions (Long and Short), Obstetric Case Discussions, Viva table discussions, Postoperative word round discussions, and MCQs are discussed in author's books:

1. **Bedside Clinic and Viva Voce:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

For further reading:

2. **Master pass in Obstetrics and Gynaecology:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

Chapter 38

Safe Motherhood, Epidemiology of Obstetrics

SAFE MOTHERHOOD

Considering the high maternal deaths in the developing countries, WHO in 1987 conceived the idea of "**Safe Motherhood Initiative**" at a conference in Nairobi, Kenya. It is a **global effort to reduce the maternal deaths by at least half by 2000 AD, now extended to 2015**. The **objectives** are to enhance the quality and safety of girls and women's lives through adoption of a combination of health and non-health strategies.

Maternal and child health promotion is one of the key commitments in the WHO constitution.

Maternal death is a tragedy for an individual woman, for her family and the community. Worldwide nearly 600,000 women between the age of 15 and 49 die every year due to complications arising from pregnancy and childbirth. **This means, almost every minute of every year, there is a maternal death.** 99% of which occur in the developing countries. **Majority (80%) of these deaths are preventable**.

In developed countries, the maternal mortality ratio is around 8–17 per 100,000 live births and in the developing countries the ratio is 20 times higher. It varies between 240 and 730 per 100,000 live births depending on the region. **The life time risk of dying from pregnancy-related complications for a woman of developing country is one in 75 compared to one in 7300 in industrialized countries**. This reflects the huge difference in national commitment.

Safe motherhood initiative (SMI) is a global effort and it is designed to operate through partners**:** (a) Government agencies, (b) Non-government agencies and (c) Other groups and individuals. Safe motherhood initiative aims to **improve women's health through social, community and economic interventions**.

What is known worldwide about **adverting maternal death** is country's overall economic wealth is not the only important determinant. Experts from WHO, UNFPA, UNICEF, IPPFF, the World Bank, the population council and other national and international agencies concerned with safe motherhood **concluded that it is possible to reduce maternal mortality significantly with limited investment and effective policy interventions** (Fig. 38.1 and Table 38.1). According to national and international human rights treaties (1948) **safe motherhood is considered a human rights issue**. Therefore, it is considered that maternal death is the reflexion of '**social disadvantage' not merely a 'health disadvantage**'.

Basic facts underlying the clinical causes of maternal deaths are:

♦ **Low social status of girls and women (gender inequality)** is considered a fundamental determinant of maternal mortality. In the developing countries girls and women face the following difficulties: (i) Limited access to economic resources, (ii) Less opportunity for basic education, (iii) Excess physical

Table 38.1: NATIONAL SOCIODEMOGRAPHIC GOALS FOR 2015
National Population Policy 2000, National Rural Health Mission (NRHM) 2005

Parameters	Current Level	2015
Total fertility rate	2.9	< 2.1
Maternal mortality ratio (per 100,000 live births)	254	< 100
Infant mortality	57	< 30
Antenatal care (%)	60	100
Institutional deliveries (%)	34	80
Deliveries by skilled personnel (%)	42	100

work, poor diet, (iv) Less ability to make decisions, (v) Unplanned childbirth that are too early, too frequently, too many or too late and (vi) Less utilization of essential obstetric services.

- **Poor nutrition** contributes to poor maternal health and results in poor pregnancy outcome.
- **Lack of** *skilled attendant during the time of delivery, appropriate referral system, emergency obstetric care (EmOC), sex education, family planning* and *safe abortion services* are the important areas. In the developing countries, only 50% women deliver with the help of a skilled attendant, only 40% deliver in a hospital or health center and about 15% face life-threatening complications.

Millennium Development Goals (MDG) 4 and 5 for Maternal, Newborn and Child Health Care (MNCH) — (WHO, UNICEF, UNPA, World Bank, Other Organizations 2007)

- Reduction of maternal mortality ratios by 75% between 1990 and 2015
- Expected annual decline in MMR from 1990 is 5.5%
- Reduction of infant mortality below 30 per 1000 live birth by the year 2015
- 90% of all births should be assisted by skilled attendants by 2015
- Access for all who need reproductive health services by 2015
- Other goals: Gender equity, Reduction of poverty, Education of girls and women

In an attempt to improve the maternal mortality situation in India, all the states have been categorized into groups: (A) **Empowered Action Group (EAG)**. The states in this group are: Bihar, Jharkhand, MP, Chhattisgarh, Orissa, Rajasthan, UP, Uttarakhand and Assam. (B) **Southern states:** AP, Karnataka, Kerala, Tamil Nadu and (C) **Other states:** The remaining states and the union territories. It is striking to note that currently MMR in India, has declined from 212 (2007–2009) to 178 (2010–2012). Decline has been observed in all the groups.

Lifetime risk is defined as the probability of dying of a woman in her reproductive age (15–49 years), due to causes in pregnancy, childbirth or within 6 weeks of childbirth. In India, presently it is 0.4%.

OBSTETRIC CARE AND THE SOCIETY: Obstetric care is by and large a preventive medicine. **Social obstetrics is defined as the obstetric care of a community that can be provided in the perspective of its social, economic, environmental and cultural background.** This will encompass many aspects of the community. Study of these factors is essential to improve the maternal and perinatal outcome. Obstetric problem of a developing country (high maternal and perinatal mortality and morbidity) is completely different from that of a developed country (congenital malformation and genetic problems).

WOMEN'S HEALTH (MDGs) BEYOND 2015: See p. 690.

REPRODUCTIVE AND CHILD HEALTH (RCH) CARE

RCH care is an integrated and composite approach to improve the health status of women and children in India. It incorporates the inputs of the Government of India (NRHM–2005, NPP–2000) and the supports of donor agencies like World Bank, WHO, European Commission and others.

The aims of RCH care are: (i) Safe motherhood, (ii) Child survival, (iii) Adolescent health, (iv) Family planning and (v) Prevention and management of infections (STI/RTI). New initiatives have been taken by Government of India (NRHM, 2005) to improve RCH care. Partnership for maternal, newborn and child health (PMNCH) was initiated to reach the **Millennium Development Goals 4 and 5**. The main objective is to reduce maternal mortality by 3/4 and child mortality by 2/3 by 2015. The new initiatives include: (a) To provide basic and comprehensive Emergency Obstetric Care (EmOc) and essential newborn care, (b) To strengthen and to make all PHCs, CHCs and FRUs operational as 24 hours delivery centers in a phased manner.

RCH INTERVENTIONS

A. SAFE MOTHERHOOD

Antenatal Care (p. 106)	Intranatal Care (p. 155)	Essential Newborn Care (p. 517)
Essential Obstetrics Care ▪ **Early registration** of pregnancy (12–14 wks) ▪ **A minimum of 4 antenatal visits (WHO), Govt. of India minimum 3 visits** (1st at 16 weeks, 2nd at 24–28 weeks, 3rd at 32 weeks and 4th visit at 36 weeks) should be carried out. **Risk approach to identify high-risk cases** during pregnancy (see p. 716) labor (see p. 717) or puerperium (see p. 717) ▪ To strengthen the referral system ▪ **Routine immunization** with tetanus toxoid and **supplementary iron-folic acid (IFA)** therapy daily for at least 100 days after the first trimester	▪ **Institutional deliveries** in 80% cases and 100% deliveries by **skilled persons**. ▪ **Three cleans** (hands, perineal area and umbilical area) must be maintained. **Postnatal Care (p. 178)** ▪ **Support** to restore the health of mother and care of the newborn ▪ **Family planning services** to prevent unplanned pregnancy. ▪ **Safe abortion services.** ▪ **Breastfeeding** — early and exclusive.	▪ **Clean delivery** ▪ **Resuscitation** at birth ▪ **Prevention** of hypothermia ▪ **Prevention** of infection ▪ **Breastfeeding**—early and exclusive ▪ **Ten baby** friendly hospital initiatives (see p. 519) ▪ **Referral** of sick newborn.

Risk assessment is not once only but a continued procedure throughout and the woman is referred to a higher level of care when needed.

B. ADOLESCENT AND REPRODUCTIVE HEALTH: 20% of the total population in India are adolescent (age group of 10–19 years), of which half are either sexually active or married. **Problems to overcome are:** (i) Early motherhood—risk of the mother and her newborn, (ii) Undernutrition and anemia (iii) Psychological immaturity and vulnerability, (iv) Consequences of unprotected sex—unwanted pregnancy, unsafe abortion, STIs and RTIs.

C. REPRODUCTIVE TRACT INFECTIONS (RTIs) AND SEXUALLY TRANSMITTED INFECTIONS (STIs): RTIs are mainly due to unsafe abortion, uncleaned delivery, poor menstrual hygiene and unhygienic IUD insertion. All these are avoidable by proper preventive and curative measures under the RCH program.

D. GENDER ISSUE: Gender inequality unfavorable to woman and girl child is an important hindrance to social development. This is evident in terms of food, education, medical care, access to financial resources and decision making. Gender discrimination is to be removed.

E. OTHERS: RCH II, SKILLED BIRTH ATTENDANT AND UNIVERSAL IMMUNIZATION TO CHILDREN: In India RCH II, highlights the following areas—(i) Community need assessment approach (CNAA), (ii) Upgradation of facilities at first referral unit (FRU) for comprehensive emergency obstetric and newborn care at the subdistrict levels, (iii) Permission for **skilled birth attendant** to administer certain **life-saving drugs** and to perform certain **life-saving interventions** under specified situations (see below).

Skilled birth attendant is an accredited health professional (midwife, doctor or nurse) who has been educated and trained to proficiency in the skills of managing normal pregnancy, labor and puerperium. She is also able to identify complications in women and newborns and to organize referral.

Life-saving drugs are: Misoprostol (prevention of PPH), Inj. Oxytocin, (Management of PPH), Inj. $MgSO_4$ (eclampsia), Ampicillin, Metronidazole (infection) and the life-saving interventions are: Digital removal of products of conception (incomplete abortion with bleeding), active management of 3rd stage of labor (see p. 165), maintaining a partograph (early diagnosis of prolonged and obstructed labor p. 606-7).

In rural India, under NRHM and Janani Suraksha Yojana (JSY) Scheme, Accredited Social Health Activists (ASHA), work as a link person (female) among the beneficiary at village level with the ANM and doctor at the FRUs. This is to improve the rate of institutional delivery with encouragement and incentives.

Emergency Obstetric Care (EmOC) is life saving, as the time between onset of an emergency during delivery and availability of treatment is very crucial.

The Comprehensive EmOC that has to be provided at FRUs are: anesthetic service, vacuum delivery, blood transfusion facilities, cesarean delivery, manual removal of placenta, safe abortion services, contraceptive services including sterilization and facilities of referral with transport.

EPIDEMIOLOGY OF OBSTETRICS

The sensitive index of the quality of the health care delivery system of a country as a whole or in part, is reflected by its maternal and perinatal mortality rates. With 16% of world's population, India accounts for over 20% of world's maternal deaths. Maintenance of accurate vital statistics (record keeping of the vital events such as births and deaths), their critical analysis and formulation of the preventive measures contributed to a great extent in the reduction of deaths in advanced countries. ***Unregulated fertility, unsafe abortion, inadequate antenatal care and lack of trained birth attendants are mainly recognized as the factors responsible for high maternal and perinatal deaths in the developing countries.***

<table>
<tr><th colspan="7">Maternal Mortality Estimates (WHO, UNICEF, UNFPA, World Bank – 2005)</th></tr>
<tr><td colspan="7">Maternal Mortality Ratios in the Developed Word</td></tr>
<tr><td>SWEDEN</td><td>USA</td><td colspan="2">NETHERLANDS</td><td colspan="2">UNITED KINGDOM</td><td>JAPAN</td></tr>
<tr><td>3</td><td>11</td><td colspan="2">6</td><td colspan="2">8</td><td>6</td></tr>
<tr><td colspan="7">Maternal Mortality Ratios in Asia</td></tr>
<tr><td>SRI LANKA</td><td>THAILAND</td><td colspan="2">INDIA (2010-12)</td><td>CHINA</td><td>BANGLADESH</td><td>NEPAL</td></tr>
<tr><td>46</td><td>44</td><td colspan="2">178</td><td>56</td><td>380</td><td>740</td></tr>
<tr><td colspan="7">Maternal Mortality Ratio in India by State (RGI; SRS: 2012-2013)</td></tr>
<tr><td>KERALA</td><td>WEST BENGAL</td><td>ORISSA</td><td>BIHAR</td><td>MP</td><td>RAJASTHAN</td><td>MAHARASHTRA</td></tr>
<tr><td>66</td><td>117</td><td>235</td><td>219</td><td>230</td><td>255</td><td>87</td></tr>
</table>

MATERNAL MORTALITY

DEFINITION OF MATERNAL DEATHS: Death of a woman while pregnant or within 42 days of the termination of pregnancy irrespective of the duration and the site of pregnancy, from any cause related to or aggravated by the pregnancy or its management but not from accidental or incidental causes.

MATERNAL MORTALITY RATIO (MMR): The MMR is expressed in terms of such maternal deaths per 100,000 live births. In most of the developed countries, the MMR varies from 4–40 per 100,000 live births. In the developing countries, it varies from 100–700 with India having about 254 per 100,000 live births. Most of the figures of the developing countries are however, based on the data from teaching hospitals as very often, the vital statistics from the whole country are not available.

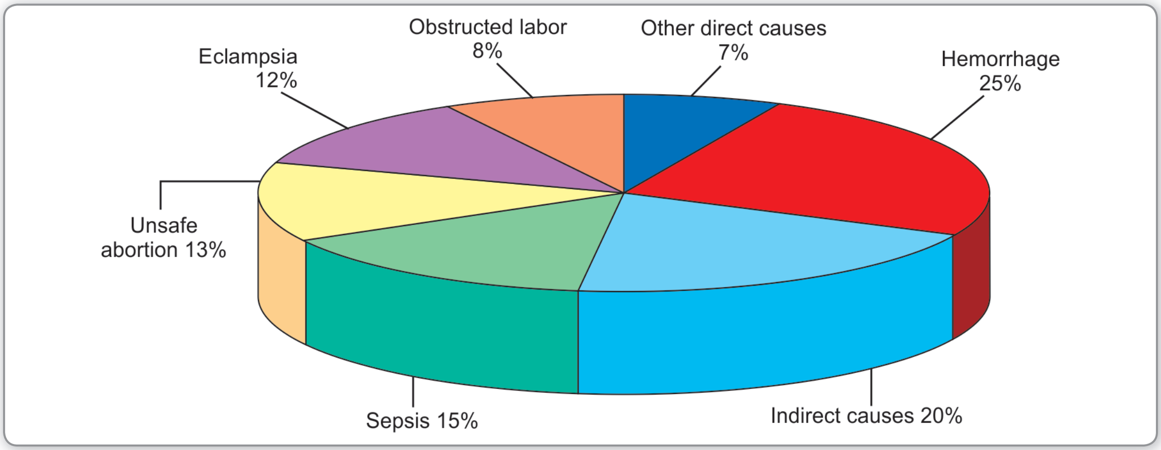


Fig. 38.1: Important causes of maternal deaths

MATERNAL MORTALITY RATE indicates the number of maternal deaths divided by the number of women of reproductive age (15–49). It is expressed per 100,000 women of reproductive age per year. In India, it is about 120 as compared to 0.5 of United States.

The term **reproductive mortality** is used currently to include maternal mortality and mortality from the use of contraceptives.

MAGNITUDE OF THE PROBLEM: Worldwide, every year approximately eight million women suffer from pregnancy-related complications. Over half a million of them, die as a result. The problems of maternal mortality and morbidity are greatest (99%) for the poor women in the developing countries. **One woman in 11 may die of pregnancy-related complications in developing countries, compared to one in 5000 in developed countries.** Here lies the major discrepancies in global health. It is further estimated that **for one maternal death at least 16 more suffer from severe morbidities**.

CLASSIFICATION: ♦ **Direct** ♦ **Indirect** ♦ **Non-obstetric**

- **Direct obstetric deaths** (75%) are those resulting from complications of pregnancy, delivery or their management. Such conditions are abortion, ectopic gestation, preeclampsia-eclampsia, antepartum and postpartum hemorrhage and puerperal sepsis (Table 38.2).
- **Indirect deaths (25%)** include conditions present before or developed during pregnancy but aggravated by the physiological effects of pregnancy and strain of labor. These are anemia, cardiac disease, diabetes, thyroid disease, etc. of which anemia is the most important single cause in the developing countries. Viral hepatitis when endemic, contributes significantly to maternal deaths.
- **Non-obstetric or fortuitous deaths:** Accidents, typhoid and other infectious diseases.

FACTORS ASSOCIATED WITH MATERNAL MORTALITY

Age: The optimum reproductive efficiency appears to be between 20–25 years. In the young adolescent, pregnancy carries a higher risk due to preeclampsia, cephalopelvic disproportion and uterine inertia. In women aged 35 years or above the risk is 3–4 times higher.

Parity: The risk is slightly more in primigravida but it is 3 times greater in para, 5 or above where postpartum hemorrhage, malpresentations and rupture uterus are more common. The risk is lowest in the second pregnancy.

Socioeconomic strata: Mortality ratios are higher in women belonging to low socioeconomic strata as these women are likely to be less privileged in the fields of nutrition, housing, education and antenatal care.

Table 38.2: Important Causes of Maternal Deaths and Main Interventions

Causes	Percentage	Proven Interventions
Hemorrhage: Mostly due to postpartum hemorrhage. Other causes are : (i) Antepartum hemorrhage (abruptio placenta, placenta previa) (ii) Retained placenta (iii) Abortion complications and ectopic pregnancy. Hemorrhage is more dangerous when the woman is anemic.	20–25	■ Treat anemia in pregnancy (see p. 308) ■ Skilled attendant at birth ■ Prevent/treat hemorrhage (see p. 476) ■ Use oxytocics in time ■ Replace fluid loss ■ Transfusion of blood, if severe hemorrhage
Infection is associated with labor and puerperium. Infections from premature rupture of membranes, prolonged and obstructed labor are still frequent in the developing world.	15–20	■ Skilled attendant at birth ■ Clean practices (three cleans) during delivery (see p. 159) ■ Antibiotics — if infection is evident
Hypertension during pregnancy preeclampsia, eclampsia	12–15	■ Early detection ■ Appropriate referral ■ Antiseizure prophylaxis/treatment with $MgSO_4$
Unsafe abortion **(see p. 192)**	10–13	■ Skilled attendant ■ Access to family planning and safe abortion services ■ Antibiotics after evacuation ■ Postabortion care (see p. 643)
Obstructed labor—due to cephalopelvic disproportion, abnormal lie or malpresentation.	8	■ Use of partograph ■ Detection in time ■ Refer for operative delivery
Anemia is an indirect cause of death. About 50% of pregnant women worldwide suffer from anemia. Anemia is commonly due to dietary deficiency (nutrition, iron, folic acid, iodine and other micronutrients) or infections.	15–20	■ Routine Iron-folic acid supplementation ■ Treat hookworm, malaria, HIV, etc. ■ Admit when Hb $\leq$ 7 g/dL
Other indirect causes: Viral hepatitis is endemic in India with high mortality. Death is mostly in the last trimester due to hepatic coma and coagulation failure and postpartum hemorrhage.	5–10	■ Safe drinking water ■ Immunization ■ Appropriate referral and supportive care

80% of these deaths can be prevented through actions that are effective and affordable in developing country settings (WHO, UNICEF and UNFPA-2001).

Antenatal care: Unfortunately, the women who have the highest mortality, like grand multipara or the patients of lower socioeconomic status are the women who often do not avail the benefits of antenatal care.

Substandard care: When the care provided is below the generally accepted level, available at that circumstances. Shortage of resources (Staff) or back up facilities (Laboratory) is also included.

In the developing countries, **avoidable social factors** are palpably evident. **These are related to**: (a) Presence of social evils—illiteracy, early pregnancy, ignorance or prejudice, (b) Unregulated fertility and unsafe abortion, (c) Poor socioeconomic condition, (d) Inadequate maternity services, (e) Underutilization of the existing services, (f) Lack of communication and referral facilities. These are most often interrelated and are responsible for increased number of avoidable deaths.

Important causes of maternal death: Whereas in the organized sector (developed countries)—hypertensive disorders, hemorrhage and pulmonary embolism are the main causes, in the developing countries—hemorrhage, sepsis and preeclampsia-eclampsia and unsafe abortion are the main causes.

STEPS TO REDUCE MATERNAL MORTALITY (ACTIONS FOR SAFE MOTHERHOOD)

It is a coordinated, long-term effort within the families, communities and the health systems. It also involves the national legislation and policy. Actions may vary in respect of an individual country. The government must make maternal mortality a priority public health issue and periodically evaluate the programs in an effort to prevent or minimize maternal deaths. **Specific actions are discussed under the following groups:**

A. Health Sector Actions

- **Basic antenatal, intranatal and postnatal care** (see RCH interventions). **Risk assessment is a continued procedure throughout and is not once only**.
- **A skilled attendant** should be present at every birth. Functioning **referral system** is essential for integration of domiciliary and institutional services.
- **Emergency obstetric care (EmOC)** is to be provided either by a field staff at the door step of a pregnant woman or preferably at the first referral unit (FRU).
- **Good quality obstetric services** at the referral centers are to be ensured. Facilities for blood transfusion, laparotomy and cesarean section must be available at the FRU level.
- **Prevention** of unwanted pregnancy and unsafe abortion. All couples and individuals should have access to effective, client oriented and confidential **family planning services**.
- **Frequent joint consultation** among specialists in the management of medical disorders in pregnancy particularly anemia, diabetes, cardiac disease, viral hepatitis, and hypertension.
- **Maternal mortality conferences** to evaluate the cause of death and the avoidable factors.
- **Periodic refresher courses** for continuing education of obstetricians, general practitioners, midwives and ancillary staff and to highlight the preventable factors.

B. Community, Society and Family Actions

These are essential to safe motherhood. Wide range of groups (women's groups), health care professionals, religious leaders and safe motherhood committees (regional, district) can help the woman to obtain the essential obstetric care.

C. Health Planners/Policy Makers' Actions

- To organize community education, motivation and formation of safe motherhood committee at the local level.
- To strengthen the referral system for obstetric emergencies.
- To develop written management protocols for obstetric emergencies in the hospitals.
- To improve the standard and quality of care by organizing refresher courses for the health care personnels.
- Periodic audit of the existing health care delivery system and to implement changes as needed.

D. Legislative and Policy Actions

- **Girl children and adolescents** should have good nutrition, education and economic opportunities. They are to be educated about the age of sex and the risks of unprotected sex.
- **Barriers to the access** of health care facilities should be removed. Policies should increase women's decision making power as regard to their own health and reproduction.
- **Decentralization** of services to make them available to all the women.
- **Safe abortion services and postabortion care** must be ensured by national policy (p. 681).
- **Social inequalities and discrimination** on grounds of gender, age and marital status, are to be removed.

MATERNAL NEAR MISS

Maternal Near Miss (MNM). Women who experienced and survived a severe health condition during pregnancy, childbirth or postpartum are considered as maternal near miss or **severe acute maternal morbidity (SAMM)** cases. **Maternal near miss is defined as:** "A woman who nearly died but survived a complication that occurred during pregnancy, childbirth or within 42 days of termination of pregnancy" (WHO). MNM incidence ratio (MNMIR) refers to the number of maternal near miss cases per 1000 live births (MNM IR = MNM/1000 LB).

MATERNAL MORBIDITY

Although considerable attention has been given to maternal mortality, very little concern has been expressed for maternal morbidity. It has been estimated that for one maternal death at least 15 more suffer from severe morbidities. As such, about an optimistic 5–7 million women suffer a severely impaired quality of life as a result of short-term or long-term disability.

Definition: Obstetric morbidity originates from any cause related to pregnancy or its management any time during antepartum, intrapartum and postpartum period usually up to 42 days after confinement. The parameters of maternal morbidity are—(1) Fever more than 100.4°F or 38°C and continuing more than 24 hours, (2) Blood pressure more than 140/90 mm of Hg, (3) Recurrent vaginal bleeding, (4) Hb% less than 10.5 g irrespective of gestational period, and (5) Asymptomatic bacteriuria of pregnancy.

Classification: I. Direct obstetric morbidity: • Temporary • Permanent II. Indirect obstetric morbidity

Direct—***Temporary:*** APH, PPH, eclampsia, obstructed labor, rupture uterus, sepsis, ectopic pregnancy, molar pregnancy, etc.

Permanent (chronic): VVF, RVF, dyspareunia, CPT, prolapse, secondary infertility, obstetric palsy, Sheehan's syndrome, etc.

Indirect: These conditions are only expressions of aggravated previous existing diseases like malaria, hepatitis, tuberculosis, anemia, etc. by the changes in the various systems during pregnancy.

Reproductive morbidity is used in a broader sense to include—(a) Obstetric morbidity, (b) Gynecological morbidity and (c) Contraceptive morbidity.

PERINATAL MORTALITY

Perinatal mortality is defined as deaths among fetuses weighing 1000 g or more at birth (28 weeks gestation) who die before or during delivery or within the first 7 days of delivery. The perinatal mortality rate is expressed in terms of such deaths per 1000 total births. The perinatal mortality rate closely reflects both the standards of medical care and effectiveness of social and public health measures. According to WHO, the limit of viability is brought down to a fetus weighing 500 g (gestational age 22 weeks) or body length (25 cm crown-heel) or more. However, for international comparisons, only deaths of fetuses or infants weighing ≥ 1000 g at birth should be included as in the developing countries many such deaths are under reported.

Worldwide nearly four million newborns die within the first week of life and another three million are born dead. Perinatal deaths could be **reduced by at least 50% worldwide** if key interventions are applied for the newborn. The perinatal mortality is less than 10 per 1000 total births in the developed countries while it is much higher in the developing countries (60/1000 in India). The national goal is between 30 and 35. **The major health problem in the developing world arises from the synergistic effect of malnutrition, infection and unregulated fertility combined with lack of adequate obstetric care.**

Majority of fetal deaths (70–90%) occur before the onset of labor. The important causes of antepartum deaths are: (a) Chronic hypoxia (30%), (b) Pregnancy complications (30%), (c) Congenital malformations (15%), (d) Infection (5%) and (e) Unexplained (20%).

PREDISPOSING FACTORS OF PERINATAL MORTALITY

Many factors influence the perinatal survival and these are briefly discussed below:

- ***Epidemiological:*** Age over 35 years, teenagers, parity above 5, low socioeconomic condition, poor maternal nutritional status—all adversely affect the pregnancy outcome.
- ***Medical disorders:*** Anemia (Hb < 8 g/dL), hypertensive disorders of pregnancy, diabetes mellitus, syphilis, acute fever (malaria) and infection (HIV) are often associated. Perinatal deaths increase due to hypoxia, intrauterine growth restriction, prematurity, congenital malformations and infection.
- ***Obstetric complications:*** (a) **Antepartum hemorrhage** particularly abruptio placenta is responsible for about 10% of perinatal deaths due to severe hypoxia, (b) **Preeclampsia-eclampsia** is associated with high perinatal loss either due to placental insufficiency or prematurity—spontaneous or induced (c) **Rh isoimmunization** (d) **Cervical incompetence**—Premature effacement and dilatation of cervix between 24 and 36 weeks is responsible for significant perinatal deaths from prematurity.
- ***Complications of labor:*** Dystocia from disproportion, malpresentation, abnormal uterine action, premature rupture of membranes may result in asphyxia, amnionitis and birth injuries contributing to perinatal deaths.
- ***Fetoplacental factors:***
  - **Multiple pregnancy** most often leads to preterm delivery and usual complications.
  - **Congenital malformation** and chromosomal abnormalities are responsible for 15% of perinatal deaths, the lethal malformations are mostly related to nervous, cardiovascular or gastrointestinal system.
  - **Intrauterine growth restriction and low-birth-weight babies**—Apart from preterm delivery, intrauterine nutritional deficiency may be responsible for such low weight babies which are more vulnerable to biochemical, neurological and respiratory complications resulting in high perinatal deaths of about 50% when the birth weight is less than 2 kg.
  - **Preterm labor and preterm rupture of the membranes** are the known leading causes of prematurity.
- ***Unexplained:*** About 20% of stillbirths have no obvious fetal, placental, maternal or obstetric causes.

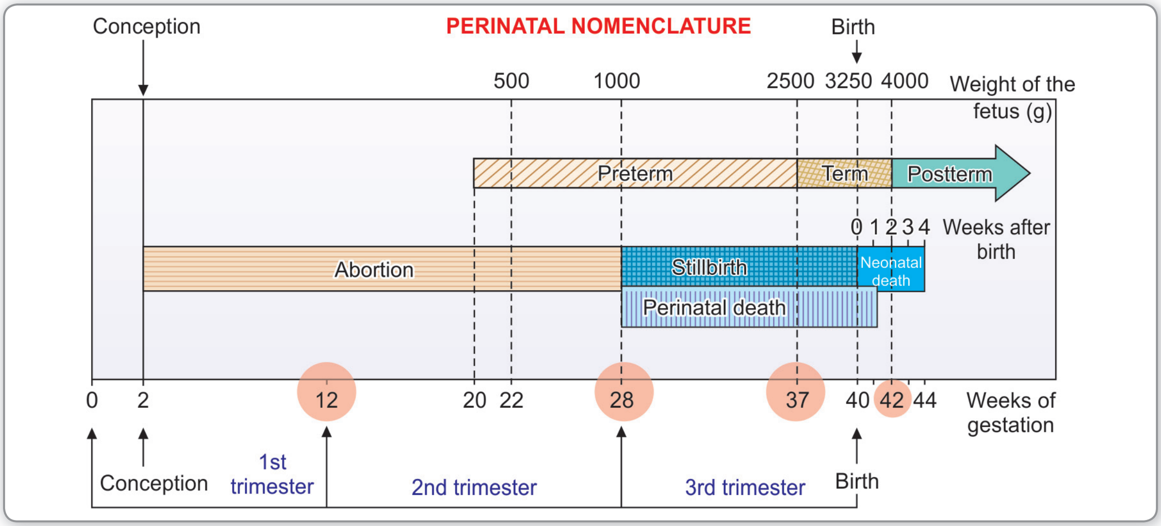
PREVENTION: **As every mother has the right to conclude her pregnancy safely so also has the baby got a right to be born alive safe and healthy**. As such, improvement of obstetric service only around delivery, will not minimize perinatal deaths appreciably. Simultaneous demographic and social changes help in reduction of perinatal mortality rate significantly. **The following measures are helpful in reducing the perinatal mortality**.

- **Prepregnancy health care and counseling** (see p. 116).
- **Genetic counseling** in high-risk cases and prenatal diagnosis (see p. 567) to detect genetic, chromosomal or structural abnormalities are essential. Termination of an affected fetus is a positive step in reduction of deaths due to congenital malformations (see p. 567).
- **Regular antenatal care**, with advice regarding health, diet and rest (see p. 112).

- **Detection and management of medical disorders in pregnancy**: anemia, diabetes, infections and preeclampsia–eclampsia. Immunization against tetanus should be done as a routine.
- **Screening of high-risk patients** those of poor socioeconomic status or high parity, extremes of age, and twins, etc. and their mandatory hospital delivery. Risk approach to RCH care is essential (see p. 718).
- **Careful monitoring** in labor (see p. 692) to detect hypoxia early and avoidance of traumatic vaginal delivery.
- **Skilled birth attendant** — To minimize sepsis, **at least three cleans are to be maintained** (see p. 159).
- **Provision of referral neonatal** service especially to look after the preterm babies.
- **Health care education of the mother about the care of the newborn**. Early and exclusive breastfeeding, prevention of hypothermia.

IMPORTANT CAUSES OF PERINATAL MORTALITY AND MAIN INTERVENTIONS

Causes	Percent	Proven Interventions
Infections (Sepsis, Meningitis, Pneumonia, Neonatal tetanus, Congenital syphilis)	33	♦ Maternal immunization against tetanus ♦ Clean delivery (three cleans) ♦ Warmth ♦ Early and exclusive breastfeeding ♦ Early recognition and management of infections ♦ Screening for infections
Birth asphyxia and trauma **Hypothermia**	28	♦ Skilled attendant at birth ♦ Detection and management of obstetric complications ♦ Warmth
Preterm birth and/or low birth weight	24	♦ Prevention of obstetric complications and management ♦ Infection control ♦ Warmth ♦ Breastfeeding ♦ Referral to special care unit
Congenital malformations and others	15	♦ Prenatal diagnosis, genetic counseling.


PERINATAL NOMENCLATURE
Conception
Birth
500
1000
2500
3250
4000
Weight of the fetus (g)
Preterm
Term
Postterm
0 1 2 3 4
Weeks after birth
Abortion
Stillbirth
Neonatal death
Perinatal death
0
2
12
20
22
28
37
40
42
44
Weeks of gestation
Conception
1st trimester
2nd trimester
3rd trimester
Birth

- **Educating the community** to utilize family planning services and also to utilize the available maternity and child health care services. **Family planning services** can prevent unwanted pregnancies.
- **Autopsy studies** of all perinatal deaths.
- **Continued study of perinatal mortality problems** by demographic studies, regular clinically allied interdepartmental meetings and pathological research.

Perinatal morbidity: It implies major illness of the neonate from birth to first 4 weeks of life. Important causes of morbidity are due to (a) Prematurity and low birth weight, (b) Birth asphyxia and birth trauma (c) Congenital malformations.

STILLBIRTHS

A stillbirth is the birth of a newborn after 28th completed week (weighing 1000 g or more) when the baby does not breathe or show any sign of life after delivery. Such deaths include antepartum deaths (macerated) and intrapartum deaths (fresh stillbirths). **Stillbirths rate is the number of such deaths per 1000 total births (live and stillbirths)** (Table 38.3).

Table 38.3: Important Causes of Stillbirths and Main Interventions

Causes	Percent	Proven Interventions
Birth asphyxia and trauma	30	Skilled attendant at birth. Effective management of obstetric complications.
Pregnancy complications (placental abruption, preeclampsia, diabetes mellitus)	30	Prepregnancy care, effective management of pregnancy complications (see p. 119).
Fetal congenital malformations and chromosomal anomalies	15	Preconceptional genetic counseling, prenatal diagnosis (see p. 127).
Infection	5	Effective care during pregnancy and labor. Clean delivery.
Cause unknown	20	

NEONATAL DEATHS

Neonatal death is the death of the infant within 28 days after birth. **Neonatal mortality rate is the number of such deaths per 1000 live births**. Majority of the deaths occur within 48 hours of birth.

Causes: The causes of death within 7 days are almost always obstetrically related and as such stillbirths and neonatal deaths within 7 days are grouped together as perinatal deaths. About two-thirds of the neonatal deaths are related to prematurity.

Women's Health (MDGs) Beyond 2015

- Good progress has been made in achieving MDG 4 and 5 to reduce child and maternal mortality. The global MMR has declined from 400 to 210 per 100,000 live births between 1990 and 2010. However the progress fell short of the target in parts of South Asia and Subsaharan Africa. Only 16 countries are estimated to achieve the MDG 5 target by 2015.
- The post 2015 agenda seeks to integrate economic development (poverty reduction), and Sustainable Development Goals (SDGs) 'ensuring universal health coverage and access'. Beyond these strategic actions, good governance must also incorporate a human rights approach which includes accountability, participation, ownership, transparency, equity and non-discrimination. Post 2015, sustainable agenda need to consider all the issues, discussed above, in the international platform to achieve improved health for 'every woman, every child'.

KEY POINTS

- **Safe motherhood** is a global effort to reduce maternal deaths by 3/4 and child mortality by 2/3 by 2015 (MDG4 and 5) see p. 680.
- **Lifetime risk of dying** from pregnancy-related complications for a woman of developing country is 1 in 11, compared to 1 in 5000 in the industrialized country (see p. 681). In India, presently it is 0.4%.
- **National sociodemographic goals for 2015** (p. 681) and international commitment are to improve maternal and newborn health (see p. 681). RCH care is an integrated and composite approach to improve the health of the women and children in India (p. 682).
- **Maternal death** (see p. 683) is expressed (MMR) per 100,000 live births. Maternal deaths are classified into—(a) Direct, (b) Indirect and (c) Fortuitous deaths (see p. 684). In India, MMR presently is 178 (2010–2012).
- **Important causes of maternal deaths are:** (i) Hemorrhage (20–25%) (ii) Hypertension (20–25%) (iii) Infection (15–20%) (iv) Unsafe abortion (10–13%) (v) Obstructed labor (8%) (vi) Anemia (15–20%) and (vii) Other indirect (viral hepatitis) causes (5–10%).
- **Maternal Near Miss** is a condition when a women who nearly died but survived from a severe health condition, during pregnancy, child birth or within 6 weeks of puerperium.
- **There are several proved interventions** that can prevent maternal deaths (see p. 683). Steps to reduce maternal mortality are a coordinated long-term effort (see p. 686).
- **Maternal morbidity (Obstetric morbidity)** develops from any cause related to pregnancy, childbirth or puerperium. Nearly 15 more women suffer from severe morbidity, when there is one maternal death.
- **Perinatal mortality** (see p. 687) is expressed per 1000 total births (see p. 687). Important causes of PNM are: (i) Infection (33%) (ii) Birth asphyxia and trauma (28%) (iii) Preterm birth and/or LBW (24%) and (iv) Congenital malformation (15%, see p. 688).
- **Important causes of stillbirths are:** Birth asphyxia and trauma (30%), pregnancy complications (30%) and others (see p. 690).

QUESTIONS

Related theory questions (Long and Short), Obstetric Case Discussions, Viva table discussions, Postoperative word round discussions, and MCQs are discussed in author's books:

1. **Bedside Clinic and Viva Voce:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

For further reading:

2. **Master pass in Obstetrics and Gynaecology:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

Chapter 39

Special Topics in Obstetrics

INTRAPARTUM FETAL MONITORING

By definition, intrapartum fetal monitoring (IFM) means simply to watch the fetal behavior during labor. Goal of IFM is to **detect hypoxia in labor and to initiate management** depending upon the severity of hypoxia. Severe hypoxia in labor when associated with metabolic acidosis can cause fetal organ damage or fetal death.

In between contractions the intraluminal pressure within the spiral artery (85 mm of Hg) is higher than the intramyometrial pressure (10 mm of Hg) to maintain the uteroplacental blood flow. **During peak uterine contractions,** myometrial pressure (120 mm of Hg) exceeds the arterial pressure (90 mm of Hg) causing temporary halting of O_2 delivery to the fetus through the placenta. Depending upon the intensity and duration of contraction, fetal hypoxia may develop.

Even in a normal labor, the baby is subjected to stress due to: (1) Uterine contractions temporarily curtailing the uteroplacental circulation. (2) Head compression affecting the function of the vital centers of the brain. A healthy fetus can withstand the stress of labor within physiological limits. But in a compromised fetus and/or in a pathological state of labor, the fetal distress may appear abruptly. The term "**Fetal distress**" has been abandoned in favor of more appropriate term "**Nonreassuring fetal status**".

METHODS OF FETAL MONITORING

A Clinical **B Biophysical** **C Biochemical**

CLINICAL: *To note the FHR*–Intermittent auscultation of FHR using an ordinary stethoscope or a fetoscope or a handheld Doppler can be done to note its rate, rhythm and intensity. FHR should be recorded at every 30 minutes interval initially followed by 15 min intervals in the first stage and at about 5 min intervals in the second stage. **The auscultation should be made for 60 sec particularly before and immediately following a uterine contraction.**

Normal fetal heart rate is at an average of 140 beats per minute (bpm) in between contractions with a variation between 110 and 160 per min (FIGO : 110-150 bpm). There may be slowing of FHR during a contraction, (vagal stimulation) which, however, comes back to normal before the contraction passes off.

Limitations—(1) As it is a periodic observation, any transient significant abnormality in between observations is likely to be overlooked (2) Inherent human error (3) Difficult at times, to count the FHR during uterine contractions or in case of obesity or hydramnios.

Evidences of distress—(1) An increase in FHR to over 160/min or a decrease in rate to less than 110/min (2) FHR takes a long time to come back to its normal rate after the contraction passes off (3) Irregularity.

Causes of Fetal Tachycardia (FHR > 160 bpm)	Causes of Fetal Bradycardia (FHR < 110 bpm)
■ **Drugs to mother:** (i) β-sympathomimetic agents used to inhibit preterm labor (isoxsuprine, ritodrine); (ii) Vagolytic : atropine ■ **Infection**—both maternal and fetal ■ **Anemia**—both maternal and fetal ■ Fetal hypoxia	♦ Fetal hypoxia, acidosis ♦ Fetal sepsis, anomalies ♦ Use of local anesthetic drugs, epidural analgesia ♦ Drugs to mother, e.g. pethidine, antihypertensives (methyldopa, propranolol), $MgSO_4$ ♦ Fetal heart conduction defect (SLE)

***Meconium in the liquor amnii*—Meconium in the liquor amnii** is a potential sign of fetal hypoxia. It acts as a toxin, if the fetus aspirates this particulate matter. **Pathogenesis:** Hypoxia →(↑) vagal response →(↑) peristaltic activity and relaxation of the anal sphincter → passage of meconium. **The vicious circle is** : Placental insufficiency → oligohydramnios → cord compression → hypoxia → thick meconium → gasping breath → meconium aspiration. Meconium staining of the liquor as observed following rupture of the membranes gives a crude idea of intrauterine fetal jeopardy. It is observed in about 10–20% of labors. **Presence of meconium and nonreassuring FHR pattern necessitates urgent intervention**. **On the other hand reassuring FHR pattern and thin meconium can be managed expectantly**.

Intermittent auscultation is recommended to monitor the fetus for a woman in labor without any complications.

A. BIOPHYSICAL—**Ultrasound:** Doppler effect is used to detect fetal pulse rate from major fetal vessels. This observation has to be rechecked when an abnormality is detected (see p. 535, 737).

B. CONTINUOUS ELECTRONIC FETAL MONITORING (EFM)

Indications of continuous EFM are: (A) Maternal conditions: Hypertension, previous cesarean delivery, induced labor, APH, PROM. (B) Fetal conditions: Small fetus (IUGR), oligohydramnios, multiple pregnancy, abnormal FHR on auscultation.

Two methods are applied: ♦ **External** ♦ **Internal**

(From maternal abdominal wall – Noninvasive) (Directly from the fetus – Invasive)

External (Fig. 35.4): Continuous tracing of FHR can be obtained using ultrasound Doppler effect. The transducers are placed on the maternal abdomen, one over the fundus and the other at a site where the fetal heart sound is best audible. Frequency of uterine contractions and uterine pressure are recorded simultaneously by tocodynamometer.

Internal: Fetal ECG tracing is made by applying a spiral pointed scalp electrode to the fetal scalp after rupturing the membranes (Fig. 39.1). Intrauterine pressure could be simultaneously measured by passing a catheter inside the uterine cavity.

Categorization of Fetal Heart Rate (FHR) Features (RCOG, NICE)					Categorization of CTG Traces
Feature	**Baseline (bpm)**	**Variability (bpm)**	**Deceleration**	**Accelerations**	**Based on 4 Features (Baseline FHR, Variability, Decelerations, Accelerations)**
Normal/ Reassuring	100–160	> 5	None or early	Present	**Normal:** **All four** features are reassuring **Suspicious:** **One** nonreassuring and the **rest** are reassuring **Pathological:** **Two or more** features nonreassuring **OR one or more** abnormal categories
Non-reassuring	161–180	< 5 for 30 to 90 minutes	**Variable decelerations:** ■ Dropping from baseline by ≤60 bpm, recovering by ≤60 secs **OR** >60 bpm, recovering >60 secs. **OR** late decelerations ■ Present up to 30 minutes ■ Occurring ≥50% of contractions	Absence of accelerations with an otherwise normal CTG is of uncertain significance	
Abnormal	> 180 or < 100	< 5 for > 90 minutes	■ Nonreassuring variable decelerations (as above) present even 30 min. after conservative measures **OR** ■ Late decelerations >30 min. with >50% of contractions **OR** ■ Bradicardia or a single prolonged deceleration lasting ≥3 minutes		

National Institute of Child and Human Development (2008), ACOG (2009); Three tier FHR interpretation system

Category I: Normal (baseline rate 110-160 bpm; FHR variability – moderate; no late or variable deceleration; early deceleration ±; acceleration ±

Category II: Indeterminate—all tracings not categorized as category I or III.

Category III: Abnormal (either absent baseline FHR variability and any of the following: recurrent late/variable decelerations, bradycardia **or** sinusoidal pattern)

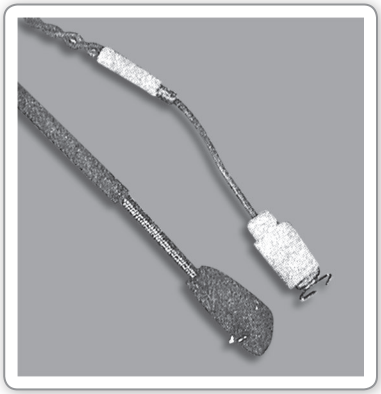


Fig. 39.1: Scalp electrodes

Advantages of EFM over clinical monitoring:

- Accurate monitoring of uterine contractions.
- Significant improvement of perinatal mortality.
- Can detect hypoxia early and can explain the mechanism of hypoxia and its specific treatment.
- Improvement of intrapartum fetal death by threefold.
- It is an important record for medicolegal purpose (see p. 727).

Drawbacks: (i) Interpretation is affected by intra- and interobserver error (ii) Due to error of interpretation, cesarean section rate may be high (iii) Instruments are expensive and trained personnel are required to interpret a trace (iv) Mother has to be confined in bed.

Interpretation of a Cardiotocography (CTG)

- Accelerations and normal baseline variability (5-25 bpm) denote a healthy fetus.
- Absence of accelerations is the first feature to denote onset of hypoxia.
- Absence of accelerations, reduced baseline variability of < 5 bpm for > 90 minutes denotes a hypoxic fetus.
- Decreased baseline variability may be due to fetal sleep, infection, hypoxia, anomalies or due to maternal medications.
- Repeated late decelerations increase the risk of low Apgar score and cerebral palsy (CP).
- Reduced baseline variability, with late or variable deceleration increases the risk of CP.
- Interpretation of the CTG should always be made in the context of clinical picture.

Baseline FHR is the mean level of FHR excluding accelerations and decelerations. It is expressed in beats per minute (bpm). Normal baseline FHR is 110-160 bpm.

Baseline variability is the oscillation of baseline FHR excluding the accelerations and decelerations. Variability is the reflex of normal cardiac behavior in response to sympathetic and parasympathetic nerve input. However, parasympathetic (vagus) has the dominant role in modulating variability. Baseline variability may be (a) **absent**, (b) **minimal** (< 5 bpm), (c) **moderate** (6-25 bpm) or (d) **marked** (> 25 bpm). Reduced baseline variability is observed in many conditions (see p. 693).

Acceleration: Transient increase in FHR by 15 bpm or more lasting for at least 15 seconds.

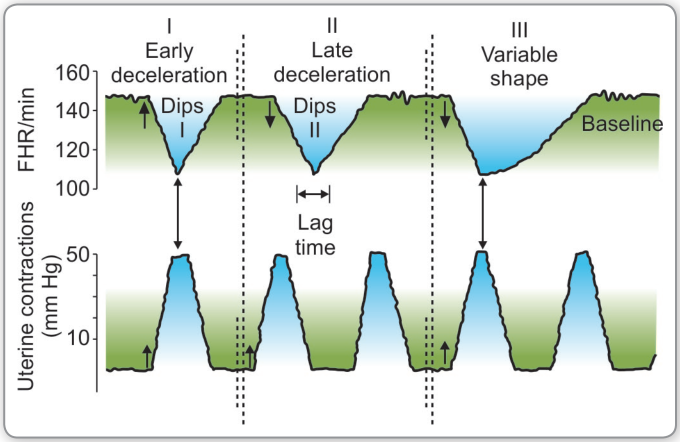


Fig. 39.2: Graphic representation showing various types of decelerations in relation to uterine contractions

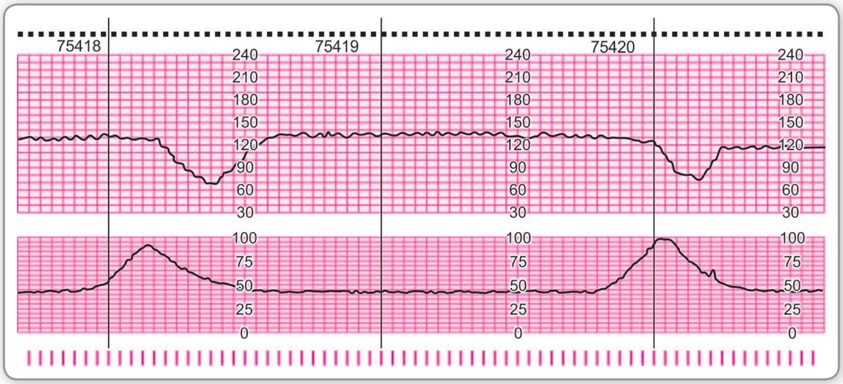


Fig. 39.3: Persistent late deceleration with loss of variability

Admission CTG is a CTG trace done for a woman on admission in the labor ward. Admission CTG are poor in predicting fetal compromises in labor. Routine admission CTG testing is not recommended in low-risk women.

Prolonged acceleration lasts ≥ 2 min but < 10 min and when it is ≥ 10 min it is a baseline change. **Acceleration denotes** an intact neurohormonal and cardiovascular activity and therefore a **healthy fetus** (Fig. 39.4).

Deceleration: Transient decrease in FHR below the baseline by 15 bpm or more and lasting ≥ 15 seconds (Fig. 39.2).

Three basic types of deceleration are observed and are called early, late and variable (Fig. 39.2).

- **Early deceleration** *(Type I Dips)*, uniform, repetitive periodic slowing of FHR and in most cases the onset, nadir and recovery of deceleration coincides with the beginning, peak and ending of uterine contraction respectively. It is due to head compression (vagal nerve activation) (Fig. 39.2).
- **Late deceleration** *(Type II Dips)*, uniform, repetitive periodic slowing of FHR. It begins after the onset of the uterine contraction. Usually, the onset, nadir and recovery of the decleration occur after the start, peak and end of the uterine contraction respectively. Nadir occurs 20 seconds after the peak of the contraction and FHR returns to normal after the contraction is over. It suggests uteroplacental insufficiency (Fig. 39.3) and fetal hypoxia (50%).

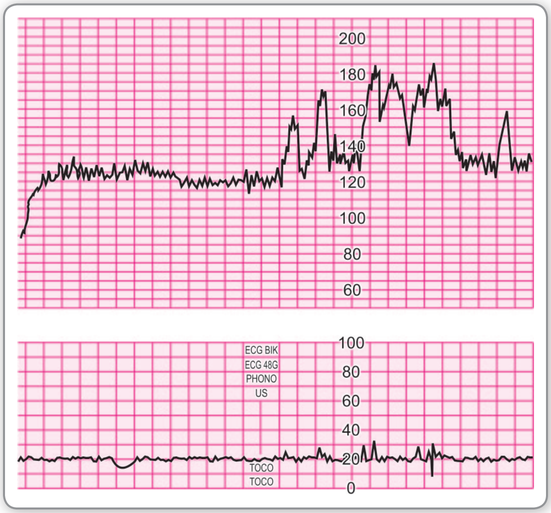


Fig. 39.4: Reactive trace with acceleration

- **Causes of late deceleration:** (i) Placental pathology (postmaturity, hypertension, diabetes, placental abruption) (ii) Excessive uterine contractions (iii) Injudicious use of oxytocin (iv) Regional anesthesia (spinal of epidural).
- **Variable deceleration:** It is the intermittent periodic slowing (variable) of FHR with rapid onset and recovery (Fig. 39.2). Decelerations are variable in all respect of size, shape, depth, duration and timing to the uterine contractions. **It is thought to indicate cord compression and may disappear with the change in position of the patient.** It is the most common type. **Accelerations often precede and follow the deceleration**.

Prolonged deceleration is the abrupt decrease in FHR to levels below the baseline and it lasts ≥ 2 min but < 10 min. If it lasts ≥ 10 min. it is a baseline change.

Lag period: It is the time taken for the FHR to reach the nadir (the lowest point of the FHR dip) from the apex of the preceding uterine contraction (Fig. 39.2). In deceleration lag period is ≥ 30 seconds.

Sinusoidal pattern: It resembles a sine wave. It has a stable baseline FHR with fixed or absent baseline variability lasting ≥ 20 min. Accelerations are absent. It is often associated with fetal anemia, fetomaternal hemorrhage, fetal hypoxia (acidosis). It may occur when narcotics are given to mother (Fig. 39.5). Such FHR patterns are called pseudosinusoidal as the fetus is well-oxygenated.

C. Induced fetal stimulation and FHR accelerations: Any FHR acceleration spontaneous or induced, indicates the absence of fetal acidosis.

Vibroacoustic stimulation (VAS) of the fetus is done using an electronic larynx placed on the maternal abdomen. Presence of FHR accelerations indicates normal blood pH.

Fetal scalp stimulation by pinching with an Allis forceps or by gentle digital stroke is done before scalp blood pH test. Presence of FHR accelerations is associated with normal scalp blood pH.

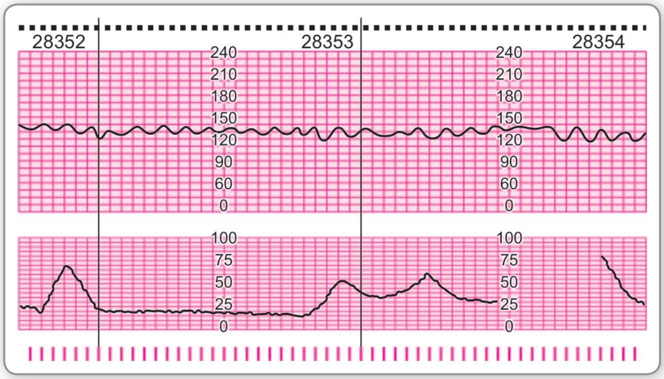


Fig. 39.5: Sinusoidal pattern showing absence of baseline variability

Evaluation of the fetus with a persistently nonreassuring FHR pattern with CTG.

Goal: Detection of fetal metabolic acidosis.

Place of auscultation of fetal heard sound versus EFM: Intermittent auscultation is an effective method for evaluation of fetal well-being. It is as effective as EFM. In the high-risk patient, auscultation should be done at every 15 minutes in the first stage and at every 5 minutes in the second stage. In the low-risk group it may be done at an interval of 30 minutes in the first stage and at every 15 minutes in the second stage. Auscultation should be done for a period of 60 seconds after a uterine contraction.

Fetuses with abnormal FHR pattern on auscultation should have EFM to detect any nonreassuring patterns.

D. BIOCHEMICAL: Fetal scalp blood sampling (SBS): To corroborate the significance of fetal CTG abnormality due to hypoxia, Saling, in 1962, demonstrated a simple and quick method to obtain fetal blood samples from the scalp to detect the **fetal blood pH**. **Fetal scalp blood pH** < 7.20 indicates fetal acidosis and urgent delivery, pH between 7.21 and 7.25 is borderline and needs to be repeated within 30 minutes. pH > 7.25 is reassuring and labor progress is monitored. pH to be repeated every 30 minutes.

Procedures: Mother is in left lateral position. An illuminated plastic cone is inserted through the dilated cervix (4–5 cm) against the fetal head. An incision of 2 mm depth is made with a lancet. Blood is collected (100 μL) with a long capillary tube for pH and PCO_2 estimation.

Indications: (i) Abnormal CTG in labor (ii) Biochemical assessment of fetal hypoxia.

Contraindications: (i) When delivery is urgently indicated or spontaneous delivery is imminent (ii) Maternal infection (HIV, hepatitis or herpes simplex virus) (iii) Fetal coagulation disorders (iv) Prematurity (< 34 weeks). **Risks involved are:** Fetal bleeding from the incision site and maternal injury.

Prediction of a compromised fetus by scalp blood pH is superior compared to CTG. False prediction is about 10%.

E. Fetal Pulse Oximetry was initially used to determine fetal oxyhemoglobin saturation. It is no longer used as its accuracy is uncertain.

F. Fetal electrocardiogram (ECG) analysis has been done with ST segment or with T/QRS ratio. An increase in T wave amplitude occurs in hypoxia. A normal fetus has a T/QRS < 0.25. A significant increase in T/QRS or more than two consecutive biphasic ST in fetal ECG complexes, combined with a nonreassuring FHR tracings (CTG) indicates fetal metabolic acidosis. It indicates intervention. Fetal ECG analysis (ST segment analysis) reduces operative delivery rates compared to CTG alone.

G. Umbilical arterial cord (or neonatal) blood samples with pH < 7 and base deficit of ≥ 2 mmol/L indicates profound metabolic acidemia and multiple organ dysfunction. **Intrapartum umbilical artery Doppler study** was poor to predict umbilical artery acidosis. A positive test increases the cesarean delivery rates. There is a correlation between NRFS and neonatal depression, but it is not related with the long-term neurologic sequelae.

NONREASSURING FETAL STATUS (NRFS)

DEFINITION: **Fetal distress is an ill-defined term, used to express intrauterine fetal jeopardy, a result of intrauterine fetal hypoxia. Nonreassuring fetal status (NRFS)** is characterized by tachycardia or bradycardia, reduced FHR variability, decelerations and **absence of accelerations (spontaneous or elicited)**. It must be emphasized that hypoxia and acidosis is the ultimate result of the many causes of intrauterine fetal compromise.

FHR patterns in labor are dynamic and **can change rapidly from normal to abnormal and vice versa**. Because of this uncertainty about the diagnosis of fetal distress, terminologies used are "**Reassuring**" and "**Nonreassuring**".

Nonreassuring fetal heart rate pattern is associated with fetal hypoxia, acidosis and therefore called fetal distress. Features to rule out metabolic acidosis are: (a) Presence of accelerations (b) Moderate variability and (c) scalp blood pH > 7.25.

PATHOPHYSIOLOGY: Under normal conditions when oxygen supply is adequate, aerobic glycolysis occurs in the fetus and glycogen is converted into pyruvic acid which is ultimately oxidized via the Kreb's cycle. During hypoxia when O_2 saturation falls below 40%, **anaerobic glycolysis** occurs, resulting in the accumulation of lactic acid and pyruvic acid leading to metabolic acidosis. H-ions first stimulate and then depress the sinoauricular node leading to tachycardia and bradycardia respectively. It also causes parasympathetic stimulation leading to hyperperistalsis and relaxation of the anal sphincter with passage of meconium. Decreased fetal oxygenation in labor → hypoxia → metabolic acidosis → asphyxia → organ damage/fetal death.

ETIOLOGY: **A. Acute** **B. Chronic**

A. ACUTE:

I. ***During pregnancy*** — less common
- Placental separation in placenta previa or abruptio placenta
- Following external cephalic version due to cord entanglement
- During oxytocin induction
- Diabetes
- Hypertension

II. ***During labor*** — common
- Uterine hyperstimulation following oxytocin for augmentation of labor
- Placental abruption
- Uterine rupture or scar dehiscence
- Cord prolapse
- Injudicious administration of oxytocin, analgesics and anesthetic agents
- Maternal hypotension – as in epidural analgesia

B. CHRONIC: The various clinical conditions which are responsible for chronic placental insufficiency and IUGR, are also linked with chronic fetal distress (see p. 539).

Fetal condition at birth is assessed by blood gas values of the umbilical artery. Normal (mean) values are: pH 7.27, PCO_2 50; HCO_3^- 23, base excess – 3.6. The correlation between the FHR and long-term neurological sequelae is poor. **In many cases asphyxia occur prior to labor**.

MANAGEMENT OF NONREASSURING FETAL STATUS (NRFS)

Ideally management should be specific to the cause of NRFS.

NONSURGICAL

Nonsurgical management is aimed to reverse the abnormality (hypoxia) by noninvasive procedures.

- **Lateral positioning** avoids compression of vena cava and aorta by the gravid uterus. This increases cardiac output and uteroplacental perfusion.
- **Oxygen is administered (6-8 L/min)** to the mother with face mask to improve fetal SaO_2.
- **Correction of dehydration** by IV fluids (crystalloids) improves intravascular volume and uterine perfusion.

- **Correction of maternal hypotension** (following epidural analgesia) with immediate infusion of 1 liter of crystalloid (Ringer's solution).
- **Stoppage of oxytocin** to improve fetal oxygenation. Fetal hypoxia may be due to strong and sustained uterine contractions. With reassuring FHR and in absence of fetal acidemia, oxytocin may be restarted.
- **Tocolytic** (Injection terbutaline 0.25 mg SC) is given when uterus is hypertonus and there is nonreassuring FHR. Tocolytics increase oxygen to the fetus.
- **Amnioinfusion** is the process to increase the intrauterine fluid volume with warm normal saline (500 mL). **Indications are:** (a) Oligohydramnios and cord compression (b) To dilute or to wash out meconium (c) To improve variable or prolonged decelerations (d) To reduce fetal gasping which is the result of hypoxia due to cord compression. ***Advantages:*** Reduces cord compression, meconium aspiration, and improves Apgar score. It also reduces cesarean section rate.

If the fetal heart rate pattern remains nonreassuring, further tests are performed to rule out metabolic acidosis.

Tests are: (i) To detect FHR accelerations (CTG)—spontaneous or induced, (ii) Scalp blood pH, (iii) Fetal pulse oximetry, (iv) Fetal ECG/ST segment analysis (STAN).

If acidosis is excluded → labor is monitored with repeated testing (every 30 min) to exclude acidosis. If the fetus is acidemic → urgent delivery by safest method (vaginal or abdominal) depending on the individual case.

Above supportive measures may result in:

A. Conversion of NRFS to a reassuring pattern and without any evidence of metabolic acidosis (presence of accelerations, variability and scalp blood pH > 7.25). These patients need to be evaluated (continuous or intermittent) at every 30 minutes to exclude acidosis. Labor progress is to be monitored also.

B. Persistence of nonreassuring pattern or presence of unusual or confusing pattern: these patients need immediate delivery.

SURGICAL: Cesarean delivery should be done with a 15° lateral tilt till the baby is delivered. **Thirty minutes has been accepted as the gold standard for decision to delivery interval in cases of confirmed fetal compromise**. *Pediatrician should be made available.*

KEY POINTS

- **EFM, scalp pH and ST analysis** are used to detect intrapartum fetal hypoxia.
- **Intermittent auscultation** is an effective method. Fetuses with abnormal FHR pattern on auscultation should have EFM.
- **EFM** has few limitations. Normal CTG indicates a healthy fetus, whereas abnormal FHR pattern in CTG does not always indicate fetal asphyxia.
- **False positive rate of EFM** for predicting fetal hypoxia is high.
- **Loss of variability**, loss of acceleration and presence of bradycardia indicate fetal compromise.
- **Presence of accelerations** of the FHR either spontaneous or induced (VAS stimulation), indicates absence of fetal aciodosis.
- **EFM is most reliable** when FHR pattern is reassuring (category - I) and when there is fetal acidosis (category III). It is most unreliable when tracings are equivocal (category - II).
- **High-risk labor** should be monitored continuously. Use of EFM is associated with an increased rate of operative interventions (vacuum, forceps, or cesarean delivery).
- **Nonsurgical measures** are used to improve or reverse hypoxia (see p. 697).
- **Persistent hypoxia** or presence of metabolic acidosis needs expeditious delivery of the baby to prevent organ damage.

SHOCK IN OBSTETRICS

DEFINITION: Shock is defined as a state of circulatory inadequacy with poor tissue perfusion resulting in generalized cellular hypoxia. Circulatory inadequacy is due to a disparity between the circulating blood volume and the capacity of the circulatory bed. The net effect of this disparity is inadequate exchange of oxygen and carbon dioxide between the intra- and extravascular compartments. The stagnation of carbon dioxide and other metabolites in the tissue leads to metabolic acidosis and cellular death. **The series of changes observed in shock and their clinical manifestations, are therefore, dependent on two sets of changes**: (a) Circulatory inadequacy at the 'filtration' level (microvascular compartment) (b) Cellular damage and ultimately death.

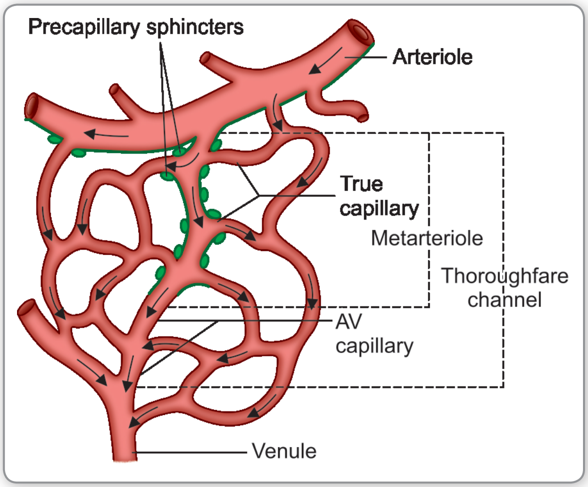


Fig. 39.6: Schematic diagram of a microcirculatory unit

Anatomy of microvascular circulation: Microvascular circulation consists of circulation of blood through a tuft of capillaries with a feeding arteriole and a draining venule at either end of the capillary bed. The flow of blood within the capillary bed is controlled by 2 sphincters – one at the arteriolar end and the other at the venular end. They are known as pre- and postcapillary sphincters. In addition to the tuft of capillaries, there is a direct communication between the arteriole and the venule and this communicating trunk bypasses the capillary bed. This is known as **metarteriole shunt or 'thoroughfare channel'**. When the sphincters are closed, the metarteriole shunt operates to divert blood for supply to the vital organs, like brain, heart and kidney. The basic pattern of microcirculation is schematically represented in Fig. 39.6.

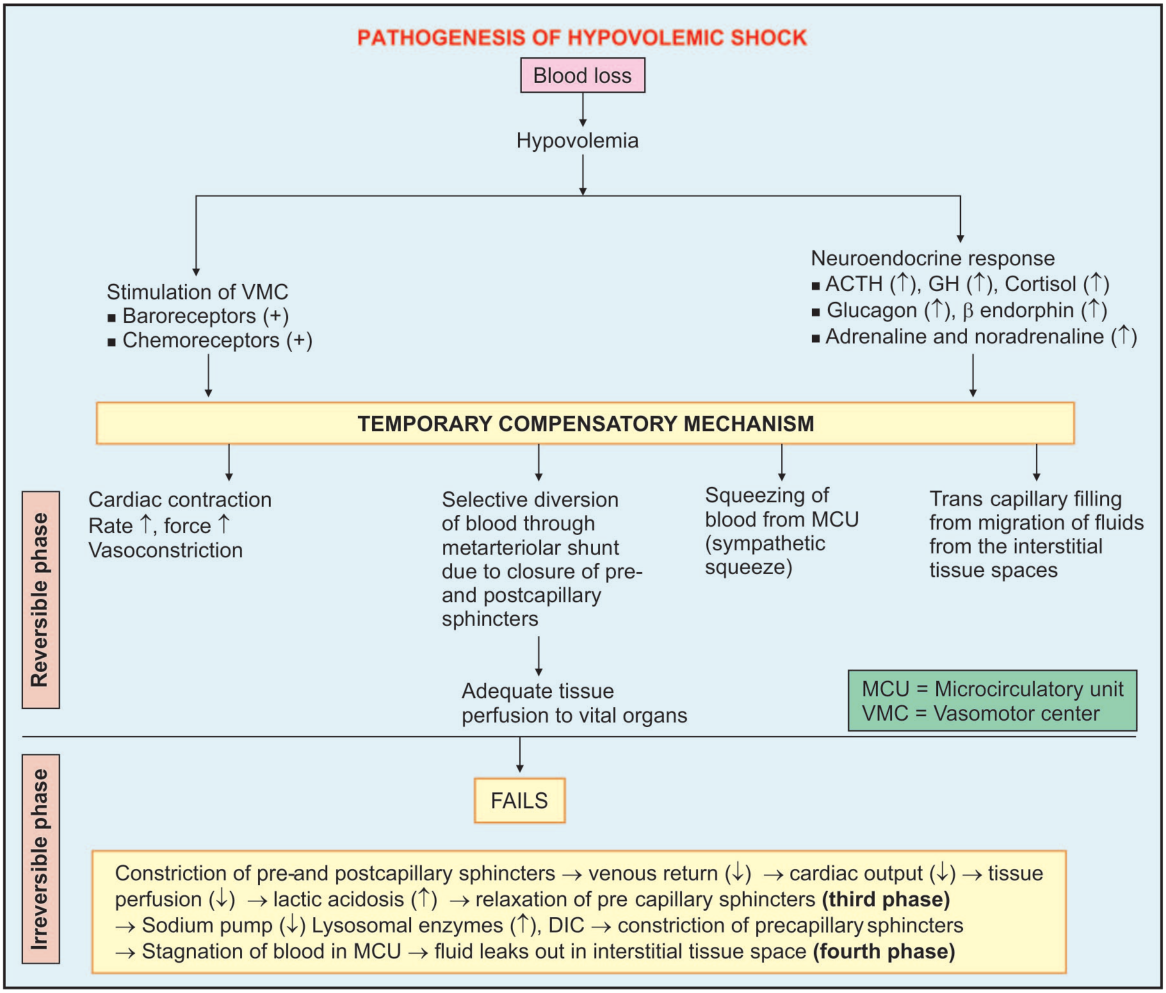
PATHOPHYSIOLOGY OF SHOCK

Pathophysiological changes in obstetric shock are predominantly associated with (a) general changes due to hypovolemia and (b) specific changes due to liberation of endotoxin.

Hypotension stimulates release of neuroendocrine mediators like adrenocorticotropic hormone (ACTH), growth hormone (GH), β endorphin, cortisol and glucagon. There is also sympathoadrenal response. Presence of endotoxin (lipopolysaccharide), in septic shock activates the leukocytes through complement system. There is release of inflammatory mediators such as proteases, superoxide (O_2^-), hydroxyl (OH^-) radicals, cytokines, prostaglandins and many cytotoxic enzymes. These interfere with the function of a number of enzyme systems and increase capillary permeability. Cytokines such as interleukines (ILS) and tumor necrosis factor (TNF) interact by autocrine and paracrine mechanism to cause cellular or organ dysfunction. In presence of hypoxia, sepsis and acidosis, lysosomal enzymes which are cytotoxic, are released. They can cause myocardial depression and coronary vasoconstriction.

Prostacyclin is a vasodilator and inhibits platelet aggregation. Thromboxane A_2 causes pulmonary vasoconstriction and platelet aggregation. Leukotrienes cause vasoconstriction, platelet activation and increased vascular permeability. Endothelium-derived relaxing factor (EDRF) which is identified as nitric oxide (NO) is found to produce sustained vasodilatation and hypotension. Thrombosis is increased due to inhibition of antithrombin III. Thrombocytopenia is common.

Metabolic changes: Hepatic glycogenolysis due to increased level of glucagon, catecholamine and cortisol leads to hyperglycemia. There is diminished peripheral utilization of glucose due to increased level of insulin antagonists like cortisol and growth hormone. Inadequate oxygen supply to tissue initiates anaerobic metabolism. Consequently there is metabolic acidosis, production of lactic acid and H^+ ions. Sodium pump fails to operate. Finally the lysosomal enzymes are released. These lead to cell death.


PATHOGENESIS OF HYPOVOLEMIC SHOCK
Blood loss
Hypovolemia
Stimulation of VMC
▪ Baroreceptors (+)
▪ Chemoreceptors (+)
Neuroendocrine response
▪ ACTH (↑), GH (↑), Cortisol (↑)
▪ Glucagon (↑), β endorphin (↑)
▪ Adrenaline and noradrenaline (↑)
TEMPORARY COMPENSATORY MECHANISM
Reversible phase
Cardiac contraction
Rate ↑, force ↑
Vasoconstriction
Selective diversion of blood through metarteriolar shunt due to closure of pre- and postcapillary sphincters
Squeezing of blood from MCU (sympathetic squeeze)
Trans capillary filling from migration of fluids from the interstitial tissue spaces
Adequate tissue perfusion to vital organs
MCU = Microcirculatory unit
VMC = Vasomotor center
Irreversible phase
FAILS
Constriction of pre-and postcapillary sphincters → venous return (↓) → cardiac output (↓) → tissue perfusion (↓) → lactic acidosis (↑) → relaxation of pre capillary sphincters (third phase)
→ Sodium pump (↓) Lysosomal enzymes (↑), DIC → constriction of precapillary sphincters
→ Stagnation of blood in MCU → fluid leaks out in interstitial tissue space (fourth phase)

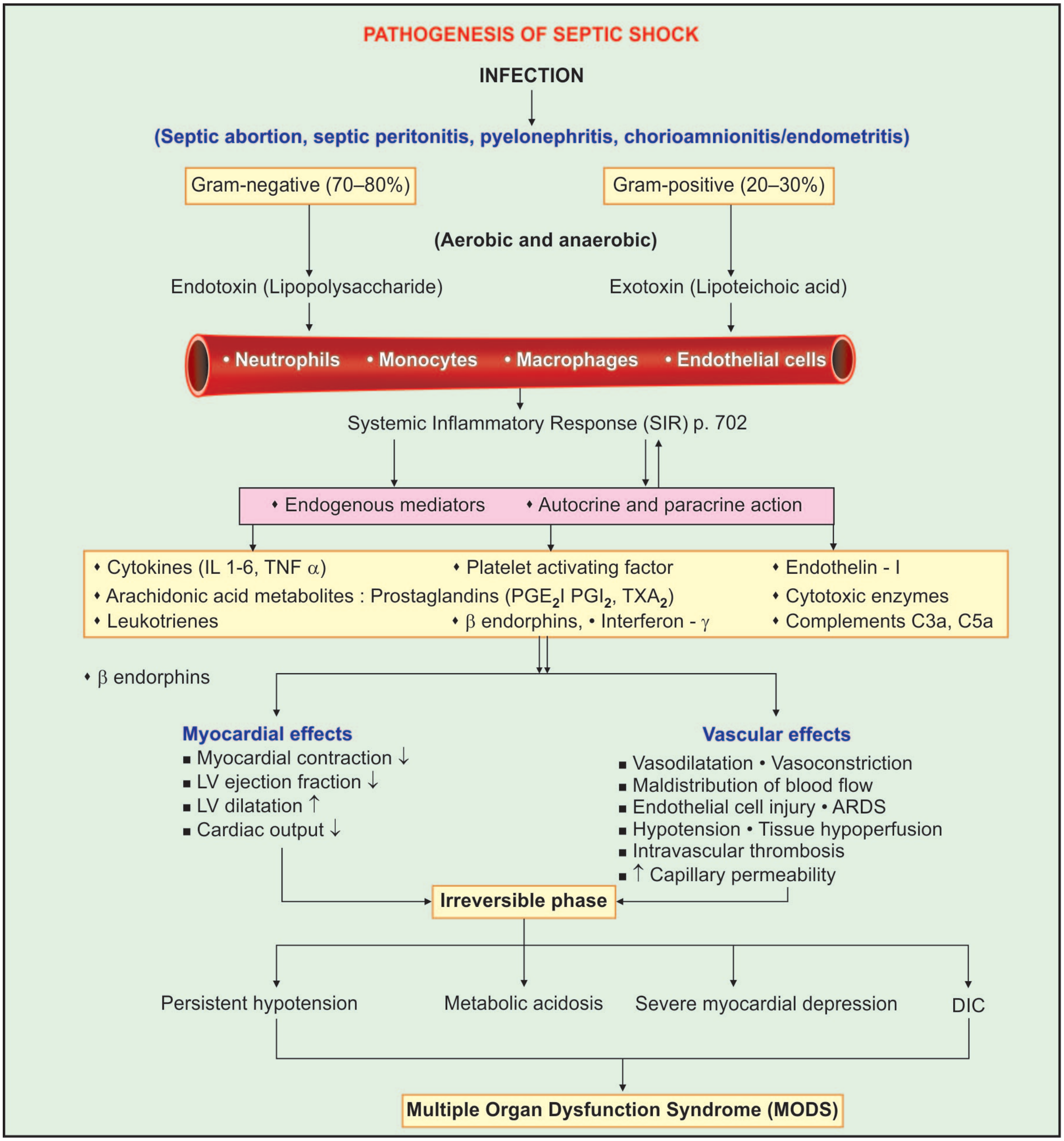
GENERAL CHANGES IN SHOCK (WITH SPECIAL REFERENCE TO HYPOVOLEMIC SHOCK)

There are four phases of changes. The first two phases are reversible; the third one probably correctable and the fourth is irreversible:

- ***First phase:*** Sympathetic impulses and the level of circulating catecholamines increase in response to hypovolemia, cardiogenic or neurogenic stimulus. Stretch receptors monitoring blood pressure in the carotid sinus and aortic arch supply information to the vasomotor center via the ninth and tenth cranial nerves. The vasomotor center responds by sending efferent impulses through the sympathetic nervous system.
- ***Second phase:*** As a result of excessive sympathetic stimulus, there is constriction of the pre- and postcapillary sphincters, resulting in inadequate venous return leading to diminished cardiac output, clinical manifestations of which are hypotension and tachycardia.

Compensatory mechanisms that operate at this stage, to maintain the blood pressure has been discussed in the scheme above.

These mechanisms attempt to correct hypovolemia, improve cardiac output and the perfusion of vital organs. **At this stage, transfusion and control of hemorrhage are usually effective in restoring the normal circulatory balance and tissue perfusion.**


PATHOGENESIS OF SEPTIC SHOCK
INFECTION
(Septic abortion, septic peritonitis, pyelonephritis, chorioamnionitis/endometritis)
Gram-negative (70–80%)
Gram-positive (20–30%)
(Aerobic and anaerobic)
Endotoxin (Lipopolysaccharide)
Exotoxin (Lipoteichoic acid)
• Neutrophils • Monocytes • Macrophages • Endothelial cells
Systemic Inflammatory Response (SIR) p. 702
• Endogenous mediators • Autocrine and paracrine action
• Cytokines (IL 1-6, TNF α)
• Platelet activating factor
• Endothelin - I
• Arachidonic acid metabolites : Prostaglandins (PGE2I PGI2, TXA2)
• Cytotoxic enzymes
• Leukotrienes
• β endorphins, • Interferon - γ
• Complements C3a, C5a
• β endorphins
Myocardial effects
■ Myocardial contraction ↓
■ LV ejection fraction ↓
■ LV dilatation ↑
■ Cardiac output ↓
Vascular effects
■ Vasodilatation • Vasoconstriction
■ Maldistribution of blood flow
■ Endothelial cell injury • ARDS
■ Hypotension • Tissue hypoperfusion
■ Intravascular thrombosis
■ ↑ Capillary permeability
Irreversible phase
Persistent hypotension
Metabolic acidosis
Severe myocardial depression
DIC
Multiple Organ Dysfunction Syndrome (MODS)

On the other hand, if bleeding continues or treatment is delayed, the changes at microcirculatory unit will continue to persist and will pass onto the third and fourth phases of shock.

♦ ***Third phase:*** Prolonged anoxia of the tissues will lead to excessive production of lactic acid (acidosis). **Lactic acid and anoxia cause relaxation of the precapillary sphincters but not the postcapillary sphincters.** In addition, thromboxane A_2 and leukotrienes (endogenous mediators) cause **damage to the endothelial cells** of the capillaries of the microcirculatory bed. These lead to formation of thrombus within the capillaries **(diffuse intravascular coagulation) and increased capillary permeability**.

♦ ***Fourth phase:*** Consequent to persistent constriction of the postcapillary sphincter, blood remains stagnant within the capillary bed. Fluid from the capillaries leaks into the tissue spaces due to increased permeability. **All fluids administered intravenously will go into the tissue spaces and circulatory blood volume cannot be restored.** Clinically, this is the stage of irreversible shock. There is severe **loss of systemic vascular resistance, severe myocardial depression (↓ cardiac output), unresponsive hypotension and ultimately multiple organ system failure**.

Systemic inflammatory response syndrome (SIRS) is manifested by two or more of the following conditions: (i) Temperature > 38°C or < 36°C (ii) HR > 90 bpm (iii) Respiratory rate > 24/min or (iv) $PaCO_2$ < 32 mm Hg or (v) WBC > 12000/µl or leukopenia: < 4000/µl or more than 10% immature forms.

CHANGES IN ENDOTOXIC SHOCK

Endotoxic shock usually follows infection with Gram-negative organisms (75-80%). The most common organism involved is *Escherichia coli* (50%). Other organisms occasionally responsible for endotoxic shock are, *Pseudomonas aeruginosa*, *Klebsiella*, *Proteus*, *Bacteroides* and *Aerobacter aerogenes*. Gram-positive organisms (*Staphylococcus*, *Streptococcus*), anaerobes (*Bacteroides fragilis*), *Clostridium* group are less common (20%).

Pathophysiology of endotoxic shock has been discussed before (see p. 701). Bacterial endotoxin causes **selective vasospasm** at the postcapillary end. Blood is pooled in the capillary bed. There is **inhibition of myocardial function and cellular damage** through complex biochemical changes (vide supra).

The patient in early septic shock feels warm due to vasodilatation. This is called **warm shock**. In the late phase, the patient feels cold due to vasoconstriction (sympathetic squeeze). This is called **cold shock or late shock**. Patient's skin becomes cold, clammy and ashen gray.

The various biochemical and pathological changes observed in endotoxic shock are: (i) Diffuse intravascular coagulation (ii) Increased capillary permeability (iii) Metabolic acidosis (iv) Release of superoxide (O_2^-) and hydroxyl (OH^-) radicals (v) Failure of sodium pump operation (vi) Water and electrolyte imbalance (vi) Excessive and uncontrolled **systemic inflammatory response (SIR)** can lead to organ changes. **Organ changes** depend on the degree of hypoperfusion and extent of the underlying pathology: (a) **Kidney**—Patchy and massive cortical necrosis leading to oliguria, anuria and azotemia. Persistent hypotension leads to acute tubular necrosis and ultimately renal failure. (b) **Liver—**Hepatocellular necrosis and degeneration ultimately leading to hepatic failure (c) **GI tract**—Hypoxic mucosal injury increases systemic sepsis by translocation of intraluminal microbes. Congestion, hemorrhage and ulceration are responsible for hematemesis (d) **Lungs—**Congestion or atelectasis leads to tachypnea or dyspnea, progressive hypoxemia and reduced pulmonary compliance. **ARDS** results from increased capillary permeability and thickening of the alveolar capillary membranes. Arterial PaO_2 is low (< 65 mm Hg). Mechanical ventilation is needed (e) **Coagulopathy (DIC)—**It is due to diffuse endothelial injury, microvascular thrombosis and thrombocytopenia. (f) **Adrenal insufficiency** is due to critical illness related corticosteroid insufficiency (CIRCI). CIRCI causes hypotension which is refractory to fluid replacement. Vasopressor therapy is needed. (g) **Heart**—Cardiac output decreases depending on the degree of hypotension, hypoperfusion and vasoconstriction. Myocardial ischemia → cardiac dysfunction → dysrhythmias → cardiac failure →↑ left ventricular end diastolic pressure (LVEDP) → pulmonary edema → tissue hypoxia (f) **Ultimately multiple organ failure develops.** Endotoxins have got special affinity for kidneys and lungs for reasons which are not very clear.

CLASSIFICATION OF SHOCK

Based on our understanding of the basic pathophysiology of shock and its clinical correlation; shock may be classified as follows:

1. **Hypovolemic shock:** Circulating blood volume is inadequate resulting from acute depletion. It may be — (i) **hemorrhagic** or (ii) **nonhemorrhagic**.

   Hemorrhagic shock: Associated with postpartum or postabortal hemorrhage, ectopic pregnancy, placenta previa, abruptio placenta, rupture of the uterus and obstetric surgery :
   - Shock associated with disseminated intravascular coagulation, Intrauterine dead fetus syndrome and amniotic fluid embolism (Table 39.1).

   Nonhemorrhagic shock:
   - Fluid loss shock — Associated with excessive vomiting, diarrhea, diuresis or too rapid removal of amniotic fluid.
   - Supine hypotensive syndrome—Due to compression of inferior vena cava by the pregnant uterus (see p. 61).

2. **Septic shock (endotoxic shock):** Hypotension (systolic BP < 90 mm Hg) is due to sepsis resulting in derangements in cellular and organ system dysfunction. Hypotension persists in spite of adequate fluid resuscitation. Associated typically with septic abortion, chorioamnionitis, pyelonephritis, and rarely postpartum endometritis

3. **Cardiogenic shock**:
   - Myocardial infarction
   - Cardiac arrest (asystole or ventricular fibrillation)
   - Cardiac tamponade

   Characterized by ↓ systolic pressure (< 80 mm Hg), ↓ cardiac index (< 1.8 L/min/m^2) and ↑ left ventricular filling pressure (> 18 mm Hg)

4. **Extracardiac shock:** Massive pulmonary embolism, amniotic fluid embolism, anaphylaxis, drug overdose, neurogenic.
   - **Chemical injury:** Associated with aspiration of gastrointestinal contents during general anesthesia **(Mendelson's syndrome)**.
   - **Drug-induced:** Associated with spinal anesthesia.

Table 39.1: Classification of Hemorrhagic Shock (Based on Total Blood Volume 6L)

Parameter	Class I	Class II	Class III	Class IV
Blood volume	≤ 15	15–30	30–40	> 40
Loss % (mL)	(< 750 mL)	(750–1500)	(1500–2000)	(> 2000)
Heart rate (bpm)	No change	Tachycardia	Moderate tachycardia	Marked tachycardia
Blood pressure	Normal	Normal	Decreased	Decreased
Respirations	Normal	Tachypnea	Tachypnea	Marked tachypnea
Mean arterial pressure	Normal	Mildly decreased	< 60 mm Hg	Decreased
Cardiac output	Normal	Mildly reduced	Reduced	Markedly reduced
Systemic vascular	Normal	Increased	Increased	Increased resistance
Urine output (mL/hr)	> 30	20–30	5–15	Anuric
Mental status	Normal	Anxious	Confused	Obtunded

CLINICAL FEATURES OF SHOCK: Clinical features of shock depend on the basic etiological factors and consequently the sequence of pathological changes occurring within the microvascular unit. **In early stages, the features of hypovolemic and septic shock are different. In the irreversible (late) phase, the clinical features are the same as the final pathology is multiple organ failure. It carries mortality of 30–100%**.

HEMORRHAGIC SHOCK

- ***Early phase (Compensatory phase):*** In the early phase there is mild vasoconstriction and with the compensatory mechanism operating, the patient has relatively normal blood pressure but tachycardia. **This phase can be easily managed by volume replacement.**
- ***Intermediate phase (Reversible phase):*** If the early phase remains untreated, the patient passes into the state of hypotension. Patient progressively becomes pale; tachycardia persists and due to intense vasoconstriction, the periphery becomes cold and there may be sweating. Due to diversion of blood to vital organs, the patient remains conscious and the urine output is within normal limits. **Still with adequate management, the shock state can be reversed**.

- ***Late stage (Irreversible):*** Hypotension continues and cannot be reversed by fluid replacement (CIRCI). Extremities become cold and clammy because of vasoconstriction due to sympathetic stimulation. Metabolic acidosis, coagulopathy and thrombocytopenia are associated. Practically imperceptible low volume pulse, oliguria, mental confusion is observed. Patient is in MODS (p. 701). **Treatment of any kind is practically useless in this phase and mortality varies between 3% and 100%.**

NEUROGENIC SHOCK: The basic pathological factors in both hemorrhagic and neurogenic shock are more or less the same except for the fact that hemorrhagic shock is hypovolemic and neurogenic shock, initially is normovolemic, though this becomes hypovolemic in the later phase due to pooling and stagnation of blood in the microvascular capillaries.

The compensatory phase, in neurogenic shock, however, is very transient. In the reversible phase, unlike hypovolemic shock, pallor is absent; on the contrary, the face may be flushed. Moreover, neurogenic shock does not show expected response to volume replacement. Temperature remains normal or subnormal.

ENDOTOXIC SHOCK: **In the initial phase** of endotoxic shock, because of precapillary dilatation and diversion of blood through metarteriolar shunt, the patient remains alert, there is marked flushing of the face and the skin feels warm. There are temperature changes, > 38°C or < 36°C, bounding pulse, heart rate > 100 beats per min, respiratory rate > 20/min, WBC > 12000/mL^3. Pathophysiology of septic shock has been described (see p. 701).

MANAGEMENT OF SHOCK

HEMORRHAGIC SHOCK: **Basic management of hemorrhagic shock is to stop the bleeding and replace the volume which has been lost** (Table 39.2). Prompt diagnosis and immediate resuscitation is essential failing which multiple organ failure develops.

- **Restore circulating volume** (*Infusion and transfusion*)**:** Blood should be transfused especially in hemorrhagic shock as soon as it is available. **Crystalloids** : Normal saline has to be infused initially for immediate volume replacement. But they are rapidly lost from circulation. **Colloids**: Polygelatin solutions (Hemaccel, Gelofusion) are iso-osmotic with plasma. They do not interfere with the coagulation system. Large volumes can be administered. They promote osmotic diuresis. Dextrans: They are polymolecular polysaccharides. They interfere with crossmatching and they are avoided. **Human albumin solutions (4.5%)**—not generally used for volume replacement.

- ***Maintenance of cardiac efficiency:*** When a large volume of fluid or blood is to be administered, the cardiac competence or efficiency should be ascertained– otherwise there is a risk of overloading the circulation and cardiac failure. 6 liters of crystalloids may be needed for loss of 1 liter of plasma volume. One or two large bore (14 or 16 gauge) cannula are inserted for volume replacement. Packed red blood cells (specific blood component), combined with normal saline, are used for hemorrhagic shock. Hemodynamic monitoring is aimed to maintain systolic BP > 90 and MAP ≥ 60 mm Hg, CVP 12-15 cm H_2O and pulmonary capillary wedge pressure 14-18 mm Hg.

- ***Administration of oxygen to avoid metabolic acidosis*:** In the initial phase, administration of oxygen by nasal cannula at a rate of 6-8 liters per minute is enough but in the later phases, ventilation by endotracheal intubation may be necessary. Oxygen delivery should be continued to maintain O_2 saturation > 92%, PaO_2 80-100 mm Hg, $PaCO_2$ 30-35 mm Hg and pH > 7.35. Endotracheal intubation and mechanical ventilation may be needed for patients with septic shock. Indications of mechanical ventilation are : severe tachypnea (RR > 40/min), altered mental status, severe hypoxemia, despite O_2 supplementation.

- ***Pharmacological agents:*** Use of vasopressor drugs should be kept to a minimum, since peripheral vasoconstriction is already present. The role of vasoactive drugs, inotropes and corticosteroids in shock has been discussed in detail in connection with management of endotoxic shock.

- ***Control of hemorrhage:*** Specific surgical and medical treatment for control of hemorrhage should start along with the general management of shock. The specific management of each variety of obstetric hemorrhage has been outlined in the related chapters.

Monitoring: Clinical parameters like skin temperature, visible peripheral veins can be helpful to assess the degree of tissue perfusion. Urine output (> 30 mL/hr) is a useful guide. Arterial blood pressure is a poor indicator to assess tissue perfusion. Invasive monitoring may not be needed in a straight forward case. In a critically ill patient, however, measurement of central venous pressure (CVP), to assess the adequacy of patient's circulating volume and the contractile state of the myocardium, is essential. Pulse oximeter and blood gas analysis are useful to assess tissue perfusion. Measurement of left atrial pressure (pulmonary artery occlusion pressure) by 'Swan-Ganz' catheters could be done in selected cases.

ENDOTOXIC SHOCK

Investigations to organize in a patient with septic shock: CBC, hematocrit, coagulation profile, (platelet count, serum fibrinogen, FDPs, PT, APTT), liver and renal function tests, chest radiograph, USG, CT or MRI may be needed for localizing pelvic pathology and also ECG monitoring.

Principles of management are: (a) to correct the hemodynamic unstability due to sepsis (endotoxin), (b) appropriate supportive care and (c) to remove the source of sepsis.

Two wide bore cannulas are sited. Foley's catheter is inserted. Oxygenation with (face mask) is to be given. Mechanical ventilation may be needed in a severe case.

Goal of hemodynamic resuscitation should be able to maintain (a) Mean arterial pressure >70 mm of Hg (b) CVP 10-15 cm H_2O, (c) Urine output 0.5 ml/kg/hour, (d) Central venous oxygen saturation >70%.

This includes administration of antibiotics, intravenous fluids, adjustment of acid base balance, steroids, inotropes, prevention and treatment of intravascular coagulation and toxic myocarditis, administration of oxygen and elimination of the source of infection.

- ***Antibiotics:*** Endotoxic shock is most commonly due to Gram-negative organisms, so proper antibiotics should be administered in adequate doses. The choice of antibiotic will depend upon the sensitivity test but before the report is available, broad spectrum antibiotics covering Gram-positive, Gram-negative and anaerobic organisms should be started. Ampicillin (2G IV every 6 hours), gentamicin (2 mg/kg IV loading dose followed by 1.5 mg/kg IV every 8 hours) and metronidazole (400 mg IV every 8 hours) is a good combination to start with. Alternative regimen is to give Imipenem – cilastatin (500 mg IV every 6 hours), meropenem (1 gm every 8 hours) or ertapenem (1 gm IV every 24 hours) ± aminoglycoside. Clindamycin 600 mg IV infusion (single dose) is an alternative to metronidazole.
- ***Intravenous fluids and electrolytes:*** Septic shock associated with hemorrhagic hypotension should be treated by liberal infusion and blood transfusion. Isotonic crystalloid (Ringer's lactate/normal saline) should be given. The amount of fluid to be administered can be precisely assessed by monitoring the pulse, BP, urine output and recording the central venous pressure. Alternatively, a rough calculation of the amount of fluid to be administered can be assessed by the volume of urinary output and its specific gravity. **Oliguria with high specific gravity is an indication for liberal fluid administration, whereas a low specific gravity indicates fluid restriction.** Impairment of renal function contraindicates administration of electrolytes. Estimation of blood electrolytes (Na, K, bicarbonate) is a helpful guide.
- ***Correction of acidosis:*** Acidosis and hypoxemia depress myocardial contractility. Bicarbonate should be administered to correct persistent metabolic acidosis (pH < 7.2) only. A reasonable first dose would be 50-100 mEq (60–110 mL of 7.5%) of sodium bicarbonate solution. Further doses will depend on the clinical state of the patient and blood gas analysis result.
- ***Maintenance of blood pressure:*** Inotropic agents—in a critically ill patient when there is hypotension (MAP < 60 mm Hg) and impaired perfusion of vital organs despite adequate volume replacement, inotropes should be used. Adrenaline, noradrenaline, dopamine and dobutamine have both inotropic and vasoconstrictive effects. Dopamine is still the drug of choice. Its main action is on β-adrenoreceptors, increasing cardiac contractility and cardiac output without change in rate. In a dose of 1-3 $\mu g\ Kg^{-1}\ min^{-1}$ it increases renal cortical plasma flow and glomerular filtration. Inotropic effect is observed with 3-10 $\mu g\ Kg^{-1}\ min^{-1}$ doses. Dobutamine (β_1 and β_2 adrenergic) is used in cardiogenic shock. Adrenaline is a very potent α and β agonist and is sometimes used in patients who do not respond to dopamine or dobutamine especially in septic shock.

Vasodilator therapy: In selected cases, (MAP > 70 mm Hg) *afterload* reduction may improve stroke volume and reduce ventricular wall tension. Sodium nitroprusside and nitroglycerin could be used for that purpose. This is done under continuous hemodynamic monitoring.

Diuretic therapy: To reduce fluid overload (*preload*) and pulmonary edema, diuretics should be used. Frusemide is the drug of choice.

- ***Corticosteroids:*** Patients with severe sepsis develop systemic inflammatory response syndrome (see p. 705) or relative adrenal insufficiency (CIRCI). Corticosteroids could be used as anti-inflammatory agents to improve mortality. The dose recommended in septic shock is 50 mg of hydrocortisone per kg body weight. **The advantages claimed are:** (i) exerts an anti-inflammatory effect at the cellular level (ii) stabilizes lysosomal membrane (iii) counteracts anaerobic oxidative mechanism (iv) improves the regional blood flow (microcirculation) and thereby reverse the metabolic acidosis (v) exerts positive inotropic effect to improve cardiac efficiency and (vi) some vasopressor effect.

- ***Treatment of diffuse intravascular coagulation:*** When there is low fibrinogen level, reduced platelet count and increased fibrin degradation products, heparin therapy should be considered. As a prophylactic measure, heparin 5000 IU subcutaneous or intravenous route at 8 hourly interval can be given safely. Alternatively, fresh frozen plasma or whole blood transfusion could be done.
- ***Treatment of myocarditis:*** Myocarditis most often is associated with septic hypotension. There is no specific treatment apart from the treatment of endotoxemia. Under exceptional circumstances when there is evidence of congestive cardiac failure or features of atrial fibrillation or flutter, digitalis may be administered.
- ***Elimination of source of infection:*** Surgical intervention should be done to eliminate the source of infection. Evacuation of the retained products of conception or hysterectomy for a case with septic abortion or puerperal sepsis should be done without delay. Removal of the source of infection may make the patient hemodynamically stable. Hysterectomy has been advocated in unresponsive endotoxic shock following septic abortion or puerperal sepsis.
- **Intensive insulin therapy** is done in patients with severe sepsis and septic shock to maintain normal blood glucose level. These patients often develop hyperglycemia which further increases the risk of septicemia and death.

Table 39.2: Presenting Features of Shock (Hemorrhagic and Septic)

Early			**Late**
Organ System	**Hemorrhagic (Hypovolemic)**	**Septic**	**Hemorrhagic and Septic)**
BP	Normotensive or hypotensive, narrow pulse pressure	Normotensive or hypotensive widened pulse pressure	Hypotension (extravascular pooling)
Pulse	Tachycardia, thready pulse	Tachycardia, bounding pulse	Tachycardia — due to myocardial ischemia, ↓ ejection fraction
Respiratory	Normal or tachypnea (sympathetic response)	Tachypnea, pulmonary edema, acidosis	Tachypnea, ARDS
Renal	Oliguia (↓ Perfusion)	Oliguria (afferent arteriolar vasoconstriction)	Oliguria due to acute renal failure
Skin	Cold, clammy (vasoconstriction) → sympathetic response	Warm (febrile response)	Cold, clammy — due to vasoconstriction
Mental status and others	Normal	Normal ● Fever or hypothermia (endotoxin)	Disorientation, obtundation due to hypoxia, cerebral edema. **Others**: Multiple organ dysfunction, anaerobic metabolism, coagulapathy, thrombocytopenia

H_2–blockers: Antacids to reduce the stress ulcer of gastric mucosa either by oral or H_2-blocking agents (IV) are used.

Nutritional support is maintained as total parenteral nutrition (TPN). Usually 20-30 Kcal/kg/day is equally distributed between fat and carbohydrate. Serum electrolytes, BUN, glucose, creatinine should be monitored on a regular basis.

Recombinant human-activated protein C therapy (Drotrecogin Alfa): Activated protein C is one endogenous protein that inhibits inflammation, thrombosis and promotes fibrinolysis. It reduces mortality in patients with severe sepsis as it reduces coagulopathy and inflammation.

ACUTE KIDNEY INJURY (AKI)

(Syn: Acute Renal Failure in Obstetrics)

The important functions of the kidney are: (1) To excrete the waste products from the body (2) To maintain the acid base equilibrium by selectively excreting the acid and base. These functions can be adequately maintained provided the blood supply to the organs and the functional integrity of the nephrons (units of the kidney) remain adequate.

DEFINITION: Acute renal injury (failure) is defined as a condition in which the urine volume falls below 400 mL in 24 hours, the minimum amount necessary for the excretion of the normal solute load. **Oliguria** is the term given to the clinical condition. **Anuria is the absence of excretion of urine in 12 hours.**

INCIDENCE: During recent years **acute renal failure in obstetrics has decreased significantly. The probable factors are:** (1) **Diminished number of septic abortion** with liberalization of abortion laws (2) **Judicious and early termination** in severe preeclampsia (3) **Better understanding** of the pathophysiology and management of shock (4) **Appropriate management of abruptio placenta** (5) **Facilities of blood transfusion**.

There is significant physiological and anatomical changes in the kidney during pregnancy (p. 63). The clinical relevance of these changes are enormous. Postpartum decrease in size should not be mistaken for renal parenchymal loss. Dilatation of the pelvicalyceal system should not be mistaken for obstructive uropathy. The atonicity of ureter, bladder results in frequent urinary tract infections. Serum bicarbonate, urea, creatinine levels are lower in pregnancy.

CAUSES OF ACUTE KIDNEY INJURY (AKI)

Causes are broadly classified into: **(A)** Causes unrelated to the pregnant state **(B)** Causes peculiar to the pregnant state. The second group may be divided into **three categories**: (1) Prerenal ARF (2) Intrinsic renal ARF and (3) Postrenal ARF (Table 39.3).

Prerenal ARF is due to hypovolemia and/or low cardiac output resulting in renal hypoperfusion.

Table 39.3: Causes of Acute Renal Injury (Failure) (Prerenal) in Pregnancy

Early Pregnancy	Late Pregnancy and Labor	Other Causes in Pregnancy
(1) **Acute and massive hemorrhage:** Abortion, ectopic pregnancy, hydatidiform mole	(1) **Acute and massive hemorrhage**: Postpartum hemorrhage, placenta previa, traumatic delivery, obstetric shock	(1) **Mismatched blood transfusion**
(2) **Severe dehydration**: Hyperemesis gravidarum, acute pyelonephritis	(2) **Abruptio placenta:** The pathological bais of ARF are: (a) Hypovolemia; (b) DIC	(2) **Renal:** Renal disease, DIC, hypoperfusion, ischemia, toxins, obstetric pathology superimposed on pre-existing renal disease (interstitial nephritis)
(3) **Septic abortion:** Septicemia, endotoxic shock hypotension	(3) **Severe preeclampsia, eclampsia,** HELLP syndrome	(3) **Postrenal** (Obstructive): Accidental ligature of the ureters during cesarean section, hysterectomy for rupture of uterus (rare)
	(4) **Severe infection:** Chorioamnionitis, IUFD, pyelonephritis	

PATHOLOGY OF ARF: Prerenal is the most common form of AKI (ARF). It is due to mild to moderate degree of renal hypoperfusion. Mild and even moderate ischemia with acute tubular necrosis are reversible. In severe ischemia, renal cortical tissue is damaged and this pathology is irreversible.

ACUTE TUBULAR NECROSIS is the most common pathology in obstetrics. The lesion begins in Henle's loop, especially in the intermediate zone, involving particularly the ascending limb and distal convoluted tubules and is fully developed after 48 hours. **Naked eye changes are** : enlarged kidneys with a pale looking cortex. The medulla is darker. Microscopically, the renal tubules undergo ischemic degeneration and necrosis and are blocked with casts and pigments. Interstitial tissues become edematous. Provided the tubules have adequate blood supply, the epithelium will slowly regenerate and the renal function will usually return to normal in 1-2 weeks if it is taken care of in time.

ACUTE CORTICAL NECROSIS: It is relatively uncommon and seen in abruptio placenta and endotoxic shock following Gram-negative septicemia. Usually diffuse ischemic necrosis occurs all over the cortex. A narrow band of cortex immediately beneath the capsule and a portion in the region of the junction of cortex and medulla are the only parts of the cortex to survive. These parts derive their blood supply from sources other than the usual afferent glomerular vessels which undergo hyaline degeneration. The glomerular afferent vessels are end arteries and thus the damage that occurs in the segment of the nephron supplied by these arteries is irreversible, hence the ultimate fatality.

CLINICAL FEATURES: When anuria is reversible, the clinical condition can be divided into four phases:

- **Incipient phase**
- **Phase of anuria**
- **Phase of diuresis**
- **Phase of recovery**

INCIPIENT PHASE: The phase is short lasting. There is marked diminution in urinary output.

PHASE OF ANURIA: This phase lasts from a few hours to as long as 3 weeks. An urinary output is less than 400 mL in 24 hours. Initially, the patient remains alert and looks well. Gradually anorexia, vomiting and diarrhea may occur. Then the patient looks toxic; blood pressure is raised and abdomen becomes distended. Still untreated–the patient becomes drowsy, has Cheyne-Stokes respiration with dry, furred tongue, twitching of muscles and mental confusion. Delirium followed by coma is the end result.

Blood biochemical changes: There is gradual rise in the concentration of plasma urea, potassium, creatinine and phosphate as a result of endogenous protein catabolism. The rise in plasma potassium is aggravated by the retention of hydrogen ions which are forced into the cells in exchange of intracellular potassium ions. The plasma concentration of bicarbonate diminishes as acidosis occurs which is due to shifting of hydrogen ions intracellularly. Simultaneously, there is rise of phosphate which leads to lowering in plasma calcium. The fall in calcium and rise in potassium level have got a combined adverse effect on the cardiac function which may cause death. A simultaneous rise of plasma magnesium potentiates the harmful effect of rising plasma potassium.

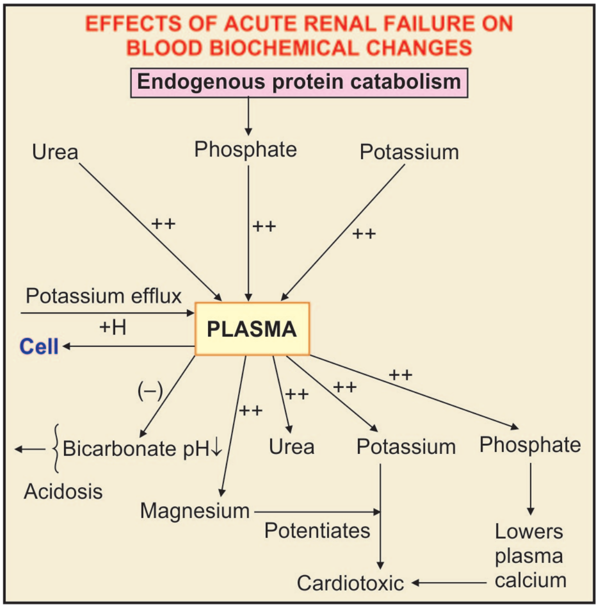
INVESTIGATIONS: ■ **Blood:** Leukocytosis may be evident and is a better index of infection than the rise of temperature.

■ **Urine:** Physical examination shows scanty and dark colored urine. Specific gravity is 1020 or more in prerenal causes and 1010 or less in renal causes. Protein is present in varying amounts. Microscopic examination reveals—presence of casts and red cells.

■ *Blood biochemical findings:* Urine sodium concentration is < 10 mmol/L in prerenal and > 20 mmol/L in renal causes. Urine osmolality is more than 500 m osmol/L in prerenal and less than 350 m osmol/L in renal causes. *Urine* **:** *Plasma* creatinine ratio > 40 in prerenal ARF and < 20 in intrinsic renal ARF.

There is raised sodium (normal 136–145 mEq/L); potassium (normal 3.5–5 mEq/L); chloride (normal 100–106 mEq/L) and urea level (normal 20–25 mg%). Standard bicarbonate level falls resulting in acidosis (normal 24–32 mEq/L). Arterial blood gases are done to detect acidosis.

■ **ECG—for evidence of rise in plasma potassium:** Serial electrocardiographic tracing is important. The findings are**:** (a) Gross peak of the 'T' waves (b) Absence of 'P' waves (c) Prolonged 'QRS' complex to 0.2 seconds.



PHASE OF EARLY DIURESIS: In this phase, tubular reabsorption is delayed up to a period usually proportionate to the period of anuria. The only favorable feature is the increased excretion of urine. But the rise of potassium, sodium, creatinine (BUN) and chloride continues and the specific gravity of the urine is still low.

THE PHASE OF LATE DIURESIS: The phase is as hazardous as the previous one. **The causes of diuresis are:** (1) Osmotic diuresis due to high blood urea (2) Functional inadequacy of tubular reabsorption (3) Release of surplus fluid and electrolytes, particularly sodium and potassium.

PHASE OF RECOVERY: Tubular epithelium regenerates and tubular function is reestablished along with the establishment of glomerular activity. The concentration of the electrolytes either in the plasma or in the urine gradually returns to normal values and so also the specific gravity of the urine. It may take about 1 year for restoration of full function.

MANAGEMENT OF AKI IN OBSTETRICS
(Prerenal)

PREVENTIVE: Prevention of ischemic ARF is of paramount importance as there is no specific therapy (see p. 709).

ACTUAL TREATMENT: The first thing is to exclude retention of urine (obstruction). The possibility of inadvertent injury to the ureters in susceptible cases should also be excluded.

IN THE VERY EARLY PHASE (INCIPIENT PHASE): Restore intravascular volume by blood transfusion, colloid or crystalloids whichever is appropriate. Severe hypovolemia due to massive hemorrhage is corrected by packed red cells. For mild to moderate hemorrhage, isotonic saline is usually optimum. Serum potassium and acid base status (bicarbonate) is monitored carefully.

Forced diuresis

- **Mannitol:** 100 mL of 20% mannitol is administered intravenously slowly taking at least 10 minutes. The diuresis is expected to occur within 1 hour. If it fails, the infusion may be repeated after 2 hours.

Mechanism: (1) Lowers blood viscosity and renal vascular resistance (2) Dilates the afferent glomerular arterioles by reducing endothelial cell swelling (3) Causes osmotic diuresis that literally washes out the renal tubules forcefully.

- **Frusemide**—Use of frusemide (Lasix) 80–120 mg intravenously, two doses at intervals of 2 hours is advocated.

PHASE OF ANURIA: The patient needs all types of biochemical studies and to be managed in collaboration with a nephrologist. ***The principles in the management* are:** (1) To control the fluid balance (2) To maintain the caloric requirement (3) To regulate the electrolyte imbalance (4) To give adequate supportive therapy (5) To prevent complications (hyperkalemia, hyperphosphatemia, hypocalcemia, metabolic acidosis).

- ***Fluid balance:*** In the anuric phase, water is eliminated through the extrarenal routes, i.e. lungs (approx 400 mL), skin (approx 600 mL) and stool. **Thus, the fluid loss is approximately 1000 mL per day.** The loss is probably a little more in the hot and humid climate of the tropics. There is production of water through endogenous oxidation of protein and fat amounting to 400-500 mL per day. This is termed as water of oxidation. It is free of electrolytes.

Thus to maintain adequate fluid balance, an estimated amount of 500 mL of fluid plus the amount equal to that of vomitus or that recovered from the gastric aspiration should be administered daily. Addition should also be made for diarrhea or excessive sweating. A further supplement equal to the volume of urine passed each day should be added. An extra 200 mL is allowed for each degree of rise of body temperature above 100° F. **It should be emphasized that the danger of slight dehydration is much less than even slight overhydration.**

- ***Nutrition:*** To meet the metabolic needs, the patient requires about 1500-2000 Kcal/day. Protein and salts are restricted. Endogenous protein catabolism is kept at minimum. The energy is provided mainly by carbohydrate diet in the form of glucose, rice or bread. Each 100 g of glucose gives 400 Kcal. Glucose not only supplies the necessary calories, but it prevents protein catabolism and thus minimizes potassium and urea production. 100 g of carbohydrate per day reduces the protein breakdown by 50%. Protein of high biological value (essential amino acids) 0.6 g/kg is given. Nutritional management is easier in nonoliguric patient and patient on dialysis.

Complications are treated depending upon the blood values and biochemical abnormalities:

- **Hyperkalemia:** ♦ Restriction of dietary K^+ intake, use of K^+ sparing diuretics, glucose (50 mL of 50% dextrose IV) and regular insulin 10 units) or dialysis.
  - **Carbohydrate intake** should be raised and fruit juices must be curtailed.
  - **Administration of 10 units of soluble insulin** SC and 50 mL of 50% dextrose by IV encourages migration of potassium from the extracellular fluid into the cells. It may be repeated after 2 to 4 hours.
  - **Calcium gluconate 10% of 10 mL** is given IV very slowly. It acts by reducing the cardiotoxic effect of potassium.
  - **Potassium binding ion exchange resins** may be given for a day or two. It absorbs potassium in the intestine from the blood and intestinal secretions and thereby removes it.
- **Hyperphosphatemia:** ♦ Restriction of dietary phosphate intake, phosphate binding agents (calcium carbonate, aluminium hydroxide).

Metabolic acidosis: Dietary protein is restricted. Sodium bicarbonate IV is given when serum HCO_3^- is < 15 m mol/L or arterial pH is < 7.2.

Hemodialysis (Artificial kidney): The principle of the artificial kidney is that of dialysis across a semipermeable (cellulose based) membrane which allows the crystalloids to diffuse down their concentration gradient but not the colloids. The crystalloids like potassium will leave the blood and enter the bath which is in continuous counter-current flow. **It is better to dialyse too early than too late**. When this method is adopted, there is little restriction as regard the intake of protein, fluids and energy. This is important for the repair of damaged nephrons. **Dialysis is no longer a last resort and the following are the accepted indications**:

- Clinical evidence of uremia
- Severe acidosis (serum HCO^-_3 < 13 mEq/L)
- Intractable intravascular volume overload
- Hyperkalemia (serum K^+ > 6.5 mEq/L)
- Blood urea nitrogen > 120 mg/dL or blood urea > 150 mg/dL
- **Prophylactic dialysis** (urea > 100 mg/dL or creatinine > 5 mg/dL)

Peritoneal dialysis: This can correct electrolyte imbalance and is equally effective as the hemodialysis. The peritoneum acts as a semipermeable membrane. It can be carried out in centers with special nephrological expertise and facilities. Continuous renal replacement therapies (arteriovenous hemodiafiltration) are needed in cases where intermittent hemodialysis fails to control uremia. The process is hazardous in paralytic ileus and in pregnancy. It is contraindicated in the presence of peritonitis. Peritoneal dialysis could be intermittent (IPD) or continuous ambulatory (CAPD).

Hemodialysis in pregnancy often causes wide fluctuation of blood pressure. Continuous EFM (p. 693) should be continued during dialysis. Patient must have at least 70 g of protein and 1.5 g of calcium daily. Hematocrit should be above 25%. Packed red cell transfusion may be given. Risk of preterm labor is high as progesterone is removed during dialysis. Parenteral progesterone therapy is advocated in patients with dialysis. Maternal complications are placental abruption, heart failure and sepsis.

- ***Supportive therapy includes***

For control of infections: Penicillins, cephalosporins and metrogyl can be used safely. **Nephrotoxic drugs** are either avoided or the doses and the frequencies are adjusted according to serum level. Intravascular volume overload are avoided by restriction of salt and water or by diuretics (loop) or dialysis.

Blood transfusion —If the hemoglobin level is at any time below 70%, transfusion of packed cells is given, as anemia by itself can reduce renal perfusion. **Human (recombinant) erythropoietin** 2000 units weekly to be given subcutaneously to combat the anemia of erythropoietin deficiency. Additional oral calcium, vitamin D, folic acid, IV iron need to be given.

PHASE OF DIURESIS: In this phase, the intake and output chart should be maintained and serum electrolytes are measured.

- ***Fluid balance:*** Initially the fluid intake is calculated on the basis of the total amount of urinary output in previous 24 hours plus 500 mL. When the urinary output exceeds 1000 mL, oral feeding is started with high calorie low protein constituent along with fruit juice.

- ***Electrolyte balance:*** As there is loss of potassium the accumulated potassium is likely to be excreted by this time. Potassium supplementation is required at this stage.

Salt supplements are usually needed during the diuretic phase to compensate for the increased urinary loss. On an average, about 5 g of sodium chloride is given for each liter of urine passed.

PHASE OF RECOVERY: This is recognized by general features of clinical improvement and increased amount of urine with more and more concentrating power evidenced by diurnal variation of specific gravity between 1.002 and 1.020. When this stage is reached, there should not be any restriction of diet, and fluid intake should be totally guided by the patient's thirst.

PROGNOSIS: Most ARF is reversible. The kidney has got the unique ability to recover from loss of function. However, patients usually die from the sequelae of the primary illness and not from ARF itself. In obstetrics, overall mortality due to ARF is about 15% and slightly high in sepsis related ARF. Renal parenchymal injury is associated with high mortality. Prognosis of the fetus is unfavorable and there is about 50% mortality.

POSTPARTUM RENAL FAILURE (postpartum hemolytic uremic syndrome).

It is a clinical condition of acute irreversible renal failure occurring within the first 6 weeks postpartum. The exact cause is still obscure. It may be due to—(a) drug sensitivity (like ergot) (b) consumptive coagulopathy or (c) result of peripheral immunological mechanism. Therapy consists of hemodialysis with or without heparin.

OBSTRUCTIVE RENAL FAILURE: Obstructive anuria due to ureteric ligation should be dealt with promptly. If the general condition permits, delegation or implantation of the ureters into the bladder can be carried out after prior confirmation by cystoscopy and ureteric catheterization. If the general condition is too poor, bilateral nephrostomy is the life-saving procedure.

BLOOD COAGULATION DISORDERS IN OBSTETRICS

Disseminated Intravascular Coagulation (DIC), is a clinicopathologic syndrome characterized by widespread intravascular fibrin deposition in response to excessive blood protease activity that overcomes the natural anticoagulant mechanism.

Control of blood loss from the vessels depends on the following:

- **Muscular contraction**
  - Vascular contraction (vasoconstriction)
  - Myometrial contraction (adjacent to the vessels)
- **Tissue pressure**
- **Platelet functions**
- **Blood coagulation mechanism**

Physiological changes: Procoagulant factors markedly increased in pregnancy are I, VII, VIII, IX and X. Factors either unchanged or mildly increased are II, V and XII. Factors that decline are XI and XIII (see p. 59).

NORMAL BLOOD COAGULATION

Normal intravascular blood coagulation is linked with three different interrelated systems.

These are:

- Coagulation system
- Coagulation inhibitory system
- Fibrinolytic system

Pathological disturbance of one or more of the systems lead to intravascular coagulation or a tendency to bleed.

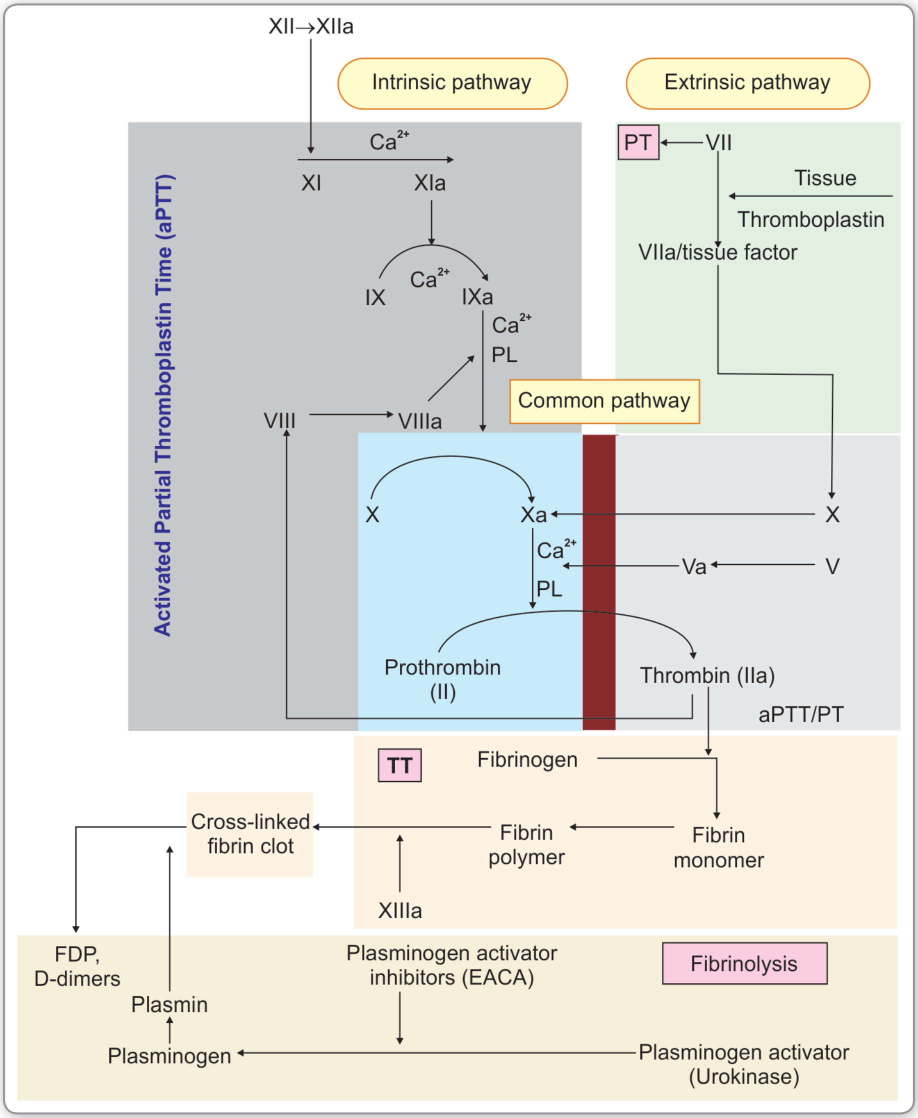
COAGULATION MECHANISM: The complex system of blood coagulation, "Enzyme cascade theory" involves two different pathways, viz. **intrinsic and extrinsic**. Both are initiated by different stimuli and ultimately they culminate into a common pathway for final conversion of inert prothrombin to thrombin.

COAGULATION INHIBITORY SYSTEM: There are number of naturally occurring anticoagulants in blood, to counterbalance the hypercoagulable state in pregnancy. Antithrombin III (AT III) is a main physiological inhibitor of thrombin and factor Xa. Protein C combined with protein S and thrombomodulin inactivates factors V and VIII. Their deficiency is associated with recurrent thromboembolism.

PLASMA FIBRINOLYTIC SYSTEM: Tissue plasminogen is activated to plasmin by tissue activators (urokinase, streptokinase). In turn, plasmin lyses fibrinogen and fibrin to fibrin degradation products (FDP). Serum FDPs are detected by immunoassays as D-dimers. Blood coagulation and fibrinolysis work side by side to maintain hemostasis and patency of microcirculation. There are several **plasminogen inhibitors** like epsilon-aminocaproic acid and (EACA) and tranexamic acid (AMCA).

PHYSIOLOGICAL CHANGES IN PREGNANCY

During pregnancy there is increase in concentration of clotting factors II, V, VII, VIII, IX, X and XII. Plasma fibrinogen level is significantly increased. There is a small decrease in platelet count, due to low grade intravascular coagulation. Plasma fibrinolytic activity is suppressed during pregnancy and labor. It returns to normal within 1 hour of delivery of the placenta. This is due to liberation of plasminogen inhibitor from the placenta.


XII→XIIa
Intrinsic pathway
Extrinsic pathway
Activated Partial Thromboplastin Time (aPTT)
Ca2+
XI
XIa
PT
VII
Tissue
Thromboplastin
VIIa/tissue factor
Ca2+
IX
IXa
Ca2+
PL
VIII
VIIIa
Common pathway
X
Xa
X
Ca2+
Va
V
PL
Prothrombin
(II)
Thrombin (IIa)
aPTT/PT
TT
Fibrinogen
Cross-linked
fibrin clot
Fibrin
polymer
Fibrin
monomer
XIIIa
FDP,
D-dimers
Plasminogen activator
inhibitors (EACA)
Fibrinolysis
Plasmin
Plasminogen
Plasminogen activator
(Urokinase)

Coagulation cascade and laboratomy assessment of clotting factor deficiency: activated partial thromboplastin time (aPTT), Prothrombin time (PT) and thrombin time (TT).

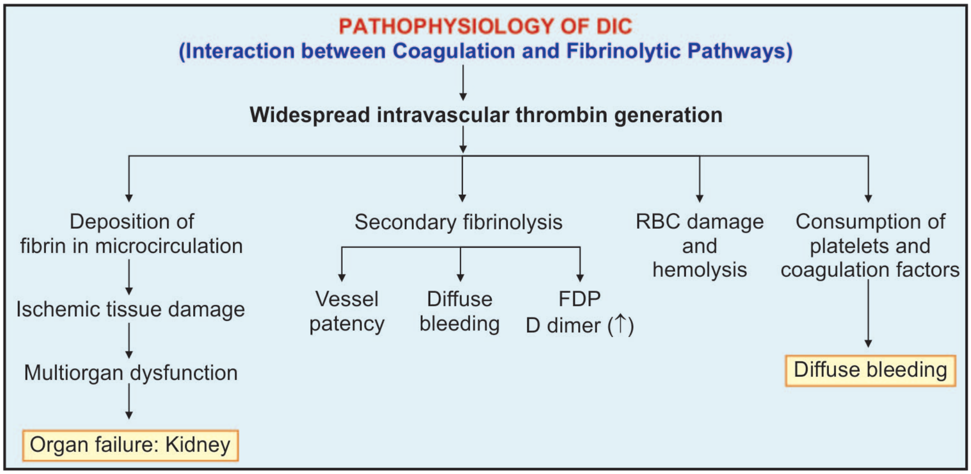
PATHOLOGICAL CONDITIONS OF ACQUIRED COAGULOPATHY

Obstetric complications and trigger factors for DIC

Endothelial Injury	Release of Thromboplastin	Release of Phospholipids
♦ Preeclampsia, eclampsia, HELLP syndrome ♦ Septicemia ● Septic abortion ● Chorioamnionitis ● Pyelonephritis ♦ Hypovolemia	♦ Amniotic fluid embolism ♦ Dead fetus syndrome ♦ Abruptio placenta ♦ Hydatidiform mole ♦ Cesarean section ♦ Intra-amniotic hypertonic saline ♦ Shock	♦ Fetomaternal bleed ♦ Incompatible blood transfusion ♦ Hemolysis ♦ Septicemia

All these clinical conditions may trigger the delicate hemostatic mechanism either by endothelial injury or by release of thromboplastin and phospholipids. It is always a secondary phenomenon and never primary. Because of the hypercoagulable state in pregnancy, presence of any provocative factor can easily upset the normal balance culminating into disseminated intravascular coagulopathy (DIC). It is sometimes called "defibrination syndrome" but because other constituent factors besides fibrin are also consumed, a better nomenclature would be "consumptive coagulopathy". **The blood fibrinogen level of 100 mg/100 mL is arbitrarily considered to be a critical level.**

Chronic DIC is a compensated state commonly observed in a case with dead fetus syndrome. Plasma levels of FDP, D-dimers are raised, aPTT, PT and fibrinogen are within the normal range. There may be mild thrombocytopenia and red cell fragmentation.


PATHOPHYSIOLOGY OF DIC
(Interaction between Coagulation and Fibrinolytic Pathways)
Widespread intravascular thrombin generation
Deposition of fibrin in microcirculation
Ischemic tissue damage
Multiorgan dysfunction
Organ failure: Kidney
Secondary fibrinolysis
Vessel patency
Diffuse bleeding
FDP D dimer (↑)
RBC damage and hemolysis
Consumption of platelets and coagulation factors
Diffuse bleeding

MECHANISM OF ACQUIRED COAGULOPATHY

ABRUPTIO PLACENTA: *Mechanism*

- **Massive retroplacental clot**—Not only the fibrinogen along with other procoagulants is consumed in the clot but after the clot retraction, the serum component is absorbed into the circulation, thereby further reducing the circulatory procoagulant.
- **Thromboplastin liberated** from the clot, damaged decidua and uterine musculature enters into the circulation and **produces DIC**.
- Because of precipitating shock, **synthesis of the essential coagulation factors fails to occur promptly**.
- **Fibrinolysis—**It serves as a protective mechanism to dissolve the fibrin clot so as to restore patency in the microcirculation.
- **Level of fibrin degradation products (EDP)** is raised. It inhibits myometrial contraction.

AMNIOTIC FLUID EMBOLISM

***Mechanism*:** Liquor amnii is forced into the maternal circulation either through a rent in the membranes or placenta. **Thromboplastin rich liquor amnii containing the debris, blocks the pulmonary arteries and triggers the complex coagulation mechanism leading to DIC.** There is massive fibrin deposition distributed throughout the entire pulmonary vascular tree. If the patient survives from the severe cardiopulmonary embarrassment which stimulates thromboembolic phenomenon, there will be severe clotting defect with profuse bleeding per vaginam or through the venopuncture sites due to consumption of coagulation factors.

From the damaged endothelium of the pulmonary arteries, massive fibrinolytic activators are produced which excite the fibrinolytic system converting the plasminogen to plasmin which in turn produces lysis of fibrin, fibrinogen and even the factor V and factor VIII. **Thus, there is secondary fibrinolysis on top of primary fibrinogen depletion arising out of DIC.**

ENDOTOXEMIA—***Mechanism:*** (1) **Prior to development of shock,** there appears a hypercoagulable state which adversely reacts with the presence of endotoxin leading to DIC. The phenomenon is comparable to generalized Shwartzman reaction; the difference is that a single stimulus instead of two is enough during the pregnant state to produce the reaction. There is extensive DIC including deposition of fibrin in the renal vascular system.

(2) **In the shock stage,** the endothelium of the capillaries in the microcirculation is damaged due to anoxia; associated stasis of blood favors DIC.

(3) **Increased production of activators from the damaged capillary endothelium** triggers the fibrinolytic activity and causes defibrination.

RETAINED DEAD FETUS: The fall in fibrinogen level is gradual and usually becomes evident following retention of the dead fetus for more than 4 weeks. Defibrination is likely to be more following retention of dead fetus due to Rh-incompatibility. **There is gradual absorption of thromboplastin liberated either from the placenta or from amniotic fluid or decidua.** This results in depletion not only of fibrinogen but also the factor VIII and platelets. **In response to DIC, there is enhanced fibrinolytic activity which in turn reduces the fibrinogen level further.** Dead fetus with living twin is a hazard for the living fetus rather than the mother. Single fetal death with a living fetus in a monochorionic twin pregnancy having vascular anastomoses is associated with fall in the level of plasma fibrinogen and rise in FDPs.

INSTILLATION OF INTRA-AMNIOTIC HYPERTONIC SALINE: Thromboplastin released from the placenta, fetus and the decidua due to necrobiotic effect gains access into the circulation and causes defibrination.

CESAREAN SECTION: Primary defibrination following cesarean section may be due to: (1) Entry of thromboplastin or amniotic fluid into the circulation through the open vessels on the uterine wound (2) Excess production of plasminogen activators from the injured uterine site.

CLINICAL MANIFESTATIONS: The manifestations of blood coagulation disorder are evidenced by hemorrhage from various sites.

Before delivery: There are signs of bruising, prolonged bleeding at the injection sites (venopuncture or intramuscular), gum bleeding or hemorrhage from the gastrointestinal tract and hypotension.

After delivery: Apart from the manifestations already described, there is postpartum hemorrhage in spite of a hard and well contracted uterus (traumatic bleeding being excluded). The hemorrhage usually occurs 1-2 hours following delivery. There may be bleeding from the suture sites (episiotomy wound) or hematoma formation in the abdominal wound following cesarean section or formation of a vulval hematoma following vaginal delivery.

INVESTIGATIONS

Bedside tests to evaluate the blood coagulation disorders can give useful information that help in tackling the crisis. Detailed laboratory investigations require an equipped set up to give round the clock service.

Bedside tests that may be done are: (1) Bleeding time (2) Coagulation time (3) Clot observation test (4) Peripheral smear (5) Circulatory fibrinolysis test.

Clot observation test (Weiner)—It is an useful bedside test. It can be repeated at 2–4 hours intervals. 5 mL of venous blood is placed in a 15 mL dry test tube and kept at 37°C. Usually, blood clot forms within 6-12 minutes. This test provides a rough idea of blood fibrinogen level. If the clotting time is less than 6 minutes, fibrinogen level is more than 150 mg percent. If no clot forms within 30 minutes, the fibrinogen level is probably less than 100 mg percent.

Peripheral blood smear—Peripheral blood smear when stained with Wright's stain may be of help. (i) If less than four platelets per high power field are seen, thrombocytopenia is diagnosed. Thrombocytopenia is a feature of DIC but not of fibrinolytic process. (ii) RBC morphology—In DIC, the cell shape will be 'helmet shaped' or fragmented whereas in fibrinolytic process, the cell morphology will be normal.

Essential Laboratory tests to know the specific defects in the coagulation mechanisms are: (1) Platelet count (2) Activated partial thromboplastin time (intrinsic coagulation) (3) Prothrombin time (extrinsic coagulation) (4) Thrombin time (5) Fibrinogen estimation (6) Fibrin degradation products (FDP) (7) D–dimer (see p. 744).

Measurement of FDP is an indirect evidence of fibrinolysis. The determination of a low platelet count is of far more diagnostic significance than the finding of a raised FDP level in the fibrinolytic process. The most valuable and rapid clotting screen is thrombin time, where thrombin is added to citrated plasma. Thrombin time of normal plasma is 10-15 seconds. Thrombin time is prolonged where fibrinogen is depleted. Normal values of blood coagulation profile are given in page 744.

TREATMENT

PREVENTIVE: Blood coagulation disorders in obstetrics of sufficient magnitude to cause hemostatic failure have been reduced to a great extent. ***The responsible factors in prevention are* the changes in the trends of obstetric management**:

- **Abruptio placenta**: (a) Massive blood transfusion (b) To expedite early delivery by low rupture of the membranes supplemented by oxytocin drip (c) Liberalization of cesarean section.
- **Intrauterine death**: (a) Expectant attitude is reduced (b) Availability of potent oxytocics (prostaglandins) to empty the uterus (p. 378).
- **Better understanding of the pathophysiology of shock** and early institution of appropriate therapy.
- **Acute fulminating preeclampsia, eclampsia and HELLP syndrome have been substantially reduced** by effective care (see p. 267).
- **Avoiding instillation of hypertonic saline** for induction of abortion.
- **Infusion of polymolecular gelatin** (hemaccel or gelofusion) as plasma expander avoiding dextran.
- **Emptying the uterus and controlling the infection early** with antibiotics.

Adjuvant therapies (Vitamin K)—The vitamin K dependent factors II, VII, IX, X are consumed in DIC. 5-10 mg of Inj Vit K given (IM), can help to replenish these procoagulants.

CURATIVE: The management goal is to identify and to correct the underlying pathology with priority. Women with severe DIC are treated for hemodynamic parameters, respiratory support and surgical intervention when needed. In most cases, delivery of the fetus brings the resolution of coagulopathy. The other part of the management is to achieve a platelet count > 50,000/μL and a fibrinogen level > 100 mg/dL.

ACTUAL MANAGEMENT

♦ **Volume replacement** ♦ **Blood component therapy** ♦ **Heparin** ♦ **Fibrinolytic inhibitors**

- **Volume replacement** by crystalloids (Ringer's solution) or by colloids (haemaccel or gelofusine or Human albumin 5%) will reduce the amount of whole blood needed to restore the blood volume. The crystalloids remain in the vascular compartment less compared to Colloids. Dextran should be avoided as they adversely affect platelet function and blood crossmatching tests. Two large bore IV catheters are sited.
- **Whole blood transfusion** is the sheet anchor to replenish not only the fibrinogen but also the other procoagulants. **500 mL of fresh blood raises the blood volume, the fibrinogen level approximately by 12.5 mg /100 mL and adds 10,000–15,000 platelets per cumm.** Whole blood is rarely used in modern obstetrics due to its disadvantages.
- **Fresh frozen plasma (FFP)** is extracted from whole blood. It contains fibrinogen, antithrombin III, clotting factors V, XI, XII. FFP transfusion provides both volume replacement and coagulation factors. One unit of FFP (250 mL) raises the fibrinogen by 5-10 mg/dL. FFP needs to be ABO or Rh compatible.
- **Cryoprecipitate** is obtained from thawed FFP. It is rich in fibrinogen, factor VIII, Von Willebrand's factor, and XIII. Cryoprecipitate provides less volume (40 mL) compared to FFP (250 mL). So it should not be used for volume replacement. One unit of cryoprecipitate increases the fibrinogen level by 5-10 mg/dL.
- **Platelet concentrates** may be given to a patient with very low platelet count (< 50,000/mL) and persistent bleeding. Platelets should be given rapidly over 10 minutes. It should be ABO and Rh specific. Transfusion of a single unit of platelets is expected to raise the count between 5,000 and 10,000/mL. In case of sensitization Rh-immunoglobulin 300 μg is given. Several units (5-10 units) of platelet concentrates are to be transfused, as one unit (50 mL) raises the platelet count by 7,500/mL. Single donor concentrates are preferred as the immunogenic and antigenic risks are low.
- **Packed red blood cells** (PRBC) are most effective to improve oxygen carrying capacity. Oxygen carrying capacity is reduced when hemoglobin level is < 8 g/dL even in a euvolemic patient. Transfusion reactions are less compared to whole blood transfusion. Each unit contains about 300 mL (250 mL RBC and 50 mL of plasma). One unit of PRBC will raise the hemoglobin by 1 g/dL and hematocrit by 3%. It must be ABO compatible.

- **Recombinant activated factors VIIA (rFVIIA):** (60-100 μg/kg IV) can reverse DIC within 10 minutes as it is a precursor for extrinsic clotting cascade (p. 712) which is replaced. It also activates platelets and the coagulation cascade.
- **Autotransfusion** is the collection of blood from the operative field (blood salvage), filter the blood and then transfusing the red cells back to the patient. The device for autotransfusion is called **cell saver**. The advantages are : less risks of infectious disease transmission and immunological reactions.
- **Alternative oxygen carriers or artificial hemoglobin solutions** have short intravascular half-life. Recent meta-analysis has revealed a significant risk of mortality and myocardial infarction.

Heparin: It should be used when the vascular compartment remains intact. In acute condition such as **amniotic fluid embolism,** intravenous heparin 5000 units repeated 4-6 hours intervals is useful to stop DIC and may be life saving. In retained dead fetus, there is progressive but slow defibrination due to DIC. In such cases, the process can be arrested by intravenous heparin. In acute DIC, heparin may aggravate bleeding.

Fibrinolytic inhibitors: Place of fibrinolytic inhibitors is very limited. Fibrinolysis may be a protective phenomenon. Commonly available antifibrinolytic agents are—(1) EACA—inhibits plasminogen and plasmin (2) Trasylol—inhibits plasmin (3) Aprotinin—nonspecific enzyme inhibitor. Fibrinolytic inhibitors are mainly indicated in postpartum hemorrhage following abruptio placenta in spite of a firm and contracted uterus and when blood fibrinogen level is 200 mg% or more. However these drugs can increase the risk of thrombosis.

Conclusion: **Prompt replacement of blood volume and coagulation factors** is an important step in the management of coagulation disorders in obstetrics. **Management of the triggering factor** (e.g. immediate delivery in a case of abruptio placenta) should be done along with. This will improve the hemostatic competence in vast majority of cases. With adequate perfusion of vital organs, not only the activated coagulation factors and FDP are promptly removed by the reticuloendothelial system but there is accelerated synthesis of procoagulants especially by the liver.

Risks of Blood Transfusion: The **adverse reactions** are: (A) Immune mediated reactions: (a) Febrile (b) Allergic (c) Anaphylactic (d) Hemolytic and (e) Transfusion related lung injury (TRALI) which is an immune mediated condition and causes ARDS. (B) Transfusion related infections: Virus (HBV, HCV, CMV, HIV), parasites (malaria), bacteria (C) Others: (a) Fluid overload, (b) Hypothermia, (c) Electrolyte imbalance (K↑, Ca↓) and (d) Acidosis.

HIGH-RISK PREGNANCY

DEFINITION: High-risk pregnancy is defined as one where pregnancy is complicated by factor or factors that adversely affects the outcome—maternal or perinatal or both.

All pregnancies and deliveries are potentially at risk. However, there are certain categories of pregnancies where the mother, the fetus or the neonate is in a state of increased jeopardy. About 20–30% pregnancies belong to this category. If we desire to improve obstetric results, this group must be identified and given extra care. Even with adequate antenatal and intranatal care, this small group is responsible for 70–80% of perinatal mortality and morbidity.

Majority (70-90%) of fetal deaths occur before the onset of labor due to chronic asphyxia (30%), congenital malformation (15%) and with some superimposed complications of pregnancy (30%). Twenty percent of stillbirths have no obvious fetal, placental or maternal or obstetric etiology.

In the developed countries, the maternal deaths have been brought down to irreducible minimum and as such it may be prudent to consider only perinatal morbidity and mortality in identifying high-risk cases. But in the developing countries with a high maternal and perinatal mortality, the maternal factors should also be considered. The risk factors may be preexisting prior to or at the time of first antenatal visit or may develop subsequently in the ongoing pregnancy labor or puerperium. It must be remembered that over 50% of all maternal complications and 60% of all primary cesarean sections arise from the high-risk group of cases.

SCREENING OF HIGH-RISK CASES

The cases are assessed at the initial antenatal examination, preferably in the first trimester of pregnancy. This examination may be performed in a big institution (teaching or nonteaching) or in a peripheral health center. In rural areas, the initial screening may be done by properly trained paramedical personnel. From the peripheral areas the high-risk cases are sent to referral hospitals in subdivisions, districts or territory care centers for management by specialists. Some risk factors may later appear and are detected at subsequent visits. The cases are also reassessed near term and again in labor for any new risk factors (Table 39.4). The neonates are also assessed very soon after delivery for any high-risk factor. It is obvious that all abnormalities do not carry the same risk; some have a lower risk as compared to others carrying a very high-risk for the mother or the fetus.

INITIAL SCREENING

HISTORY

- ***Maternal age:*** **Pregnancy is safest between the ages of 20-29 years**. Age < 16/> 30 years and pregnancy following a long period of infertility, after induction of ovulation are of high risks.
- ***Reproductive history:*** Second and third pregnancies after a normal first delivery carry the low risk.

Table 39.4: The High-risk Factors in Pregnancy

Reproductive History	Medical Disorders in Pregnancy (p. 303)	Previous Surgery (p. 354)
▪ Two or more previous miscarriage or previous induced abortion. These cases run the risk of further abortion or preterm delivery ▪ Previous stillbirth, neonatal death or birth of babies with congenital abnormality ▪ Previous preterm labor or birth of a IUGR or, macrosomic baby ▪ Grand multiparity ▪ Previous cesarean section or hysterotomy ▪ Third stage abnormalities (PPH) – this has a particular tendency to recur ▪ Previous infant with Rh-isoimmunization or ABO incompatibility	**Diseases —** ▪ Pulmonary disease – tuberculosis (see p. 341) ▪ Renal disease (Pyelonephritis) (see p. 346) ▪ Thyroid disorders (see p. 334) ▪ Psychiatric illness (see p. 512) ▪ Cardiac disease (se p. 319) ▪ Epilepsy (see p. 338) ▪ Viral hepatitis (see p. 336) ▪ Preeclampsia, eclampsia ▪ Anemia (see p. 304) ▪ Infections in pregnancy (Malaria, HIV) (see p. 344, 350)	▪ Myomectomy ▪ Repair of complete perineal tear ▪ Repair of vesicovaginal fistula ▪ Repair of stress incontinence ▪ In all these conditions, fetal or maternal outcome or both may be affected **Family History** ▪ **Socioeconomic status** — Patients belonging to low socioeconomic status have a higher incidence of anemia, preterm labor, growth retarded babies ▪ **Family history** of diabetes, hypertension or multiple pregnancy and congenital malformation.

According to WHO, risk approach for MCH care is to identify the high-risk cases from a large group of antenatal mothers. These cases are:

During pregnancy: (1) Elderly primi (≥30 years) (2) Short statured primi (≤140 cm) (3) Threatened abortion and APH (4) Malpresentations (5) Preeclampsia and eclampsia (6) Anemia (7) Elderly grand multiparas (8) Twins and hydramnios (9) Previous stillbirth, IUD, manual removal of placenta (10) Prolonged pregnancy (11) History of previous cesarean section and instrumental delivery (12) Pregnancy associated with medical diseases.

During labor: (1) PROM (see p. 369) (2) Prologned labor (3) Hand, feet or cord prolapse (4) Placenta retained more than half an hour (5) PPH (6) Puerperal fever and sepsis

EXAMINATION:

General Physical Examination	Pelvic Examination
▪ Height: Below 150 cm, particularly below 145 cm in our country ▪ Weight: Overweight or underweight Body mass index (BMI) : Weight/(height)^2 BMI : 20–24 is accepted as normal (see p. 400) ▪ High blood pressure ▪ Anemia ▪ Cardiac or pulmonary disease ▪ Orthopedic problems	▪ Uterine size – disproportionately smaller or bigger ▪ Genital prolapse ▪ Lacerations or dilatation of the cervix ▪ Associated tumors ▪ Pelvic inadequacy

COURSE OF THE PRESENT PREGNANCY: After the initial visit, the cases should be reassessed at each antenatal visit to detect any abnormality that might have arisen later. Few examples are—preeclampsia, anemia, Rh-isoimmunization, high fever, pyelonephritis, hemorrhage, diabetes mellitus, large uterus; lack of uterine growth, postmaturity (both the conditions may be associated with poor placental function); abnormal presentation, twin and history of exposure to drugs or radiation, acute surgical problems.

COMPLICATIONS OF LABOR: The cases should be reassessed during late pregnancy and labor. Attention is turned to detect the risks that may develop during labor. **Some important points to be considered are:**

- Patients having no antenatal care
- Anemia, preeclampsia or eclampsia
- Premature or prolonged rupture of membranes
- Amnionitis
- Meconium-stained liquor
- Abnormal presentation and position
- Disproportion, floating head in labor
- Multiple pregnancy
- Premature labor
- Abnormal fetal heart rate
- Patients admitted with prolonged or obstructed labor
- Rupture uterus
- Patients having induction or acceleration of labor

Certain complications may arise during labor and place the mother or baby at a high risk. Examples are:

- Intrapartum fetal distress
- Difficult forceps or breech delivery
- Postpartum hemorrhage or retained placenta
- Delivery under general anesthesia
- Failed forceps
- Prolonged interval from the diagnosis of fetal distress to delivery. If more than 30 minutes elapse from the recognition of fetal distress to delivery, the mortality increases threefolds.

POSTPARTUM COMPLICATIONS: An uneventful labor may suddenly turn into an abnormal one in the form of PPH retained placenta, shock or inversion or sepsis may develop later on. **The condition of the neonate** should be assessed after delivery. **The following categories of neonate are at high risk:**

- Apgar score below 7
- Birth weight less than 2500 gm or more than 4 kg
- Convulsions
- Respiratory distress syndrome
- Hypoglycemia
- Fetal infection
- Persistent cyanosis
- Anemia
- Major congenital abnormalities
- Jaundice
- Hemorrhagic diathesis.

Some workers have introduced a scoring system for screening of high-risk cases; such a scoring system is not essential. However, if one wants to introduce a scoring system, local factors and experience of previous management should be taken into consideration before attaching a particular score to any abnormality.

MANAGEMENT OF HIGH RISK CASES

If we desire to improve our obstetric results, the high-risk cases should be identified and given proper antenatal, intranatal and neonatal care. This is not to say that healthy uncomplicated cases should not get proper attention. But in general they need not be admitted to specialized centers and their care can be left to properly trained midwives and medical officers in health centers, or general practitioners. It is necessary that all expectant mothers are covered by the obstetric service of a particular area. The service of trained community health workers and assistant nurse-cum-midwife of health centers should be utilized to provide the primary care and screening in rural areas and urban and semiurban pockets. **A simple checklist** should be prepared for them to fill up; arrangement should be made for early examination of the high-risk cases by medical officers of health centers in the health center itself or in small community antenatal clinics situated in different rural area, catering to a small group of population. The health centers of clinics should have periodic specialist cover from teaching or nonteaching hospitals, as well as district and subdivisional hospitals. The general practitioner or medical officer of health centers, in collaboration with the specialists will decide what type of cases (with a comparatively lower risk) can be managed at home or health centers. Cases with a significantly higher risk should be referred to specialized referral centers. Cases from rural areas may be kept at maternity waiting homes close to the referral centers. **The organizational aspect may be summarized as follows:**

- Strengthen midwifery skills, community participation and referral (transport) system
- Proper training of resident, nursing personnel and community health workers
- Arranging periodic seminars, refresher courses with participation of workers involved in the care of these cases

- Concentration of cases in specialized centers for management
- Community participation, proper utilization of health care manpower and financial resources where it is mostly needed
- Availability of perinatal laboratory for necessary investigations; availability of a good pediatric service for the neonates
- Lastly, improvement of literary rate, health awareness of the community and economic status.

Cases having a previous unsuccessful pregnancy should be seen and investigated before another conception occurs. Investigations like hysterography, hysteroscopy, laparoscopy or transvaginal ultrasonography should be performed to rule out Mullerian abnormality. Complete investigations for hypertension, diabetes, kidney disease or thyroid disorders should be undertaken and proper treatment instituted in the nonpregnant state. Sexually transmitted diseases should be treated before embarking on another pregnancy. Cervical tears should also be repaired in the nonpregnant state. Serology for toxoplasma IgG, IgM and antiphospholipid antibodies should be done and corrected appropriately when found positive (see p. 186).

Folic acid (4 mg/day) therapy should be started in the prepregnant state and is continued throughout the pregnancy. Early in pregnancy after the initial clinical examination, routine and special laboratory investigations should be undertaken. Necessary advice should be given regarding diet, activities, rest and medicines. Minimum medicines should be taken during pregnancy, particularly in the early months.

Assessment of maternal and fetal well-being: This should be done at each antenatal visit according to the guidelines given in the appropriate chapter; maternal complications should be looked for and treated, if necessary.

Cases for Antepartum Fetal Monitoring (Biophysical and Electronic) p. 693

Cases with uteroplacental insufficiency	Prolonged pregnancy	Diabetes mellitus
IUGR	Maternal age: < 15, > 30 years	Isoimmunization in pregnancy
Hypertension	Previous stillbirth	Polyhydramnios/ oligohydramnios
Antepartum hemorrhage	Multiple pregnancy	Hematological disorders in pregnancy

MANAGEMENT OF LABOR: It is evident that elective cesarean section is necessary in a high-risk case. Some cases may need induction of labor after 37-38 completed weeks of gestation. Those cases who go into labor spontaneously or after induction, need close monitoring during labor for the assessment of progress of labor or for any evidence of the fetal hypoxia.

The condition of the fetus can be assessed by—

- Fetal heart rate monitoring**:** By stethoscope, fetoscope or Doppler—Continuous electronic monitoring (see p. 692)
- Passage of meconium in the liquor (see p. 693)
- Examination of fetal scalp blood for pH values (see p. 696)

If there is any evidence of fetal anoxia in the first stage or there is failure to progress, cesarean section is necessary. The condition of the neonate is assessed immediately after delivery. Many of these babies need expert neonatal care. Delivery is conducted in an institute with equipped neonatal care unit.

IMMUNOLOGY IN OBSTETRICS

Many obstetric problems are now explained with the complexity of immunology. In this chapter a short review of the selected areas will be made highlighting the immunological explanations.

BASIC IMMUNOLOGY OF PREGNANCY: One tissue that is repeatedly grafted and repeatedly tolerated is the fetus. The mysterious mechanism of the immune system that prevents rejection of fetus remains unknown to the immunobiologists.

Why the fetal allograft that receives half of its histocompatibility antigens from the father is not rejected? Successful pregnancy itself is an immunological paradox. Some complications in pregnancy are associated with underlying immunological etiology. For better understanding, some areas of basic immunology are discussed first.

The immune system is generally divided into two arms: (i) **Innate** and (ii) **Adaptive**.

The **innate immune system** is the (a) First line of host defense against infection. (b) It works fast once it recognizes the pathogens. (c) It cannot identify the self vs non-self (nonspecific). (d) It involves complement system. The immune cells involved are : phagocytic (neutrophils, monocytes and macrophages) cells, natural killer cells (NK cells), eosinophils and basophils. The immune responses are by the release of **cytokines** (TNFα, IL-1, IL-6) and **chemokines** (IL-8, MIP-1α, MCP-1). The phagocytic cells ingest and digest microorganisms with lysosomal enzyme. The **NK cells** can recognize and distinguish between normal cells and cells infected with a virus or tumor (self or non-self) through the expression of MHC Class I antigens. The NK cells destroy cells that are deficient in HLA Class I molecules.

Adaptive Immune System: (a) It works as a second line defense against infection (b) It has delayed response (c) It can discriminate 'self' from 'non-self' (d) It prevents re-infection through 'immunological memory'. It is contributed mainly by two types of cells. Lymphocytes (B and T lymphocytes) and antigen presenting cells (APC). T lymphocytes are classified into T helper (Th) and T cytotoxic (Tc) cells. Th cells have two subtypes– Th 1 and Th 2. Th 1 cells activate macrophages and are involved in cell mediated immunity. Th 2 cells activate B cell differentiation and are involved in humoral immunity. When pathogens (viruses) replicate inside the cells and are inaccessible to antibodies, are destroyed by T cells.

Major Histocompatibility Complex (MHC) is a complex of genes with multiple loci. It is located on the chromosome 6. MHC encodes two types of membrane proteins. Those are known as **Human Leukocyte Antigens (HLA)** Class I and Class II. The process of graft rejection generally involves recognition of foreign MHC molecules by host T l hymphocytes.

(1) IMMUNOLOGY IN PREGNANCY

- Pregnancy is not an immunodeficient state. Women are able to respond to both humoral and cell mediated immunity against the paternal antigen.
- Specific types of NK cells (large granular lymphocytes) are present in the decidua mainly at the site of implantation. These NK cells (Uterine NK cells) are different from blood NK cells. These U-NK cells control the trophoblast proliferation, invasion when they interact with the trophoblast HLA Class I molecule. The U-NK cells depend on progesterone for survival. U-NK cells contribute to maternal tolerance of the fetus and maintenance of pregnancy. NK cells share many antigenic and functional similarities to 'T' cells. There is high (30%) proportion of NK cells in fetal circulation (13 weeks). Due to their high number, early presence and the ability to kill cells, it is likely that these NK cells are very important in fetal innate immune system. Uterine macrophages are the major source of nitric oxide and prostaglandins.
- Trophoblast cells are derived from the fetal tissue and invade the decidua. These trophoblast cells (placenta) form the interface between the fetus and the mother. Thus the placenta forms an efficient barrier against the transmission of immunocompetent cells between the fetus and the mother.
- The trophoblast covering the chorionic villi (villous trophoblasts) comes in contact with the maternal blood in the intervillous space and interacts with maternal systemic immune response. It does not express HLA Class I or Class II molecules.
- The trophoblast in contact with the decidua (extravillous trophoblast) expresses HLA Class I molecules but not HLA Class II molecule. This interacts with maternal local uterine immune response.
- Placenta presents no immunocompetent cells due to lack of HLA antigens. Placenta acts as an efficient immunological barrier.
- During pregnancy, maternal immune response is shifted (immunomodulation) from Th 1 (cell mediated) to Th 2 (humoral mediated) type. Th 2 type response is beneficial due to the production of anti-inflammatory cytokines. **Immunomodulation** results in improvement of woman with rheumatoid arthritis in pregnancy.
- During pregnancy there is production of antibodies of paternal antigens. These are anti–HLA antibodies and antibodies against sensitized T cells. These antibodies have no major effect on pregnancy outcome.
- Immunological mechanisms involved in pregnancy are not the same as that of organ transplantation. Immunological tolerance through complement and cytokines regulation is protective for pregnancy.
- The relationship between HLA matching between the partners and fetal loss is not based on any evidence.

Other postulations are—

- **Maternal fetal cell trafficking and microchimerism** : Maternal tolerance of fetus is due to bidirectional cell trafficking between the mother and fetus. Cell free fetal DNA (Cff DNA) and also infant fetal cells are detected in maternal circulation during entire pregnancy. The existence of two cell populations in a single person is known as microchimerism. It is likely that microchimerism may have beneficial effects.
- **Immunosuppressive factors** that operate in pregnancy are: estradiol, progesterone, hCG and prolactin. Fetal tolerance is probably due to the presence of alpha fetoprotein. A number of pregnancy associated glycoproteins, e.g. α_2 macroglobulin and placental interferon have immunosuppressive properties. Amniotic fluid is rich in immunosuppressive phospholipids.

Immune Tolerance:

- Immune tolerance of normal pregnancy at the maternal-fetal interface is maintained by the interaction of HLA-G with uterine NK cells. This effect predominates in a normal pregnancy.
- The levels of complements and cytokines (proinflammatory factors) are often raised during pregnancy. Inhibition of such complements and cytokines by the placenta reduces the immune mediated pregnancy complications.

(2) ABO HEMOLYTIC DISEASE OF THE NEWBORN (p. 388): Jaundice in newborn infants within 24 hours of birth may be due to ABO isoimmunization of the mother. The incidence is higher in group 'O' mothers carrying group A_1 fetuses. Except for the fact that A_1 antigen is strong, it has not been clearly known as to why the A_1 fetuses mostly take the brunt. It is postulated that IgG anti-A/anti-B are formed more commonly in group 'O' mothers. IgM anti-A/anti-B maternal antibodies are also known to play some role in bringing about ABO hemolytic diseases of the newborn (p. 388).

(3) Rh-ISOIMMUNIZATION (p. 386): Though entry of fetal blood in maternal circulation can take place at any time during pregnancy, fetomaternal bleed is common in the third trimester, particularly during separation of the placenta. 0.1 mL of Rh-positive fetal blood is sufficient to bring about immunization in Rh-negative mother. Immunization against RhD antigen is most frequent as 'D' is more potently antigenic than the other Rh-antigens like C, E, etc. Maternal immunization against Non-D Rh and other blood group antigens like 'Kell', 'Duffy', etc. are also in record. Fetal RhD positive red cells gaining entry into the circulation of Rh-negative mother take several weeks to immunize her. Under these circumstances, the mother will form anti RhD agglutinin which will pass again through the placental barrier into the fetus giving rise to agglutination or hemolysis of fetal erythrocytes which ultimately may lead to dangerous situations like hydrops fetalis, icterus gravis neonatorum or kernicterus.

(4) PREECLAMPSIA/ECLAMPSIA: In preeclampsia the abnormal immunological response develop in two stages:

A. Abnormal placentation and spiral artery remodeling: This is due to decreased placental HLA-G expression. HLA-G has a major role in placentation and blood flow development as observed in a normal pregnancy. There is failure of extravillous trophoblasts invasion and spiral artery remodeling. This is due to failure of interaction of extravillous trophoblasts with uterine NK cells and HLA-C receptors.

B. Preeclampsia is associated with widespread systemic inflammation and endothelial dysfunction. The immune dysfunction in preeclampsia are as follows:

- There is decrease in regulatory T cells both in number and function.
- There is insufficient shift from Th-1 to Th-2 as opposed to normal pregnancy where Th-2 predominance is observed.
- There is a higher level of cytokine abnormalities with increased concentration of TNFα, IL-6, IL-1β, IL-8 and lower concentration of IL-10.
- In eclampsia there is abnormal maternal-fetal immune interactions.

(5) SPORADIC AND RECURRENT MISCARRIAGE: There are some observations suggesting an immunological interaction in cases with sporadic and recurrent miscarriages.

- Cytokines are immune molecules. Th-1 cells produce proinflammatory cytokines whereas Th-2 cells produce anti-inflammatory cytokines. In a normal pregnancy there is a shift of Th-1 response to Th-2 response. Progesterone has an immunomodulatory role to induce a pregnancy protective shift from Th-1 cytokine response to more favorable Th-2 cytokine response.
- Women with recurrent miscarriage produce low levels of Th 2 cytokines (IL–4 and IL–10).
- Women with recurrent miscarriage have a decreased population of NK cells in the decidua. This indicates an altered immune environment within the decidua.

However, evidences to support the following hypothesis for successful pregnancy have been found to be insufficient (RCOG), currently such tests are not recommended.

(a) Incompatibility of HLA antigens between the couples, (b) Absence of leukocytokines antibodies in the mother, (c) Absence of blocking antibodies in the mother, (d) Testing for peripheral blood natural killer cells

(6) ANTIPHOSPHOLIPID SYNDROME: See p. 199, 399, 508: In SLE antiphospholipid antibodies, e.g. lupus anticoagulant β_2 glycoprotein, and anticardiolipin are important. These antibodies act by **dysregulation of coagulation pathways. This causes thrombosis of uteroplacental vessels and poor placental perfusion.** Obstetric complications are due to this pathology (see p. 399).

(7) MATERNAL AUTOIMMUNE DISEASE AND FETUS

- Incidence of neonatal thyrotoxicosis is higher in babies born of a thyrotoxic mother.
- A baby born to a mother with ITP will in all probability suffer from the same disease through transplacental transfer of antiplatelet antibodies.
- Myasthenia gravis also has some such relationship due to transplacental transfer of acetylcholine-blocking factor.

Babies born of mothers suffering from systemic lupus erythematosus often develop congenital heart block due to transplacental transmission of anti-Ro and anti LA (anti-SS–A and anti-SS-B) antibodies. SLE patients very often have exacerbation of disease activity during pregnancy or in the early postpartum phase. All the diseases listed in this group manifest transiently in the newborn.

(8) IMMUNOLOGICAL CONTROL OF FERTILITY

(a) **Placental hormones**—Human chorionic gonadotropin (hCG) that cross-reacts with luteinizing hormone (LH), thyroid stimulating hormone and follicle stimulating hormone has the β subunit, that is nonimmunogenic so as to be coupled with a hapten for antibody production. Thus the anti-placental antigen has been focused on anti-hCG vaccine. Of the many anti-hCG vaccine, most effective being the one that is directed against the C terminal peptide on the beta subunit of hCG. This antibody does not cross-react with the LH and thought to be effective up to 12 months.

(b) **Human sperm antigen:** Of the numerous sperm antigens, antibodies against LDH–X is being currently evaluated, and there is a significant reduction of viable sperm, as well as improper implantation of ovum in experimental animals, when anti-LDH–X antibody is injected into them.

(c) **Zona pellucida (ZP)** is the most extensively studied potentially oocyte target antigen. Antisera to ZP block sperm penetration as it coats the zona surface. Sera from infertile women with anti-ZP antibodies can block *in vitro* fertilization.

(d) **Antisperm antibodies (IgG, IgM or IgA)** are present either in the serum or in the reproductive tract. Antibodies present in the female reproductive tract that binds the sperm surface antigens affect the motility of sperm and may cause infertility.

CRITICAL CARE IN OBSTETRICS

Pregnant women with multisystem pathology need special care with improved technology and expertise of critical care obstetrics. Women need ICU admission, when they need cardiovascular, or pulmonary support following trauma or with multiorgan pathology.

Overall 1-3% obstetric patients are admitted in traditional intensive care unit (ICU). Among these patients, the risk of death ranges from 2% to 11%.

Selection Criteria of Obstetric Women for ICU Admission

ICU beds are a scarce resource. Therefore, ICU admission should be restricted to a critically ill woman who is likely to be benefited. Often these women are assessed clinically, based on specific abnormalities in physical examination of vital parameters, including laboratory values and imaging studies.

Some institutes have their own guideline for transfer to ICU (ACOG). Antenatal transfer rather than with newborn transfer is preferred except in a situation, where maternal transport is unsafe or impossible.

The essential requirements during transfer for such a critically ill patient are: continuous pulse oximetry, ECG monitoring, venous access, and confirmed position of endotracheal tube when a woman is under mechanical ventilation.

Organization of a Critical Care Unit

A qualified intensive care physician is to manage ICU, though it is not mandatory. However, it is observed that high-intensity ICU physician staffing is associated with lower ICU mortality and decreased hospital stay, when compared with low-intensity ICU physician staffing.

Critical care unit involves multidisciplinary approach: The team members involve physicians, anesthetists, cardiologists, pulmonologists, intensivists, respiratory therapists, pharmacists and nurses. Obstetric critical care unit involves obstetricians, obstetric nurses and neonatologists.

There are **three levels** of adult critical care (ACOG)

Level 1: Highest level of care: Severely ill patients are managed with the involvement of multidisciplinary team members.

Level 2: Intermediate care or high dependency care units (HDU): This is the post ICU step down unit. These are within the labor ward. Care is provided by the obstetricians and nurses who are experienced.

Level 3: Other intensive care units: For patients requiring long-term ventilator support.

Common Indication for Admission to Obstetric ICU

A. **Hemorrhage**
- APH
- PPH

Nearly 75% of obstetric patients admitted in ICU are postpartum

B. **Sepsis syndrome**
- Postabortal
- Pregnancy (Chorioamnionitis, pyelonephritis)

C. **Trauma**

D. **Hypertensive disorders**
- Severe Preeclampsia
- Eclampsia
- HELLP syndrome

E. **Cardiopulmonary**
- Heart disease in pregnancy
- Thromboembolism

F. **Puerperal sepsis**

Table 39.5: Arterial Blood Gases (ABG) during Nonpregnant State and Pregnancy

ABG Variables	Nonpregnant State	Pregnancy
pH	7.35–7.43	7.40–7.47
PCO_2 (mm Hg)	37–40	27–34
PO_2 (mm Hg)	103	101–104
HCO_3^- (mEq/L)	22–26	18–22
Base deficit mEq/L)	1	3

Arterial blood gases (ABG) values during nonpregnant state and pregnancy varies (see table above). It is important while managing a woman during pregnancy in ICU.

OBJECTIVE PARAMETERS (SELECTED) FOR ADMISSION OF A PATIENT (NONPREGNANT) IN AN ICU

(Laboratory values and physiologic parameters are changed in pregnancy)

A. Vital sings:	**C. Laboratory values:**
▪ Heart rate (HR) < 40 bpm or >150 bpm ▪ BP < 80 mm Hg systolic or > 120 mm Hg diastolic ▪ Mean arterial pressure < 60 mm Hg ▪ Respiratory rate > 35 breaths/minute	▪ Serum Na^+ < 110 or > 170 mEq/L ▪ Serum K^+ < 2 or >7 mEq/L ▪ PaO_2 < 50 mm Hg ▪ pH < 7.1 or > 7.7
B. Physical findings:	**D. Imaging studies:**
▪ Anuria ▪ Coma ▪ Uncontrolled seizures ▪ Cardiac arrest ▪ Cyanosis	▪ CT/MRI: Cerebral hemorrhage ▪ Electrocardiography: Complete heart block, complex arrhythmia, CCF

Hemodynamic changes in a normal nonpregnant and pregnant woman at term are significant: These values are important while managing an obstetric patient in ICU. Cardiovascular changes (see

p. 60) and respiratory system changes (see p. 62) are important. Use of pulmonary artery catheter is informative specially, in cases with severe preeclampsia, eclampsia, respiratory distress syndrome and amniotic fluid embolism.

Pulmonary artery catheter values: Normally, pulmonary capillary wedge pressure (mm Hg) at term pregnancy is 7.5 (+18% rise from nonpregnant state) and CVP is 3.6 mm Hg (–2%). There is fall in systemic vascular resistance (–21%) and pulmonary vascular resistance (–35%) at term pregnancy compared to a nonpregnant adult.

Decision Making and Patient Care in ICU

For a pregnant woman, ICU team members should plan for management including delivery which may be needed long before the EDD. Safe delivery of a woman needs consideration of period of gestation (fetal survival), place and mode of delivery (vaginal or cesarean). Vaginal delivery or operative vaginal delivery (forceps, ventouse) after at least 34 weeks of gestation within the ICU set up when possible is always beneficial. Cesarean delivery in the ICU is often faced with the problem of space for anesthesia, operative facilities, neonatal resuscitation arrangements and the risk of infection. Cesarean delivery in ICU may have to be done where transport of patient is not possible or for perimortem procedures.

Fetal Care in ICU

- Fetal gestational age assessment is essential to estimate the approximate fetal survival rate following delivery. Effects of obstetric medications need to be carefully judged in terms of risks and benefits.
- Drug-related side effects that may arise are: beta agonists (tachycardia), indomethacin (platelet dysfunction, reduced renal perfusion), beta blockers (IUGR). Benefits of antenatal corticosteroids are established and it is to be given in the event of preterm delivery (< 34 weeks).
- Maternal drugs (sedatives), acidemia, hypoxia, blood pH, may alter the CTG tracings. Correction of maternal hypoxia, acidemia may improve fetal condition. However, fetal interest comes second and essential medications should not be withheld to the pregnant woman.

Place of perimortem cesarean delivery: There is no such clear guideline regarding this issue. However, it is observed that cesarean delivery should be considered for both maternal or fetal benefits about 4 minutes after a pregnant woman has experienced total cardiopulmonary arrest in the third trimester.

KEY POINTS

- **Women with multisystem pathology** need improved care with technology and expertize of critical care obstetrics.
- **Common indications** for admission in ICU (based on objective parameters see p. 723) are: need of cardiac, circulatory, pulmonary or multiorgan support arising out of obstetric complications (hemorrhage, hypertensive disorders or sepsis)
- **The comparative values** of hemodynamic changes in nonpregnant and pregnant women at term are important in the management. Pulmonary artery catheterization are of immense value in the management (see above)
- **Critical care unit management involves multidisciplinary approach** (see p. 723). High-intensity ICU staffing can reduce ICU mortality and decrease hospital stay. Obstetrician, intensivist, speciality nurses, and neonatologist are involved.
- **Acute respiratory distress syndrome (ARDS)** may be due to pneumonia, sepsis, preeclampsia, embolism or drugs. Vigorous antimicrobial therapy, oxygen delivery (early intubation and ventilation for woman with respiratory failure) and support of circulatory volume (IV crystalloids and blood) are essential considerations.
- **Fetal care in ICU** needs considerable fetal gestational age, drug related side effects, and timing, place and mode of delivery (see p. 724).

contd...

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- **Benefit of antenatal corticosteroids** in the event of preterm delivery (< 34 weeks) is established and should be used.
- **Nearly 75% of obstetric ICU patients** admitted are postpartum.
- Hemorrhage, hypertension and sepsis are the **most common causes of admission** in obstetric ICU.
- **Necessary medications should not be withheld** from a pregnant woman because of fetal concerns. So also necessary imaging studies. However, attempts should be made to limit fetal exposure (drugs/radiation) as much as possible.

QUESTIONS

Related theory questions (Long and Short), Obstetric Case Discussions, Viva table discussions, Postoperative word round discussions, and MCQs are discussed in author's books:

1. **Bedside Clinic and Viva Voce:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

For further reading:

2. **Master pass in Obstetrics and Gynaecology:** 1st Ed. Jaypee Brothers Medical Publishers (P) Ltd.; New Delhi.

Chapter 40

Current Topics in Obstetrics

ANTIBIOTIC PROPHYLAXIS IN CESAREAN SECTION

Prophylactic use of antibiotics effectively reduces postoperative infectious morbidity both for the mother and the neonate. Postoperative morbidity like fever, endometritis, wound infection, peritonitis, and also pelvic abscess can significantly be reduced. However an institution, where infection rate is high, should primarily improve the surgical and aseptic technique. Emergency cesarean section is associated with higher rate of infection than the elective procedure. Similarly cases with prolonged rupture of membranes and in prolonged labor are at higher risk of infection.

Infective agents are mostly polymicrobial, including Gram-positive, Gram-negative aerobes and anaerobes. Generally antibiotics with broad spectrum activity are better. Use of ceftriaxone (1 g), Cefuroxime (1.5 g) or co-amoxiclav (1.2 g) by intravenous route which is a reasonable choice. Shorter courses of 1-3 doses are to be given. This can reduce the cost compared to a full 7 days course. First dose to the mother is given before the skin incision is made. Ideally the antibiotic infusion should be timed so that a bactericidal serum level is reached by the time skin incision is made. It is recommended that prophylactic antibiotic should be administered within 60 minutes of the start of the cesarean delivery. When this is not possible it should be started as soon as possible (ACOG). This avoids antibiotic exposure to the baby. Bacteriology pattern and antibiotic sensitivity need to be monitored regularly by the microbiology laboratory. Antibiotic prophylaxis has no deleterious effects on the mother or the neonate.

DAY CARE OBSTETRICS

It is designed to provide inpatient care to a pregnant woman, on an outpatient basis throughout the day. It is a new concept. This is similar to today's care surgery, as done for minor operations.

DEVELOPMENT OF A DAY CARE UNIT: Significant number of antenatal inpatient load is due to pregnancies complicated by hypertension. Objective is to provide rest, risk assessment and treatment to avoid any complication. When such a patient is seen in the day care unit, repeated blood pressure measurement is done. Examination of urine for protein, blood for uric acid and platelets, and LFT are also done. Fetal well-being is assessed by clinical examination and also with cardiotocograph and ultrasonography for liqor volume and fetal weight. Finally patient's risk is assessed and management is done accordingly.

Similarly women with diminished fetal movements could be assessed in a day care unit. She could be assessed with all parameters of history, clinical examination (fetal growth, liqor volume, auscultation of FHR), ultrasonographic study (BPP), and Doppler flow study of umbilical artery and ductus venosus depending upon the case.

PROCEDURES FOR RISK EVALUATION IN DAY CARE OBSTETRICS

- It requires an organized setup with quick access to laboratory and other monitoring parameters.
- It is essential that an experienced obstetrician should assess the pregnant women in a day care unit.
- A high-risk patient should be admitted from the day care unit for subsequent management.
- A moderate-risk patient could be seen for repeat day care assessment.
- A low-risk patient without any maternal or fetal compromise is referred back to routine care.

Advantages: (i) This acts as a safety net for assessment of obstetric complaints, (ii) Reduces inpatient overcrowding and workload specially in a busy hospital, (iii) Reduces the stress of the woman due to separation from the family, (iv) It reduces concomitant costs.

LEGAL AND ETHICAL ISSUES IN OBSTETRIC PRACTICE

Currently there is growing concern in the relationship of care giver (Doctor) and the care receiver (Client) in medical practice, in terms of mutual trust and understanding. This is due to great expectations of the society with progressive technological advancement. Medicolegal problems in obstetric practice are, therefore, rising both in the developed and in the developing world.

The doctor owes to his patient a duty of care. Care and attention must be according to the established norms available at that time and place. When the doctor fails to exercise that duty properly, he is found to be negligent. The failure to perform the proper duty to patient care may be due to his incompetence or malpractice or mere negligence. The failure to provide a standard care may again be either by acts of omission or commission.

Adverse outcomes of medical care are often due to: (i) System errors (inadequate staff, physician or operating room, etc.) or (ii) Health care personnel's error.

Once the act of substandard care due to system error, negligence, malpractice or incompetence is proved in the court of law, the plaintiff has to be compensated.

COMMON AREAS OF LEGAL THREAT IN OBSTETRICS: There are certain areas where claims are frequent and sometimes very high. These are in the field of (a) Perinatal injury, (b) Maternal injury or (c) Both.

(a) Perinatal injury: (i) Stillbirth and neonatal death, (ii) Brain damage to a baby, (iii) Injury following vaginal breech delivery (Ch. 26), (iv) Operative vaginal delivery (Ch. 37).

(b) Maternal injury: (i) Maternal trauma, (ii) Maternal death, (iii) Episiotomy, (iv) Forgotten packs in abdominal cavity or within the vagina.

(c) Both: (i) Instrumental delivery, (ii) Operative delivery, (iii) Anesthesia.

MEASURES TO MINIMIZE THE MEDICOLEGAL PROBLEMS

(a) Communication—must be made in a clear and understandable way to the client (patient) and the relatives about the management decision.

(b) Informed and written consent—must be taken before any agreed management decision or investigations.

(c) ***Legal and Ethical***

- Her consent must be following a clear understanding of the proposed procedures or therapy, its risks to herself and her fetus, the alternatives, success rates and the likely problems or complications.
- Obstetrician should not perform any procedure that is refused by the pregnant woman. Surgery without consent is an assault.
- The physician must provide information to the parents in relation to genetic counseling and prenatal diagnosis.
- Patients privacy and autonomy must be protected. No information, obtained in genetic counseling and screening, should be disclosed to any third party without the patient's authorization.
- Where conflicts arise, the doctor should seek help of and advice from other professional colleagues.

(d) Proper documentation of facts in the patient's file clearly and legibly in respect of date and time.

(e) Strict adherence to established management protocol (evidence based) is essential. When there is any deviation, it must be documented showing sufficient reasons.

(f) Careful record maintenance in institution as it may be required later on.

(g) Adequate training and supervision of juniors, specially involved in labor ward patient care. Seniors must be available for consultation or direct involvement as and when asked for.

(h) Consultation with another physician in the speciality when any difficulty is faced as regard the patient care.

(i) Regular audit and meetings—should be done to update the knowledge of all the staff involved in patient care. Audit will help to improve the quality of care.

AUDIT IN OBSTETRICS

Progress in clinical care cannot be achieved without a change. Many outdated practices in clinical medicine should be removed for betterment. Audit (clinical review) is an effective tool to indicate that change is essential.

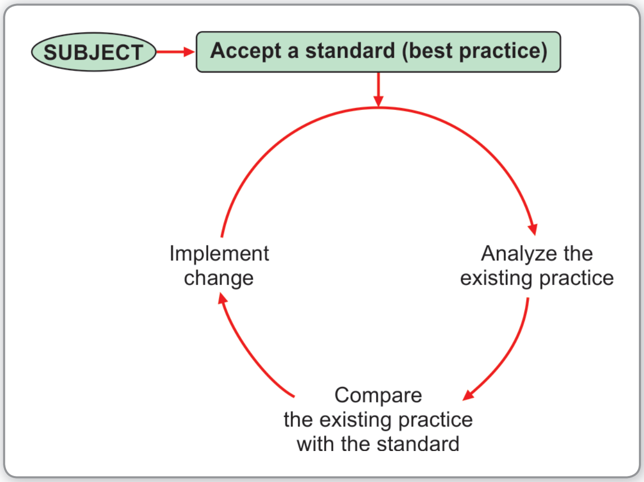
DEFINITION: Audit is defined as the systematic and critical analysis of the quality of medical care. **Objective** of carrying out an audit is to improve the quality of clinical care. It is done by changing and strengthening many aspects of hospital practice and administration. Audit should not be confused with research which involves new experiments, investigations or treatment.

Audit could be medical where scrutiny is done over the medical aspect of the work performed by the doctors. It could be clinical, where scrutiny is done over the work done by all health professionals including the doctors.

STRUCTURING AN AUDIT: Audit has to be structured beforehand. It should be based on available resources including personnel and finance. Important aspect to organize an obstetric audit is motivation of all doctors, midwives and other health professionals. Proper documentation of facts and figures must be there. Audit should be kept confidential and is considered as an educational tool.

EXAMPLE: Subject—'Hypertensive pregnant women must have their blood pressure (BP) checked at least 6 hours interval.' With this subject a standard (best practice) has to be adopted. The indicators are: BP measurement, hypertensive pregnant women and the time, so staff and equipment (resources) should be made available. A target is set up to include maximum number (95%) of the patients in the study. Monitoring methods should be strictly followed. The components are documentation and data collection. There must be an individual (Registrar/Lecturer) assigned for carrying out the audit. Finally this existing practice is critically analyzed, interpreted and then compared with the standard. Use of computers is helpful in data processing. Once the problems are identified and solved, better clinical care would emerge.

AUDIT CYCLE:


SUBJECT
Accept a standard (best practice)
Analyze the existing practice
Compare the existing practice with the standard
Implement change

IMPORTANCE OF CARRYING OUT AN AUDIT

1. A well-structured and efficient audit is based on scientific evidences with facts and figures.
2. It can replace the out of date clinical practice with the better one.
3. It can remove the disbelieving and agnostic attitudes between hospital management and professionals and also amongst the professionals.
4. It improves awareness between doctors and patients.
5. It is an efficient educational tool.

LIMITATIONS: Unless the audit is a simple one, it requires lot of time, staff commitment and technology.

THE PRECONCEPTION AND PRENATAL DIAGNOSTIC TECHNIQUES

Prohibition of Sex Selection Act, 1994 amended up to Feb. 2003

The act (PC & PNDT) is enforced to prohibit sex selection before or after conception to prevent its misuse that leads to female feticide. The act has got several chapters. It also covers the regulation of genetic counseling centers, genetic laboratories and genetic clinics. **The act permits such procedure** to detect any of the following abnormalities only : (i) Chromosomal abnormalities, (ii) Genetic metabolic diseases, (iii) Hemoglobinopathies, (iv) Sex-linked genetic diseases, (v) Congenital anomalies, (vi) Any other abnormalities or diseases as may be specified by the central supervisory board.

The person qualified to do the procedure must be satisfied for reasons to fulfil the following conditions and it must be recorded in writing : (i) Age of the pregnant woman is above 35 years, (ii) The pregnant woman has undergone two or more miscarriages or fetal loss, (iii) The pregnant woman had been exposed to potentially teratogenic agents, e.g. drugs, radiation, infection or chemicals, (iv) The pregnant woman or her spouse has a family history of mental retardation or physical deformities such as spasticity or any other genetic disease, (v) Any other conditions as may be specified by the central supervisory board. Written consent of the pregnant woman is obtained and there is prohibition of communicating the sex of fetus.

- The physician must provide accurate information to the parent while involved in genetic counseling and prenatal diagnosis.
- No information, should be disclosed to any third party without written permission of the patient.

UMBILICAL CORD BLOOD BANKING

Cord blood banking of hemopoietic stem cells (HSC) from umbilical cord blood gives benefit to store baby's own HSC for a long time. This cord blood stem cell can be transplanted for the treatment, in case that child or his/her siblings ever develop a metabolic, immunological, hematological, neurological or cardiovascular disease.

Umbilical cord blood contains HSC that they have greater proliferative and colony forming capacity and are more responsive to some growth factors. This cord blood is an useful source of stem cells for mesenchymal (cartilage, fat, hepatic or cardiac) cells and neural precursor cells.

The major clinical use of umbilical cord blood is for hematological malignancy (leukemia) in children. Compared to bone marrow transplant, the advantages of HSC transplant are: (i) Faster availability, (ii) Better tolerance between donor and the recipient, (iii) Lower incidence and severity of graft versus host disease, (iv) Low incidence of viral transmission (CMV, EBV), (v) Lack of donor attrition (bone marrow donor may not be available). Blood collection is made from the ex-utero separated placenta.

STEM CELLS AND THERAPIES IN OBSTETRICS

Reproductive tissues are the important source of stem cells (progenitor cells). Stem cells have the potential to be used in the field of regenerative medicine. A stem cell has the ability to renew (reproduce) itself for long periods.

Potentials for the use of stem cells in Regenerative Medicine

1. Treatment of inherited genetic disorders
2. Treatment of hematological diseases.

Properties of Stem Cells

a. Ability to self-renew (undergoing numerous cell divisions) maintaining the undifferentiated state.

b. Multipotency: Capacity to differentiate into a mature cell type.

Sources of Stem Cells

a. Embryonic tissues, b. Fetal tissues, c. Extrafetal tissues, d. Adult gonads

A. ***Embryonic tissues:*** Inner cell mass (ICM) of the blastocyst, embryo and yolk sac.

B. ***Fetal Stem Cells:*** Human fetal hematopoietic stem cells (hf HSC) are primarily obtained from bone marrow and liver. Virtually every part of the developing fetus has higher proliferative capacity. These cells have higher amount of telomerase activity and have longer telomeres compared to their adult counterparts. Moreover, these tissues can differentiate efficiently into neuronal, muscle and osteogenic lineages.

   Primitive hf MSC are transduced by integrating vectors and they do not express HLA-II. They can be used for ex vivo gene therapy as well as postnatal bone tissue engineering.

C. ***Extrafetal tissues:*** Amniotic membranes, placenta, trophoblasts, amniotic fluid cells, all contain progenitor cells. These mesenchymal stem cells (MSC) can differentiate into most cell types of mesodermal lineages.

Stem cell sample collection and banking

Currently the use of stem cells in regenerative medicine is regulated through institutional regulatory boards.

- ***Umbilical cord blood (UCB)*** collection and banking is an established source of HSC and MSC. This is used for treatment of hematological diseases like leukemia and bone marrow failure.
- ***Fetal tissues*** can be obtained following medical termination of pregnancy. Stem cells from fetal tissues can be harvested. Intrauterine transplantation of hf MSC, collected from liver, can be used for the treatment of hemoglobinopathies.

Intrauterine stem cell transplantation (IUSCT) can be used to correct genetic disorders (monogenic diseases).

Use of hf MSC has been explored for diseases having mesenchymal origin. hf MSC undergo site specific differentiation and contribute to repair tissues in such diseases (muscular dystrophy, osteogenesis imperfecta).

Place of IUSCT

- Hemoglobinopathies (α thalassemia, β thalassemia, Sickle cell anemia).
- Mucopolysaccharidoses (MPS)
- Inherited immune deficiencies
- Osteogenesis imperfecta

Allogeneic transplantation of HSC in the treatment of monogenic disorder has certain advantages. It has high tolerance and less rejection rate as it is done before the onset of fetal immune maturity (first trimester).

Autologous stem cells from fetal cord blood sampling or fetal liver biopsy in early pregnancy is done and the cells are harvested. An ex-vivo gene transfer may be done thereafter. This also reduces the risk of immune rejection. However, fetal HSC in first trimester has favorable engraftment kinetics.

In the first trimester fetal hematopoietic stem cells are highly proliferative and they circulate in significant numbers. Therefore these cells are the important source of autologous HSC.

Fetal mesenchymal stem cells can be bioengineered and used for the disease of bone, skin, liver and heart.

The potential to use stem cells for the fabrication of tissues or organ implants may prove helpful in the treatment of several diseases like genetic, immunodeficiency syndromes, urinary incontinence, infertility and structural repair.

However, till date it is essential to understand its known limitations, putative benefits and the unknown risks. Until there is sufficient evidence on the efficacy of therapy, each case should be considered on an individual basis.

Chapter 41

Imaging in Obstetrics (USG, MRI, CT, Radiology), Amniocentesis and Guides to Clinical Tests

Imaging in obstetrics is indicated for the purpose of diagnosis and/or therapy to the fetus or the mother. Most of the imaging studies are harmless or associated with minimal fetal risks. Of these radiography is of most concern.

PRINCIPLES OF DIAGNOSTIC IMAGING IN OBSTETRICS

- Ultrasound is the most valuable diagnostic tool in obstetrics.
- Benefits of radiation must outweigh the risks of the procedure.
- Minimum radiation dose (<5 rad) to be used.
- Appropriate fetal shielding should be done.
- First trimester should preferably be avoided.
- Benefits and safety of ultrasonography or MRI must be considered as an alternative.

ULTRASOUND IN OBSTETRICS

The ultrasound is a **sound wave beyond the human audible range of frequency greater than 2 MHz (cycles per second)**. SONAR stands for "Sound, Navigation and Ranging". The clinical application of ultrasound in obstetrics was introduced and popularized by **Ian Donald in Glasgow in 1958**.

Ultrasound is produced by the vibration of a synthetic piezoelectric crystal in response to a rapidly altering electrical potential situated in the transducer of an ultrasound machine probe. The transducer converts electrical energy to mechanical energy (ultrasound) and vice versa. The commonly used **frequency range in obstetrics is 3–5 MHz for abdominal transducers and 5–7 MHz for vaginal transducers**. When the frequency (number of ultrasound waves per second) increases there is improvement in image resolution but due to rapid wave attenuation, deeper structures are not properly visualized. This is due to poor penetration. In medical imaging, the transducer both sends and receives ultrasound waves (pulse echosonography). Sound travels through the tissues of the body at 1,540 meters per second.

The echo strength (strength of the reflected sound) depends mainly on the following four factors: (a) acoustic impedance mismatch (e.g. soft tissue—bone interface causes maximum ultrasound reflection producing bright echogenic structure), (b) the angle at which the ultrasound beam strikes a reflecting interface (more the ultrasound beam is perpendicular to the reflector, more echogenic the structure), (c) the strength of the ultrasound and (d) size of the reflector (fetal femur is more echogenic whereas renal pelvises scatter the ultrasound to give speckle).

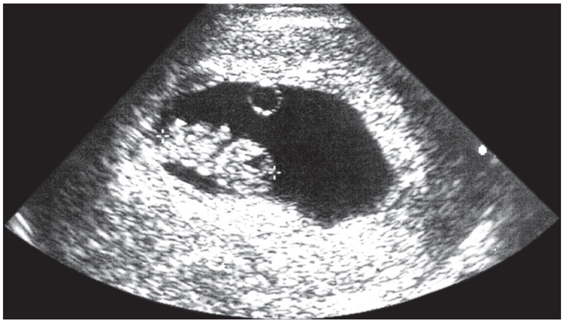


Fig. 41.1: Sonogram demonstrating crown-rump length (between crosses) of an 8-week fetus. Yolk sac is in the near field

In clinical practice, standard **ultrasound images** are:

- **B-mode** (brightness mode display)—two-dimensional (2D) images (width and brightness) are obtained.
- **M-mode** is used to study the moving organs, e.g. fetal heart. This results in a wavy pattern in the presence of motion.
- **Colour Doppler and pulse wave ultrasound** (Christian J Doppler–1942) is based on the principle of **Doppler frequency shift** which means there is a change in frequency and wave length between the incident wave (from the transducer) and the reflected wave (from the moving object) when the wave interacts with a moving structure (red blood cells in the umbilical artery). The Doppler shifted audible signals can be converted to visual signals and are known as **flow velocity waveform (FVW)**. **Doppler USG** is primarily used to demonstrate the presence, direction and velocity of blood flow. Usually flow toward the USG transducer is displayed in red and flow away in blue.
- **Three-dimensional (3D)** images.

THREE-DIMENSIONAL ULTRASONOGRAPHY (3D SCANNING)

3D can produce more lifelike images of the fetus in utero. The ultrasound beam is swept in two orthogonal planes to capture a block or volume of echoes (depending on the required volume) which are digitally stored. This volume of echoes can be resliced in any plane. Reconstruction of a 3D image from a subvolume of images can be made using computer software. **3D images have multiple advantages**: (a) Complex structure can be viewed in a single image, e.g. no need of mental reconstruction to define a defect. (b) The stored volume hypoplasia can be reviewed at any plane later on without needing the patient. This helps to get second opinion if required. (c) Prenatal diagnosis of certain anomalies is improved. (d) Lifelike photo of 3D images improves antenatal parental bonding. (e) It calculates tissue and fluid volumes, e.g. fetal lung volume measurement could be done to predict pulmonary hypoplasia. (f) This is also an important teaching tool. **Use of 3D images** for evaluation of fetal faces for clefting; brain for corpus callosum, cerebellar vermis; fetal heart for congenital defects. 3-D USG should be an adjunct to but not a replacement for 2D ultrasonography (ACOG).

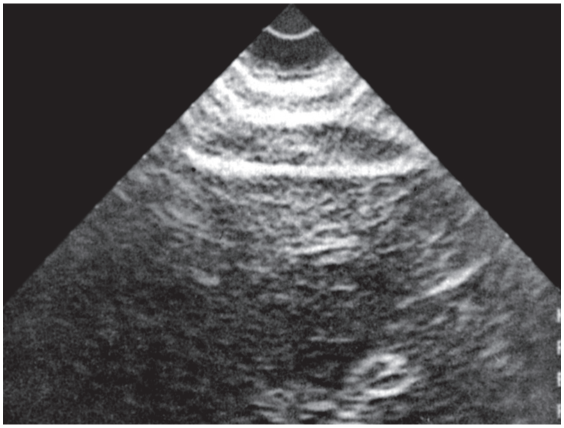


Fig. 41.2: Echogram showing femur length

Safety of ultrasound: Ultrasound is an essential tool in the management of almost every pregnancy. The effects of ultrasound on tissues are: temperature elevation, formation of microbubbles and cavitation. However, there is no clear evidence till date that ultrasound examination during pregnancy is harmful. Ultrasound should be done with valid medical indications and with shortest duration possible to avoid unnecessary exposure especially with the Doppler. Doppler ultrasound requires high power output. The upper limit of power what is considered safe is 1,000 mW/cm^2. Therefore, ultrasound should be judiciously used especially the Doppler mode and its casual use should be avoided.

Transvaginal ultrasound (TVS) is superior to transabdominal ultrasound for early pregnancy evaluation (when uterus is within the pelvis). There is very little attenuation of sound waves because the distance between the probe and the concepts is very close. This makes tissue resolution better.

Ultrasound examination may be—(a) standard (basic), (b) limited and (c) specialized (detailed).

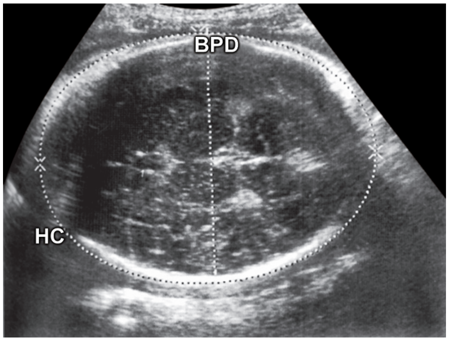


Fig. 41.3: Biparietal diameter at the level of cavum septum pellucidum

Indication of First Trimester Ultrasonography (ACR 2007) (To Evaluate, Estimate or to Confirm)

- Intrauterine pregnancy (see p. 77)
- Suspected ectopic pregnancy
- Vaginal bleeding (in all trimesters)
- Fetal anomalies (anencephaly)
- Suspected molar pregnancy
- Gestational age (see p. 77)
- Multiple pregnancy (chorionicity)
- To confirm cardiac activity
- Screening of aneuploidy
- Evaluation of pelvic/adnexal masses (all trimesters)

Indication of Second and Third Trimester USG (ACR 2007) (For Estimation/Evaluation/Confirmation)

- Gestational age
- Cervical insufficiency
- Suspected multiple pregnancy
- Liquor volume (poly/oligohydramnios)
- Placenta previa/abruption
- Suspected uterine malformation
- Cases with PROM
- Fetal growth (FGR)
- Fetal presentation (breech, face)
- Fetal well-being (BPP) assessment
- Screening fetal anomalies
- As an adjunct to: amniocentesis, CVS, cordocentesis, fetal therapy, ECV
- Uterine size: Either > dates or < dates

A complete examination includes survey of fetal life, fetal anatomy, fetal measurements, intrauterine environment and maternal structures.

FIRST TRIMESTER ULTRASONOGRAPHY

Common indications are mentioned in p. 119.

An **intrauterine GS** should be seen by TVS when the maternal serum β-hCG level is 1,000–1,200 mIU/mL and by TAS with the level of β-hCG 6,000 mIU/mL. **Yolk sac** is seen at the level of 7,000 mIU/mL and the embryo at 11,000 mIU/mL. **Definite diagnosis** of intrauterine pregnancy is possible as early as 29–35 days of menstrual age. **True Gestational Sac (GS)** is eccentric in position within the endometrium of fundus or body of the uterus. **Double decidua sign** of the gestational sac is due to the interface between the decidua and the chorion which appears as two distinct layers of the wall of the gestation sac. **Presence of yolk sac or fetal pole within the gestation sac confirms pregnancy**. True gestational sac size increases 1 mm/day. **Pseudogestational sac or pseudosac** is irregular in outline, usually centrally located in the uterus, has no double decidua sign and the sac remains empty (see p. 78). The rate of early (<12 weeks) pregnancy loss (miscarriage) diminishes steeply with the progressive appearance of fetal structures (e.g. with only GS = 11.5% and with embryo > 10 mm = 0–5%).

Fetal anatomy and viability

Fetal Features on Transvaginal Sonography (TVS) for Dating in Pregnancy

Mean Sac Diameter (MSD)	Findings
5–8 mm	Yolk sac
12 mm	Embryo
15–18 mm	Cardiac activity
Embryo CRL $\geq$ 4 mm	Cardiac activity

GS should increase by 1.1 mm in diameter per day

Gestational Age and Embryonic Structures Identified by Transvaginal Sonography (TVS)

Menstrual Age (weeks)	Fetal Structures
4	Choriodecidual thickness, chorionic sac
5	Gestation sac, embryo yolk sac
6	Fetal pole, cardiac activity
7	Lower limb buds, midgut herniation (physiological)
8	Upper limb buds, stomach
9	Spine, choroid plexus

Gestational age dating in pregnancy: Ultrasound examination is the best method to estimate the gestational age dating. The error with LMP is due to late ovulation (>14 days after LMP). **CRL (Fig. 41.1) is most accurate** with an error of 2.1 days in the first trimester. BPD, FL, HC and AC are commonly used for dating thereafter. **Transcerebellar diameter (TCD)** is an accurate predictor of gestational age when measured between 14 weeks and 28 weeks. **IVF pregnancy** is dated by the date of embryo transfer minus 14 days to get LMP and to calculate EDC by Naegele's rule.

One can determine the gestational age (in days) by adding 42 days to the crown rump length CRL, e.g. CRL (in mm) + 42 = gestational age in days (approx). After 12 weeks, accuracy of CRL decreases due to progressive curvature of the fetus. Gestational age in days can be determined by adding 30 days to the MSD (in mm), e.g. MSD (in mm) + 30 = gestational age in days. The embryo should increase its CRL by 1 mm per day. Failure to visualize an embryo when the MSD is 6 mm, it indicates pregnancy loss.

Ultrasound markers for fetal anomalies:

Nuchal translucency—Increased fetal nuchal skin thickness (in the first trimester) >3 mm by TVS is a strong marker for chromosomal anomalies (trisomy 21, 18, 13, triploidy and Turner's syndrome). Others (see p. 738).

Multiple pregnancy: Identification of two gestational sacs indicates twin birth in 52–63% of cases. **Anembryonic pregnancy (blighted ovum)**—see p. 188.

Ectopic pregnancy—TVS can detect 90% of tubal ectopic pregnancy. The double decidual sac sign differentiates normal pregnancy from pseudogestational sac of an ectopic pregnancy. Presence of echogenic fluid in the pouch of Douglas (blood) suggests probable presence of ectopic pregnancy. Color Doppler helps to identify the echogenic ring (ring-of-fire) of an ectopic gestational sac outside the uterine cavity (see p. 213). **Hydatidiform mole**—see p. 222.

MIDTRIMESTER ULTRASONOGRAPHY

- **Fetal growth**—is calculated on the basis of an accurate gestational age and is expressed in percentiles. Normal fetal weight should be between the 10th and 90th percentiles. Weight less than 10th percentile is considered small for gestational age (SGA). Whereas more than 90th percentile is large for gestational age. On the basis of biometric data, computer software can calculate fetal weight using formula. **Hadlock formula** which is commonly used, is sensitive to 7.5% of the actual fetal weight using fetal biometric data (BPD, HC, AC and FL). The availability of the estimate is ± 16–20%.
- **Gestational age assessment:** Nearly 20% of pregnant women are uncertain about the last menstrual period. In the second trimester, optimum time for most accurate assessment of gestational age is between 14 weeks and 20 weeks. **The parameters used are** biparietal diameter **(BPD)**, head circumference **(HC)** (Fig. 41.3), abdominal circumference **(AC)** and the femur length **(FL)** (Fig. 41.2). **The fetal head is imaged in a transverse axial section at the level falx cerebri, cavum septum pellucidum and the thalamic nuclei (Fig. 41.4). BPD (Fig. 41.3) is recorded from outer skull edge of the proximal skull to the inner edge on the distal skull. Femur length is measured** when the beam from the transducer is perpendicular to the shaft. **AC is measured** preferably at the level of the junction of the left and right portal veins, umbilical vein and as round as possible (Fig. 41.5). **AC is the single most sensitive parameter for assessment of fetal growth.**

Transcerebellar diameter (TCD) is an accurate predictor of gestational age when done between 14 weeks and 28 weeks. It is rarely affected by fetal growth aberrations.

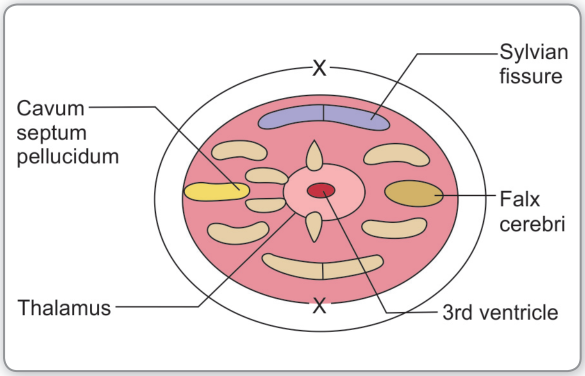


Fig. 41.4: Level for the measurement of head circumference (HC) and biparietal diameter (BPD)

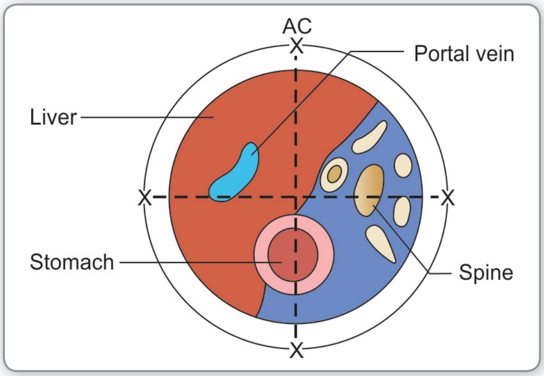


Fig. 41.5: Level for fetal abdominal circumference (AC)

Averaging the measurements of BPD, HC, AC and FL the variation of estimated gestational age to true gestational age is:

In the first trimester: When the LMP and US-based dating difference is ≥7 days. It is better to accept US-based date.

In the second trimester (13–28 *weeks*): Difference may be between 10 days and 14 days.

In the third trimester (29–42 *weeks*): Difference may be ≥21 days.

Dating ultrasound done before 22 weeks should be used in preference to menstrual dates irrespective of the reliability or closeness with menstrual dates.

Essential Elements in Standard Examination of Fetal Anatomy (ACR–2007)

- **Head, Face, Neck**
  - Cerebellum,
  - Choroid plexus,
  - Cisterna magna
- **Abdomen**
  - Stomach,
  - Kidneys,
  - Bladder,
  - Umbilical cord insertion,
  - Cord vessel number
- **Chest, Heart**
  - A four-chamber view of the heart
  - To view the outflow tract, often feasible
- **Spine:** Entire extent
  - Extremities: Legs, arms
  - Sex: When medically indicated

Neural tube defects (NTDs) commonly include spina bifida, anencephaly, exencephaly. **Important observations on ultrasonography are**:

A. **Cranial abnormalities (Fig. 41.6)**—(i) Obliteration of cisterna magna. (ii) Compression or flattening of the posterior cerebellar surface making a **crescentic appearance (banana sign).** (iii) Concave deformity of the frontal bones **(lemon sign)** in NTD. (iv) **Absence of calvarium**—anencephaly. (v) **Hydrocephaly** may be suspected when the HC or BPD is enlarged. Ventriculomegaly is diagnosed when the width of the lateral ventricle is >10 mm and the choroid plexus is seen dangling.

B. **Fetal face**—for cleft lip or palate.

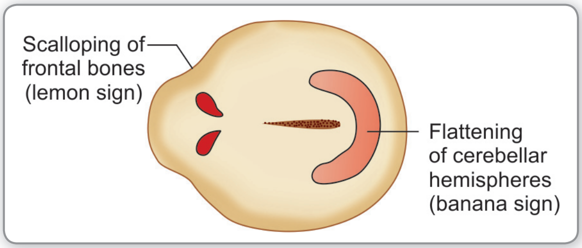

Scalloping of frontal bones (lemon sign)
Flattening of cerebellar hemispheres (banana sign)

Fig. 41.6: Cranial abnormalities associated with open spina bifida and Arnold-Chiari malformation

Anencephaly is diagnosed by the absence of cranial vault (calvarium) and telencephalon. Brain tissue is angiomatous. Early diagnosis is possible at about 13 weeks. Encephalocele is the protrusion of brain and/or meninges through a cranial defect. **Choroid plexus cysts**, majority are benign, but some of them may be associated with chromosomal abnormalities (trisomy 18, 21).

C. **Spinal anomalies** may be (i) **Spina bifida occulta** characterized by a vertebral schisis covered by normal soft tissues. (ii) **Spina bifida aperta** is a full thickness defect of the skin and vertebral arches. The neural canal is exposed. This defect may be covered by thin meninges (meningocele) and with neural tissue (myelomeningocele). **Fetal heart**: Four-chamber view of the heart and evaluation of outflow tracts are done for screening of congenital heart disease. Fetal echocardiography is more informative. **Fetal abdomen and abdominal wall**—Stomach bubble is seen normally by 20 weeks of gestational age. Its absence suggests fetal anomaly (esophageal atresia). **Hyperechoic bowel** has been associated with chromosomal abnormalities. Kidneys are usually hypoechoic. **Fetal kidney** for any obstructive or dysplastic abnormalities.

Gastroschisis	Omphalocele
■ Paraumbilical defect	■ Usually midline defect
■ Usually on the right side of the cord insertion	■ Cord insertion is on the herniated mass
■ Herniated small bowel loops floating free in amniotic fluid	■ Hernia contains intestines, liver or both
■ Not covered by any membrane	■ Covered by a membrane (peritoneum and amnion)
■ Not associated with chromosomal anomaly	■ Associated chromosomal anomaly 40–60%
■ Prognosis—good	■ Prognosis—often poor

Omphalocele and gastroschisis are rare (1 in 4,000 live births) abdominal wall defects. They should be differentiated (Table 41.1).

Hydrops fetalis (see p. 388). **Hydatidiform mole** (see p. 222).

Fetal gender identification is confirmed by detection of testes within the scrotum in the third trimester. Fetal perineal examination for external genitalia may be incorrect in the second trimester in 1% of cases.

Placenta and umbilical cord—Placenta is a echogenic discoid mass. Placental thickness at term is about 30 mm. **Placental thickness more than 45 mm at any period of gestation is considered abnormal**. The relationship of placenta to the internal cervical os is important to define low lying placenta and placenta previa (see p. 282). False-positive diagnosis may be due to focal uterine contraction or maternal bladder (too full/too empty). Transperineal or transvaginal imaging may be necessary in that case. **When the distance between the internal os and placental edge is more than 20 mm placenta previa is excluded and vaginal delivery is allowed. Only 5% of placenta previa identified in the second trimester will persist to term**.

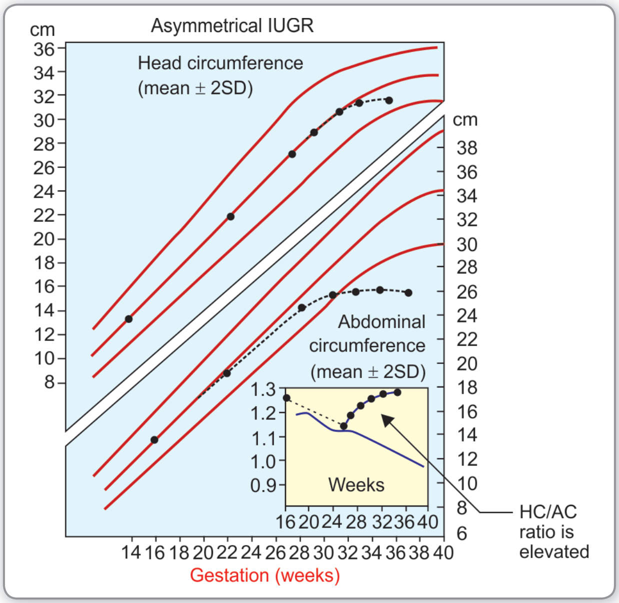


Fig. 41.7: Ultrasonographic growth profile in a case of asymmetric IUGR shows, head circumference (HC) is maintained but abdominal circumferences (AC) falls off around 30 weeks. The HC/AC ratio is elevated.

Placenta of multifetal pregnancy (Chorionicity)—**Dizygotic twins** have always diamniotic-dichorionic **(DiDi)** placenta whereas DiDi may be observed in 20–30% of monozygotic twins. **DiDi placenta is characterized ultrasonographically by twin peak sign** (see p. 236). The proliferating placental villi grow into the interchorionic space of the two placentae. This projected placental tissue is shaped like a triangle and has the same echogenicity as that of the placenta. The base of the triangle is toward the chorionic surface and the apex is toward the dividing membrane (Figs 17.3A and B). However, **twin peak sign** is neither present entirely over the placenta nor it is a consistent feature.

The diagnosis of DiDi twins is made when thickness of the dividing membrane is more than 2 mm (Figs 17.3A and B; p. 236). However, with the progress of pregnancy, the membranes become attenuated.

Placenta accreta may be diagnosed with loss of retroplacental sonolucent zone beneath the previous cesarean section scar. In cases with increta, the placenta invades the myometrium and in percreta, it invades the serosa and even the bladder. It is evident as focal exophytic masses.

3D Power Doppler: Hypervascularity at serosa bladder interface and numerous coherent vessels involving the uterine serosa-bladder junction are seen. Color Doppler shows vascular lakes, with turbulent flow with peak systolic velocity >15 cm/s.

Umbilical cord: Single umbilical artery (SUA) is associated with a higher rate of fetal anomalies (30–70%). The major anomalies observed are: Cardiac, CNS, Kidneys. **Umbilical cord insertion:** Velamentous insertion, (see p. 252, 253) and vasa previa (see p. 301) can be diagnosed with Doppler ultrasound.

Assessment of amniotic fluid (see p. 44): AFI >25 cm constitutes polyhydramnios. Largest vertical pocket >8 cm and is considered as polyhydramnios.

Fetal weight: Hadlock formula. Commonly uses four variables: BPD, HC, AC and FL. The absolute error for birth weight prediction is about 8–10%. Ultrasound and clinical examinations have similar accuracy for predicting birth weight.

Genetic USG—in the second trimester is a noninvasive way to elect fetal chromosomal anomalies.

Doppler: Direction as well as velocity of blood flow can be measured by Doppler ultrasound. Color flow Doppler converts velocity information into color code. **Blood flowing toward the transducer is shown in shades of red, flow away from the transducer is shown in shades of blue**. Brighter shades indicate high velocity. This helps to study all major blood vessels in the placenta and fetus. Arterial flow is pulsatile and venous flow is usually constant. **Reduced diastolic flow indicates high resistance in the downstream vessel and low tissue perfusion. Presence of "notch" in the early diastole waveform** also indicates high resistance to the flow. The muscular spiral arteries are the downstream from the uterine artery. Presence of notch in the **uterine artery** waveform indicates high resistance to flow in the downstream vessels. This waveform with a notch implies that trophoblast invasion to these arteries is incomplete and inadequate (see p. 37). **Presence of notch in the uterine artery** when confirmed bilaterally at 24 weeks indicates the **possible development of preeclampsia and fetal growth restriction**. After 30 weeks, the S/D ratio of the **umbilical artery** should be less than 3. **Absent end-diastolic flow in the umbilical artery was associated with 16% fetal death rate and reverse diastolic flow with 50% fetal death rates**. Increased fetal diastolic flow in the middle cerebral artery (centralization of flow) with absent diastolic flow in the aorta implies fetal acidemia. Increase in diastolic flow in the fetal middle cerebral artery (MCA) is a preferential adaptive mechanism intended to preserve the brain (brain-sparing effect). Abnormal venous waveforms (ductus venosus, inferior vena cava) indicate fetal cardiac dysfunction (failure). Abnormal venous Doppler parameters are directly related to fetal death and stillbirth. The indices for Doppler study are: Systolic/Diastolic ratio, resistance index (Rl) or pulsatility index (PI). Following equivocal biophysical profile (see p. 122), umbilical cord Doppler study is an important investigation.

Routine ultrasonography—at 18–22 weeks gestation has the following effects: (i) Reduces the incidence of post-term pregnancy (39%) and rates of induction of labor for post-term pregnancy. (ii) Increases detection of multiple pregnancy (92%). (iii) Increases detection of major fetal anomalies when termination is possible. (iv) No significant differences in the clinical outcomes such as perinatal mortality. (v) Reduce neonatal admission to special care baby unit (14%).

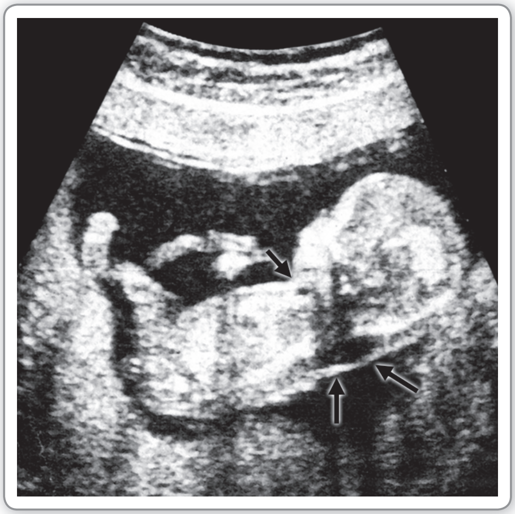


Fig. 41.8: Longitudinal view of a 12-week fetus with a thickened nuchal translucency (arrows). This is due to dilatation of the lymphatic capillaries and edema. Fetuses with nuchal abnormalities need amniocentesis

Table 41.1: Ultrasound Markers of Chromosomal Abnormalities (Genetic Sonography)

Observation	Chromosomal Abnormality	Observation	Chromosomal Abnormality
(a) **Head**		(f) **Hands/feet**	
■ Choroid plexus cyst	Trisomy 18, 13, triploidy	■ Flexed overlapping fingers	
■ Strawberry skull		■ Rockerbottom/club foot	Trisomy 18
■ Hydrocephalus		■ Polydactyly	Trisomy 13
■ Holoprosencephaly		■ Wide gap between 1st and 2nd toes	
(b) **Face**			
■ Cleft lip/palate	Trisomy 13, 18,	■ Clinodactyly	Trisomy 21
■ Micrognathia	Meckel-Gruber syndrome	■ Short femur	
■ Low set ears	Triploidy		
(c) **Nuchal translucency**	Trisomy 21, 18, 13,	(g) **GI system**	
>3 mm	Turner syndrome	■ Echogenic bowel	Trisomy 13, 18
(d) **Cardiac defects**	Trisomy 13, 18, 21	■ Omphalocele	Trisomy 21
		■ Duodenal atresia	
(e) **Renal anomalies**		(h) **General**	
■ Horseshoe kidney		■ Early IUGR	Trisomy 13, 18, 21
■ Bilateral dilatation of renal pelvis	Trisomy 13, 18, 21	■ Hydrops	Triploidy, 45XO
■ Cystic dysplasia	Triploidy		

Needs fetal karyotyping for confirmation, see p. 130

THIRD TRIMESTER ULTRASONOGRAPHY

All the information of second trimester sonography can be obtained in third trimester.

- **A detailed anatomical survey** should be done now even if the previous survey was normal. Achondroplastic dwarfism is diagnosed in this trimester.
- **Estimated fetal weight (EFW)** is determined from the average of three readings for each of the following: FL, AC and BPD. AC is most important. Sonographic EFW has an error risk of 15–20%.

Growth profile (Fig. 41.7): (i) In asymmetric IUGR, the HC is maintained but the AC falls off around 30 weeks. The HC:AC ratio is therefore elevated. (ii) In symmetric IUGR, both the HC and AC are affected early. Therefore, HC:AC ratio remains normal.

MAGNETIC RESONANCE IMAGING (MRI) AND COMPUTED TOMOGRAPHY (CT) IN OBSTETRICS

MRI is useful to obtain high soft-tissue contrast and acquisition of images in axial, sagittal and coronal planes. Powerful magnets are used to alter temporarily the static tissue proteins (mainly hydrogen protons). Radio waves are used to deflect the magnetic vector. The hydrogen protons return to their normal state once the radiofrequency source is turned off. During this phase, they emit radio waves of different frequencies which are received by radio coils wrapped around the body part. An image is constructed from these pulse sequences using their location and characteristics.

MRI is devoid of any ionizing radiation and found to have no fetal harmful effect at any gestational age with 1.5 tesla strength.

The gadolinium contrast MRI in pregnancy should be used with caution as gadolinium crosses the placenta. It is a category C drug (see p. 588) and should be avoided in first trimester. Rapid sequence MRI is preferable to conventional MRI as the exposure is brief. MRI is preferable to CT scanning during pregnancy as MRI is devoid of ionizing radiation.

Indications—(A) *Fetal:* (i) Fetal anatomy survey, (ii) Fetal biometry, (iii) Fetal weight estimation (superior to sonography), (iv) Evaluation of complex abnormalities (brain, chest, genitourinary system) (v) As a complement to sonography. (B) *Maternal:* (i) Cerebral vascular flow study (eclampsia) and detection of thrombosis, (ii) Angiography, (iii) Evaluation of maternal tumors, (iv) Evaluation of placenta

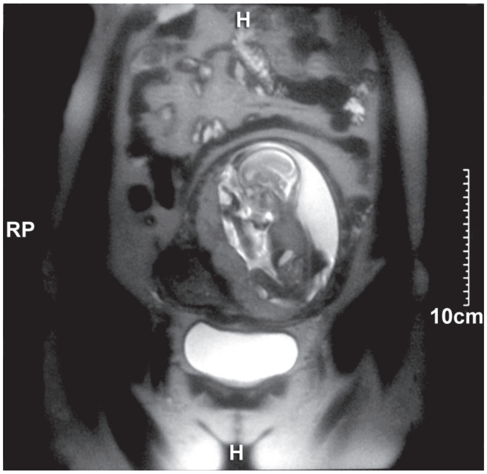


Fig. 41.9: MRI of the abdomen and pelvis showing a fetus in one horn of a bicornuate Uterus and the nonpregnant horn is seen by the side. (*Courtesy:* Dr. Rewa Tripathy, Professor and Head, Dept. OB-GYN, MAMC, New Delhi)

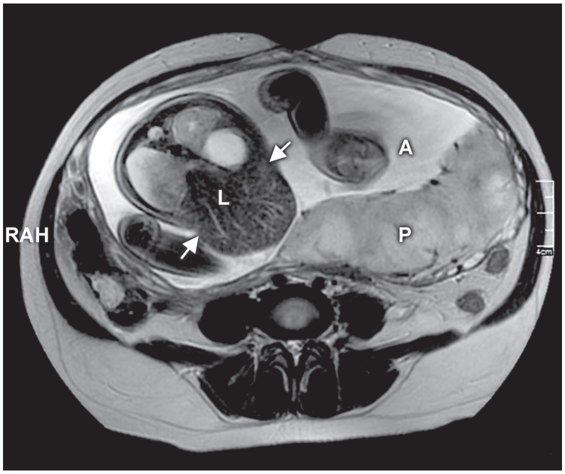


Fig. 41.10: Axial (T2) image through the abdomen of a fetus at 34 weeks of gestation. Arrows denote the area of abdominal wall deficiency through which the liver herniates (Omphalocele). **L:** Fetal liver; **P:** Placenta; **A:** Amniotic fluid

previa accreta. MRI is better compared to USG to detect depth of trophoblasts penetration within the uterus/bladder in cases with placenta previa accreta. MRI reveals uterine bulging, heterogenous signal intensity/placental bands on T2-weighted imaging.

MRI is used as an **adjunct to fetal therapy**. Fetal abnormalities are outlined preoperatively because of its precision in diagnosis. MR imaging is helpful before laser ablation of TTTS and also before the surgical procedures of spina bifida, sacrococcygeal teratoma and neck surgery.

Contraindications of use are: Internal cardiac pacemaker, implanted defibrillator, implants or other metals in the body.

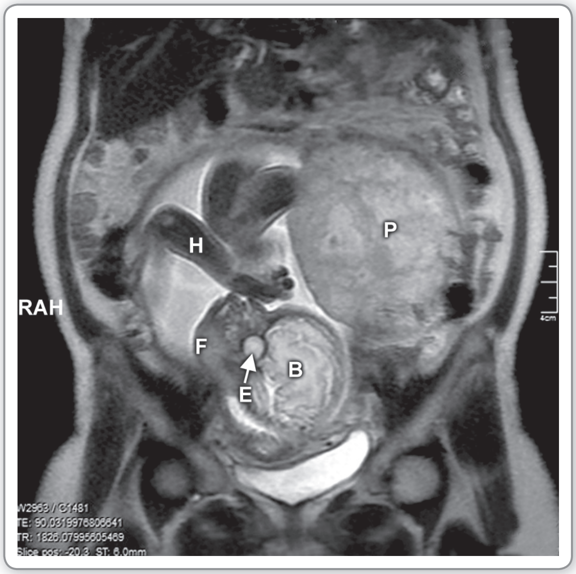


Fig. 41.11: Coronal (T2) image of a fetus, showing fetal face, brain, eyeball, hand and the placenta. Normal sulcation and gyration in the brain are seen. **F:** Face; **B:** Brain; **E:** Eyeball; **H:** Hand; **P:** Placenta

COMPUTED TOMOGRAPHY (CT): CT imaging is done by obtaining a spiral 360° images that are processed in multiple planes. Multidetector CT (MDCT) is now used. MDCT has got increased dosimetry compared to traditional CT. Due to its radiation risks, use of CT should be avoided in all trimesters of pregnancy unless absolutely essential. Fetal dose during CT pelvimetry varies between 0.25 rad and 1.5 rad. CT pelvimetry may be obtained with little or no exposure to the fetus. Chest CT scan is done in cases with suspected pulmonary embolism and cranial CT scan is done in cases with eclampsia with neurological features.

RADIOLOGY IN OBSTETRICS

With the advent of ultrasonography, magnetic resonance imaging (MRI) and computed tomography (CT) the importance of radiology is declining. Currently there are very few conditions where radiology may be of help during pregnancy.

INDICATIONS: ♦ **Fetal** ♦ **Maternal**

FETAL: Congenital malformation of the fetus and neonates, skeletal malformations, birth injuries like fracture or dislocations (see p. 331).

MATERNAL: Patient having cardiopulmonary disease may require X-ray chest during pregnancy and that should be done beyond 12 weeks.

RADIATION HAZARDS: Risk is primarily based on the estimated dose and the period of gestation (Table 41.2).

Table 41.2: Absorbed Radiation by the Fetus in Different Diagnostic Radiation Procedures

Procedure	Dose (cGy)
Abdominal X-ray	0.1–0.3
Pelvic X-ray	0.5–1.1
Chest X-ray	<0.001
Abdominal CT	0.4–0.8
Chest CT	0.002–0.02
Abdominopelvic CT	2.5–3.5
Ventilation scan	0.007–0.05
PET scan	1–1.5

Radiation can cause fetal morbidity (FGR, genetic mutations, neurologic abnormalities, mental retardation, childhood leukemia) and mortality. Radiation risks are high with radiation after the first 2 weeks and within the first trimester (period of organogenesis). Exposure >15 rad during second and third trimester or >5 rad in the first trimester needs patient counseling. Elective termination of pregnancy may be considered.

- **Teratogenicity:** Diagnostic range of radiation exposure (less than 5 rad) is not associated with any significant congenital malformation either in human or in animal.
- **Oncogenicity:** Dividing cells particularly in the first trimester are more sensitive to injury from radiation. Diagnostic radiation with fetal exposure is associated with an increased risk of malignancy.

- **Genetic damage:** No radiation-induced transmissible gene mutations have been seen in human.
- **Intrauterine death:** Low-dose radiation (1–5 rad) is not associated with any fetal death.

CAUTION: Radiation during pregnancy can damage the early phase of embryo but the risk can be minimized by using the "10-day rule". **This rule states that no woman should be exposed to X-ray for a nonurgent indication outside 10 days from her last period during reproductive period unless pregnancy is excluded.**

KEY POINTS OF IMAGING IN OBSTETRICS

- **Before any imaging in obstetrics**, benefits and safety of different modalities (USG, MRI, CT, Radiology) must be carefully considered.
- **Ultrasonography is the preferred** modality of initial imaging for evaluation of a pregnant woman with abdominal pain.
- **Ultrasound is the most valuable** diagnostic tool in pregnancy and is safe when used appropriately (see p. 732).
- **Benefits of USG in all the trimesters of pregnancy** are many (see p. 734 Table). Gestational age is accurately determined in the first half of pregnancy and fetal anomalies are determined.
- **The optimal timing** for a single USG examination is at 18–20 weeks.
- **MRI has got some added benefits** to USG (see p. 739).
- **MRI is safe in pregnancy**; however, it is a good practice to avoid MRI during pregnancy particularly for elective studies or in the first trimester (ACOG).
- **MRI is devoid of any ionizing radiation.**
- **IV gadolinium is contraindicated in pregnancy**. It should be used only if absolutely essential.
- **CT of the fetus should be avoided** in all the trimesters of pregnancy as the main concern is carcinogenesis. Risks of teratogenesis is less. CT pulmonary angiogram is the preferred modality for imaging of suspected pulmonary embolism.
- **Use of radiology** in obstetrics is very limited.
- **Exposure or radiation >15 rad** during second or third trimester or >5 rad in first trimester is hazardous. Patient needs to be counseled for elective termination of pregnancy.

AMNIOCENTESIS

DEFINITION: **Amniocentesis is the deliberate puncture of the amniotic fluid sac per abdomen**.

INDICATIONS: ♦ **Diagnostic** ♦ **Therapeutic**

DIAGNOSTIC:

- ***Early months (15–20 weeks):*** **Genetic amniocentesis** antenatal diagnosis of chromosomal and genetic disorders: (i) Sex-linked disorders. (ii) Karyotyping. (iii) Inborn errors of metabolism. (iv) Neural tube defects. (Details in Ch. 13).
- ***Later months:***
  - (i) **Fetal maturity** (see p. 372).
  - (ii) **Degree of fetal hemolysis in Rh-sensitized mother**—Spectrophotometric analysis of amniotic fluid and deviation bulge of the optical density at 450 nm is obtained (see p. 392).
  - (iii) **Meconium staining of liquor**—an evidence of fetal distress.

THERAPEUTIC:

- ***First half:*** (1) Induction of abortion by instillation of chemicals such as hypertonic saline, urea or prostaglandins. (2) Repeated decompression of the uterus in acute hydramnios.
- ***Second half:*** (1) Decompression of uterus in unresponsive cases of chronic hydramnios producing distress or to stabilize the lie when it is not axial prior to induction. (2) To give intrauterine fetal transfusion in severe hemolysis following Rh isoimmunization. (3) *Amnioinfusion*: Infusion of warm normal saline into the amniotic cavity is done transabdominally or transcervically to increase the volume of amniotic fluid. *Indications of amnioinfusion*: A. Oligohydramnios—(i) to prevent fetal lung hypoplasia, (ii) to minimize umbilical cord compression during labor. B. to dilute meconium-stained amniotic fluid.

PROCEDURE: (1) After emptying the bladder, the patient remains in dorsal position. (2) The abdominal wall is prepared aseptically and draped. (3) The proposed site of puncture is infiltrated with 2 mL of 1% lignocaine.

A 20- or 22-gauge spinal needle with stylet in about 4" in length is inserted into the amniotic cavity **under real-time sonographic control** (Fig. 41.12). Injury to the placenta, umbilical cord and fetus is to be avoided. Continuous visualization of the needle under USG guidance reduces the risks of injury, bloody or dry tap and need of multiple insertion. The stilette is withdrawn and few drops of liquor are discarded. Initial 1–2 mL of fluid is either used for AFP or is discarded as it is contaminated with maternal cells. Rest is used for fetal karyotyping. About 30 mL of fluid is collected in a test tube for diagnostic purposes. Fetal cardiac motion is to be seen after the procedure. Patient is asked to report for any uterine cramps, vaginal bleeding or leakage of liquor.

PRECAUTIONS: (i) Prior sonographic localization of placenta is desirable to prevent bloody tap and fetomaternal bleeding. (ii) Prophylactic administration of 100 mg of anti-D immunoglobulin in Rh-negative nonimmunized mother. Hazards are reduced significantly when it is done **"under direct ultrasound control"** compared to the blind procedure.

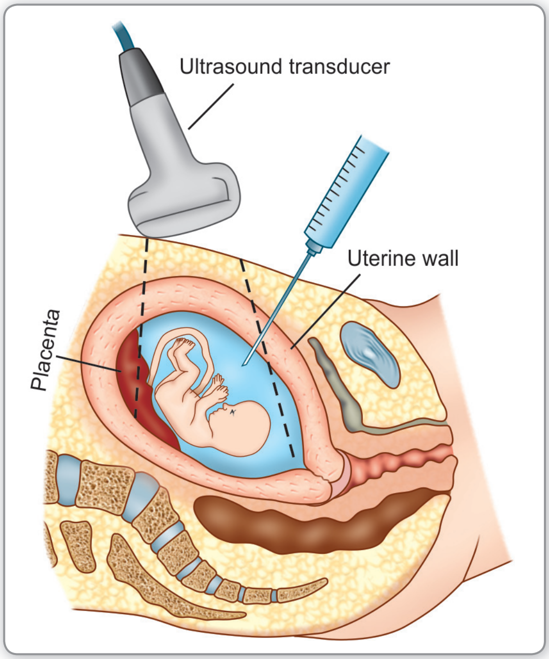


Fig. 41.12: Amniocentesis—direct ultrasound control

HAZARDS: (A) **Maternal complications are:** (1) Infection. (2) Hemorrhage (placental or uterine injury). (3) Premature rupture of the membranes and premature labor. (4) Maternal isoimmunization in Rh-negative cases.

(B) **Fetal hazards are:** (1) Fetal loss (1 in 400 procedures). (2) Trauma. (3) Fetomaternal hemorrhage. (4) Oligohydramnios due to leakage of amniotic fluid and that may lead to: (i) Fetal lung hypoplasia. (ii) Respiratory distress. (iii) Talipes. (iv) Amnionitis (rare).

Amniocentesis should be avoided for HIV-positive women and noninvasive tests (NT, MSAFP, anatomic USG) are preferred. However in women with HBV, HCV may be done with counseling.

Early amniocentesis (11–14 weeks) not to be done for genetic indications as the cell culture failure rate is high. Less fluid is withdrawn. Rates of complications are high.

GUIDES TO CLINICAL TESTS

EXAMINATION OF URINE

TESTS THAT ARE DONE: (1) Physical. (2) Biochemical. (3) Microscopical. (4) Bacteriological. (5) Immunological. (6) Hormonal.

COLLECTION OF URINE: For items 1, 2 and 3, first morning urine is collected in a clean container. But for bacteriological examination, "clean-catch" midstream urine is collected in a sterile container. Alternatively, a catheter sample may be taken.

Tests for Protein: • **Qualitative** • **Quantitative**

Qualitative: (i) Heat and acetic acid test. (ii) Dipsticks.

Heat and Acetic Acid Test

Experiment	Observation	Inference
Three-fourth of a test tube is filled with urine. The tube is held obliquely and the upper part of the urine is heated	A cloudy precipitate is formed	Presence of protein or phosphate
Add few drops of 5% acetic acid to it	The cloudiness persists or increases	Presence of protein

Report: Protein—absent, trace (+), (++), or (+++)

Dipsticks (Albustix): These reagent strips provide quick and simple standardized color tests to detect protein. The dipsticks available may be specific for albumin or may cover other urinary constituents like pH, acetone, glucose and specific gravity **(multistix)**. *Procedure:* Dip the test end of the reagent strip in urine and take it out immediately. Now compare any change of color of the test end with the color chart supplied by the manufacturer. **If the test end turns green or blue, the test is positive**.

Interpretation: Trace = 0.1 g/L; 1 + = 0.3 g/L; 2 + = 1.0 g/L; 3 + = 3.0 g/L; 4 + = 10.0 g/L.

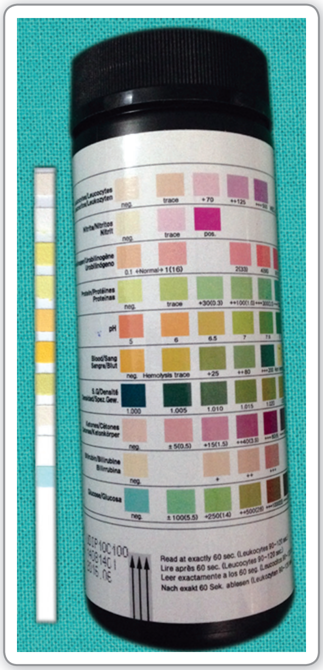


Fig. 41.13: Uro-dip Reagent Strips: For rapid determination of Urobilinogen, Glucose, Bilirubin, Ketones, Specific Gravity, Blood, pH, Protein, Nitrite and Leukocytes and Ascorbic Acid in Urine

Test for Sugar: ♦ Benedict's test ♦ Dipsticks

Qualitative: Benedict's test

Experiment: (a) 5 mL of Benedict's reagent is taken in a test tube and is heated to boiling. (b) Add 8–10 drops of urine to it. (c) Boil the mixture vigorously for 2 minutes and allow it to cool.

Interpretation: Presence of reducing substance is indicated by change of color in the precipitate. Approximate glucose concentration in urine is as follows: Pale green: 0–100 mg%; Green: 100–500 mg%; Greenish yellow: 500–1,000 mg%; Yellowish orange: 1,000–2,000 mg%; Brick red: >2,000 mg%.

***Dipsticks (Diastix)*:** These reagent strips provide quick, specific, simple qualitative and semiquantitative color tests to detect the presence of glucose in urine. They do not react with other reducing substances found in urine. The strip is of absorbent cellulose, one end of which is impregnated with buffered mixture of glucose oxidase and peroxidase.

Procedure: Dip the test end of the reagent strip in urine and take it out immediately. Now observe the color of the test end exactly 1 minute later. **If there is change in color of the test end, the test is positive.** The instructions of the manufacturers are to be followed strictly. The dipsticks available may be only used for glucose or may cover other urinary constituents (Multistix).

Urine dipstick test is based on the detection of leukocyte esterase and nitrites in a freshly collected sample of urine. Positive dipstick test (change in color) indicates infection.

Test for Acetone: ♦ Rothera's test ♦ Ketostix

Rothera's test:

Experiment	Observation	Inference
Take 5 mL of urine in a test tube. Add ammonium sulfate to make it saturated To it, mix 3–4 drops of freshly prepared sodium nitroprusside solution (or a few crystals of sodium nitroprusside). Then overlay with 1 mL of concentrated ammonium hydroxide and allow it to stand for 1–2 minutes	A reddish-purple ring appears at the junction of the two liquids (A brown ring is of no significance)	Presence of acetone

Ketostix: The reagent strips provide quick, simple standardized color test for detection of ketone bodies in urine. The strip is impregnated with a buffered mixture of sodium nitroprusside and glycine.

Procedure: Dip the test end of the reagent strip in fresh specimen. Compare the color of the test end with the supplied color chart as per instruction of manufacturer.

TESTS FOR BLOOD COAGULATION DISORDERS

Bedside tests are given in Ch. 39. Only the laboratory tests for blood coagulopathy are mentioned here.

LABORATORY TESTS

- ***Prothrombin time*** (PI)—Quick's prothrombin time assay measures the time required for clotting by the extrinsic pathway and is carried out by adding tissue thromboplastin to plasma. **Normal value is 11–17 seconds**. In coagulation failure, the time is prolonged.

Procedure: Blood is to be sent in a vial containing 3.8% of sodium citrate solution in proportion of 9:1 (0.5 mL sodium citrate and 4.5 mL blood).

- ***Estimation of fibrinogen:*** Blood sample is to be collected in EDTA vial. Critical level is considered to be 100 mg% (plasma).
- ***Platelets:*** Blood is to be sent in EDTA vial containing disodium or dipotassium salt of ethylenediaminetetraacetic acid powder. A count less than 70,000 per mm^3 is usually found in DIC. Thrombocytopenia is characteristic of intravascular coagulation but it is not found in a pure fibrinolytic syndrome.
- ***Fibrin degradation products*** (FDPs) is estimated by latex agglutination test. Blood sample is to be sent in a special tube. In normal pregnancy, it is usually absent. **In DIC, its presence to the extent of 80 µg/mL is significant.**
- ***D-dimer*** is a specific component of fibrin breakdown. It is detected by latex agglutination method. The latex beads are coated with monoclonal antibodies. Level more than 200 mg/mL is found in DIC. Blood sample is to be collected as that of fibrinogen.
- ***Euglobulin clot lysis time (ECLT)***—Blood sample is to be sent as that of prothrombin time. The time is very much shortened in fibrinolytic syndrome.

Table 41.3: Normal Values of Blood Coagulation Profile

	Test	Value
1.	Bleeding time (Duke's method)	1–3 min
	(Ivy's method)	1–9 min
2.	Coagulation time—	
	(Wright's tube method)	3–7min
	(Lee and White method)	4–9 min
3.	Clot observation test (Weiner's)	6–12 min
4.	Clot retraction time	30 min
5.	Fibrindex or Thrombin test	Formation of a clot within 1 min
6.	Prothrombin time	8.5–13.1 sec
7.	Thrombin time	16–17 sec
8.	Platelet count	1.5–4 lac/mm^3
9.	Euglobulin clot lysis time (ECL T)	2–4 hours
10.	Fibrinogen	300–600 mg%
11.	D-dimer (breakdown product of fibrin)	0.1–1.8 (mcg/mL)

COLLECTION OF BLOOD SAMPLE

MATERIAL: Table 41.4.

EDTA/Glucose vials containing recommended quantity of anticoagulants are available.

CORD BLOOD SAMPLES: Cord blood sample should be collected from the placental end of the severed cord. **It should not be squeezed out,** otherwise Wharton's jelly may contaminate the blood, which may vitiate the result. To get a true picture, the sample should be taken by opening the Kocher's forceps from the placental end of the cord as early as possible. Samples are taken especially of babies born of Rh-negative mothers. 10 mL of cord blood should be collected in a heparinized tube. Alternatively, about 5 mL of blood (2 mL EDTA and 3 mL clotted) should be collected for the following tests:

Arterial Blood and Gas Values in Pregnancy (3rd Trimester at Normal Altitude)		Coagulation Parameters in Normal Pregnancy (3rd Trimester)		Newborn Umbilical Cord Blood at Delivery (Umbilical Artery)	
Test	**Value**	**Test**	**Value**	**Test**	**Value**
Arterial pH	7.44 ± 0.04	INR	0.87 ± 0.07	Hb (g/dL)	15.3–17.2
Arterial PO_2 (mm Hg)	85 ± 5	PTT (sec)	27.5 ± 2.8	Hematocrit (%)	45.2–50.9
O_2 saturation (%)	96 ± 1	Antithrombin III (77–122% of normal 22–39 mg/dL)	102.8 ± 13.5	pH	7.06–7.36
Arterial PCO_2 (mm Hg)	29.7 ± 2.8	Protein C (%) (71–142% of increased functional activity)	94.9 ± 25.5	PCO_2 (mm Hg)	27.8–68.3
Sodium bicarbonate (mEq/L)	22.0 ± 2.1	Protein S (%)	51.7 ± 17.9 (70–120% of normal)	PO_2 (mm Hg)	9.8–41.2

Liver and Pancreatic Function Tests

Parameter	Value (Term)
AST	4–36 (IU/L)
ALT	2–30 (IU/L)
Total bilirubin	0.1–1.1 mg/dL
LDH	80–500 U/L
■ Amylase	10–100 IU/L
■ Lipase	20–150 IU/L

Osmolarity, Electrolytes, BUN (Term)

Parameter	Value
Osmolarity	270–285 (mOsm/kg)
Na^+	124–140 (mEq/L)
K^+	3.4–5.5 (mEq/L)
HCO_3	17–25 (mEq/L)
Urea	4–15 (mg/dL)
Creatinine	0.5–1.1 (mg/dL)
Uric acid	2.0–4 (mg/dL)

- **Clotted blood**—ABO and Rh grouping, direct Coombs' test and serum bilirubin.
- **EDTA blood**—Hemoglobin estimation and blood smear for the presence of immature RBC.

DETECTION OF FETAL HEMOGLOBIN FROM VAGINAL BLEEDING (in case of antepartum hemorrhage)

Table 41.4: Collection of Blood Samples for Laboratory Tests

Test	Method of Collection of Blood in Test Tube or Vial	Amount of Blood
Hb%/PCV	EDTA vial	2 mL
ABO and Rh-group	Plain (clotted) and 3.8% sodium citrate solution	2 mL Few drops
VDRL	Plain (clotted)	2 mL
Direct or indirect Coombs' test	Plain (clotted)	2 mL
Prothrombin time	0.5 mL of 3.8% sodium citrate solution	4.5 mL
Fibrinogen	EDTA vial	2 mL
Platelet	EDTA vial	2 mL
EDP	Special tube supplied with kits	
ECLT	Citrate solution	4.5 mL
D-dimer	EDTA vial	2 mL
Blood glucose	Oxalate-fluoride	2 mL
Blood culture	Glucose broth media 50 mL	5 mL
Urea, NPN, creatinine	Plain (clotted)	5 mL
Bilirubin	Plain (clotted)	3 mL
Plasma protein	EDTA vial	3 mL

Alkali denaturation test (Singer's test): Blood to be sent—2 mL blood is to be collected from the vagina in an EDTA vial.

Detection of fetal red cells in the maternal blood (in case of Rh isoimmunization)

Modified Kleihauer-Betke acid elution test (Fig. 23.6)

Blood to be sent—A blood film is drawn over a dry slide from maternal blood. The slide is fixed by immersing it in a solution of ethanol (80%) for 3 minutes and after drying in air, it is to be sent to the laboratory. *Alternatively*—2 mL of maternal venous blood may be sent to laboratory in an EDTA vial.

SAMPLES FOR BLOOD SUGAR ESTIMATION

GLUCOSE TOLERANCE TEST: *Procedure*—Patient should be on normal carbohydrate diet for 3 days prior to the test. Patient should not eat after dinner and no breakfast is given. In the morning, urine is collected before commencement of the test. A fasting venous blood sample is taken and then patient is given 100 g (or 75 g) of glucose in 250 mL of water flavored with lemon or orange. Further venous blood samples are collected at half-hour intervals for 3 hours. The patient should be at rest during the period. Urinary samples are taken at 1 hour, 2 hours and 3 hours. 2 mL venous blood are taken in a oxalate-fluoride vial. Sodium fluoride is used to destroy glycolytic enzyme. If the test is to be delayed, the blood should be stored in a refrigerator. **The concentration of glucose in plasma or serum is typically about 15% greater than that in whole blood** (True glucose estimated by glucose oxidase-peroxidase method).

DEXTROSTIX: These reagent strips provide a rapid, convenient, specific and semiquantitative method for approximating blood glucose level. The strip is composed of a firm plastic, one end of which is impregnated with chromogen system which turns into oxidized chromogen, based on the action of the enzyme, glucose oxidase.

Procedure: Dip the entire test end of the reagent strip in a large drop of blood either from finger tip or ear lobule. Just after 60 seconds, wash off the blood quickly with a sharp stream of water or to wipe off the blood with a tissue. Read the result within 1–2 seconds after washing, by comparing with the color chart supplied by the manufacturer. The results are provided directly in mg of glucose per 100 mL of blood.

Pulmonary Function Tests (Term)

Parameter	Value
Resp. rate	17/min (unchanged)
Total lung capacity (VC + RV)	4.0 (L)↓ 5%
Vital capacity (VC)	3.20 (L) (unchanged)
Tidal volume (TV)	600 (mL)↑ 30–40%
Residual volume (RV)	800 (mL)↓ by 20–25%
Inspiratory capacity (IC)	2.6 (L) ↑ by 5–10%
Functional residual capacity (ERV + RV)	1,350 (mL) ↓20%

CERVICAL AND VAGINAL CYTOLOGY

While cervical cytology is done as a routine protocol to every woman attending the antenatal clinic in advanced countries, it is selectively used in the developing countries. The indications are:

- **Cervical:** Suspected cervix to exclude premalignant or malignant lesion (see p. 357).
- **Vaginal:** ♦ Vaginitis to know the specific pathogen ♦ Cytohormonal study to know the progesterone status (rarely done nowadays).

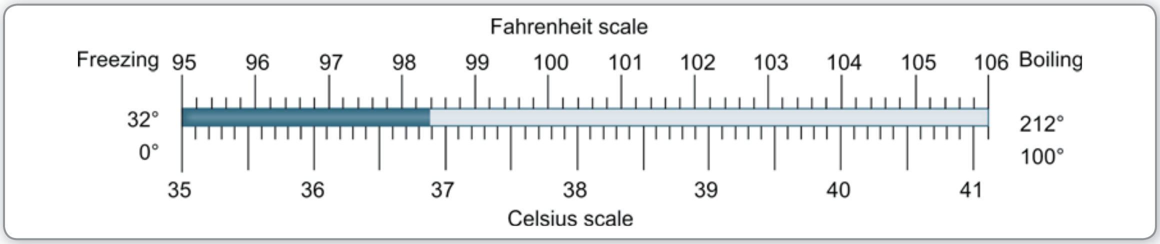
Hormonal effect is studied on the cells obtained from the upper third of lateral vaginal wall by Papanicolaou staining. Normally, combined effects of estrogen and progesterone result in marked thickening of the intermediate layer with practically no superficial layer. This cytohormonal effect is due to progesterone. The vaginal smear shows preponderance of navicular cells (intermediate cells with folded margins). Hence, progesterone deficiency is to be considered, when there is replacement of these cells by superficial cells with rise in karyopyknotic index or a shift to right of maturation index. Clinical conditions are: (i) Threatened abortion. (ii) Missed abortion. (iii) Postmaturity. (iv) Placental insufficiency.

Chapter 42

Practical Obstetrics

• INSTRUMENTS • SPECIMENS • IMAGING STUDIES (USG, MRI) AND DRUGS

Fig. 42.1 CLINICAL THERMOMETER: Celsius temperature scale (°C) is SI derived unit and is known as Celsius after the name of the scientist who introduced it. **Conversion of Fahrenheit to Celsius**—Subtract 32, multiply by 5 and then divide by 9. **Conversion of Celsius to Fahrenheit**—Multiply by 9, divide by 5 and then add 32.



OBSTETRIC INSTRUMENTS

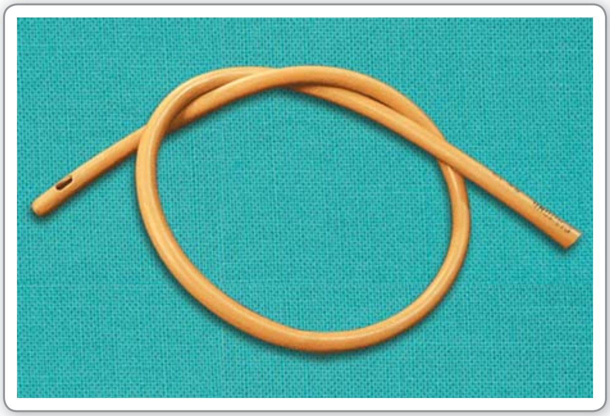


Fig. 42.2 SIMPLE RUBBER CATHETER: It is used to empty the bladder in cases with **retention of urine:** A. during—(a) **Pregnancy** (Retroverted Gravid Uterus p. 361). (b) **Labor—** (i) when the woman fails to pass urine by herself, (ii) before and after any operative interventions (forceps delivery p. 444, destructive operations p. 666). (c) **Postpartum—**(i) during management of postpartum hemorrhage (p. 474), (ii) retained placenta (p. 484). B. **Other uses**—(a) as a tourniquet, (b) to administer O_2 when nasal catheter is not available, (c) as a mucus sucker—when it is attached to a mechanical or electric sucker.

Self-assessment: (i) Length of female urethra (p. 15). (ii) Causes of retention of urine during pregnancy, labor and puerperium. (iii) Why a metal catheter is not used in obstetrics? **Ans:** To avoid trauma to the soft and vascular urethra.

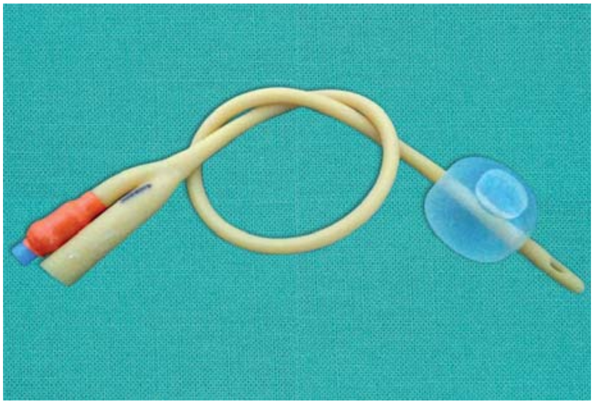


Fig. 42.3 FOLEY's Catheter: It is used for continuous drainage of bladder in cases with: (i) Eclampsia (p. 268). (ii) Retroverted gravid uterus (p. 361). (iii) To give rest to the bladder following any destructive operation and/or in a case with suspected bladder injury (p. 667). It is usually kept for 7–10 days. (iv) In the management of atonic PPH (p. 481). (v) To control atonic PPH. The catheter is inserted within the uterine cavity and the catheter balloon is inflated with normal saline. The balloon provides a **tamponade** (p. 480, 482) to the uterine surface. The catheter drains the blood from the uterine cavity if there is any.

Self-assessment: (i) Indications of continuous bladder drainage (p. 362, 677), (ii) Causes of atonic PPH (p. 498).

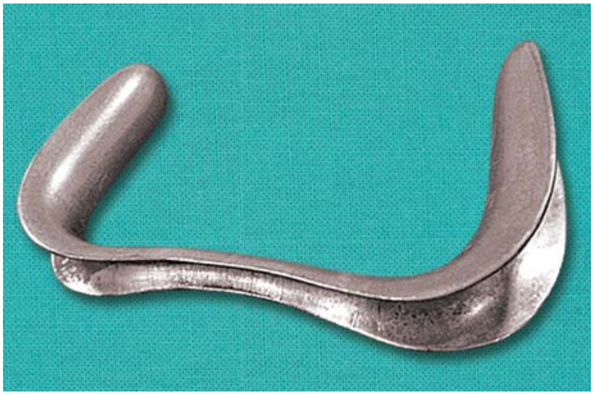


Fig. 42.4 SIMS' DOUBLE BLADED POSTERIOR VAGINAL SPECULUM: The blades are of unequal breadth to facilitate introduction into the vagina depending upon the space available (narrow blade in nulliparous and the wider blade in parous women). **It is used in obstetrics:** (1) To inspect the cervix and vagina and to detect any injury following delivery. (2) To clean the vagina following delivery. (3) To inspect the cervix and vagina to exclude any local cause for bleeding in APH (Cusco's speculum preferred). (4) During D & E operation (p. 642, 643).

Self-assessment: (i) Common sites of traumatic PPH (p. 483), (ii) Diagnosis of traumatic PPH (p. 476), (iii) Indications of D + E (p. 642), (iv) What are the local (extra placental) causes of APH (p. 282), (v) What is Sims' position and what is Sims' triad? *See author's Text Book of Gynecology (p. 103, 423).*

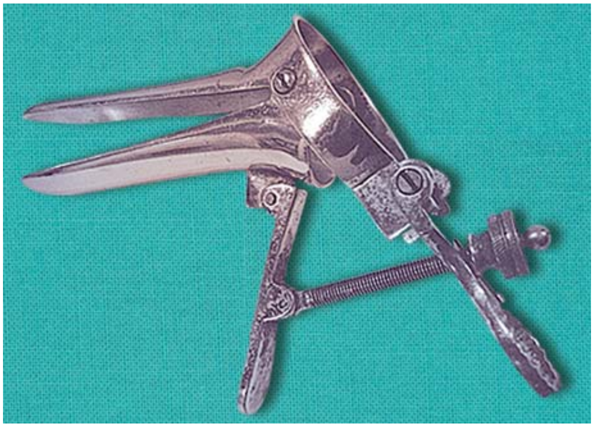


Fig. 42.5 CUSCO'S BIVALVE SELF RETAINING VAGINAL SPECULUM

Uses:

(i) To visualize the cervix and vaginal fornices for any local cause (polyp, ectopy) of APH (p. 287, 291).

(ii) To inspect the cervix and to prepare cervical smear for cytology screening.

(iii) To detect leakage of liqor from the cervical os in a case of suspected PROM (p. 369).

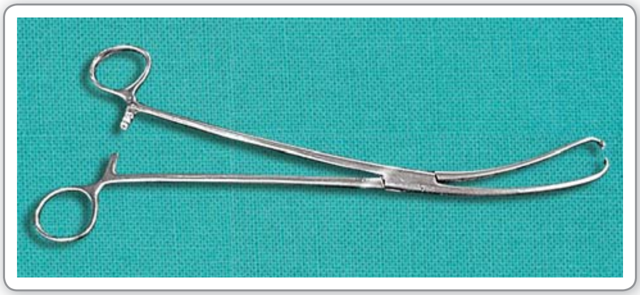


Fig. 42.6 MULTIPLE TOOTHED VULSELLUM:

It is used to catch hold the anterior lip of the cervix in (a) D + E operation, (b) suction evacuation (p. 645). As it produces trauma to the soft and vascular cervix, Allis tissue forceps is used instead.

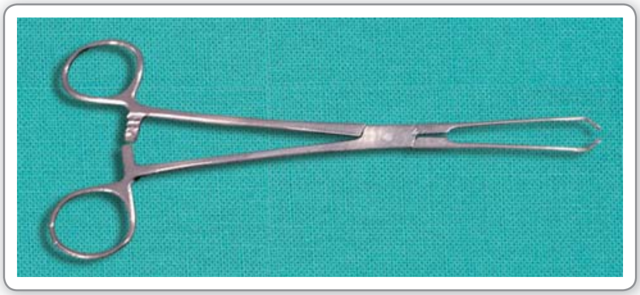


Fig. 42.7 ALLIS TISSUE FORCEPS: Uses: (1) To catch hold the anterior lip of the cervix in D + E operation. (2) To hold the apex of the episiotomy wound during repair. (3) To catch hold of the margins of the peritoneum, rectus sheath, vaginal mucosa during repair. (4) To catch hold of the torn ends of the sphincter ani externus prior to suture in repair of complete perineal tear. (5) To catch hold the margins and angles of the uterine flaps in LSCS after the delivery of the baby as an alternative to Green-Armytage hemostatic clamp.

Self-assessment: (i) **Episiotomy**—See p. 647. (ii) What are the obstetric causes of perineal tear? See p. 489 (iii) What are the different degrees of perineal tear? See p. 490 (iv) When and how a recent perineal tear is repaired? See p. 490.

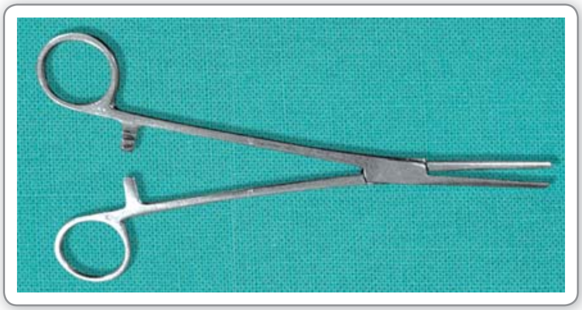


Fig. 42.8 LONG STRAIGHT HEMOSTATIC FORCEPS: Use: This is not commonly used in obstetrics. It can be used to clamp the pedicle while removing the uterus as in rupture uterus. The umbilical cord may be clamped as an alternative to Kocher's.

Self-assessment: (i) What are the causes of rupture uterus? See p. 494 (ii) How to suspect scar dehiscence? See p. 383, 496 (iii) How a case of rupture uterus is managed? See p. 498.

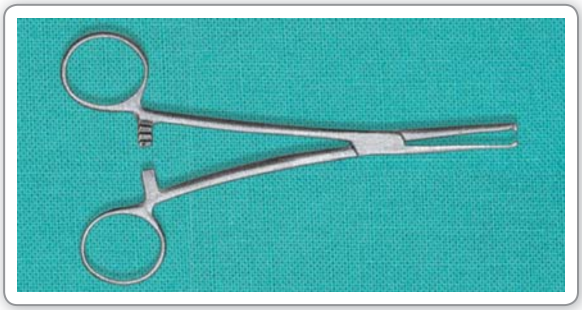


Fig. 42.9 KOCHER'S HEMOSTATIC FORCEPS: Uses: (1) To clamp the umbilical cord—for better grip and effective crushing effect to occlude the vessels. (2) In low rupture of the membranes as surgical induction of labor or augmentation of labor (see also Figs 35.2, 35.3).

Self-assessment: (i) Structures of umbilical cord (p. 44) (ii) Significance of single umbilical artery (p. 254) (iii) Indications of induction of labor (p. 598) (iv) Indications of surgical induction of labor (p. 601, 602, Table 35.6) (v) Dangers of induction of labor (medical and surgical) p. 600 (vi) What is the pre-induction cervical scoring system? See p. 600 (vii) What immediate attention we should pay following ARM? See p. 601-602.

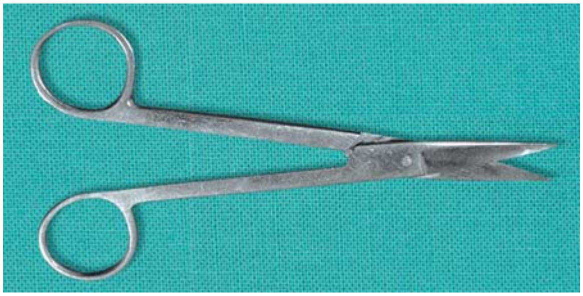
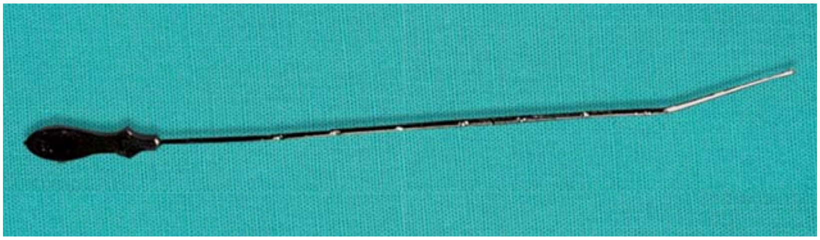


Fig. 42.10 LONG STRAIGHT SCISSORS

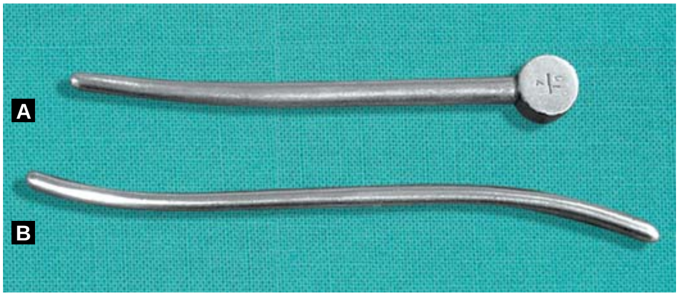
Uses: It is commonly used to cut the (i) umbilical cord (ii) to make episiotomy (iii) to cut suture materials as in cesarean section.

Self-assessment: (i) When the umbilical cord should be clamped and cut? See p. 162. (ii) What are the indications of early cord clamping and cutting? See p. 162. (iii) At what distance from the umbilicus the cord is clamped and cut? See p. 162.

Fig. 42.11 UTERINE SOUND: It is an olive pointed, graduated, malleable, metallic uterine sound.



Uses: (i) To know the position of the uterus and the length of the uterine cavity prior to dilatation of the cervix in D + E operation. (ii) To sound the uterine cavity to detect any foreign body (IUCD). (iii) It acts as a first dilator of the cervical canal. ***Self-assessment:*** (i) What are the instruments required for D + E or Suction Evacuation? See p. 642, 645. (ii) What are the important steps of S + E or D + E? See p. 642, 643, 644, 645. (iii) What are the complications of S + E or D + E operation? See p. 644.



Figs 42.12A and B CERVICAL DILATORS: HAWKIN-AMBLER (Fig. 42.12A) AND DAS OR HEGAR'S DILATORS (Fig. 42.12B)

Hawkin-Ambler: It has got 16 sizes, the smallest one being 3/6 and the largest one being 18/21. The number is arbitrary in the scale of Hawkin-Ambler. The smaller one denotes measurement at the tip and the larger one measures the maximum diameter at the base in mm.

Das or Hegar's dilators is a double ended one. The minimum size is 1/2 and the maximum size is 11/12. The number represents the diameter in mm. Both the sides are used with the lower number first. **Use:** It is used in dilatation of the cervical canal prior to evacuation operation.

Degree of dilatation required: (i) Incomplete abortion—sufficient to introduce the index finger (usually 16/19). (ii) In suction evacuation—one size smaller than the size of the suction cannula. (iii) In MTP by D + E—sufficient dilatation to introduce ovum forceps (usually 9/12). ***Self-assessment:*** (i) How to know the end point of suction procedure? See p. 646, (ii) What is the management protocol when there is uterine perforation? See p. 645, (iii) What are the indications of laparotomy following perforation? **Ans.** Laparoscopy is helpful to assess the situation. (i) Lateral uterine wall injury with intraperitoneal hemorrhage or broad ligament hematoma, (ii) Suspected injury to bowel and/or omentum (iii) Deterioration of vital signs during the period of observation, (iv) Perforation prior to complete evacuation.

Fig. 42.13 FLUSHING CURETTE:

It is a blunt curette used in the operation of D + E. Previously, it was used to flush the uterine cavity with lukewarm antiseptic solution—passing through the communicating channel.

Self-assessment: Questions are similar as in Figures 42.11 and 42.12.

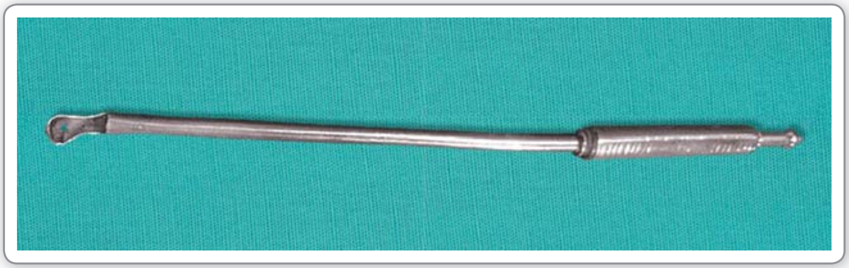
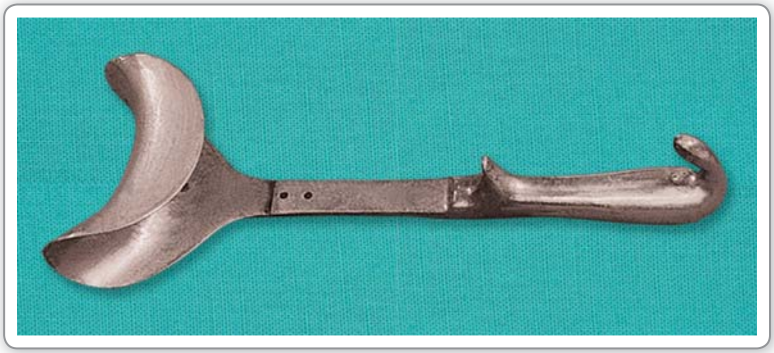


Fig. 42.14 DOYEN'S RETRACTOR:

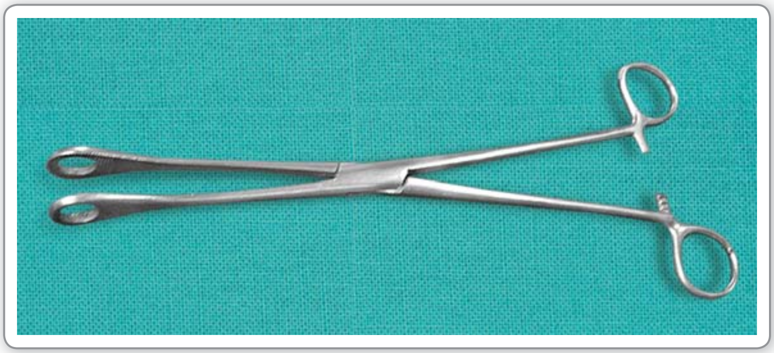
It is used to retract the abdominal wall as well as the bladder for proper exposure of lower uterine segment during LSCS. It is to be **introduced** after opening the abdomen; to be temporarily **taken off** while the baby is delivered, to be **reintroduced** after delivery of the baby and finally to be removed after toileting the peritoneal cavity.



Self-assessment: (i) Types of CS (p. 671), (ii) Common indications of LSCS (p. 670), (iii) Principal steps of LSCS (p. 671), (iv) Merits and demerits of LSCS over classical (p. 676), (v) Complications of CS (p. 677), (vi) Measures to reduce cesarean delivery (p. 679).

Fig. 42.15 SPONGE HOLDING FORCEPS

Uses: (1) Toileting the vulva, vagina and perineum prior to and following delivery. (2) Antiseptic painting of the abdominal wall prior to cesarean section. (3) To catch hold the membranes if it threatens to tear during delivery of the placenta. (4) To catch hold the cervix (2 pairs are needed) for inspection in suspected cervical tear. (5) To catch hold the cervix during encirclage operation.



Self-assessment: (i) What antiseptic solutions are commonly used to clean the vulva and vagina prior to and following delivery? See p. 155. (ii) How the antiseptic painting of the abdominal wall is done before CS and what antiseptic solution is commonly used? See p. 672. (iii) What happens if bits of placental tissue or membranes are left behind? See p. 483. (iv) How a cervical tear is repaired? See p. 491.

Fig. 42.16 OVUM FORCEPS: It has got no catch and the blades are slightly bent and fenestrated. Absence of catch minimizes uterine injury, if accidentally caught. It prevents crushing of the conceptus. It is to be introduced with the blades closed, to open up inside the uterine cavity, to grasp the products and to take out the instrument with a slight rotatory movement. The rotatory movements not only facilitate detachment of the products from the uterine wall but also minimize the injury of the uterine wall, if accidentally grasped.

Self-assessment: (i) How to differentiate it from a sponge holding forceps. (ii) How the absence of catch made it advantageous? See above. (iii) What are the indications of its use? See p. 643.

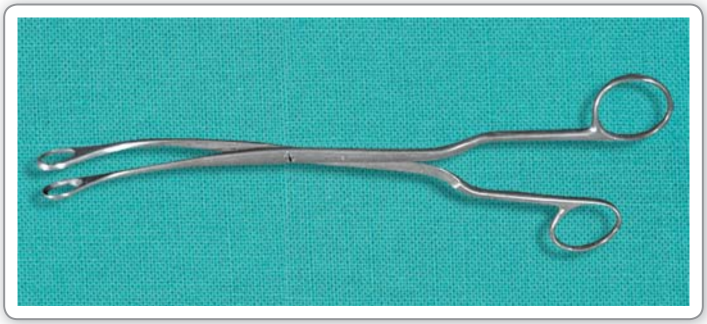


Fig. 42.17 UTERINE CURETTE: It may be sharp at both ends or sharp at one end and blunt at the other. Its common use in obstetrics is in the operation of D + C for incomplete abortion. In D + E operation, the curettage is done by blunt curette as the uterine wall is very soft. It can also be used in D + C operation one week following evacuation of hydatidiform mole.

Self-assessment: (i) Questions as in Figures 42.11, 42.12, 42.13, 42.15 and 42.16. (ii) Place of curettage following evacuation of H. mole (p. 227). (iii) Drawbacks of vigorous curettage. (iv) What are the post-abortion care? See p. 643.

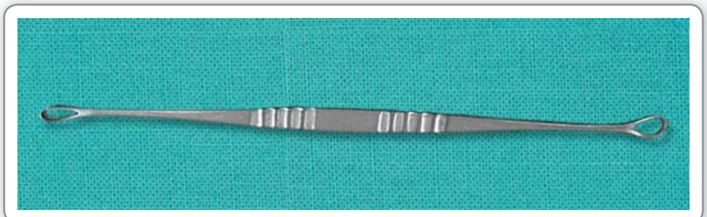


Fig. 42.18 UTERINE DRESSING FORCEPS:

The instrument is most often confused with laminaria tent introducing forceps. The blades are transversely serrated while in the latter, there is a groove on either blade. **It is used:** (a) To swab the uterine cavity following D + E with small gauze pieces (b) To dilate the cervix in lochiometra or pyometra.

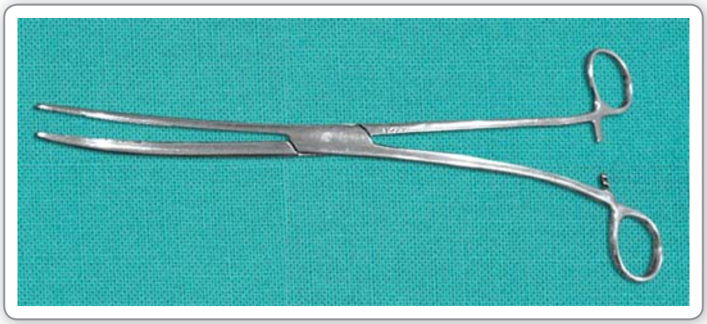


Fig. 42.19 LAMINARIA TENT AND THE TENT INTRODUCING FORCEPS WITH LAMINARIA TENT

The instrument is almost similar to uterine dressing forceps. There is a groove on either blade to catch the laminaria tent.

Laminaria tent: It is dehydrated, compressed, Chinese sea-weeds. It is sterilized by keeping it in absolute alcohol at least for 24 hours. Usually more than one tent is to be introduced to prevent dumbling of the ends. It produces slow dilatation of the cervical canal, as it swells up due to hygroscopic action (see also Fig. 37.1).

Isabgol tents (Isogel): It is dried granules prepared from the husks of "certain mucilaginous tropical seeds".

Self-assessment: (i) Steps of introduction of tents (p. 643), (ii) What are the other alternatives of tent used for slow dilatation of the cervix? See p. 643.

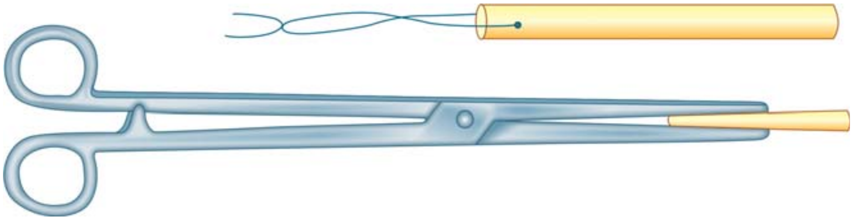
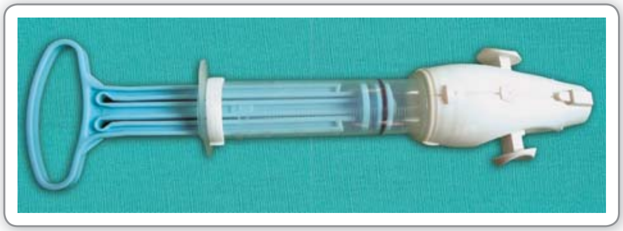


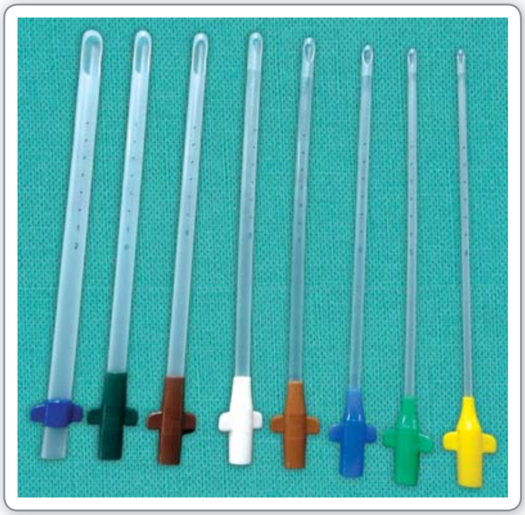
Fig. 42.20 MANUAL VACUUM ASPIRATION (MVA) SYRINGE



Use: This is used for evacuation of the uterus by creating a vacuum. It is used up to 12 weeks of pregnancy (see p. 646). **Advantages of MVA:** (i) It is simple, (ii) safe, (iii) can be done as an outpatient basis, (iv) with local anesthesia, (v) effective (98%), (vi) less traumatic and (vii) it takes less time (10–15 min).

Self-assessment: (i) Methods of termination of pregnancy in the first trimester (p. 203), (ii) Complications of MVA (p. 644), (iii) How one can ensure that the procedure is completed (p. 646), (iv) What are the precautions that we should take (p. 646).

Fig. 42.21 PLASTIC SUCTION CANNULA (KARMAN'S TYPE):



These are of different sizes (4, 5, 6, 7, 8, 9, 10 and 12 mm). Appropriate size of the cannula (diameter in mm) needed for a particular case, is same to the duration of pregnancy in weeks. In general, the size of the pregnant uterus (weeks) and the size of the cannula (mm) are: 4–6 weeks size with 4–7mmcannula; 7–9 weeks size with 5–10 mm cannula and for 9–12 weeks with 8–12 mm size cannula. The plastic cannula has got advantages over the metallic one—as it causes less damage to the uterine wall and the product sucked out is visible. **The vacuum must be broken before it is withdrawal.** It is used for S + E (p. 645) and MVA (p. 646). Cannulas are used for S+ E (p. 645) when attached with MVA syringe.

Self-assessment: (i) How the size of the cannula is determined? See p. 645, (ii) During S + E procedure how the cannula is to be moved? See p. 645, (iii) How much suction pressure is generally used? See p. 645.

Fig. 42.22 LONG CURVED OBSTETRIC FORCEPS

It is commonly used in low forceps operation (see also Fig. 36.12). ***Self-assessment:*** (i) Different types of obstetric forceps (p. 651), (ii) Different parts and the curvatures (p. 652), (iii) Identification of blades (p. 653), (iv) Types of forceps application (p. 654), (v) Functions of the obstetric forceps (p. 654), (vi) Common indications of forceps delivery (p. 654), (vii) Conditions to be fulfilled before application of forceps (p. 654), (viii) Steps of forceps application (p. 655), (ix) Direction of pull during delivery (p. 657, Fig. 37.14), (x) Complications of forceps delivery (p. 659), (xi) Indications of elective (Prophylactic) forceps delivery (p. 659), (xii) Trial of forceps (p. 660), (xiii) What is failed forceps? See p. 660.

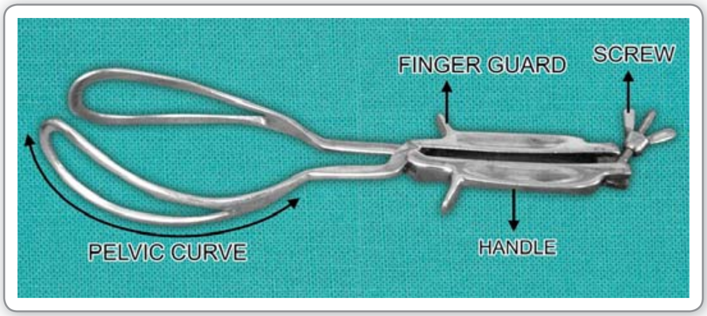


Fig. 42.23 SHORT CURVED OBSTETRIC FORCEPS (WRIGLEY'S FORCEPS)

It can only be used as outlet forceps for extraction of the head. ***Self-assessment:*** (i) Difference with long curved forceps (p. 653), (ii) Define outlet forceps (p. 653), (iii) What is the direction of pull? (p. 657).

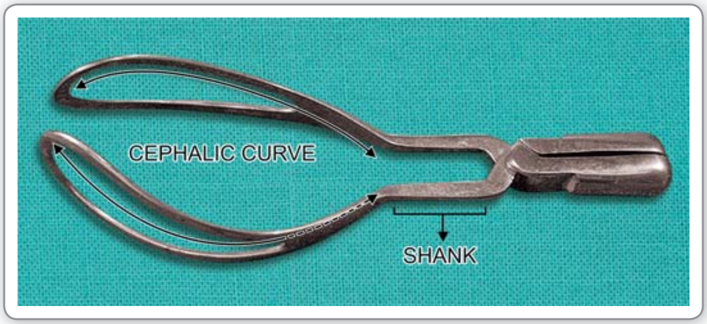


Fig. 42.24 KIELLAND'S FORCEPS: It is usually used as rotation forceps in deep transverse arrest of occipito-posterior position of the head or in unrotated vertex or face presentation.

Self-assessment: (i) Identification of blades (p. 658), (ii) Special advantages over the long curved forceps (p. 653), (iii) Methods of application (p. 658), (iv) Hazards of its use (p. 659).

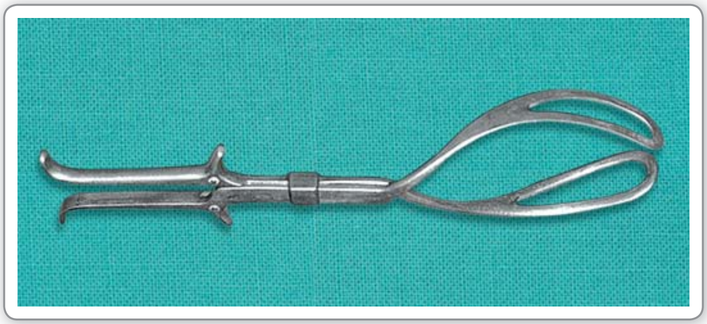
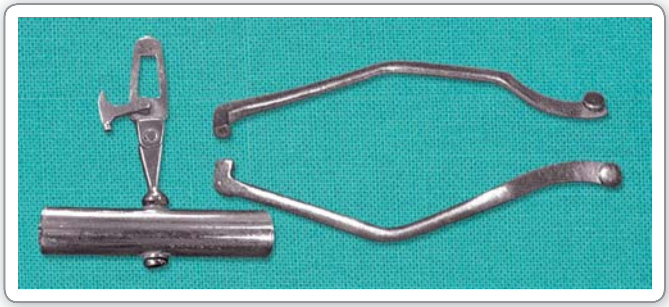


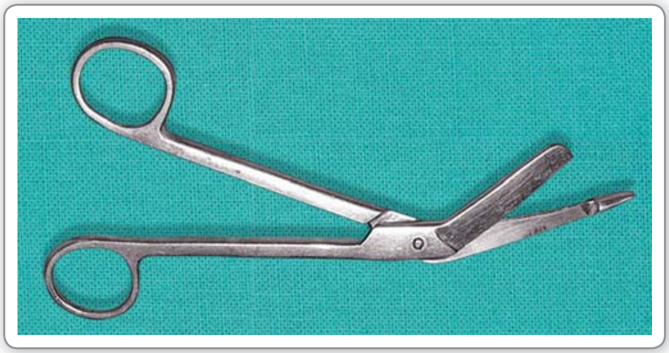
Fig. 42.25 FORCEPS' AXIS TRACTION DEVICES



It includes axis traction rods (right and left) and handle. The rods are assembled in the blades of long-curved obstetric forceps prior to introduction and lastly the handle is attached to the rods. The devices are required where much forces are necessary for traction as in mid forceps operation. These are less commonly used now.

Self-assessment: (i) Identification of traction rods (p. 653). (ii) Indications of use (p. 656).

Fig. 42.26 EPISIOTOMY SCISSORS



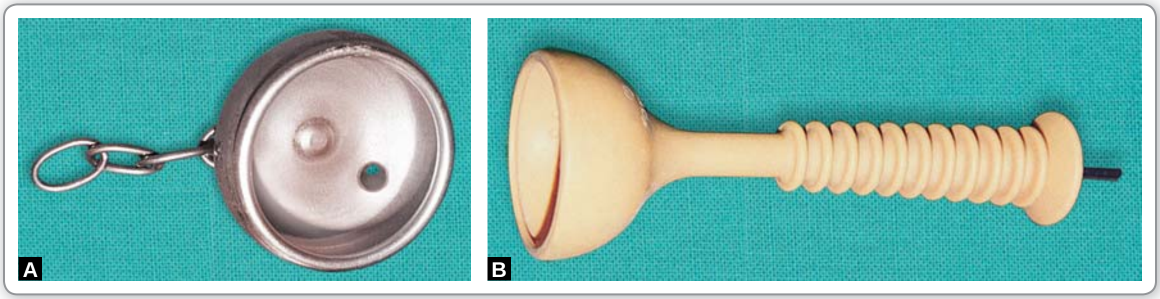
It is bent on edge. The blade with blunt tip goes inside the vagina.

Self-assessment: Common indications of episiotomy (p. 647), (ii) Should episiotomy be made in all cases? See p. 648, (iii) Types of episiotomy (p. 648), (v) Complications of episiotomy (p. 651).

Fig. 42.27 VENTOUSE CUP WITH TRACTION DEVICE

Use: It is used in the operation of vacuum extraction of the head. The cup is to be fitted to the scalp of the forecoming head by producing "chignon" with the help of vacuum. The cup has got various sizes (see p. 660).

Self-assessment: (i) Indications of its use (p. 660), (ii) Advantages over forceps (p. 661), (iii) Conditions to be fulfilled for its application (p. 654), (iv) Methods of its use (p. 661), (v) Hazards of ventouse delivery (p. 663), (vi) Advantages of a silastic cup over the metallic one (p. 660), (vii) What is flexion point? **Ans.** p. 661.



Figs 42.27A and B: Ventouse cup—(A) Metal (B) Silastic

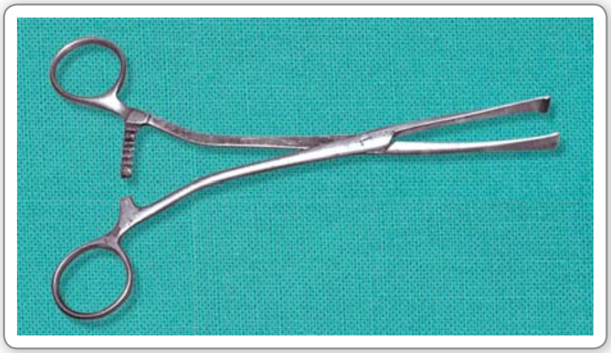


Fig. 42.28 GREEN–ARMYTAGE HEMOSTATIC FORCEPS

This forceps is used in lower segment cesarean section. Total four forceps are ordinarily required—one for each angle and one for each flap. Its functions are hemostasis and to catch hold of the margins so that they are not missed during suture. It cannot be used in classical cesarean section. Alternative to this Allis tissue forceps may be used.

Self-assessment: (i) Factors for rise in CS rate (p. 669), (ii) Methods of suturing the uterine wound (p. 674), (iii) Criteria for VBAC (p. 384), (iv) Intraoperative complications of CS (p. 677).

Fig. 42.29A and B MUCUS SUCKER

(A) Disposable, (B) Metal—It is used to suck out the mucus from the naso-oropharynx following delivery of the head of the baby. To be of value, the mucus should be sucked prior to the attempt of respiration, otherwise the tracheobronchial tree may be occluded leading to inadequate pulmonary aeration and development of asphyxia neonatorum. The metal sucker requires a **sterile simple rubber catheter** to be fitted at one end and a sterile piece of gauze to the other end. Currently electric or the disposable sucker is being used.

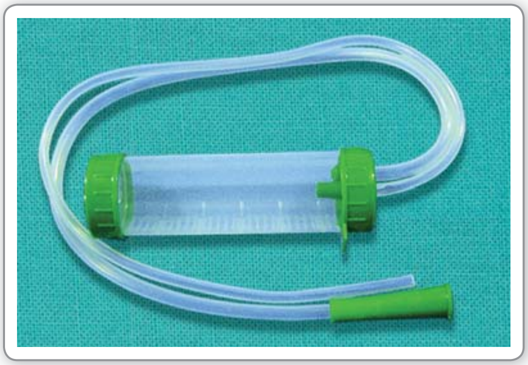


Fig. 42.29A: Mucus sucker—Disposable

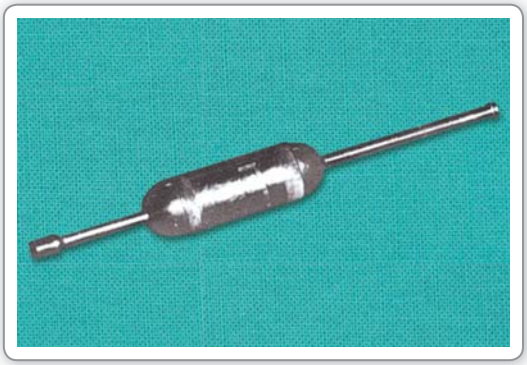


Fig. 42.29B: Mucus sucker—Metal

Self-assessment: (i) Immediate care of the newborn (p. 161). (ii) Management of the cord round the neck (p. 162). (iii) Causes of asphyxia neonatorum (p. 542). (iv) How Apgar Scoring is done? See p. 543. (v) How do you manage an asphyxiated neonate? See p. 544, 545.

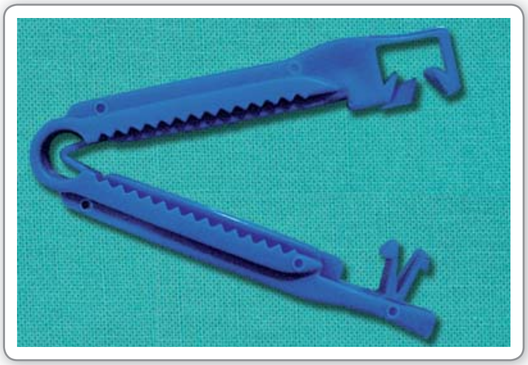
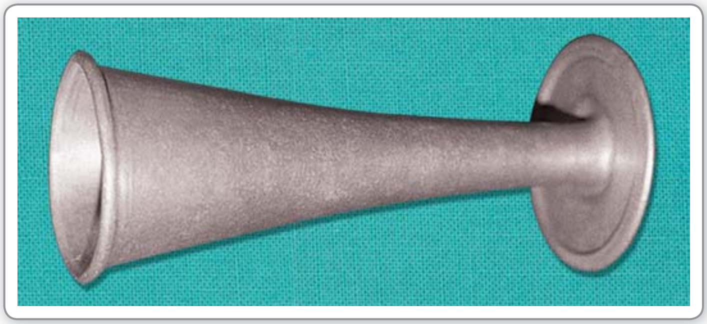


Fig. 42.30 CORD-CLAMP (DISPOSABLE)

It is made of plastic and is supplied in a sterile pack. The serrated surface and the lock make its grip firm. It occludes the umbilical vessels effectively. The cord clamp is to be kept in place until it falls off together with the detached stump of umbilical cord.

Self-assessment: See also Figure 42.9. (i) What is the purpose of the cord-clamp that is applied on the maternal end (p. 162)? (ii) What are the different abnormalities of cord attachment? See p. 253. (iii) What is the significance when the cord is unduly long or short? See p. 253.

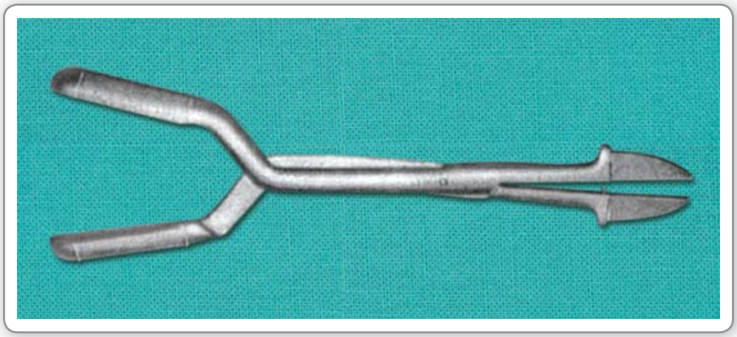
Fig. 42.31 PINARD'S STETHOSCOPE



Use: It should be held firmly at right angle to the point on the abdominal wall. The ear must be firmly closed to the aural end. **It should not be touched by hand while listening.**

Self-assessment: (i) Earliest at what weeks, FHS could be detected with a stethoscope? See p. 79. (ii) What are the different sites where maximum intensity of FHS is obtained in relation to fetal presentation and position? See p. 91. (iii) What are the clinical conditions where FHS may not be audible? See p. 81, 90, 91.

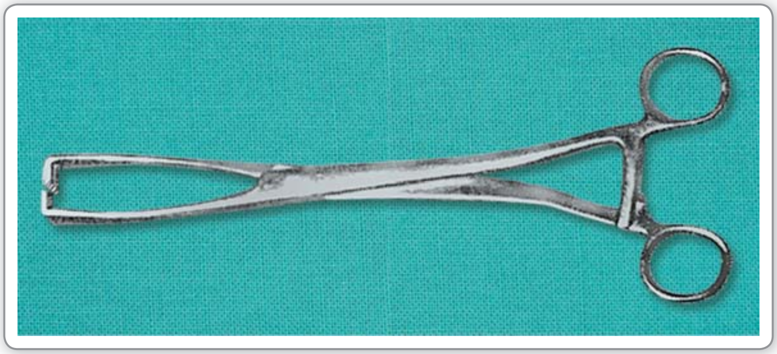
Fig. 42.32 PERFORATOR (OLDHAM'S)



The instrument is required in craniotomy to perforate the skull bone for decompression of the fetal head.

Self-assessment: (i) Indications of craniotomy (p. 666), (ii) Contraindications of craniotomy (p. 666), (iii) Conditions to be fulfilled prior to craniotomy (p. 666), (iv) What specific postoperative care is essential in such a case? See p. 669, (v) Important steps of the operation (p. 667), (vi) Procedure to do after delivery of the placenta (p. 668), (vii) Complications of destructive operations (p. 669).

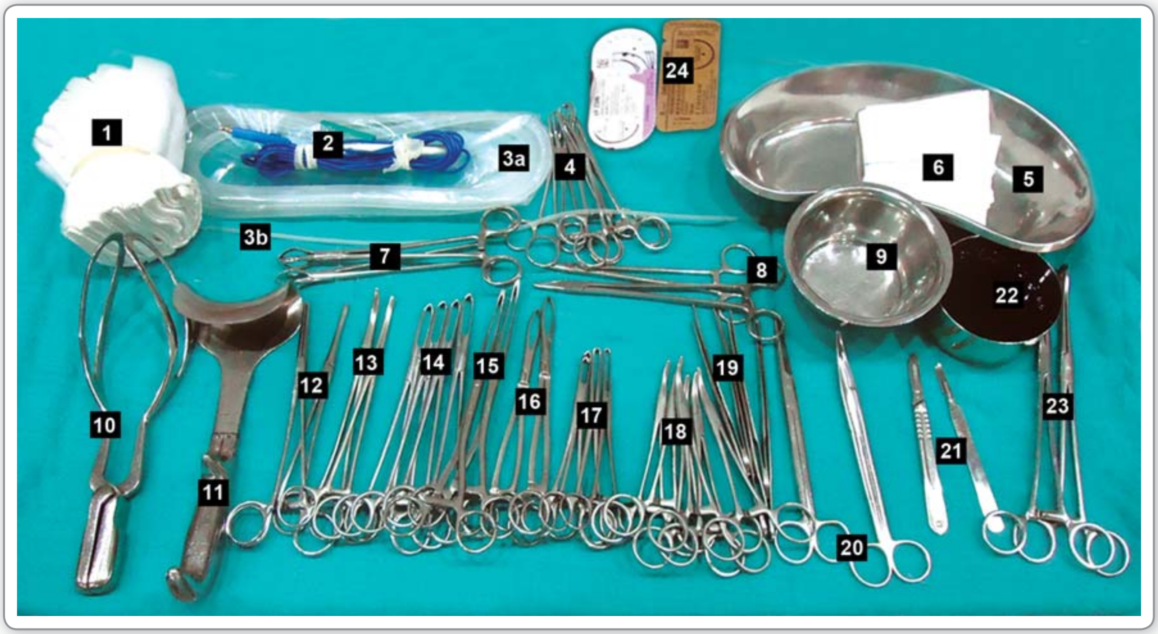
Fig. 42.33 GIANT VULSELLUM



It is used in destructive operation specially in evisceration to have a good grip of the fetal parts for giving traction.

Self-assessment: (i) Indications of use. (ii) What is meant by neglected shoulder presentation (p. 457)? (iii) Mention the postoperative care following any destructive operation (p. 669).

Fig. 42.34 TROLLY WITH INSTRUMENTS PREPARED FOR CESAREAN SECTION OPERATION

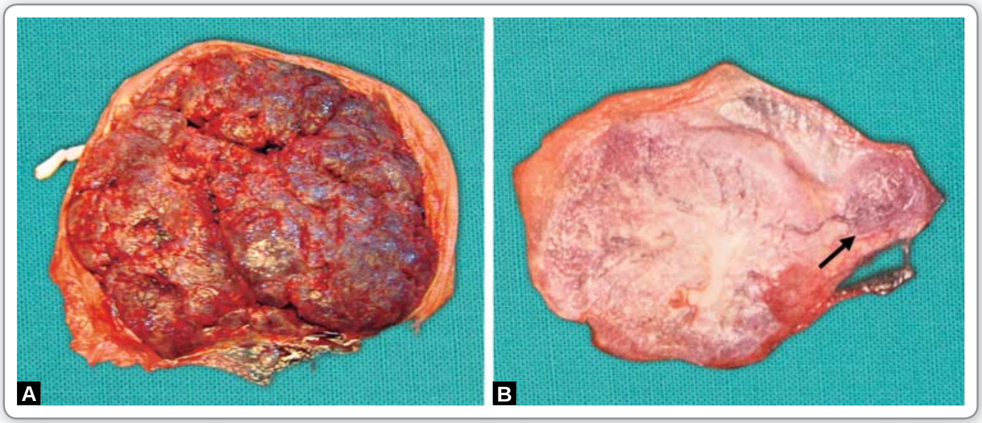


Instruments are (From left to right and top to down): (1) Mops (large swabs), (2) Electrodiathermy set, (3a) Suction tube with cannula, (3b) Baby suction catheter, (4) Towel clips, (5) Kidney dish, (6) Gauze pieces, (7) Lanes tissue forceps, (8) Needle holders, (9) Empty bowl, (10) Obstetric Forceps (Wrigley's), (11) Doyen's retractor, (12) Kocher's clamps, (13) Long artery forceps, (14) Allis tissue forceps (long variety), (15) Green Armytage forceps, (16) Little wood's forceps, (17) Allis tissue forceps (short variety), (18) Artery forceps (short variety), (19) Dissecting forceps toothed and non toothed, (20) Scissors (straight and curved variety), (21) Knives (two), (22) Bowl with povidone iodine lotion, (23) Sponge holding forceps (two), (24) Suture packets.

SPECIMENS

Fig. 42.35 NORMAL PLACENTA AND PLACENTA SUCCENTURIATA

Photographs showing maternal surface of a normal placenta (Fig. 42.34A) and the fetal surface of a placenta succenturiata. Fig. 42.34B: Maternal surface looks rough and shaggy. It shows 15–20 convex



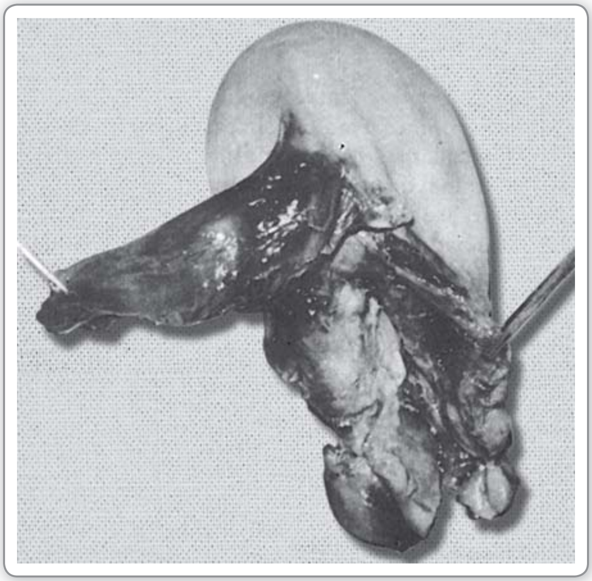
Figs 42.35A and B: (A) Normal placenta; (B) Placenta succenturiata

polygonal areas as lobes or cotyledons bounded by fissures. Membranes are seen at the margin. Fig. 42.34B shows the fetal surface of a placenta succenturiata. The fetal surface looks smooth and shiny as it is covered by amnion layer. The umbilical cord is attached at/or near the centre. There is one small lobe (size of a cotyledon) situated at a distance from the main placental margin. It has vascular communication (arrow mark). It is a placenta with a succenturiate lobe.

Self-assessment: (i) What are the surfaces of a normal placenta and how they could be identified? (p. 33). (ii) What is the weight of a normal placenta? (iii) What is the normal attachment of a placenta (p. 34)? (iv) Clinical significance of placenta succenturiata: **Ans.** p. 252. (ii) What other abnormalities of placenta are commonly seen? **Ans.** P. 251.

Fig. 42.36 RUPTURE UTERUS

It is a specimen of gravid uterus showing ragged, irregular, blackish necrosed margin along the lateral wall of the uterus. Cervix is not seen. It is a specimen of rupture uterus. Sub-total hysterectomy with conservation of ovaries had been done.

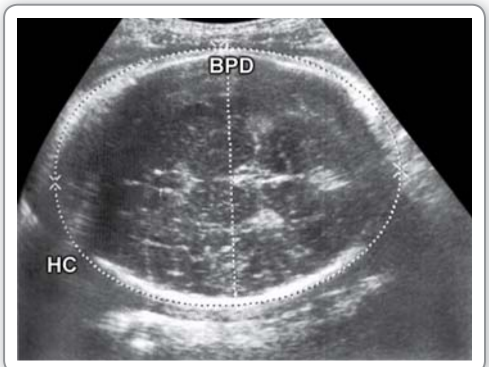


Self-assessment:

(i) What are the common causes of rupture uterus? **Ans.** p. 494. (ii) Differentiation of scar dehiscence from scar rupture (p. 496). (iii) How the diagnosis of rupture uterus (spontaneous as well as scar) is made? **Ans.** p. 497. (iv) How do you manage a case of rupture uterus? **Ans.** Resuscitation and Laparotomy. Laparotomy → Hysterectomy (commonly subtotal). Repair of uterus may be done in selected case with scar rupture. Repair and bilateral tubal ligation may also be done (see details p. 498).

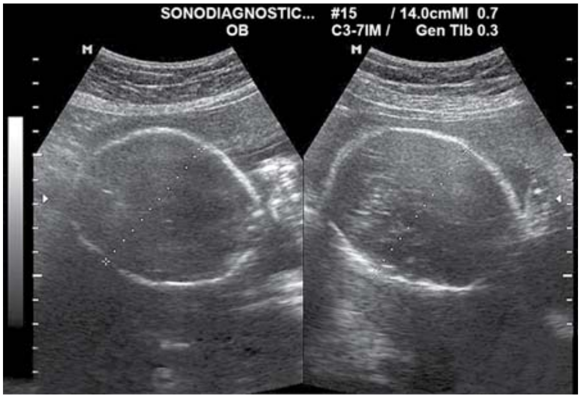
IMAGING STUDIES (USG PLATES)

Fig. 42.37 BPD and HC: It is an ultrasonogram showing the fetal head. In this view the biparietal diameter (BPD) and the head circumference (HC) had been measured as a part of fetal biometry (see p. 735).



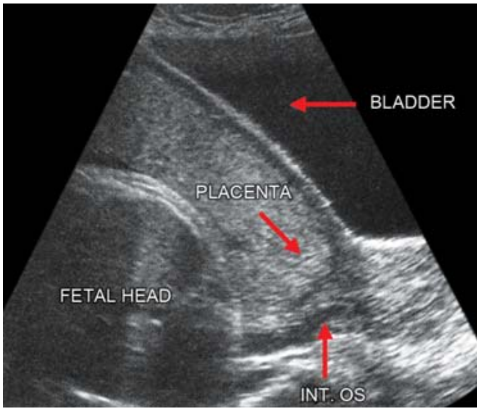
Self-assessment: (i) What is the importance of BPD measurement? **Ans**. BPD is most accurate for assessment of fetal gestational age from 14 to 24 weeks (variation ± 8 days). (ii) How BPD is measured? **Ans.** It is measured from outer edge of proximal skull to the inner edge of the distal skull. It is measured at the level of thalami and cavum septum pellucidum (Fig. 41.4). (iii) What is the importance of HC? **Ans**. Head shape dolichocephaly (flattened) or rounded (brachycephaly) is known. HC is more reliable than BPD. (iv) What other fetal parameters are measured to determine fetal gestational age? **Ans**. Femur length (FL) and abdominal circumference (AC). AC is measured at the level of fetal stomach and umbilical vein.

Fig. 42.38 ULTRASONOGRAM SHOWING TWO FETAL HEADS IN TWIN PREGNANCY



Self-assessment: (i) How the diagnosis of twin pregnancy can be made clinically? **Ans.** p. 237. (ii) What other information can be obtained from sonography besides the confirmation of diagnosis? **Ans.** p. 237. (iii) What are the complications (maternal & fetal) of twin pregnancy? **Ans.** p. 237. (iv) Outline the management of twin pregnancy in labor. **Ans.** p. 243.

Fig. 42.39 ULTRASONOGRAM: PLACENTA PREVIA



It is an ultrasonogram showing placenta previa where the placenta is seen implanted over the anterior wall of the lower segment approaching the internal os.

Self-assessments:

(1) What are the common causes of antepartum hemorrhage? **Ans.** p. 282
(2) What are the types of placenta previa? **Ans.** p. 284
(3) How do you differentiate a case of placenta previa from abruptio placenta? **Ans.** p. 287
(4) What are the complications of placenta previa? **Ans.** p. 288
(5) How do you manage a case of placenta previa? **Ans.** p. 289, 292

Fig. 42.40 ULTRASONOGRAM: HYDATIDIFORM MOLE

Fig. 42.40(A) showing grape like vesicles of varying sizes. These are the tissues of molar pregnancy. Fig. 42.40(B) is an ultrasonogram showing snowstorm appearance of a molar pregnancy.

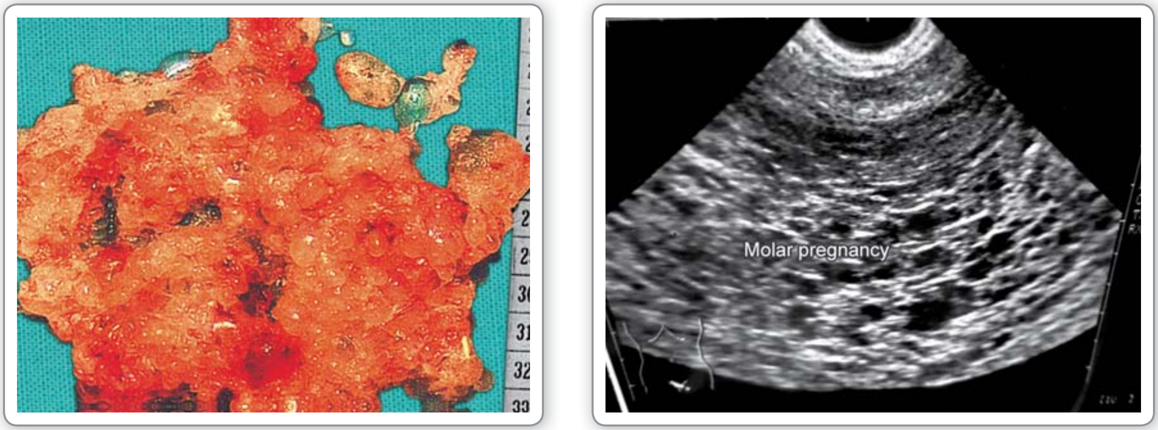
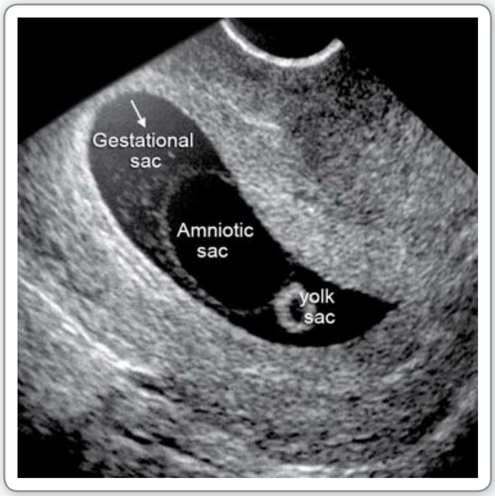


Fig. 42.40(A)

Fig. 42.40(B)

Self-assessment: (i) How a case of molar pregnancy commonly presents? **Ans.** 223. (ii) What are the complications of a molar pregnancy? **Ans.** Hemorrhage, shock, pre-eclampsia, sepsis, acute pulmonary insufficiency and rarely coagulation failure. The late complications are: development of persistent trophoblastic neoplasia and choriocarcinoma (p. 226). (iii) How do you manage a case of hydatidiform mole? **Ans.** p. 226. Principles of management are: (a) Supportive therapy (blood transfusion) (b) Suction evacuation of the uterus and (c) follow up (p. 227).

Fig. 42.41 ULTRASONOGRAM: GESTATIONAL SAC

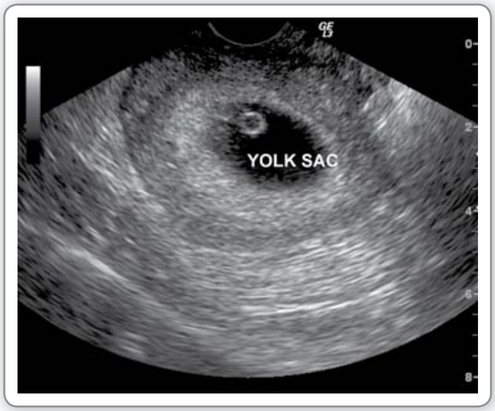


Presence of an intrauterine gestational sac is a reliable evidence of intrauterine pregnancy. The sac is eccentrically located. The gestation sac could be seen as early as 4.5 to 5 weeks with the use of TVS. The normal gestational sac appears round in the early stages and gradually it becomes oval in shape. The gestational sac is filled with chorionic sac fluid which is more echogenic than the amniotic fluid as it contains more protein materials. A gestational sac diameter ≥16mm without an embryo is a strong sign of early pregnancy failure. Abnormal size of the gestational sac is an indication of abnormal outcome.

Clinical Importance of GS

(1) TVS with color flow Doppler Study is helpful to identify a normal pregnancy (2) It can differentiate a normal pregnancy from a failed intrauterine pregnancy (3). It can diagnose ectopic pregnancy (extra uterine). (4) Fetal gestational age can be estimated measuring the mean gestational sac diameter (MSD). (5) Abnormal size of the gestational sac is an indicator of abnormal outcome.

Fig. 42.42 ULTRASONOGRAM: YOLK SAC



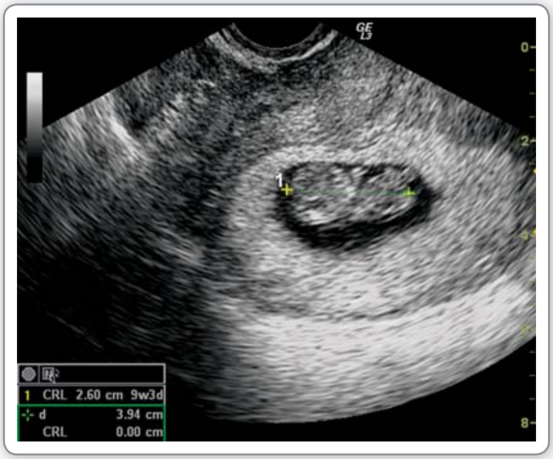
Yolk sac is the first structure seen normally within the Gestational Sac (GS). Yolk sac is seen using TAS when the mean GS diameter (MSD) is 10–15mm and using TVS, with MSD of 8 mm.

Clinical Importance of the Presence of Yolk Sac

(1) It can differentiate an early intrauterine GS from a pseudosac. (2) It is diagnostic of intrauterine pregnancy. (3) It plays an important role of embryonic development by transfer of nutrients. (4) Fetal angiogenesis starts in the wall of the yolk sac in the 5th week. (5) Fetal hematopoiesis occurs first in the yolk sac. (6) Primitive gut is formed from the dorsal wall of the yolk sac.

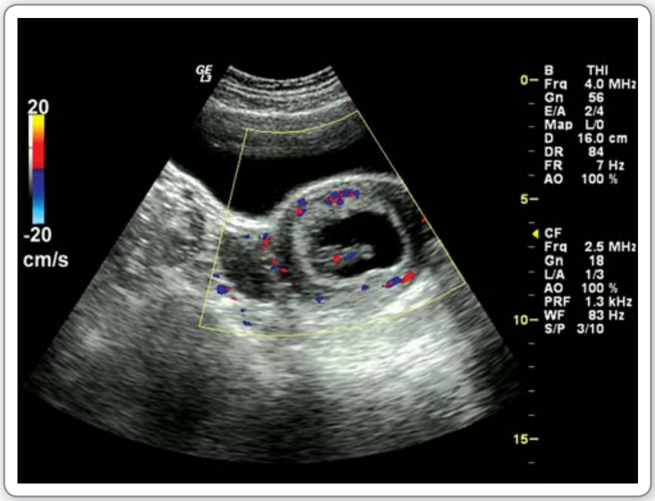
The number of yolk sacs and the number of amniotic sacs are the same. The number of yolk sacs is helpful to determine amnionicity of a multifetal pregnancy. In monochorionic, monoamniotic (MCMA) twin pregnancy there are: (a) Two embryos, (b) One chorionic sac, (c) One amniotic sac and (d) One yolk sac.

Fig. 42.43 ULTRASONOGRAM: CROWN RUMP LENGTH (CRL)



Gestational sac is visualized between 4.5 and 5 weeks of menstrual age. MSD is about 5 mm at 5 weeks. At 5.5 weeks the yolk sac appears. At 6 weeks, an embryo first appears adjacent to the yolk sac. A CRL length when correctly measured in the first trimester is accurate to measure the fetal gestational age to ±5–7 days. The fetal measurement from the top of the head (see calipers) to the end point of the bottom (excluding the limbs) is called CRL. Currently late first trimester sonography (11–13 weeks) is considered to be the ideal time for screening for aneuploidy, when combined with maternal serum screening and fetal nuchal translucency (see p. 735).

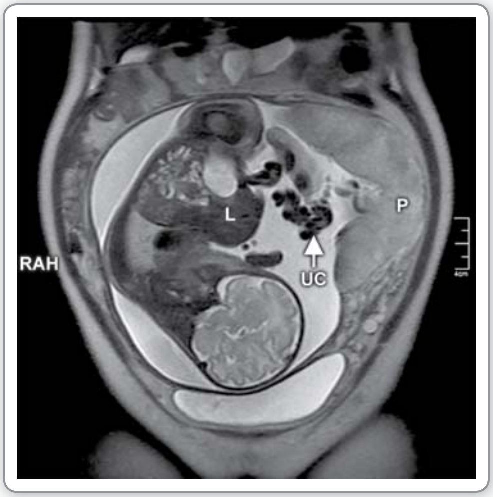
Fig. 42.44 EMBRYONIC CARDIAC ACTIVITY



Using TVS and an embryo can be identified almost always with a MSD of 16–18 mm. The tubular embryonic heart begins to beat at 36–37 days gestational age. In general, cardiac activity can be visualized in normal embryos of greater than 5 mm CRL. Normal embryonic cardiac activity is >100 beats per minute. Absent cardiac activity in embryos is the most important factor in predicting poor pregnancy outcome.

TVS: Embryonic color flow Doppler study showing cardiac activity of a normally growing fetus.

Fig. 42.45



Coronal (T2) image of a fetus with herniation of liver covered with peritoneum, in the amniotic cavity is seen. Fetal brain, foot (Lt), Placenta, attachments of the umbilical cord over the placenta and the herniated mass are seen (omphalocele). Genetic amniocentesis was done. FISH for aneuploidy screen (chromosome—13, 18, 21, X and Y) probes, on amniotic fluid was normal.

Levels: L: Liver; **P:** Placenta; **UC:** Umbilical Cord

Q. How fetal evaluation with MRI is helpful?

Ans: Thorough fetal anatomic survey could be done using fast acquisition MR protocols. This new technology can significantly reduce motion artifact. Volume acquisition, 3D processing and real time MRI for moving organs are the other special benefits.

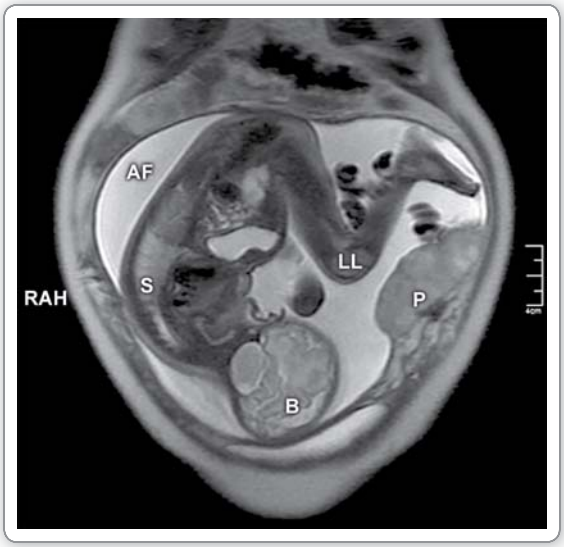
Q. What is the place of MRI compared to Sonography?

Ans: MRI is an adjunct to sonography. MRI is useful in evaluation of complex fetal abnormalities of all the organs. Images of MR are superior to USG, as it is not affected by maternal obesity, oligohydramnios or due to bony interfaces. Limitations of MRI are: (a) not portable and, (b) it is time consuming (ACOR-2010).

Fig. 42.46

Coronal image (T2) of a fetus at 33 2/7 weeks with the placenta, amniotic fluid, fetal brain, spine and the lower limb (Lt) are seen. Attachment of the umbilical cord to the placenta is seen.

Levels: P: Placenta; **AF:** Amniotic Fluid; **FS:** Fetal Spine; **LL:** Lower Limb; **B:** Brain



Q. What are the common indications for fetal MRI?

Ans: Evaluation of fetal anatomy for diagnosis and as an adjunct to therapy.

A. Brain, Spine (ventriculomegaly, vertebral anomalies)
B. Skull, face, neck
C. Thorax
D. Abdomen, retro peritoneum
E. Monochorionic twins (vascular anatomy p. 240), Conjoined twins
F. Fetal surgery (before & after)

Q. How safe MRI is in Pregnancy?

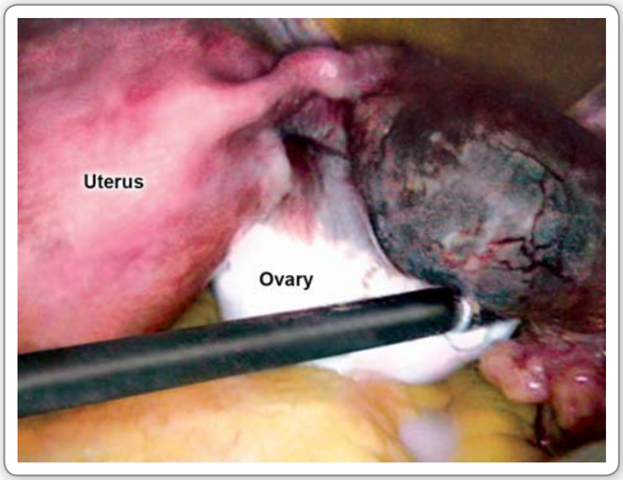
Ans: MRI uses a fluctuating electromagnetic fields (Telsa) and high sound intensity levels. There is no ionizing radiation. MRI studies during pregnancy using 1.5T or less are found safe to the fetus (ACOR-2013)

Q. What are the adverse effects of MRI?

Ans: Presence of metallic implants, pace makers, any other metal or iron devices in the body may alter the study result. Maternal anxiety, fear, claustrophobia has been observed occasionally.

Fig. 42.47 UNRUPTURED TUBAL ECTOPIC PREGNANCY

Laparoscopic panaromic view of an unruptured tubal ectopic pregnancy. Ectopic pregnancy is seen at the region of ampulla of the tube (Rt.).



Self-assessment: (i) How a case of acute (ruptured) tubal ectopic pregnancy is diagnosed? **Ans.** p. 212. (ii) How a case of unruptured tubal ectopic pregnancy is diagnosed? **Ans.** p. 211 (estimation of serum β hCG, TVS and Laparoscopy). (iii) Management of acute tubal ectopic pregnancy (p. 215). (iv) Management of an unruptured tubal pregnancy (p. 216).

PROCESSING OF INSTRUMENTS

A. Disinfection is done by any one of the methods: Immersing instruments in (i) boiling water for 20 minutes, (ii) 2% glutaraldehyde (cidex) solution for 20 minutes, or (iii) 0.5% chlorine solution for 20 minutes (0.5% of chlorine solution is made by adding 3 teaspoons (15 g) of bleaching powder in one liter of water).

B. Cleaning: Instruments are disassembled and washed on all surfaces in running (*preferably warm*) water. The cannulas should be flushed repeatedly.

C. Sterilization: Either by (i) Autoclaving at 121°C (250°F), under pressure of 15 lbs/in^2 (106 kPa) for 30 minutes or (ii) Immersing in 2% glutaraldehyde (cidex) solution for 10 hours.

Fig. 42.48 OXYTOCICS: OXYTOCIN, METHERGINE, MISOPROSTOL (PGE$_1$); CARBOPROST (PGF$_{2\alpha}$), PROSTIN (PGE$_2$)

Self-assessment: (i) What are oxytocics? **Ans.** p. 573, 575, 576. (ii) Indications of use (p. 574). (iii) Contraindication of use (p. 481, 574, 578, 580). (iv) How the different oxytocics differ in the action? **Ans.** p. 578, 579, 601. (v) What are the merits and demerits of oxytocin and prostaglandins when used for medical induction of labor. **Ans.** p. 601.

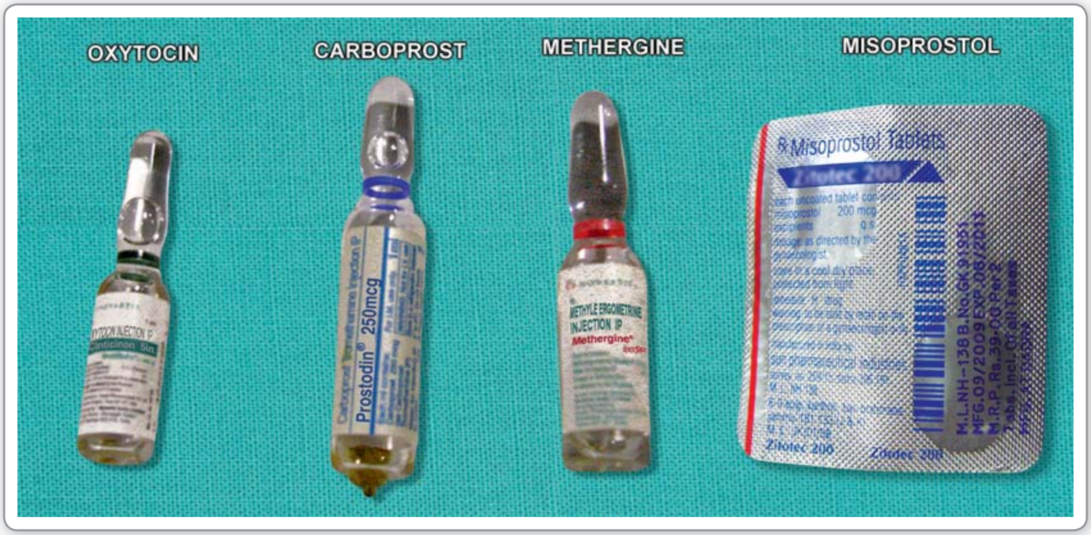
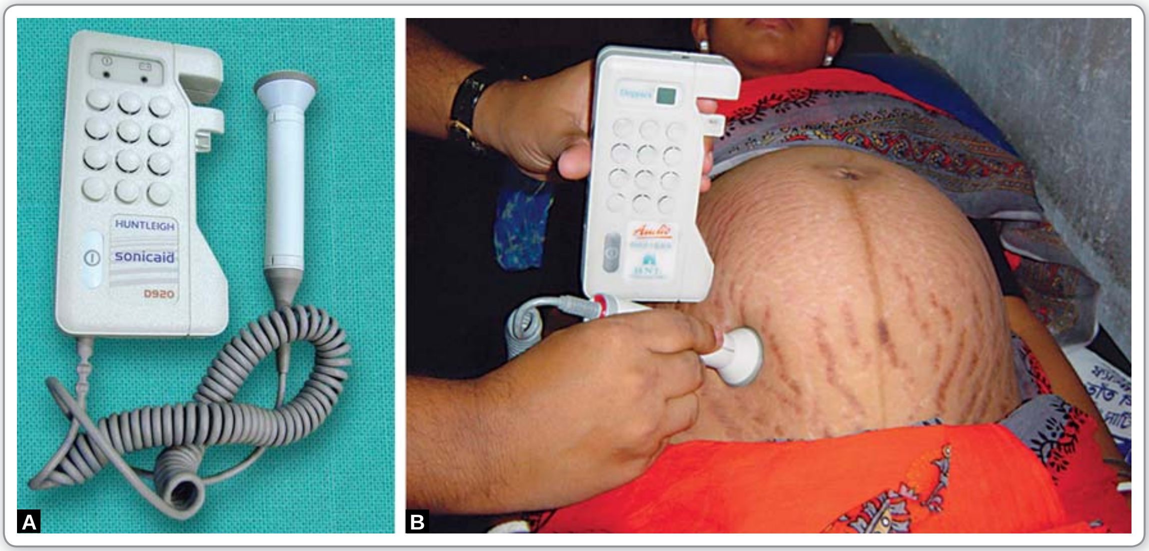

OXYTOCIN
CARBOPROST
METHERGINE
MISOPROSTOL
Prostodin® 250mcg
METHYLE ERGOMETRINE INJECTION IP
Methergine
℞ Misoprostol Tablets
Zitotec 200

Fig. 42.49 DOPPLER (ULTRASOUND) FETAL MONITOR—(A) Device and (B) In use

Self-assessment: (i) Earliest at what weeks the FHS could be detected with a Doppler (p. 78). (ii) What are the other alternatives when FHS is not audible with a stethoscope? **Ans.** (a) Doppler (p. 693) (b) CTG (p. 694) (c) Ultrasound for cardiac motion (p. 78). (iii) What are the advantages of EFM over the clinical? See p. 694. (iv) What are the causes of fetal bradycardia? (p. 693). (v) What are the characteristics of an abnormal CTG? (p. 695). (vi) What other tests could be done when a CTG is abnormal? See p. 696. (vii) What is meant by non-reassuring fetal status (fetal distress)? See p. 697.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