OB Obstetrics

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Acronyms

AC	abdominal circumference
ACOG	American College of Obstetricians and Gynecologists
AFI	amniotic fluid index
AFLP	acute fatty liver of pregnancy
AFV	amniotic fluid volume
AP	anteroposterior
APGAR	Appearance, pulse, grimace, activity, and respiration
aPTT	activated partial thromboplastin time
APS	antiphospholipid antibody syndrome
ARDS	acute respiratory distress syndrome
β-hCG	beta human chorionic gonadotropin
BPP	biophysical profile
C/S	Cesarean section
CHF	congestive heart failure
CMV	cytomegalovirus
CPD	cephalopelvic disproportion
CT	computed tomography scan
CTG	cardiotocography
CVS	chorionic villus sampling
CXR	chest X-ray
D&C	dilatation and curettage
DIC	disseminated intravascular coagulation
DM	diabetes mellitus
DVT	deep vein thrombosis
ECV	external cephalic version
EDD	estimated date of delivery
EFM	electronic fetal monitoring
eFTS	enhanced first trimester screen
EFW	estimated fetal weight
FDP	fibrin degradation products
FHR	fetal heart rate
FISH	fluorescence in situ hybridization
FL	femur length
FM	fetal movement
FPG	fasting plasma glucose
FTS	first trimester screen
GA	gestational age
GBS	Group B *Streptococcus*
GDM	gestational diabetes mellitus
GTN	gestational trophoblastic neoplasia
HC	head circumference
HELLP	hemolysis, elevated liver enzymes, low platelets
IMM	intramyometrial
IOL	induction of labour
IPS	integrated prenatal screen
ITP	idiopathic thrombocytopenic purpura
IUFD	intrauterine fetal death
IUGR	intrauterine growth restriction
IVH	intraventricular hemorrhage
L/S	lecithin-sphingomyelin ratio
LFT	liver function test
LLDP	left lateral decubitus position
LMP	last menstrual period
LMWH	low molecular weight heparin
MSAFP	maternal serum α-fetoprotein
MSS	maternal serum screening
MTX	methotrexate
N/V	nausea/vomiting
NIPT	non-invasive prenatal testing
NPO	nothing by mouth
NST	non-stress test
NTD	neural tube defects
NTUS	nuchal translucency ultrasound
OA	occiput anterior
OGCT	oral glucose challenge test
OGTT	oral glucose tolerance test
ONTD	open neural tube defect
OP	occiput posterior
OT	occiput transverse
PAPP-A	pregnancy-associated plasma protein A
PCR	polymerase chain reaction
PE	pulmonary embolism
PG	plasma glucose
PPD	postpartum depression
PPH	postpartum hemorrhage
PPROM	preterm premature rupture of membranes
PROM	premature rupture of membranes
PTL	preterm labour
QF-PCR	quantitative fluorescence-polymerase chain reaction
RDS	respiratory distress syndrome
RhIG	Rh immune globulin
ROM	rupture of membranes
SFH	symphysis fundal height
SOGC	Society of Obstetricians and Gynaecologists of Canada
SVD	spontaneous vaginal delivery
TENS	transcutaneous electrical nerve stimulation
TOLAC	trial of labour after Cesarean section
T1	first trimester
T2	second trimester
T3	third trimester
TB	tuberculosis
TPN	total parenteral nutrition
TTP	thrombotic thrombocytopenic purpura
U/S	ultrasound
UTI	urinary tract infection
V/Q	ventilation/perfusion lung scan
VBAC	vaginal birth after Cesarean
vWD	von Willebrand disease
VTE	venous thromboembolism

Basic Anatomy Review

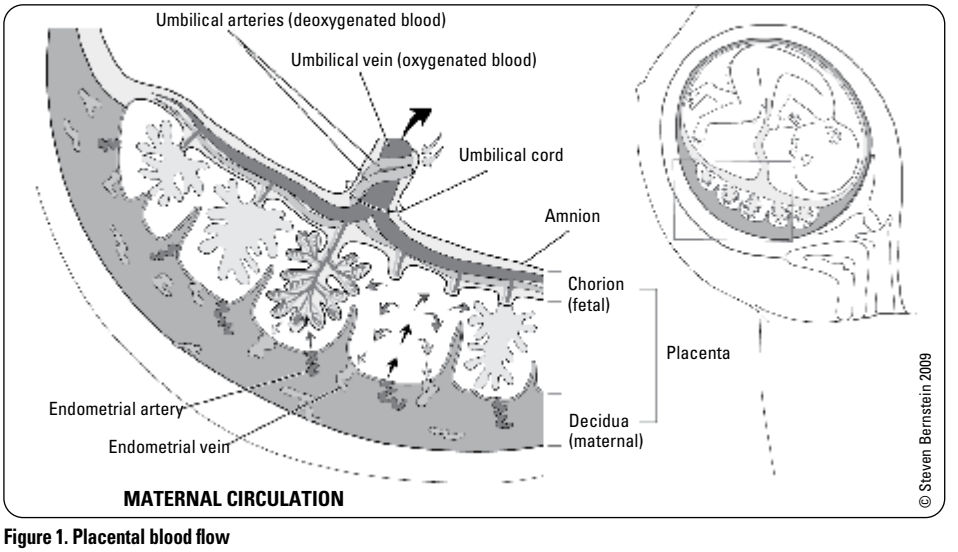

Figure 1. Placental blood flow

Placenta

- site of fetal nutritive, respiratory, and excretory function
- discoid mass composed of fetal (chorion frondosum) and maternal (decidua basalis) tissues divided by fissures into cotyledons (lobules) on the uterine side
- produces hormones such as progesterone, placental lactogen, estrogen, relaxin, β-hCG, and infant growth factors
- poor implantation can lead to spontaneous abortion
- abnormal location, implantation, or detachment can lead to antepartum hemorrhage (see *Obstetrical Hemorrhage*, OB13)

Pregnancy

Diagnosis of Pregnancy

History

- symptoms: amenorrhea, nausea and/or vomiting, breast tenderness, urinary frequency, and fatigue
- obstetrical and gynecological history: year, location, mode of delivery, duration of labour, sex, gestational age, birth weight, and complications of every pregnancy; organize into GTPAL format
  - Gravidity (G)
    - **G**: total number of pregnancies of any gestation (multiple gestation=one pregnancy)
    - includes current pregnancy, abortions, ectopic pregnancies, and hydatidiform moles

- Parity (TPAL)
  - **T**: number of term deliveries (>37 wk)
  - **P**: number of premature deliveries (20-36+6 wk)
  - **A**: number of abortions (ending <20 wk)
    - induced (therapeutic) and spontaneous (miscarriage)
  - **L**: number of living children

Physical Signs
- uterine enlargement
- breast engorgement, areola darkening, and prominent vascular patterns
- Goodell's sign: softening of the cervix (4-6 wk)
- Chadwick's sign: bluish discolouration of the cervix and vagina due to pelvic vasculature engorgement (6 wk)
- Hegar's sign: softening of the cervical isthmus (6-8 wk)

Investigations
- β-hCG: peptide hormone composed of α and β subunits produced by placental trophoblastic cells – maintains the corpus luteum during pregnancy
  - positive in serum 9 d post-conception, positive in urine 28 d after 1st day of LMP
  - plasma levels usually double every 1.4-2.0 d, peak at 8-12 wk, then fall, but continue to be measurable until delivery
- levels less than expected suggest ectopic pregnancy, abortion, inaccurate dates, or some normal pregnancies
- levels greater than expected suggest: multiple gestation, molar pregnancy, Trisomy 21, or inaccurate dates
- U/S:
  - transvaginal
    - 5 wk GA: gestational sac visible
    - 6 wk GA: fetal pole visible
    - 7-8 wk GA: fetal heart activity visible
  - transabdominal
    - 6-8 wk GA: intrauterine pregnancy visible

β-hCG Rule of 10s
10 IU at time of missed menses
100,000 IU at 10 wk (peak)
10,000 IU at term

Trimesters
- T1 (first trimester): 1-14 wk
- T2 (second trimester): 14-28 wk
- T3 (third trimester): 28-42 wk
- Normal pregnancy term: 37-42 wk

Maternal Physiologic Adaptations to Pregnancy

Table 1. Physiologic Changes During Pregnancy

Skin	Increased pigmentation of perineum and areola, chloasma (pigmentation changes under eyes and on bridge of nose), linea nigra (midline abdominal pigmentation), spider angiomas, palmar erythema due to increased estrogen, and striae gravidarum due to connective tissue changes
Cardiovascular	Hyper-dynamic circulation Increased cardiac output, heart rate, and blood volume Decreased blood pressure: decreased PVR and decreased venous return from enlarging uterus compressing IVC and pelvic veins Increased venous pressure leads to risk of varicose veins, hemorrhoids, and leg edema
Hematologic	Hemodilution causes physiologic anemia and apparent decrease in hemoglobin and hematocrit Increased leukocyte count but impaired function leads to improvement in some autoimmune diseases Gestational thrombocytopenia: mild (platelets >70,000/μL) and asymptomatic, normalizes within 2-12 wk following delivery Hypercoagulable state: increased risk of DVT and PE but also decreased bleeding at delivery
Respiratory	Increased incidence of nasal congestion Increased O_2 consumption to meet increased metabolic requirements Elevated diaphragm (i.e. appears more "barrel-chested") Increased minute ventilation leads to decreased CO_2 resulting in mild respiratory alkalosis that helps CO^2 diffuse across the placenta from fetal to maternal circulation Decreased total lung capacity (TLC), functional residual capacity (FRC), and residual volume (RV) No change in vital capacity (VC) and FEV_1
Gastrointestinal	GERD due to increased intra-abdominal pressure and progesterone (causing decreased sphincter tone and delayed gastric emptying) Increased incidence of gallstones due to progesterone causing increased gallbladder stasis Constipation due to progesterone causing decreased GI motility and hemorrhoids as a result of constipation and increased intra-abdominal pressure
Genitourinary	Increased urinary frequency due to increased total urinary output Increased incidence of UTI and pyelonephritis due to urinary stasis (see *Urinary Tract Infection*, OB29) Glycosuria that can be physiologic especially in the T3; consider testing for GDM if noted in first 2 trimesters Ureters and renal pelvis dilation (R>L) due to progesterone-induced smooth muscle relaxation and uterine enlargement Increased CO and thus increased GFR leads to decreased creatinine (normal in pregnancy 35-44 mmol/L), uric acid, and BUN
Neurologic	Increased incidence of carpal tunnel syndrome and Bell's palsy
Endocrine	Thyroid: moderate enlargement (not clinically detectable) and increased basal metabolic rate Increased total thyroxine and thyroxine binding globulin (TBG) Normal free thyroxine index and FSH levels Adrenal: increased maternal cortisol throughout pregnancy (total and free) Calcium: decreased total maternal Ca^{2+} due to decreased albumin Free ionized Ca^{2+} (i.e. active) proportion remains the same due to parathyroid hormone (PTH), resulting in increased bone resorption and gut absorption, and increased bone turnover (but no loss of bone density due to estrogen inhibition) (see *Diabetes Mellitus*, OB26)

PVR – pulmonary vascular resistance; FEV_1 – forced expiratory volume in 1 second; CO – cardiac output; GFR – glomerular filtration rate; BUN – blood urea nitrogen

Antepartum Care

- can be provided by an obstetrician, family physician, midwife, or multidisciplinary team (based on patient preference and risk factors)

Preconception Counselling

- 3-8 wk GA is a critical period of organogenesis, so early preparation is vital
- **past medical history**: optimize medical conditions and review medications prior to pregnancy (see *Medical Complications of Pregnancy*, OB26, and *Medications*, OB49)
- supplementation
  - folic acid: encourage diet rich in folic acid and consider supplementation from 8-12 wk pre-conception until end of T1 to prevent NTD
    - 0.4-1 mg daily in all women; 5 mg if previous NTD, antiepileptic medications, DM, or BMI >35 kg/m^2
  - iron supplementation (in cases of iron deficiency anemia), prenatal vitamins
- **risk modification**
  - lifestyle/social: smoking, alcohol, drug use, domestic violence, occupational risks, poor social support, balanced nutrition, and physical fitness (see Family Medicine)
  - medications: discuss teratogenicity of medications so they may be adjusted, replaced, or stopped if necessary
  - infection screening: rubella, HBsAg, VDRL, Pap smear, gonorrhea/chlamydia, HIV, TB testing based on travel and working in health care, history of varicella or vaccination, parvovirus immunity if exposed to small children, cytomegalovirus immunity if health care worker, and toxoplasmosis serology in case of proximity to cats or gardening
  - genetic testing as appropriate for high risk groups (see *Prenatal Screening*, Table 2); consider genetics referral in known carriers, recurrent pregnancy loss/stillbirth, family members with developmental delay, birth anomalies, genetic diseases, and consanguinity

Family doctors and midwives to consider OB consultation if:
- Insulin-dependent GDM
- VBAC
- HTN
- Multiple gestation
- Malpresentation
- Active antepartum hemorrhage
- PTL/PPROM
- Failure to progress/descend
- Induction/augmentation if high risk
- Tears: 3rd or 4th degree
- Retained placenta

Note: Guidelines vary by institution and by provincial midwifery colleges

Advise all women capable of becoming pregnant to supplement their diet with 0.4 mg/d of folic acid (CTFPHC Grade II-2-A Evidence)

Prenatal and genetic screening are voluntary and require proper counselling and informed consent before proceeding HIV is done automatically in some provinces as opt-out testing; need to inform patient of this

Initial Prenatal Visit

- usually within 8-12 wk of the 1st day of LMP or earlier if <20 or >35 yr old, bleeding, very nauseous, or other risk factors present

History

- gestational age by dates from the 1st day of the LMP
  - Naegele's rule: 1st day of LMP + 1 year + 7 d – 3 mo
  - e.g. LMP = 1 Apr 2014, EDD = 8 Jan 2015 (modify if cycle >28 d by adding number of d >28)
  - EDD by LMP not reliable if irregular menstrual cycle, or if patient unsure of the LMP
- if LMP unreliable, get a dating U/S which could coincide with nuchal translucency at ~12 wk
- EDD by T1 U/S more reliable than LMP if difference is greater than 5 d from LMP due date
- history of present pregnancy (e.g. bleeding, N/V) and all previous pregnancies
- past medical, surgical, and gynecological history
- prescription and non-prescription medications
- family history: genetic diseases, birth defects, multiple gestation, and consanguinity
- social history: smoking, alcohol, drug use, and domestic violence (see Family Medicine)

In history of previous pregnancies, **ALWAYS** ask:
GTPAL
Year
Sex
Weight
Gestational age
Mode of delivery
Length of labour
Complications

Physical Exam

- complete physical exam to obtain baseline patient information – BP and weight important for interpreting subsequent changes
- BMI for risk stratification (risk of DVT, GDM, and pre-eclampsia all increase with greater BMI)

Ask every woman about abuse – not just those whose situations raise suspicion of abuse AND ask as early as possible in pregnancy

Investigations

- blood work
  - CBC, blood group and Rh status, antibody screen, and infection screening as per preconception counselling
- urine R&M, midstream urine C&S
  - screen for bacteriuria and proteinuria
- pelvic exam
  - Pap smear (only if required according to patient history and provincial screening guidelines), cervical or urine PCR for *N. gonorrhoeae* (GC) and *C. trachomatis*

Estimated Date of Delivery (EDD) Determination
- By LMP if menses regular, patient reliable historian
- By T1 U/S if irregular menses
- By embryo age and date of transfer if IVF

- T1 U/S up to 13+6/7 weeks GA is most accurate method of establishing GA
- Changes to the EDD must be documented and discussed with the patient
- Pregnancy without U/S confirming or revising the EDD prior to 22+0/7 GA is considered sub-optimally dated

Nausea and Vomiting

Epidemiology

- affects 50-90% of pregnant women
- often limited to T1 but may persist beyond this

Management
- rule out other causes of N/V especially if refractory to initial therapy
- weigh frequently, assess level of hydration, and test urine for ketones
- non-pharmacological
  - frequent small meals (bland, dry, salty are better tolerated), encourage any safe appealing foods
  - electrolyte oral solutions (Pedialyte®, Gatorade®)
  - stop prenatal vitamins and if T1, substitute with folic acid or adult/children's vitamins that are low in iron
  - increase sleep/rest
  - ginger (maximum 1000 mg/d)
  - acupuncture, acupressure, and mindfulness-based cognitive therapy
- pharmacological
  - first line: pyridoxine (B6) monotherapy or doxylamine/pyridoxine (Diclectin) combination 4 tablets PO daily (1 q am, 1 q lunch and 2 qhs) up to maximum of 8 tablets/d
  - H1 receptor antagonists should be considered for acute or chronic episodes of N/V in pregnancy
  - metoclopramide and phenothiazines can be used as an adjunctive therapy for severe N/V in pregnancy
  - Ondansetron if severe N/V and other anti-emetics have failed
- severe/refractory
  - consider homecare with IV fluids and parenteral anti-emetics, hospitalization

Hyperemesis Gravidarum

Definition
- intractable N/V, usually presents in T1 then diminishes; occasionally persists throughout pregnancy
- affects ~1% of pregnancies

Etiology
- multifactorial with hormonal, immunologic, and psychological components
- rapidly rising β-hCG ± estrogen levels may be implicated

Investigations
- rule out systemic causes: GI, pyelonephritis, thyrotoxicosis
- rule out other obstetrical causes: multiple gestation, GTN, HELLP syndrome
- CBC, electrolytes, BUN, creatinine, LFTs, urinalysis
- U/S

Management
- thiamine supplementation may be indicated
- non-pharmacological (see *Nausea and Vomiting*, OB4)
- pharmacological options
  - doxylamine/pyridoxine (for dosage, see *Nausea and Vomiting*, OB4)
  - dimenhydrinate can be safely used as an adjunct to Diclectin® (1 suppository bid or 25 mg PO qid)
  - other adjuncts: hydroxyzine, pyridoxine, phenothiazine, metoclopramide
  - also consider: ondansetron or methylprednisolone (avoid steroids in T1 due to increased risk of oral clefting)
  - if severe: admit to hospital, NPO initially then small frequent meals; correct hypovolemia, electrolyte disturbance, and ketosis; TPN (if very severe) to reverse catabolic state

Complications
- maternal
  - dehydration, electrolyte, and acid-base disturbances
  - Mallory-Weiss tear
  - Wernicke's encephalopathy, if protracted course
  - death
- fetal: usually none, IUGR is 15x more common in women losing >5% of pre-pregnancy weight

Subsequent Prenatal Visits

Timing
- for uncomplicated pregnancies, SOGC recommends q4-6 wk until 30 wk, q2-3 wk from 30 wk, and q1-2 wk from 36 wk until delivery

Assess at Every Visit
- estimated GA
- history: fetal movements, uterine bleeding, leaking, cramping, questions, concerns
- physical exam: BP, weight gain, SFH, Leopold's maneuvers (T3) for lie, position, and presentation of fetus
- investigations: urinalysis for proteinuria in high risk women; fetal heart rate starting at 10-12 wk using Doppler U/S

Symphysis Fundal Height

Symphysis Fundal Height (SFH)

12 wk	Uterine fundus at pubic symphysis
20 wk	Fundus at umbilicus
20-36 wk	SFH should be within 2 cm of GA

SFH< Dates
- Date miscalculation
- IUGR
- Fetal demise
- Oligohydramnios
- Early engagement

SFH> Dates
- Date miscalculation
- Multiple gestation
- Polyhydramnios
- large for gestational age (familial, DM)
- Fibroids

Leopold's Maneuvers
- performed after 30-32 wk gestation
- first maneuver: to determine which fetal part is lying furthest away from the pelvic inlet
- second maneuver: to determine the location of the fetal back
- third maneuver: to determine which fetal part is lying above the pelvic inlet
- fourth maneuver: to locate the fetal brow

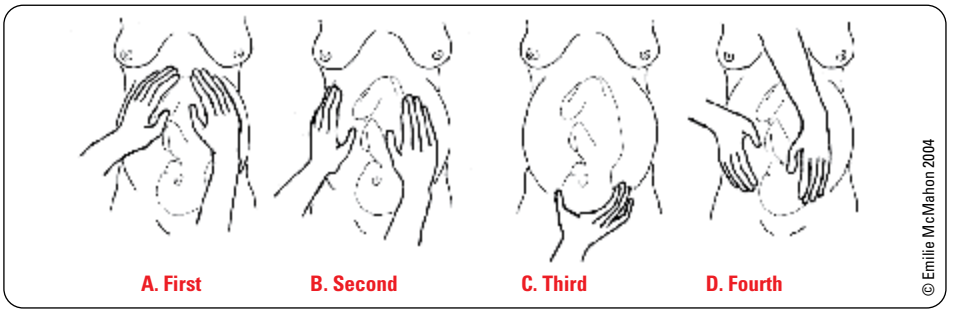

Figure 2. Leopold's maneuvers (T3)
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Prenatal Screening and Diagnostic Tests

Screening Tests
- testing should only occur following counselling and with informed consent from the patient

Table 2. High-Risk Population Screening Tests

Disease (Inheritance)	Population(s) at Risk	Screening Test(s)
Thalassemia (AR)	Mediterranean, South East Asian, Western Pacific, African, Middle Eastern, Caribbean, South American	CBC (MCV and MCH), Hb electrophoresis, or HPLC
Sickle Cell (AR)	African, Caribbean, Mediterranean, Middle Eastern, Indian, South American	CBC (MCV and MCH), Hb electrophoresis, or HPLC
Cystic Fibrosis (CF) (AR)	Family history of CF in patient or partner or medical condition linked to CF like male infertility	CFTR gene DNA analysis
Tay Sachs Disease (AR)	Ashkenazi Jewish*, French Canadians, Cajun	Enzyme assay HEXA, or DNA analysis HEXA gene
Fragile X Syndrome (X-linked)	Family history – confirmed or suspected	DNA analysis: *FMR-1* gene

AR = autosomal recessive; HEXA = hexosaminidase A; HPLC = high performance liquid chromatography
*If both partners are Ashkenazi Jewish, test for Canavan disease and Familial Dysautonomia (FD); if family history of a specific condition, look for carrier status: e.g. Gaucher, CF, Bloom syndrome, Niemann-Pick disease, etc. In all cases, if both partners are positive, refer for genetic counselling.

Table 3. Gestation-Dependent Screening Investigations

Gestational Age (wk)	Investigations	Details
8-12	Dating U/S, possible Pap smear, chlamydia/gonorrhea testing, urine C&S (detect asymptomatic bacteriuria), HIV, VDRL, HepBsAg, Rubella IgG, Parvovirus IgM or IgG if high risk (small child at home or daycare worker/primary teacher), Varicella IgG if no history of disease/immunization, CBC, blood group and screen, urine C&S (detect asymptomatic bacteriuria)	
>10	NIPT	Measures cell-free fetal DNA in maternal circulation
10-12	CVS	
11-14	Enhanced FTS IPS Part 1	Measures 1. Nuchal translucency on U/S 2. β-hCG 3. PAPP-A 4. Placental growth factor (enhanced FTS only) 5. MSAFP (enhanced FTS only)
11-14	Nuchal translucency U/S	
15-16 to term	Amniocentesis	
15-20	IPS Part 2	Measures 1. MSAFP 2. β-hCG 3. Unconjugated estrogen (estriol or μE3) 4. Inhibin A

Routine T2 U/S at 18-22 wk Helps Determine
- Number of fetuses
- GA (if no prior U/S)
- Location of placenta
- Fetal anomalies

Table 3. Gestation-Dependent Screening Investigations (continued)

Gestational Age (wk)	Investigations	Details
15-20	MSS	Measures 1. MSAFP 2. β-hCG 3. Unconjugated estrogen (estriol or μE3) 4. Inhibin A
18-20 to term	Fetal movements (quickening)	
18-20	U/S for dates, fetal growth, and anatomy assessment	
24-28	Gestational Diabetes Screen 50 g OGCT	See *Diabetes Mellitus*, OB26
28	Repeat CBC RhIG for all Rh-negative women	
35-37	GBS screen	See *Group B Streptococcus*, OB28
6 wk postpartum	Discuss contraception, menses, breastfeeding, depression, mental health, support Physical exam: breast exam, pelvic exam including Pap smear (only if due as per provincial screening)	

Maternal serum screen is also referred to as Triple Screen; if Inhibin A is also tested, it is referred to as Quadruple Screen

DDx of Increased MSAFP
- Incorrect GA
- >1 fetus (e.g. twins)
- Fetal demise
- ONTD
- Abdominal wall defects (e.g. omphalocele)

ULTRASOUND SCREENING
- 8-12 wk GA: dating U/S (most accurate form of pregnancy dating)
- measurement of crown-rump length (margin of error: ± 5 d)
- EDD should be based on T1 U/S if available
- 11-14 wk GA: NTUS
- measures the amount of fluid behind the neck of the fetus
- early screen for Trisomy 21 (may also detect cardiac and other aneuploidies like Turner syndrome)
- NT measurement is necessary for the FTS and IPS Part 1
- 18-20 wk GA: growth and anatomy U/S (margin of error: ± 10 d)
- earlier or subsequent U/S performed when medically indicated

NON-INVASIVE PRENATAL TESTING (NIPT)
- analyses maternal blood for circulating cell-free fetal DNA (ccffDNA) at 9-10 wk GA onwards. Requires dating U/S for accuracy

Advantages
- increased accuracy (high detection rate (DR), low false positive rate [FPR]) highly sensitive for Trisomy 21 (DR 99%, FPR 0.1- can also look for Trisomy 18 (DR 96%, FPR 0.1%), 13 (DR 91%, FPR 0.1%), Turner syndrome (DR 90%, FPR 0.2%), and some other disorders (DiGeorge syndrome, Cri Du Chat, Prader-Willi, Angelman syndrome, XY disorders)
- increased positive predictive value
- earlier timing with results available in 1-2 weeks where parents can potentially have a CVS at 11-13 weeks for diagnosis over an amniocentesis after 15 weeks

Disadvantages
- does not screen for ONTD
- high cost to patient (only covered in some provinces [ON and BC] in certain cases)
- need to confirm with invasive testing (it is a screening test, not a diagnostic test)
- does not test for all aneuploidies
- gives no result in 1-5% of cases (insufficient fetal fraction more common with elevated BMI)
- not applicable to donor eggs

Table 4. Comparison of FTS, MSS, and IPS

eFTS	MSS	IPS
11-14 wk	15-20 wk	11-13 wk U/S-nuchal translucency 11-14 wk: eFTS blood 15-20 wk: MSS blood including inhibin A
Risk estimate for 1. Down syndrome (Trisomy 21): increased NT, increased β-hCG, decreased PAPP-A 2. Trisomy 18: increased NT, decreased PAPP-A, decreased β-hCG Note: does not measure risk of ONTD and should be combined with MSAFP at 15-20 wk Useful when patient wants results within the T1 More accurate estimate of Down syndrome risk than MSS, sensitivity ~85% (when combined with age) 5% false positive rate Patients with positive screen should be offered CVS, amniocentesis, or NIPT (covered in some provinces, self-pay in others)	Risk estimate for 1. ONTD: increased MSAFP (sensitivity 80-90%) 2. Trisomy 21: decreased MSAFP, increased β-hCG, decreased μE3 (sensitivity 65%) 3. Trisomy 18: decreased MSAFP, decreased β-hCG, decreased μE3, decreased inhibin A (sensitivity 80%) Only offered alone if patient missed the time window for IPS or eFTS 8% baseline false positive rate for Trisomy 21, lower for NTD and Trisomy 18 Patients with positive screen should be offered U/S, amniocentesis, or NIPT (covered in some provinces, self-pay in others)	Risk estimate for ONTD, Trisomy 21, Trisomy 18 Sensitivity ~85-90% 2% false positive rate Patients with positive screen should be offered U/S and/or amniocentesis or NIPT (covered in some provinces, self-pay in others)

Note: In twins, eFTS, MSS, and IPS are not applicable; screen with NT, NIPT for chromosomal abnormalities and MSAFP for ONTDs

Diagnostic Tests
- Diagnostic tests available:
  - amniocentesis
  - chorionic villus sampling

Indications
- age >35 yr (increased risk of chromosomal anomalies)
  - risk factors in current pregnancy
  - abnormal U/S
- abnormal prenatal screen (IPS, eFTS, or MSS)
- past history/family history of:
  - chromosomal anomaly or genetic disease
  - either parent a known carrier of a genetic disorder or balanced translocation
  - consanguinity
  - >3 spontaneous abortions

AMNIOCENTESIS
- U/S-guided transabdominal extraction of amniotic fluid performed as early as 15 weeks GA

Indications
- identification of genetic and chromosomal anomalies (15-16 wk gestation) as per indications above
- confirmation of positive NIPT testing
- positive eFTS/IPS/MSS
- assessment of fetal lung maturity (T3) via the L/S ratio (lecithin:sphingomyelin)
  - if >2:1, RDS is less likely to occur

Advantages
- also screens for ONTD (acetylcholinesterase and amniotic AFP) – 96% accurate
- in women >35 yr, the risk of chromosomal anomaly (1/180) is greater than the risk of miscarriage from the procedure
- more accurate genetic testing than CVS

Disadvantages
- 1/200 to 1/900 risk of procedure-related pregnancy loss, depending on local experience
- results take 14-28 d; QF-PCR or FISH can be done on chromosomes X, Y, 13, 18, 21, 22 to give preliminary results in 48 h

Compared to CVS, amniocentesis has a higher accuracy of prenatal cytogenetic diagnosis (99.8% vs. 97.5%) and lower risk of spontaneous abortion (0.5% vs. 1-2%)

Risk Factors for Neural Tube Defects

GRIMM
Genetics: family history of NTD (risk of having second child with NTD is increased to 2-5%), consanguinity, chromosomal (characteristic of Trisomy 13, 18, and 21)
Race: European Caucasians > African Americans, 3-fold higher in Hispanics
Insufficient vitamins: zinc and folate
Maternal chronic disease (e.g. DM)
Maternal use of antiepileptic drugs

General population risk for NTD is 0.1%

CHORIONIC VILLUS SAMPLING
- biopsy of fetal-derived chorion using a transabdominal needle or transcervical catheter at 10-12 wk

Advantages
- enables pregnancy to be terminated earlier than with amniocentesis
- rapid karyotyping and biochemical assay within 48 h, including FISH analysis
- high sensitivity and specificity

Disadvantages
- 1% risk of procedure-related pregnancy loss
- does not screen for ONTD
- 1-2% incidence of genetic mosaicism "false negative" results

ISOIMMUNIZATION SCREENING

Definition
- isoimmunization: antibodies (Ab) produced against a specific RBC antigen (Ag) as a result of antigenic stimulation with RBC of another individual

Etiology
- maternal-fetal circulation normally separated by placental barrier, but sensitization can occur and can affect the current pregnancy, or more commonly, future pregnancies
- anti-Rh Ab produced by a sensitized Rh-negative mother can lead to fetal hemolytic anemia
- risk of isoimmunization of an Rh-negative mother with an Rh-positive ABO-compatible infant is 16%
- sensitization routes
  - incompatible blood transfusions
  - previous fetal-maternal transplacental hemorrhage (e.g. ectopic pregnancy, abruption)
  - invasive procedures in pregnancy (e.g. prenatal diagnosis, cerclage, D&C)
  - any type of abortion
  - labour and delivery
  - trauma (e.g. car accident, fall, etc.)

Rh Antibody Titre
A positive titre (≥1:16) indicates an increased risk of fetal hemolytic anemia

Investigations
- screening with indirect Coombs test at first visit for blood group, Rh status, and antibodies
- Kleihauer-Betke test used to determine extent of fetomaternal hemorrhage by estimating volume of fetal blood volume that entered maternal circulation
- detailed U/S for hydrops fetalis
- middle cerebral artery Dopplers are done to assess degree of fetal anemia; if not available, bilirubin is measured by serial amniocentesis to assess the severity of hemolysis
- cordocentesis for fetal Hb should be used cautiously (not first-line)

Standard dose of 300 µg of Rhogam® sufficient for 30 mL of fetal blood. Give additional 10 µg of Rhogam® for every mL of fetal blood over 30 mL

Prophylaxis
- exogenous Rh IgG (Rhogam® or WinRho®) binds to Rh antigens of fetal cells and prevents them from contacting maternal immune system
- Rhogam® (120-300 µg) given to all Rh negative and antibody screen negative women in the following scenarios:
  - routinely at 28 wk GA (provides protection for ~12 wk)
  - within 72 h of the birth of a Rh positive fetus
  - with any invasive procedure in pregnancy (CVS, amniocentesis)
  - as part of management of ectopic pregnancy
  - with miscarriage or therapeutic abortion
  - with an antepartum hemorrhage
  - with trauma
- Rhogam® 300 µg provides sufficient prophylaxis for 30 mL fetal Rh positive whole blood
- a Kleihauer-Betke test or flow cytometry can be used to measure the relative quantity of fetal blood in maternal circulation to determine if additional Rhogam® is indicated (if >30 mL fetal blood)
- if Rh negative and Ab screen positive, follow mother with serial monthly Ab titres throughout pregnancy + U/S ± serial amniocentesis as needed (Rhogam® has no benefit, as B cells sensitized antibodies already in circulation)

Treatment
- falling biliary pigment warrants no intervention (usually indicative of either unaffected or mildly affected fetus)
- intrauterine transfusion between 18-35 wk GA of O-negative packed RBCs may be required for severely affected fetus
- early delivery of the fetus for exchange transfusion following 35 wk GA

Complications
- anti-Rh IgG can cross the placenta and cause fetal RBC hemolysis resulting in fetal anemia, CHF, edema, ascites
- severe cases can lead to hydrops fetalis (edema in at least two fetal compartments due to fetal heart failure secondary to anemia) or erythroblastosis fetalis (moderate to severe immune-mediated hemolytic anemia)

Fetal Surveillance

DDx of Decreased Fetal Movements

DASH
Death of fetus
Amniotic fluid decreased
Sleep cycle of fetus
Hunger/Thirst

- patients will generally first notice fetal movement ("quickening") at 18-20 wk in primigravidas; can occur 1-2 wk earlier in multigravidas; can occur 1-2 wk later if placenta is implanted on the anterior wall of uterus
- if the patient is concerned about decreased fetal movement, she is counselled to choose a time when the fetus is normally active to count movements (usually recommended after 26 wk)
- all high-risk women should be told to do FM counts
  - ≥6 movements in 2 h expected
  - If there is a subjective decrease in fetal movement, time how long it takes to feel 10 discreet movements, laying on the left in a quiet setting may facilitate feeling subtle movements
  - if 10 movements take more than 2 h, further assessment is indicated, and patient should present to labour and delivery triage for non-stress test

NON-STRESS TEST

Normal NST: 2 accels, >15 bpm from baseline, lasting >15 s in 20 min

Definition
- FHR tracing ≥20 min using an external Doppler to assess FHR and its relationship to fetal movement (see *Fetal Monitoring in Labour*, OB33)

Describe NST: baseline rate, absent/minimal/moderate/marked variability, accelerations present/not present, decelerations early/late/variable

Indication
- any suggestion of uteroplacental insufficiency or suspected compromise in fetal well-being

Table 5. Classification of Intrapartum EFM Tracings

	Normal Tracing (Category 1)	Atypical Tracing* (Category 2)	Abnormal Tracing* (Category 3)
Baseline	110-160 bpm	100-110 bpm or >160 bpm for <30 min Rising baseline	Bradycardia <100 bpm Tachycardia >160 for >30 min Erratic baseline
Variability	6-25 bpm (moderate) ≤5 (absent or minimal) for <40 min	5 (absent or minimal) for 40-80 min	≤5 for 80 min Sinusoidal 25 bpm for >10 min
Decelerations	None or occasional variable <30 s	Variable decelerations 30-60 s duration	Variable decelerations >60 s Late deceleration(s)
Accelerations in Term Fetus	2 accelerations with acme of ≥15 bpm, lasting 15 s over <40 min of testing	2 accelerations with acme of ≥15 bpm, lasting 15 s in 40-80 min	<2 accelerations with acme (peak) of contraction of ≥15 bpm, lasting 15 s in >80 min
Accelerations in Preterm Fetus (<32 wk)	>2 accelerations with acme of >10 bpm, lasting 10 s in <40 min	<2 accelerations with acme of >10 bpm, lasting 10 s in 40-80 min	<2 accelerations with acme of >10 bpm, lasting 10 s in >80 min
Action	FURTHER ASSESSMENT OPTIONAL, based on total clinical picture	FURTHER ASSESSMENT REQUIRED	URGENT ACTION REQUIRED An overall assessment of the situation and further investigation with U/S or BPP is required; some situations will require delivery

Adapted from: SOGC, Fetal Health Surveillance: Antepartum and Intrapartum Consensus Guideline, September 2007

Reassuring BPP (8/8)

LAMB
Limb extension + flexion
AFV 2 cm x 2 cm
Movement (3 discrete)
Breathing (one episode x 30 s)

Overlap >20 mm to the internal os in the T3 of pregnancy is highly predictive of the need for a C/S. Any degree of overlap after 35 wk is an indication for a C/S

Operating Characteristics
- false positive rate depends on duration; false negative rate = 0.2-0.3%

Interpretation
- normal: at least 2 accelerations of FHR >15 bpm from the baseline lasting >15 s in 20 min
- abnormal: <2 accelerations of FHR in 40 min
- if no observed accelerations or fetal movement in the first 20 min, stimulate fetus (fundal pressure, acoustic/vibratory stimulation) and continue monitoring for 30 min

BIOPHYSICAL PROFILE

Definition
- U/S assessment of the fetus ± NST

Indications
- post-term pregnancy
- decreased fetal movement
- IUGR
- any other suggestion of fetal distress or uteroplacental insufficiency

Table 6. Scoring of the BPP

Parameter	Reassuring (2 points)
Tone	At least one episode of limb extension followed by flexion
Movement	Three discrete movements
Breathing	At least one episode of breathing lasting at least 30 s
Amniotic Fluid Volume (AFV)*	Fluid pocket of 2 cm in 2 axes

*AFV is a marker of chronic hypoxia, all other parameters indicate acute hypoxia

Interpretation
- 8: perinatal mortality rate 1:1000; repeat BPP as clinically indicated
- 6: perinatal mortality 31:1000; repeat BPP in 24 h
- 0-4: perinatal mortality rate 200:1000; deliver fetus if benefits of delivery outweigh risks

Counselling of the Pregnant Woman

Nutrition

- Canada's Food Guide to Healthy Eating suggests
  - eating a varied diet with plenty of vegetables and fruits, whole grains, dairy products, and lean meats or plant proteins
  - caloric increase of ~100 kCal/d in the T1, ~300 kCal/d in the T2 and T3, and ~450 kCal/d during lactation (less if BMI >25)
  - daily multivitamin with folic acid should be continued during pregnancy

Nutrients in Pregnancy

- folate: 0.4-1 mg/d for first 12 wk (5 mg/d if high risk)
  - supports increase in blood volume, growth of maternal and fetal tissue, and decrease in incidence of NTD
  - foods rich in folic acid include: spinach, lentils, chick peas, asparagus, broccoli, peas, brussels sprouts, corn, and oranges
- calcium: 1200-1500 mg/d
  - maintains integrity of maternal bones, skeletal development of fetus, and breast milk production
- vitamin D: 1000 IU
  - promotes calcium absorption
- iron: 0.8 mg/d in T1, 4-5 mg/d in T2, and >6 mg/d in T3
  - supports maternal increase in blood cell mass, supports fetal and placental tissue
  - required amounts exceed normal body stores and typical intake, and therefore need supplemental iron
  - iron is the only known nutrient for which requirements during pregnancy cannot be met by diet alone (see *Iron and Folate Deficiency Anemia*, OB26)
- essential fatty acids – supports fetal neural and visual development
  - contained in vegetable oils, margarines, peanuts, and fatty fish

Caffeine

- diuretic and stimulant that readily crosses placenta
- less than 300 mg/d is considered safe
- relationship between caffeine and IUGR is unknown (ACOG)
- SOGC states 1-2 cups/d are safe during pregnancy

Sources of Caffeine
- 5 oz cup coffee: 40-180 mg
- 5 oz brewed tea: 20-90 mg
- 12 oz cola: 46 mg
- Red Bull®: 67 mg
- Dark chocolate bar: 10 mg
- 8 oz hot chocolate: 5 mg

Herbal Teas and Preparations

- not enough scientific information about safety of various herbs and herbal products to recommend their use during pregnancy
- some herbal teas can have toxic or pharmacological effects on the mother or fetus
- raspberry leaf tea often used at term to promote labour
- herbal teas considered safe in moderation (2-3 cups/d): citrus peel, ginger, lemon balm, linden flower (unless cardiac condition), orange peel, and rose hip

Foodborne Illnesses

- microbiological contamination of food may occur through cross-contamination and/or improper food handling
  - listeriosis (*Listeria monocytogenes*) and toxoplasmosis (*Toxoplasma gondii*) are of concern during pregnancy
  - avoid consumption of raw meats and fish, raw hotdogs, raw eggs, raw sprouts (especially alfalfa), and unpasteurized dairy products or juices
  - avoid unpasteurized soft cheeses, deli meats, smoked salmon, and pâtés as they may be sources of Listeria
- chemical contamination of food
  - current guideline for mercury of 0.5 ppm in fish is not considered harmful for the general population, including pregnant women
  - Health Canada advises pregnant women to limit consumption of top predator fish such as shark, swordfish, king mackerel, and tilefish

Lifestyle

- exercise under physician guidance; "talk test" = should be able to speak while exercising; avoid supine position after 20 wk GA
- absolute contraindications
  - ruptured membranes, preterm labour, hypertensive disorders of pregnancy, incompetent cervix, IUGR, multiple gestations (>3), placenta previa after 28 wk, persistent T2 or T3 bleeding, uncontrolled type I DM, uncontrolled thyroid disease, serious cardiovascular or respiratory disease, and other systemic disorders

- relative contraindications
  - recurrent pregnancy loss, gestational hypertension, history of spontaneous preterm birth, mild/moderate cardiovascular or respiratory disease, symptomatic anemia, malnutrition, eating disorder, twin pregnancy after 28 wk, and other significant medical conditions
- weight gain: optimal gain depends on pre-pregnancy BMI (varies from 6.8-18.2 kg)
- work: strenuous work, extended hours and shift work during pregnancy may be associated with greater risk of low birth weight, prematurity, and spontaneous abortion
- air travel acceptable in T2; airline cut off for travel is 36-38 wk gestation depending on the airline, to avoid giving birth on the plane
- sexual intercourse: may continue, except in patients at risk for: abortion, preterm labour, or placenta previa; breast stimulation may induce uterine activity, and is discouraged in high-risk patients near term
- smoking: assist/encourage to reduce or quit smoking
  - increased risk of decreased birth weight, placenta previa/abruption, spontaneous abortion, preterm labour, and stillbirth
- alcohol: no amount of alcohol is safe in pregnancy; encourage abstinence from alcohol during pregnancy; alcohol increases incidence of abortion, stillbirth, and congenital anomalies
  - fetal alcohol syndrome (see Pediatrics, P24)
- cocaine: microcephaly, growth retardation, prematurity, and abruptio placentae
- marijuana: smoking associated with low birth weight infants
- biopsychosocial considerations: discuss birth plan, offer community maternal resources

Weight Gain in Pregnancy

BMI	Total Gain	Weekly Gain in T2 & T3
<18.5	28-40 lb	1-1.3 lb/wk
18.5-24.9	25-35 lb	1 lb/wk
>25-29.9	15-25 lb	0.5-0.7 lb/wk
>30	11-20 lb	0.4-0.6 lb/wk

Medications

- most drugs cross the placenta to some extent
- very few drugs are teratogenic, but very few drugs have proven safety in pregnancy
- use any drug with caution and only if necessary
- analgesics: acetaminophen preferable to ASA or ibuprofen

Table 7. Documented Adverse Effects, Weigh Benefits vs. Risks, and Consider Medication Change

Contraindicated Medication	Adverse Effect
ACE Inhibitor	Fetal renal defects, IUGR, oligohydramnios
Carbamazepine	ONTD in 1-2%
Chloramphenicol	Grey baby syndrome (fetal circulatory collapse 2° to toxic accumulation)
Lithium	Ebstein's cardiac anomaly, goitre, hyponatremia
Misoprostol	Mobius syndrome (congenital facial paralysis with or without limb defects), spontaneous abortion, preterm labour
NSAIDs	Premature closure of the ductus arteriosus after 30 wk GA (prior to that, indomethacin used for tocolysis)
Phenytoin	Fetal hydantoin syndrome in 5-10% (IUGR, mental retardation, facial dysmorphogenesis, congenital anomalies)
Retinoids (e.g. Accutane®)	CNS, craniofacial, cardiac, and thymic anomalies
Sulpha drugs	Anti-folate properties, therefore theoretical risk in T1; risk of kernicterus in T3
Tetracycline	Stains infant's teeth, may affect long bone development
Valproate	Congenital malformation (including ONTD) up to 9%
Warfarin	Increased incidence of spontaneous abortion, stillbirth, prematurity, IUGR, fetal warfarin syndrome (nasal hypoplasia, epiphyseal stippling, optic atrophy, mental retardation, intracranial hemorrhage)

Drug Resources During Pregnancy and Breastfeeding
- Hale T. Medications and mothers' milk, 11th ed. Pharmasoft Publishing, 2004
- Lactmed: https://toxnet.nlm.nih.gov/newtoxnet/lactmed.htm

Immunizations

Intrapartum
- administration is dependent on the risk of infection vs. risk of immunization complications
- safe: tetanus toxoid, diphtheria, influenza, hepatitis B, and pertussis
- avoid live vaccines (risk of placental and fetal infection): polio, measles/mumps/rubella, and varicella
- contraindicated: oral typhoid
- the Public Health Agency of Canada recommends:
  - all pregnant women receive the influenza vaccine
  - all pregnant women should be given Tdap every pregnancy irrespective of immunization history ideally between 27-32 weeks but can be given at 13-26 weeks if high risk of preterm labour. If Tdap was given in T1 (i.e. prior to pregnancy recognition), it does not need to be repeated

Postpartum
- rubella vaccine for all non-immune mothers. If they have had an adult booster and remain non-immune, they should not be revaccinated and pregnancy should be deferred for at least 1 mo following vaccination
- hepatitis B vaccine should be given to infant within 12 h of birth if maternal status unknown or positive – follow-up doses at 1 and 6 mo
- any vaccine required/recommended is generally safe postpartum

Radiation

- ionizing radiation exposure is considered teratogenic at high doses
  - if indicated for maternal health, should be done
- imaging not involving direct abdominal/pelvic high dosage radiation is not associated with adverse effects
  - higher dosage to fetus: plain x-ray of lumbar spine/abdomen/pelvis, barium enema, CT abdomen/pelvis/lumbar spine
- radioactive isotopes of iodine are contraindicated
- no known adverse effects from U/S or MRI (long-term effects of gadolinium unknown, avoid if possible)

Table 8. Approximate Fetal Doses from Common Diagnostic Procedures

Examination	Estimated Fetal Dose (cGy)	Number of Exams Safe in Pregnancy
Plain Film		
Abdomen	0-14	35
Pelvis	0-11	45
Lumbar spine	0-17	29
Thoracic spine	0.009	555
Chest (2 views)	<0.001	5000
CT	0-8	6
Abdomen	2-5	2
Pelvis	0-24	20
Lumbar spine	0.006	833
Chest		

Adapted from: Cohen-Kerem, et al. 2005 and Valentin 2000

Radiation in Pregnancy
- Necessary amount to cause miscarriage: >5 cGy
- Necessary amount to cause malformations: >20-30 cGy

Obstetrical Hemorrhage

Definition
- vaginal bleeding from 20 wk to term

Differential Diagnosis
- bloody show (represents cervical changes/early stages of dilation) – most common etiology in T3
- placenta previa
- abruptio placentae – most common pathological etiology in T3
- vasa previa
- cervical lesion (cervicitis, polyp, ectropion, cervical cancer)
- uterine rupture
- other: bleeding from bowel or bladder, abnormal coagulation

Table 9. Comparison of Placenta Previa and Abruptio Placentae

	Placenta Previa	Abruptio Placentae
Definition	Abnormal location of the placenta near, partially, or completely over the internal cervical os	Premature separation of a normally implanted placenta after 20 wk GA
Etiology	Idiopathic	Idiopathic
Epidemiology	0.5-0.8% of all pregnancies	1-2% of all pregnancies
Risk Factors	History of placenta previa (4-8% recurrence risk) Multiparity Increased maternal age Multiple gestation Uterine tumour (e.g. fibroids) or other uterine anomalies Uterine scar due to previous abortion, C/S, D&C, myomectomy	Previous abruption (recurrence rate 5-16%) Maternal HTN (chronic or gestational HTN in 50% of abruptions) or vascular disease Cigarette smoking (>1 pack/d), excessive alcohol consumption, cocaine Multiparity and/or maternal age >35 yr PPROM Rapid decompression of a distended uterus (polyhydramnios, multiple gestation) Uterine anomaly, fibroids Trauma (e.g. motor vehicle collision, maternal battery)
Bleeding	PAINLESS	Usually PAINFUL

Placenta Previa

Definition
- placenta implanted in the lower segment of the uterus, presenting ahead of the leading pole of the fetus
- placental position is described in relation to the internal os as "mm away" or "mm of overlap"

Clinical Features
- PAINLESS bright red vaginal bleeding (recurrent), may be minimized and cease spontaneously but can become catastrophic
- mean onset of bleeding is 30 wk GA, but onset depends on degree of previa
- physical exam
  - do not perform digital vaginal exam until ruling out placenta previa
  - uterus soft and non-tender
  - presenting fetal part high or displaced
  - FHR usually normal
  - shock/anemia correspond to degree of apparent blood loss
- complications
  - fetal
    - perinatal mortality low but still higher than with a normal pregnancy
    - prematurity (bleeding often dictates early C/S)
    - intrauterine hypoxia (acute or IUGR)
    - fetal malpresentation
    - PPROM
    - risk of fetal blood loss from placenta, especially if incised during C/S
  - maternal
    - <1% maternal mortality
    - hemorrhage and hypovolemic shock, anemia, acute renal failure, and pituitary necrosis (Sheehan syndrome)
    - placenta accreta – especially if previous uterine surgery or anterior placenta previa
    - hysterectomy

Investigations
- transvaginal U/S is more accurate than transabdominal U/S at diagnosing placenta previa at any gestational age
- spontaneously resolution is likely with increasing uterine distention if the placenta obscures the internal os by less than 20 mm at 20 wk GA
- transvaginal U/S should be repeated in the T3 as continued change in the placental location is likely

Do NOT perform a vaginal exam until placenta previa has been ruled out by U/S

Management
- goal: keep pregnancy intrauterine until the risk of continuing pregnancy outweighs the risk of preterm delivery
- stabilize and monitor
  - maternal stabilization: large bore IV with hydration, O_2 for hypotensive patients
  - maternal monitoring: vitals, urine output, blood loss, blood work (hematocrit, CBC, INR/PTT, platelets, fibrinogen, FDP, type and cross match)
  - electronic fetal monitoring
  - U/S assessment: when fetal and maternal conditions permit, determine fetal viability, GA, and placental position
- Rhogam® if mother is Rhnegative
- Kleihauer-Betke test to determine extent of fetomaternal transfusion and administer Rhogam® at adequate dose
- GA <37 wk and minimal bleeding: expectant management
  - admit to hospital
  - limited physical activity, no douches, enemas, or sexual intercourse
  - consider corticosteroids for fetal lung maturity
  - delivery when fetus is mature or hemorrhage indicating maternal or fetal compromise
- GA ≥37 wk – deliver by C/S

Abruptio Placentae

Definition
- partial or total placental detachment that is premature and caused by bleeding at the decidual-placental interface
- occurring >20 wk gestation

Clinical Features
- classification
  - total (fetal death inevitable) vs. partial
  - external/revealed/apparent: blood dissects downward toward cervix
  - internal/concealed/occult (20%): blood dissects upward toward fetus, may or may not present with vaginal bleeding
  - most are mixed
- presentation
  - usually PAINFUL (80%) vaginal bleeding (bleeding not always present if abruption is concealed), uterine tenderness, uterine contractions/hypertonus
  - pain: sudden onset, constant, localized to lower back and uterus
  - shock/anemia out of proportion to apparent blood loss
  - ± fetal distress, fetal demise (15% present with demise), bloody amniotic fluid (fetal presentation typically normal)
  - ± coagulopathy

Abruptio placentae is the most common cause of DIC in pregnancy

Complications
- fetal complications: perinatal mortality 25-60%, prematurity, intrauterine hypoxia
- maternal complications: <1% maternal mortality, DIC (in 20% of abruptions), acute renal failure, anemia, hemorrhagic shock, pituitary necrosis (Sheehan syndrome), or amniotic fluid embolus

Investigations
- clinical diagnosis, U/S not sensitive for diagnosing abruption (sensitivity = 15%)

Management
- maternal stabilization: large bore IV with hydration, O_2 for hypotensive patients
- maternal monitoring: vitals, urine output, blood loss, blood work (hematocrit, CBC, PTT/PT, platelets, fibrinogen, FDP, type and cross match)
- electronic fetal monitoring
- blood products on hand (red cells, platelets, cryoprecipitate) because of DIC risk
- Rhogam® if Rh negative
  - Kleihauer-Betke test may confirm abruption
- abruption without fetal/maternal compromise (mild abruption)
  - GA <37 wk: use serial Hct to assess concealed bleeding, deliver when fetus is mature or when hemorrhage dictates
  - GA ≥37 wk: stabilize and deliver
- abruption with fetal/maternal compromise (moderate to severe abruption)
  - hydrate and restore blood loss and correct coagulation defect if present
  - vaginal delivery if no contraindication and no evidence of fetal or maternal distress
  - C/S if live fetus and fetal or maternal distress develops with fluid/blood replacement, labour fails to progress, or if vaginal delivery otherwise contraindicated

Kleihauer-Betke Test
Quantifies fetal cells in the maternal circulation

Vasa Previa

Definition
- unprotected fetal vessels pass over the cervical os; associated with velamentous insertion of cord into membranes of placenta or succenturiate (accessory) lobe

Epidemiology
- 1 in 5000 deliveries – higher in twin pregnancies

Clinical Features
- PAINLESS vaginal bleeding and fetal distress (tachy-to-bradyarrhythmia in a sinusoidal pattern)
- if undiagnosed, 50% perinatal mortality, increasing to 75% if membranes rupture (most infants die of exsanguination)
- if diagnosed antenatally on U/S without labour or symptoms, then 97% survival

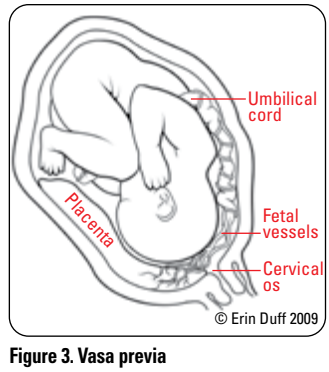


Figure 3. Vasa previa

Investigations
- Apt test (NaOH mixed with the blood) can be done immediately to determine if the source of bleeding is fetal (supernatant turns pink) or maternal (supernatant turns yellow)
- Wright's stain on blood smear and look for nucleated red blood cells (in cord, not maternal blood)

Management
- planned C/S (35-36 weeks) or if bleeding, emergency C/S (since bleeding is from fetus, a small amount of blood loss can have catastrophic consequences)

Obstetrical Complications

Preterm Labour

Definition
- labour between 20 and 37 wk gestation

Etiology
- idiopathic (most common)
- maternal: infection (recurrent pyelonephritis, untreated bacteriuria, chorioamnionitis), HTN, DM, chronic illness, mechanical factors (previous obstetric, gynecological, and abdominal surgeries); socio-environmental (poor nutrition, smoking, drugs, alcohol, stress), pre-eclampsia
- maternal-fetal: PPROM (common), polyhydramnios, placenta previa, abruptio placentae, or placental insufficiency
- fetal: multiple gestation, congenital abnormalities, fetal hydrops
- uterine: excessive enlargement (hydramnios, multiple gestation), malformations (intracavitary leiomyomas, septate uterus, and Müllerian duct abnormalities)

Epidemiology
- preterm labour complicates about 10% of pregnancies

Risk Factors
- prior history of spontaneous PTL is the most important risk factor
- prior history of large or multiple cervical excisions (cone biopsy) or mechanical dilatation (D&C)
- cervical length: measured by transvaginal U/S (cervical length >30 mm has high negative predictive value for PTL before 34 wk)
- identification of bacterial vaginosis and ureaplasma urealyticum infections
  - routine screening not supported by current data, but it is reasonable to screen high-risk women
- family history of preterm birth
- smoking
- late maternal age
- multiple gestation

Prevention of Preterm Labour
A. Cervical Cerclage
- **definition**: placement of cervical sutures at the level of the internal os, usually at the end of the T1 or in the T2 and removed in the T3
- indications: cervical incompetence (i.e. cervical dilation and effacement in the absence of increased uterine contractility)
- diagnosis of cervical incompetence
  - obstetrical Hx: silent cervical dilation, recurrent T2 losses, cervical procedures such as loop excisions
  - ability of cervix to hold an inflated Foley catheter during a hysterosonogram
  - transvaginal U/S of cervical length is recommended only for high-risk pregnancies and only before 30 wk GA
- proven benefit in the prevention of PTL in women with primary structural abnormality of the cervix (e.g. conization of the cervix, connective tissue disorders)

B. Progesterone
- progesterone thought to maintain uterine quiescence; however exact mechanism of action is unclear
- if previous PTL: 17-alphahydroxyprogesterone 250 mg IM weekly from 16+0 to 36 wk GA
- if short cervix: 200 mg daily vaginally from time of diagnosis to 36 wk GA
- superior to cerclage in preventing preterm labour of singletons not due to cervical incompetence

C. Lifestyle Modification
- smoking cessation, substance use reduction, treatment of GU infections (including asymptomatic UTIs), and patient education regarding risk factors

Predicting PTL
- fetal fibronectin: a glycoprotein in amniotic fluid and placental tissue
  - positive if >50 ng/mL; NPV > PPV
  - done if 1 or more signs of preterm labour (regular contractions >6/h, pelvic pressure, low abdominal pain and/or cramps, low backache)
  - done only if: 24-34 weeks, intact membranes, <3 cm dilated, established fetal well being
  - contraindicated as well if: cerclage, active vaginal bleeding, vaginal exam, or sex in last 24 h
  - if negative, not likely to deliver in 7-14 d (>95% accuracy); if positive increased risk of delivery, may need admission/transfer to centre that can do delivery ± tocolysis and/or corticosteroids

Clinical Features
- regular contractions (2 in 10 min, >6/h)
- cervix >1 cm dilated, >80% effaced, or length <2.5 cm

Preterm labour is the most common cause of neonatal mortality in the US

Positive fetal fibronectin in cervicovaginal fluid (>50 ng/mL) at 24 wk gestation predicted spontaneous PTL at <34 wk with sensitivity of 23%, specificity of 97%, PPV of 25%, NPV of 96%

Ultrasonographic Cervical Length Assessment in Predicting Preterm Birth in Singleton Pregnancies
J Obstet Gynaecol Can 2018;40(2);154-61
Recommendations:
1. Transabdominal ultrasonography should not be used for cervical length assessment to predict preterm birth (II-2D).
2. Transvaginal ultrasonography is the preferred route for cervical assessment to identify women at increased risk of spontaneous preterm birth and may be offered to women at increased risk of preterm birth (II-2B).
3. Transperineal ultrasonography may be offered to women at increased risk of preterm birth if transvaginal ultrasonography is either unacceptable or unavailable (II-2B).
4. Because of poor positive predictive values and sensitivities and lack of proven effective interventions, routine transvaginal cervical length assessment is not recommended in women at low risk (II-2E).
5. In women presenting with suspected preterm labour, transvaginal sonographic assessment of cervical length may be used to help in determining who is at high risk of preterm delivery and may be helpful in preventing unnecessary intervention. It is unclear whether this information results in a reduced risk of preterm birth (II-2B).
6. In asymptomatic women with a history of spontaneous preterm birth and an ultrasonographically diagnosed short cervical length (<25 mm) prior to 24 weeks of gestation, cervical cerclage should be considered to reduce the risk of preterm birth (I-B).
7. In all asymptomatic women who present with membranes at or protruding past the external cervical os, an emergency cerclage should be considered to reduce the risk of preterm delivery (I-B).

Management

A. Initial

- transfer to appropriate facility if stable
  - tocolysis and first dose of antenatal steroids prior to transfer
- hydration (normal saline at 150 mL/h)
- bed rest in left lateral decubitus position to reduce aortocaval compression and improve cardiac output
- sedation (morphine)
- avoid repeated pelvic exams (increased infection risk)
- U/S examination of fetus (GA, BPP, position, placenta location, estimated fetal weight)
- prophylactic antibiotics; (for GBS) important to consider if PPROM (e.g. erythromycin controversial, but may help to delay delivery)

B. Tocolysis (Suppression of Labour)

- does not inhibit preterm labour completely, but may delay delivery (used for <48 h) to allow for betamethasone valerate (Celestone®) and/or transfer to appropriate centre for care of the premature infant
- requirements (all must be satisfied)
  - preterm labour
  - live, immature fetus, intact membranes, cervical dilatation of <4 cm
  - absence of maternal or fetal contraindications
- contraindications
  - maternal: bleeding (placenta previa or abruption), maternal disease (HTN, DM, heart disease), preeclampsia or eclampsia, chorioamnionitis
  - fetal: erythroblastosis fetalis, severe congenital anomalies, fetal distress/demise, IUGR, multiple gestation (relative)
- agents
  - calcium channel blockers: nifedipine
    - 20 mg PO loading dose followed by 20 mg PO 90 min later
    - 20 mg can be continued q3-8h for 72 h or to a max of 180 mg
    - 10 mg PO q20min x 4 doses
    - relative contraindications: nifedipine allergy, hypotension, hepatic dysfunction, concurrent beta-mimetics or magnesium sulfate use, transdermal nitrates, or other antihypertensive medications
    - absolute contraindications: maternal congestive heart failure, aortic stenosis
  - prostaglandin synthesis inhibitors: indomethacin
    - 1st line for early preterm labour (<30 wk GA) or polyhydramnios
    - 50-100 mg PR loading dose followed by 25-50 mg q6h x 8 doses for 48 hours

C. Antenatal Corticosteroids

- betamethasone valerate (Celestone®) 12 mg IM q24h x 2 doses or dexamethasone 6 mg IM q12h x 4 doses
  - given between 24 to 33+6 wk GA if expected to deliver in the next 7 d
  - women between 22+0 and 23+6 wk GA at high risk of preterm birth within the next 7 d should be provided with multidisciplinary consultation regarding high likelihood for severe perinatal morbidity and mortality and associated maternal morbidity – consider antenatal corticosteroids therapy if early intensive care is requested and planned
  - specific maternal contraindications: active TB
- enhance fetal lung maturity, reduce perinatal death, reduce incidence of severe RDS, and intraventricular hemorrhage, necrotizing enterocolitis, and neonatal sepsis

D. Neuroprotection

- $MgSO_4$ 4 g bolus followed by 1 g/h infusion for at least 4 h if imminent delivery expected and <32 wk GA

Prognosis

- prematurity is the leading cause of perinatal morbidity and mortality
- 24 wk = 50% survival (may be higher in tertiary care centres with level 3-4 NICU)
- 30 wk or 1500 g (3.3 lb) = 90% survival
- 33 wk or 2000 g (4.4 lb) = 99% survival
- morbidity due to asphyxia, hypoxia, sepsis, RDS, intraventricular cerebral hemorrhage, thermal instability, retinopathy of prematurity, bronchopulmonary dysplasia, necrotizing enterocolitis

Cerclage for Short Cervix on Ultrasonography in Women with Singleton Gestations and Previous Preterm Birth
Obstet Gynecol 2011;117:663-71
Purpose: To determine if cerclage prevents preterm birth (<35 wk gestation) and perinatal mortality and morbidity among women with previous spontaneous preterm birth, asymptomatic singleton gestation, and short cervical length (<25 mm before 24 wk gestation) on transvaginal ultrasonography.
Methods: Meta-analysis of randomized trials identified using searches on MEDLINE, PUBMED, EMBASE, and the Cochrane Library.
Results: 5 trials included. Preterm birth was significantly lower among women receiving cerclage vs. those not receiving (RR = 0.70, 95% CI 0.55-0.89). Cerclage also significantly reduced preterm birth before 24, 28, 32, and 37 wk gestation. Perinatal mortality and morbidity were significantly lower in the cerclage group (RR = 0.64, 95% CI 0.45-0.91).
Conclusions: Cerclage significantly prevents preterm birth and perinatal mortality and morbidity in this specific group of women.

Tocolytics for Preterm Premature Rupture of Membranes
Cochrane DB Syst Rev 2014;2:CD007062
Purpose: To assess the potential benefits and harms of tocolysis in women with PPROM.
Selection Criteria: Pregnant women with singleton pregnancies and PPROM (23-36 wk and 6 d GA).
Results: 8 studies with 408 women total. Prophylactic tocolysis with PPROM was associated with increased overall latency, without additional benefits for maternal/neonatal outcomes. For women with PPROM before 34 wk, there was a significantly increased risk of chorioamnionitis in women who received tocolysis. Neonatal outcomes were not significantly different.
Conclusion: Although there are limitations to the studies, there is currently insufficient evidence to support tocolytic therapy for women with PPROM, as there was an increase in maternal chorioamnionitis without significant benefits to the infant.

Premature Rupture of Membranes

Definitions

- PROM: premature (pre-labour) rupture of membranes at any GA
- prolonged ROM: >24 h elapsed between rupture of membranes and onset of labour
- preterm ROM: ROM occurring before 37 wk gestation
- PPROM: preterm (before 37 wk) AND premature (pre-labour) rupture of membranes

Membrane status determined by

- Pooling of fluid on speculum exam
- Increased pH of vaginal fluid (nitrazine test)
- Ferning of fluid under light microscopy
- Decreased AFV on U/S

Risk Factors

- maternal: multiparity, cervical incompetence, infection (cervicitis, vaginitis, STI, UTI), family history of PROM, low socioeconomic class/poor nutrition
- fetal: congenital anomaly, multiple gestation
- other risk factors associated with PTL

Clinical Features

- history of fluid gush or continued leakage

Investigations
- sterile speculum exam (avoid introduction of infection)
  - pooling of fluid in the posterior fornix
  - cascading: fluid leaking out of cervix with cough/valsalva
- nitrazine (basic amniotic fluid turns nitrazine paper blue)
  - low specificity as it can also be positive with blood, urine, or semen
- ferning: salt in amniotic fluid evaporates, giving amniotic fluid the appearance of ferns on microscopy
- U/S to rule out fetal anomalies; assess GA, presentation, and BPP

Management
- admit for expectant management and monitor vitals q4h, daily NST, WBC count, increased surveillance
- avoid introducing infection by minimizing examinations
  - consider administration of betamethasone valerate (Celestone®) to accelerate maturity if <34 weeks if no evidence of infection
  - consider tocolysis for 48 h to permit administration of steroids if PPROM induces labour
- screen women for UTIs, STIs, GBS infection and treat with appropriate antibiotics if positive (treat GBS at time of labour)
- if not in labour or labour not indicated, consider antibiotics: penicillins or macrolide antibiotics are the antibiotics of choice
- deliver urgently if evidence of fetal distress and/or chorioamnionitis

Table 10. PROM Management

Degree of Prematurity	Management
22-25 wk	Individual consideration with counselling of parents regarding risks to preterm infants
26-34 wk	Expectant management as prematurity complications are significant
34-36 wk	"Grey zone" where risk of death from RDS and neonatal sepsis is the same
≥37 wk	Induction of labour since the risk of death from sepsis is greater than RDS

Prognosis
- varies with gestational age
- 90% of women with PROM at 28-34 wk GA go into spontaneous labour within 1 wk
- 50% of women with PROM at <26 wk GA go into spontaneous labour within 1 wk
- complications: cord prolapse, intrauterine infection (chorioamnionitis), premature delivery, limb contracture, and pulmonary hypoplasia especially at very early gestation

Antibiotic Therapy in Preterm Premature Rupture of the Membranes
J Obstet Gynaecol Can 2017;39(9):207-12
Recommendations:
1. Following PPROM at 32 weeks' gestation, antibiotics should be administered to women who are not in labour in order to prolong pregnancy and to decrease maternal and neonatal morbidity.
2. The benefit of antibiotics is greater at earlier gestational ages.
3. Antibiotics of choice are penicillins or macrolide antibiotics (erythromycin) in parenteral and/or oral forms. In patients allergic to penicillin, macrolide antibiotics should be used alone.
4. Two possible regimen options from large PPROM RCTs are: (1) ampicillin 2g IV q6h and erythromycin 250 mg IV q6h for 48 hours followed by amoxicillin 250 mg orally q8h and erythromycin 333 mg orally q8h for 5 d; (2) erythromycin 250 mg orally q6h for 10 d.
5. Amoxicillin/clavulanic acid should not be used because of an increased risk of necrotizing enterocolitis in neonates. Amoxicillin without clavulanic acid is safe.
6. Women presenting with PPROM should be screened for urinary tract infections, sexually transmitted infections, and group *B streptococcus*.

Postterm Pregnancy

Definition
- pregnancy >42 wk GA

Epidemiology
- 41 wk GA: up to 27%
- >42 wk GA: 5.5%

Etiology
- most cases idiopathic
- anencephalic fetus with no pituitary gland
- placental sulfatase deficiency (X-linked recessive condition in 1/2000-1/6000 infants) – rare

Management (for singleton, cephalic fetus, otherwise uncomplicated)
- GA 39 wk with advanced maternal age (>40 y): consideration should be given to IOL due to increased risk of stillbirth
- GA 40-41 wk: expectant management
  - no evidence to support IOL or C/S unless other risk factors for morbidity are present (see prognosis)
- GA >41 wk: offer IOL if vaginal delivery is not contraindicated
  - IOL shown to decrease C/S, fetal heart rate changes, meconium staining, macrosomia, and death when compared with expectant management
- GA >41 wk and expectant management elected: serial fetal surveillance
  - fetal movement count by the mother
  - BPP q3-4d
- if AFI is decreased, labour should be induced

Prognosis
- if >42 wk, perinatal mortality 2-3x higher (due to progressive uteroplacental insufficiency)
- with increasing GA, higher rates of: intrauterine infection, asphyxia, meconium aspiration syndrome, placental insufficiency, placental aging and infarction, macrosomia, dystocia, fetal distress, operative deliveries, pneumonia, seizures, requirement of NICU admission, stillbirth
- morbidity increased with HTN in pregnancy, DM, abruption, IUGR, and multiple gestation

Intrauterine Fetal Demise

Definition
- fetal demise in utero after 20 wk GA (before 20 wk GA called spontaneous abortion)

Epidemiology
- occurring in 1% of pregnancies

Etiology
- 50% idiopathic
- 50% secondary to HTN, DM, erythroblastosis fetalis, congenital anomalies, umbilical cord or placental complications, intrauterine infection, and APS

Clinical Features
- decreased perception of fetal movement by mother
- SFH and maternal weight not increasing
- absent fetal heart tones on Doppler (not diagnostic)
- high MSAFP
- on U/S: no fetal heart rate. Depending on timing of death, may see skull collapse, brain tissue retraction, empty fetal bladder, non-filled aorta, or poor visualization of midline flax

Management
- diagnosis: absent cardiac activity and fetal movement on U/S (required)
- determine secondary cause
  - maternal: HbA1c, fasting glucose, TSH, Kleihauer-Betke, VDRL, ANA, CBC, anticardiolipins, antibody screens, INR/PTT, serum/urine toxicology screens, cervical and vaginal cultures, and TORCH screen
  - fetal: karyotype, cord blood, skin biopsy, genetics evaluation, autopsy, amniotic fluid culture for CMV, parvovirus B19, and herpes
  - placenta: pathology, bacterial cultures

Treatment
- <12 wk: dilation and curettage
- 13-20 wk: dilation and evacuation or sometimes IOL
- >20 wk: IOL
- monitor for maternal coagulopathy (10% risk of DIC)
- parental psychological care/bereavement support as per hospital protocol
- comprehensive discussion within 3 mo about final investigation and post-mortem results, help make plans for future pregnancies

DIC: Generalized Coagulation and Fibrinolysis Leading to Depletion of Coagulation Factors

Obstetrical Causes
- Abruption
- Gestational HTN
- Fetal demise
- PPH

DIC-specific Blood Work
- Platelets
- aPTT and PT
- FDP
- Fibrinogen

Treatment
- Treat underlying cause
- Supportive
- Fluids
- Blood products
- FFP, platelets, cryoprecipitate
- Consider anti-coagulation as VTE prophylaxis

Intrauterine Growth Restriction

Definition
- estimated fetal weight <10th percentile for GA on U/S, has not reached biologically determined growth potential

Etiology/Risk Factors
- 50% unknown
- maternal causes
  - malnutrition, smoking, drug abuse, alcoholism, cyanotic heart disease, type 1 DM, SLE, pulmonary insufficiency, previous IUGR (25% risk, most important risk factor), and chronic HTN
- maternal
  - gestational HTN, chronic renal insufficiency, prolonged gestation, substance abuse, and poor nutrition
- placental
  - any disease that causes placental insufficiency
  - gross placental morphological abnormalities (infarction, hemangiomas, placenta previa, ansd abnormal cord insertion)
- fetal causes
  - TORCH infections, multiple gestation, and congenital anomalies/chromosomal abnormalities (10%)

Clinical Features
- symmetric/type I (25-30%): occurs early in pregnancy
  - reduced growth of both head and abdomen
  - head:abdomen ratio may be normal (>1 up to 32 wk; =1 at 32-34 wk; <1 after 34 wk GA)
  - usually associated with congenital anomalies or TORCH infections
- asymmetric/type II (70%): occurs late in pregnancy
  - fetal abdomen is disproportionately smaller than fetal head
  - brain is spared; therefore head:abdomen ratio increased
  - usually associated with placental insufficiency
  - more favourable prognosis than type I

TORCH
Toxoplasmosis
Others: e.g. syphilis
Rubella
CMV
HSV
• See Table 15, OB29

- complications
  - prone to meconium aspiration, asphyxia, polycythemia, hypoglycemia, hypocalcemia, hypophosphatemia, hyponatremia, and mental retardation
  - greater risk of perinatal morbidity and mortality

Investigations
- SFH measurements at every antepartum visit (ensure accurate GA)
- if mother at high risk or SFH lags >2 cm behind GA
  - U/S for biparietal diameter, head and abdominal circumference ratio, femur length, fetal weight, AFV (decrease associated with IUGR), and decrease in the rate of growth
  - ± BPP
  - Doppler analysis of umbilical cord blood flow

Management
- prevention via risk modification prior to pregnancy is ideal
- modify controllable factors: smoking, alcohol, nutrition, and treat maternal illness
- serial BPP (monitor fetal growth) and determine cause of IUGR, if possible
- delivery when extrauterine existence is less dangerous than continued intrauterine existence (abnormal function tests, absent growth, severe oligohydramnios) especially if GA >34 wk
- as IUGR fetuses are less likely to withstand stresses of labour, they are more likely to be delivered by Cesarean section

Macrosomia

Definition
- infant weight ≥90th percentile for a particular GA or >4000 g

Etiology/Risk Factors
- maternal obesity, GDM, past history of macrosomic infant, prolonged gestation, multiparity

Clinical Features
- increased risk of perinatal mortality
- CPD and birth injuries (shoulder dystocia, fetal bone fracture) more common
- complications of DM in labour (see Table 14, OB28)

Investigations
- serial SFH
- further investigations if mother at high risk or SFH >2 cm ahead of GA
- U/S predictors
  - polyhydramnios
  - T3 abdominal circumference >1.5 cm/wk
  - head circumference/abdominal circumference ratio <10th percentile
  - femur length/abdominal circumference ratio <20th percentile

Management
- prevent hyperglycemia in women with DM, optimize pre-pregnancy weight, and limit pregnancy weight gain
- prophylactic C/S is a reasonable option where EFW >5000 g in non-diabetic woman and EFW >4500 g in diabetic woman
- risks and benefits of early induction (risk of C/S vs. risk of dystocia) must be weighed in diabetic mothers, need for person-centred and shared decision-making

Polyhydramnios/Oligohydramnios

Table 11. Polyhydramnios and Oligohydramnios

	Polyhydramnios	Oligohydramnios
Definition	AFI >25 cm U/S: single deepest pocket >8 cm	AFI <5 cm U/S: single deepest pocket ≤2 cm
Etiology	Idiopathic most common Maternal Type 1 DM: abnormalities of transchorionic flow Maternal-fetal Chorioangiomas Multiple gestation Fetal hydrops (increased erythroblastosis) Fetal Chromosomal anomaly (up to 2/3 of fetuses have severe polyhydramnios) Respiratory: cystic adenomatoid malformed lung CNS: anencephaly, hydrocephalus, meningocele GI: tracheoesophageal fistula, duodenal atresia, facial clefts (interfere with swallowing)	Idiopathic most common Maternal Uteroplacental insufficiency (preeclampsia, nephropathy) Medications (ACEI) Fetal Congenital urinary tract anomalies (renal agenesis, obstruction, posterior urethral valves) Demise/chronic hypoxemia (blood shunt away from kidneys to perfuse brain) IUGR Ruptured membranes: prolonged amniotic fluid leak Amniotic fluid normally decreases after 35 wk
Epidemiology	Occur in 0.2-1.6% of all pregnancies	Occur in ~4.5% of all pregnancies Severe form in <0.7% Common in pregnancies >41 wk (~12%)
Clinical Features and Complications	Uterus large for dates, difficulty palpating fetal parts and hearing FHR Maternal complications Pressure symptoms from overdistended uterus (dyspnea, edema, hydronephrosis) Obstetrical complications Cord prolapse, placental abruption, malpresentation, preterm labour, uterine dysfunction, and PPH	Uterus small for dates Fetal complications 15-25% have fetal anomalies Amniotic fluid bands (T1) can lead to Potter's facies, limb deformities, abdominal wall defects Obstetrical complications Cord compression Increased risk of adverse fetal outcomes Pulmonary hypoplasia (late-onset) Marker for infants who may not tolerate labour well
Management	Determine underlying cause Screen for maternal disease/infection Complete fetal U/S evaluation Depends on severity Mild to moderate cases require no treatment If severe, hospitalize and consider therapeutic amniocentesis	Always warrants admission and investigation Rule out ROM Fetal monitoring (NST, BPP) U/S Doppler studies (umbilical cord and uterine artery) Maternal hydration with oral or IV fluids to help increase amniotic fluid Injection of fluid via amniocentesis will improve condition for ~1 wk – may be most helpful for visualizing any associated fetal anomalies Consider delivery if term Amnio-infusion may be considered during labour via intrauterine catheter
Prognosis	2-5 fold increase in risk of perinatal mortality	Poorer with early onset High mortality related to congenital malformations and pulmonary hypoplasia when diagnosed during T2

Multi-Fetal Gestation and Malpresentation

Epidemiology

- incidence of twins is 1/80 and triplets 1/6400 in North America
- 2/3 of twins are dizygotic (fraternal)
  - risk factors for dizygotic twins: IVF, increased maternal age, newly discontinued OCP, and ethnicity (e.g. certain African regions)
- monozygous twinning occurs at a constant rate worldwide (1/250)
- determine zygosity by number of placentas, thickness of membranes, sex, and blood type

Clinical Features

Table 12. Complications Associated with Multiple Gestation

Maternal	Uteroplacental	Fetal
Hyperemesis gravidarum GDM Gestational HTN Anemia Increased physiological stress on all systems Increased compressive symptoms C/S	Increased PROM/PTL Polyhydramnios Placenta previa Placental abruption PPH (uterine atony) Umbilical cord prolapse Cord anomalies (velamentous insertion, 2 vessel cord)	Prematurity IUGR Malpresentation Congenital anomalies Twin-twin transfusion Increased perinatal morbidity and mortality Twin interlocking (twin A breech, twin B vertex) Single fetal demise

Management
- U/S determination of chorionicity must be done within T1 (ideally 8-12 wk GA)
- increased antenatal surveillance
  - serial U/S q3-4wk from 22 wk GA to assess growth (uncomplicated diamniotic dichorionic)
  - increased frequency of U/S in monochorionic diamniotic and monochorionic monoamniotic twins
  - Doppler flow studies weekly if discordant fetal growth (>30%)
  - BPP as needed
- may attempt vaginal delivery if twin A presents as vertex, otherwise C/S (40-50% of all twin deliveries, 10% of cases have twin A delivered vaginally and twin B delivered by C/S)
- mode of delivery depends on fetal weights, GA, and presentation

The Ps of Multiple Gestation Complications

Increased rates of
Puking
Pallor (anemia)
Preeclampsia/**P**IH
Pressure (compressive symptoms)
PTL/**P**ROM/**PP**ROM
Polyhydramnios
Placenta previa/abruption
PPH/A**P**H
Prolonged labour
Cord **p**rolapse
Prematurity
Mal**p**resentation
Perinatal morbidity and mortality
Parental distress
Postpartum depression

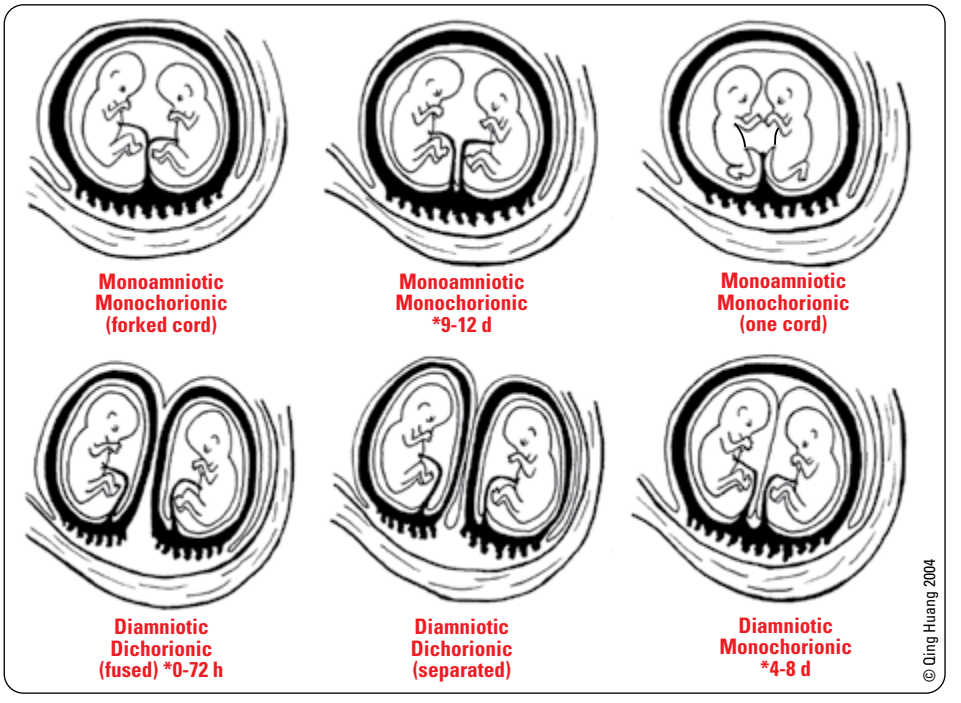


Figure 4. Classification of twin pregnancies ***Indicates time of cleavage**

Twin-Twin Transfusion Syndrome

Definition
- formation of placental intertwin vascular anastomoses causes arterial blood from donor twin to pass into veins of the recipient twin

Epidemiology
- 10% of monochorionic twins
- concern if >30% discordance in estimated fetal weight

Clinical Features
- donor twin: IUGR, hypovolemia, hypotension, anemia, and oligohydramnios
- recipient twin: hypervolemia, HTN, CHF, polycythemia, edema, polyhydramnios, and kernicterus in neonatal period

Investigations
- detected by U/S screening, Doppler flow analysis

Management
- therapeutic serial amniocentesis to decompress polyhydramnios of recipient twin and decrease pressure in cavity and on placenta
- intrauterine blood transfusion to donor twin if necessary
- laparoscopic occlusion of placental vessels
- fetoscopic laser ablation of placental vascular anastomoses when indicated and if available

Breech Presentation

Definition
- fetal buttocks or lower extremity is the presenting part as determined on U/S
- complete (10%): hips and knees both flexed
- frank (60%): hips flexed, knees extended, buttocks present at cervix
  - most common type of breech presentation
  - most common breech presentation to be delivered vaginally
- incomplete (30%): both or one hip partially flexed and both or one knee present below the buttocks, feet or knees present first (footling breech, kneeling breech)

Epidemiology
- occurs in 3-4% of pregnancies at term (25% <28 wk)

Risk Factors
- maternal: pelvis (contracted), uterus (shape abnormalities, intrauterine tumours, fibroids, previous breech), pelvic tumours causing compression, and grand multiparity
- placental: placenta previa
- fetal: prematurity, amniotic fluid (poly-/oligohydramnios), multiple gestation, congenital malformations (found in 6% of breeches; 2-3% if in vertex presentations), abnormalities in fetal tone and movement, aneuploidy, hydrocephalus, and anencephalus

Management
- ECV (external cephalic version): repositioning of singleton fetus within uterus under U/S guidance
  - overall success rate of ~60%
  - criteria: >36 wk GA, singleton, unengaged presenting part, reactive NST, not in labour
  - absolute contraindications: where C/S is required (placenta previa, previous classical C/S), previous myomectomy, PROM, uteroplacental insufficiency, nuchal cord, non-reactive NST, multiple gestation
  - relative contraindications: mild/moderate oligohydramnios, suspected IUGR, HTN, previous T3 bleed
  - risks: abruption, cord compression, cord accident, ROM, labour, fetal bradycardia requiring C/S (<1% risk), alloimmunization, fetal death (1:5000)
  - method: tocometry, followed by U/S guided transabdominal manipulation of fetus with constant fetal heart monitoring
  - if patient Rh negative, give Rhogam® after the procedure
  - better prognosis if multiparous, good fluid volume, small baby, skilled obstetrician, and posterior placenta
- pre-or early-labour U/S to assess type of breech presentation, fetal growth, estimated weight, placenta position, attitude of fetal head (flexed is preferable); if U/S unavailable, recommend C/S
- ECV and elective C/S should be presented as options with the risks and benefits outlined; obtain informed consent
- method for vaginal breech delivery
  - encourage effective maternal pushing efforts
  - at delivery of head (after feet), assistant must apply suprapubic pressure to flex and engage fetal head
  - delivery can be spontaneous or assisted; avoid fetal traction
  - apply fetal manipulation only after spontaneous delivery to level of umbilicus
- contraindications to vaginal breech delivery
  - cord presentation
  - clinically inadequate maternal pelvis
  - fetal factors incompatible with vaginal delivery (e.g. hydrocephalus, macrosomia, fetal growth restriction)
- C/S recommended if: the breech has not descended to the perineum in the second stage of labour after 2 h, in the absence of active pushing, or if vaginal delivery is not imminent after 1 h of active pushing

Prognosis
- regardless of route of delivery, breech infants have lower birth weights and higher rates of perinatal mortality, congenital anomalies, abruption, and cord prolapse

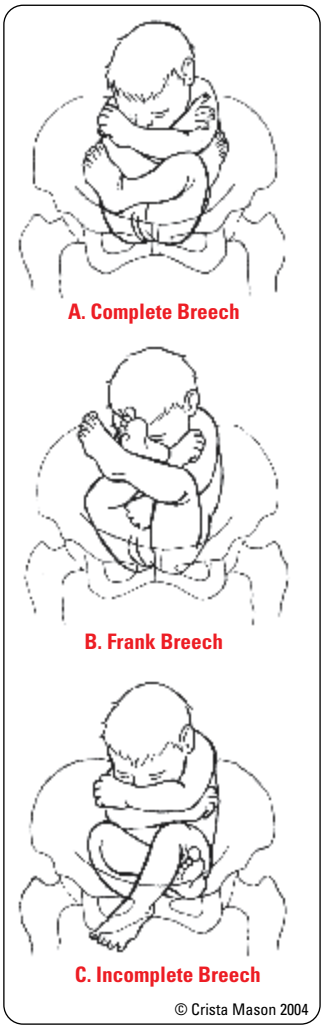


Figure 5. Types of breech presentation

Criteria for Vaginal Breech Delivery
- Frank or complete breech, GA >36 wk
- EFW 2500-3800 g based on clinical and U/S assessment (5.5–8.5 lb)
- Fetal head flexed
- Continuous fetal monitoring
- 2 experienced obstetricians, assistant, and anesthetist present
- Ability to perform emergency C/S within 30 min if required

Hypertensive Disorders of Pregnancy

Hypertension in Pregnancy

- hypertensive disorders of pregnancy are classified as either pre-existing or gestational HTN. Pre-eclampsia and eclampsia are included in the spectrum of hypertensive disorders of pregnancy

Ominous Symptoms of HTN in Pregnancy
RUQ pain, headache, and visual disturbances

PRE-EXISTING HYPERTENSION

Definition
- BP ≥140/90 prior to 20 wk GA; BP should be elevated on ≥2 occasions at least 15 minutes apart
- essential HTN is associated with an increased risk of gestational HTN, abruptio placentae, IUGR, and IUFD

GESTATIONAL HTN

Definition
- sBP ≥140 or dBP ≥90 after 20 wk GA without proteinuria in a woman known to be normotensive before pregnancy

PREECLAMPSIA
Definition
- pre-existing or gestational HTN with new onset proteinuria or adverse conditions (end organ dysfunction)

ECLAMPSIA
Definition
- the occurrence of ≥1 generalized convulsions and/or coma in the setting of preeclampsia and in the absence of other neurologic conditions

Note
Eclampsia prior to 20 wk of gestation is rare and should raise the possibility of an underlying molar pregnancy or antiphospholipid syndrome

Epidemiology of Eclampsia
- an eclamptic seizure occurs in approximately 0.5% of mildly preeclamptic women and 2-3% of severely preeclamptic women

Clinical Manifestation of Eclampsia
- eclampsia is a clinical diagnosis
- typically tonic-clonic and lasting 60-75 s
- symptoms that may occur before the seizure include persistent frontal or occipital headache, blurred vision, photophobia, right upper quadrant or epigastric pain, and altered mental status
- in up to one third of cases, there is no proteinuria or blood pressure >140/90 mmHg prior to the seizure
- in general, women with typical eclamptic seizures who do not have focal neurologic deficits or prolonged coma do not require diagnostic evaluation including imaging

Risk Factors for Hypertensive Disorders in Pregnancy
- maternal factors
  - primigravida (80-90% of gestational HTN), first conception with a new partner, PMHx or FHx of gestational HTN, or preeclampsia/eclampsia
  - DM, chronic HTN, or renal insufficiency
  - obesity
  - antiphospholipid syndrome or inherited thrombophilia
  - extremes of maternal age (<18 or >35 yr)
  - previous stillbirth or IUFD
  - vascular or connective tissue disease
- fetal factors
  - IUGR or oligohydramnios
  - GTN
  - multiple gestation
  - fetal hydrops "mirror syndrome"
  - abruptio placentae

Hypertension in Pregnancy

Adverse Maternal Conditions
- sBP >160 mmHg
- dBP >100 mmHg
- HELLP
- Cerebral hemorrhage
- Renal dysfunction: oliguria <500 mL/d
- Left ventricular failure, pulmonary edema
- Placental abruption, DIC
- **Symptoms**
  - Abdominal pain, N/V
  - Headaches, visual problems
  - SOB, chest pain
  - Eclampsia: convulsions

Adverse Fetal Conditions
- IUGR
- Oligohydramnios
- Absent/reversed umbilical artery end diastolic flow
- **Can result in:**
  - Fetal disability and/or death

Clinical Evaluation of Hypertensive Disorders in Pregnancy
- in general, clinical evaluation should include the mother and fetus
- evaluation of mother:
  - body weight
  - central nervous system
    - presence and severity of headache
    - visual disturbances – blurring, scotomata

        - tremulousness, irritability,and somnolence
        - hyperreflexia
    - hematologic
        - bleeding, petechiae
    - hepatic
        - RUQ or epigastric pain
        - severe N/V
    - renal
        - urine output and colour
- evaluation of fetus:
    - fetal movement
    - fetal heart rate tracing – NST
    - U/S for growth
    - BPP
    - Doppler flow studies

Laboratory Evaluation of Hypertensive Disorders in Pregnancy

- CBC
- PTT, INR, fibrinogen – if abnormal LFTs or bleeding
- ALT, AST
- creatinine, uric acid
- 24 h urine collection for protein or albumin:creatinine ratio
- may consider placental growth factor (PlGF) testing as an early screening test for suspected preeclampsia

Complications of Hypertensive Disorders in Pregnancy

- maternal
    - liver and renal dysfunction
    - seizure - "eclampsia"
    - abruptio placentae
    - left ventricular failure/pulmonary edema
    - DIC (release of placental thromboplastin consumptive coagulopathy)
    - HELLP syndrome
    - hemorrhagic stroke (50% of deaths)
- fetal (2° to placental insufficiency)
    - IUGR, prematurity, abruptio placentae, IUFD

Management of HTN

- for non-severe HTN (149-159/90-105) target a BP of 130-155/80-105 in women without comorbidities or <140/90 in women with comorbidities
- for both pre-existing and gestational HTN, labetalol 100-400 mg PO bid-tid, nifedipine XL preparation 20-60 mg PO od, or α-methyldopa 250-500 mg PO bid-qid
- for severe HTN (BP>160/110), give one of:
    - labetalol 20 mg IV then 20-80 mg IV q30min (max 300 mg) (then switch to oral)
    - nifedipine 5-10 mg capsule q30min
        - hydralazine 5 mg IV, repeat 5-10 mg IV q30min or 0.5 to 10 mg/h IV, to a maximum of 20 mg IV (or 30 mg IM)
- no ACEI, ARBs, diuretics (in cases of pulmonary edema or cardiac failure, may be used), prazosin, or atenolol
- pre-existing HTN and gestational HTN without any deterioration can be followed until 37 wk, then decide to induce shortly thereafter

Management of Preeclampsia

- if stable and no adverse factors (GA 24-33+6 wk), expectant management, ± delivery as approaching 34-36 wk (must weigh risks of fetal prematurity vs. risks of developing severe preeclampsia/eclampsia)
- antenatal corticosteroids should be considered if GA ≤ 34 wk
- if >37 wk, immediate delivery is recommended
- for severe preeclampsia, stabilize and deliver, regardless of GA
- if severe preeclampsia during labour, increase maternal monitoring: hourly input and output, urine dip q12h, hourly neurological vitals, and increase fetal monitoring (continuous FHR monitoring)
- antihypertensive therapy
    - labetalol 20 mg IV, then 20-80 mg IV q30min (max 300 mg) (then switch to oral)
    - nifedipine 5-10 mg capsule q30min
        - hydralazine 5 mg IV, repeat 5-10 mg IV q30min or 0.5-10 mg/h IV, to a maximum of 20 mg IV (or 30 mg IM)

I-A Evidence-Recommendation Highlights of SOGC Clinical Practice Guidelines Diagnosis, Evaluation, and Management of the Hypertensive Disorders of Pregnancy
J Obstet Gynaecol Can 2014;36(5):416-38

- For BP measurement, Korotkoff phase V should be used to designate the dBP.
- Calcium supplementation (of at least 1g/d, orally) is recommended for women with low dietary intake of calcium (<600 mg/d). (I-A)
- For preeclampsia prevention among increased risk women, low-dose ASA (75-100 mg/d) is recommended until delivery.
- Umbilical artery Doppler velocimetry should be part of the antenatal fetal surveillance in preeclampsia.
- Initial antihypertensive therapy for severe HTN (sBP >160 or dBP ≥110) should be with labetalol, nifedipine, or hydralazine.
- Initial antihypertensive therapy for non-severe HTN (BP 140-159/90-109 mmHg) should be with methyldopa, β-blockers, or calcium channel blockers.
- Antenatal corticosteroids for fetal lung maturation should be considered for all women with preeclampsia before 34 wk gestation.
- In a planned vaginal delivery with an unfavourable cervix, cervical ripening should be used.
- Oxytocin 5 units IV or 10 units IM should be used as part of the management during the third stage of labour, particularly in the presence of thrombocytopenia or coagulopathy.
- Magnesium sulfate is the recommended first-line treatment for eclampsia.
- Magnesium sulfate is the recommended eclampsia prophylaxis in severe preeclampsia.

Preeclampsia Investigations

- CBC
- AST, ALT
- INR and aPTT (if abn LFTs or bleeding)
- Cr
- Urine (24 h protein collection or albumin/creatinine ratio)
- Uric acid

- seizure prevention
  - magnesium sulfate: 4 g IV loading dose, followed by 1g/h
  - postpartum management
  - risk of seizure highest in first 24 h postpartum – continue $MgSO_4$ for 12-24 h after delivery
  - vitals q1h
  - consider HELLP syndrome
  - most return to a normotensive BP within 2 wk

Management of Eclampsia

- ABCs
- roll patient into LLDP
- supplemental O_2 via face mask to treat hypoxemia due to hypoventilation during convulsive episode
- aggressive antihypertensive therapy for sustained dBP ≥105 mmHg or sBP ≥160 mmHg with hydralazine or labetalol
- prevention of recurrent convulsions: to prevent further seizures and the possible complications of repeated seizure activity (e.g. rhabdomyolysis, metabolic acidosis, aspiration pneumonitis, etc.)
- $MgSO_4$ is now the drug of choice
- the definitive treatment of eclampsia is DELIVERY, irrespective of gestational age, to reduce the risk of maternal morbidity and mortality from complications of the disease
- mode of delivery is dependent on clinical situation and fetal-maternal condition

HELLP Syndrome
Hemolysis
Elevated **L**iver Enzymes
Low Platelets

Differential Diagnosis of Cause for Seizure in a Pregnant Woman
- Stroke
- Hypertensive disease (hypertensive encephalopathy, pheochromocytoma)
- Space-occupying lesion of the CNS
- Metabolic disorders (hypoglycemia, SIADH)
- Infection (meningitis, encephalitis)
- TTP or thrombophilia
- Idiopathic epilepsy
- Use of illicit drugs
- Cerebral vasculitis

Medical Complications of Pregnancy

Iron and Folate Deficiency Anemia

Table 13. Iron Deficiency and Folate Deficiency Anemia

	Iron Deficiency Anemia	Folate Deficiency Anemia
Etiology	See Hematology, H15	See Hematology, H25
Epidemiology	Responsible for 80% of non-physiologic anemia during pregnancy	Incidence varies from 0.5-25% depending on region, population, and diet
Clinical Features	See Hematology, H15	See Hematology, H25
Investigations	See Hematology, H15	See Hematology, H25
Management	Prevention (non-anemic): 30 mg elemental iron/d (met by most prenatal vitamins) Treatment (anemic): 30-120 mg elemental iron/d 325 mg ferrous fumarate = 106 mg elemental Fe; 325 mg ferrous sulfate = 65 mg elemental Fe; 325 mg ferrous gluconate = 36 mg elemental Fe Polysaccharide-Iron Complex = 150 mg elemental Fe/capsule	Prevention: 0.4-1 mg folic acid PO daily for 1-3 mo preconceptually and throughout T1, or 5 mg folic acid/d with past history of ONTD, DM, or antiepileptic medication use
Complications	Maternal: angina, CHF, infection, slower recuperation, preterm labour Fetal: decreased oxygen carrying capacity leading to fetal distress, IUGR, and low birth weight	Maternal: decreased blood volume, N/V, and anorexia Fetal: neural tube defects in T1, low birth weight, and prematurity
Notes	Mother needs 1 g of elemental iron per fetus; this amount exceeds normal stores + dietary intake Iron requirements increase during pregnancy due to fetal/placental growth (500 mg), increased maternal RBC mass (500 mg), and losses (200 mg) – more needed for multiple gestations	Minimum daily requirement is 0.4 mg Most often associated with iron deficiency anemia Folic acid is necessary for closure of neural tube during early fetal development (by day 28 of gestation)

Diabetes Mellitus

Epidemiology

- 2-4% of pregnancies are complicated by DM

Classification of Diabetes Mellitus

- type 1 and type 2 DM (see Endocrinology, E7)
- GDM: onset of DM during pregnancy (usually tested for around 24-28 wk GA)

Etiology

- type 1 and type 2 DM
- GDM: anti-insulin factors produced by placenta and high maternal cortisol levels create increased peripheral insulin resistance → leading to GDM and/or exacerbating pre-existing DM

MANAGEMENT

A. TYPE 1 and TYPE 2 DM

Preconception
- pre-plan and refer to high-risk clinic
- commence folic acid 3 mo prior
- optimize glycemic control (HbA1c <6%)
- counsel patient on potential risks and complications
- evaluate for diabetic retinopathy, neuropathy, and CAD

Pregnancy
- for Type 2 DM, if already on oral medication, generally switch to insulin therapy
  - continuing glyburide or metformin controversial
  - teratogenicity unknown for other oral anti-hyperglycemics
- tight glycemic control
  - insulin dosage may need to be adjusted in T2 due to increased demand and increased insulin resistance
- monitor as for normal pregnancy, plus initial 24 h urine protein and creatinine clearance, retinal exam, and HbA1c
  - HbA1c: >140% of pre-pregnancy value associated with increased risk of spontaneous abortion and congenital malformations
- increased fetal surveillance (fetal growth, BPP, NST) starting in the late T2 and T3, consider fetal ECHO in the T2 (if high HbA1c in T1 or just prior to pregnancy) to look for cardiac abnormalities

Monitoring Glucose Levels
- Frequent measurements of blood glucose during pregnancy are advised for women with type 1 or 2 DM to help prevent or treat both hypoglycemia and hyperglycemia, and also improves neonatal outcome
- Aim for:
  - FPG ≤5.3 mmol/L (95 mg/dL)
  - 1 h post prandial PG ≤7.8 mmol/L (140 mg/dL), 2 h post prandial PG ≤6.7 mmol/L (120 mg/dL)
- Most women can be followed with monthly HbA1c determinations

Labour
- timing of delivery depends on fetal and maternal health and risk factors (i.e. must consider size of baby, lung maturity, maternal blood glucose)
- induce by 38-39 wk, depending on glycemic control and presence of end-organ involvment
- type of delivery
  - increased risk of cephalopelvic disproportion (CPD) and shoulder dystocia with babies >4000 g (8.8 lbs)
  - consider elective C/S for predicted birthweight >4500 g (9.9 lbs) (controversial)
- monitoring
  - during labour, monitor blood glucose q1h with patient on insulin and dextrose drip
  - aim for blood glucose between 3.9-7 mmol/L to reduce the risk of neonatal hypoglycemia

Post-prandial blood glucose values seem to be the most effective at determining the likelihood of macrosomia or other adverse pregnancy outcomes

Postpartum
- insulin requirements dramatically drop with expulsion of placenta (source of insulin antagonists)
- monitor glucose q6h, restart insulin at two-thirds of pre-pregnancy dosage when glucose >8 mmol/L

B. GESTATIONAL DM

Screening and Diagnosis
- all pregnant women between 24-28 wk GA (or at any stage if high risk)
- 2 screening options
  - 2-step screening (recommended by the Canadian Diabetes Association)
    - Step 1: perform a random non-fasting 50 g OGCT
      - 1 h PG <7.8 mmol/L is normal
      - 1 h PG ≥11.1 mmol/L is GDM
      - if 1 h PG 7.8-11.0 mmol/L, proceed to Step 2
    - Step 2: perform a fasting 75 g OGTT, GDM if ≥1 of:
      - FPG ≥5.3 mmol/L
      - 1 h PG ≥10.6 mmol/L
      - 2 h PG ≥9.0 mmol/L
  - Alternative 1-step screening with fasting 75 g OGTT; GDM if ≥1 of:
    - FPG ≥5.1 mmol/L
    - 1 h PG ≥10.0 mmol/L
    - 2 h PG ≥8.5 mmol/L

Risk Factors for GDM
- Age >25 yr
- Obesity
- Ethnicity (Aboriginal, Hispanic, Asian, and African)
- FHx of DM
- Previous history of GDM
- Previous child with birthweight >4.0 kg
- Polycystic ovarian syndrome
- Current use of glucocorticoids
- Essential HTN or pregnancy-related HTN

Management
- first line: diet modification and increased physical activity
- initiate insulin therapy if glycemic targets not achieved within 2 wk of lifestyle modification alone
- glycemic targets: FPG <5.3 mmol/L, 1 h PG <7.8 mmol/L, 2 h PG <6.7 mmol/L
- oral agents can be used in pregnancy but is off-label and should be discussed with patient
- stop insulin and diabetic diet postpartum
- follow-up with 75 g OGTT between 6 wk-6 mo postpartum, counsel about lifestyle modifications

Prognosis
- most maternal and fetal complications are related to hyperglycemia and its effects

Table 14. Complications of DM in Pregnancy

Maternal	Fetal
Obstetric HTN/preeclampsia (especially if pre-existing nephropathy/proteinuria): insulin resistance is implicated in etiology of HTN Polyhydramnios: maternal hyperglycemia leads to fetal hyperglycemia, which leads to fetal polyuria (a major source of amniotic fluid)	**Growth Abnormalities** Macrosomia: maternal hyperglycemia leads to fetal hyperinsulinism resulting in accelerated anabolism IUGR: due to placental vascular insufficiency
Diabetic Emergencies Hypoglycemia Ketoacidosis Diabetic coma	**Delayed Organ Maturity** Fetal lung immaturity: hyperglycemia interferes with surfactant synthesis (respiratory distress syndrome)
End-Organ Involvement or Deterioration (occur in type 1 DM and type 2 DM, not in GDM) Retinopathy Nephropathy	**Congenital Anomalies (occur in type 1 DM and type 2 DM, not in GDM)** 2-7x increased risk of cardiac (ventricular septal defect), NTD, GU (cystic kidneys), GI (anal atresia), and MSK (sacral agenesis) anomalies due to hyperglycemia Note: Pregnancies complicated by GDM do not manifest an increased risk of congenital anomalies because GDM develops after the critical period of organogenesis (in T1)
Other Pyelonephritis/UTI: glucosuria provides a culture medium for *E. coli* and other bacteria Increased incidence of spontaneous abortion (in type 1 DM and type 2 DM, not in GDM): related to pre-conception glycemic control	**Labour and Delivery** Preterm labour/prematurity: most commonly in patients with HTN/preeclampsia Preterm labour is associated with poor glycemic control but the exact mechanism is unknown Increased incidence of stillbirth Birth trauma: due to macrosomia, can lead to difficult vaginal delivery and shoulder dystocia **Neonatal** Hypoglycemia: due to pancreatic hyperplasia and excess insulin secretion in the neonate Hyperbilirubinemia and jaundice: due to prematurity and polycythemia Hypocalcemia: exact pathophysiology not understood, may be related to functional hypoparathyroidism Polycythemia: hyperglycemia stimulates fetal erythropoietin production

Long-Term Maternal Complications
- type 1 and type 2 DM: risk of progressive retinopathy and nephropathy
- GDM: 50% risk of developing type 2 DM in next 20 yr

Early-Onset Group B *Streptococcus*

Epidemiology
- 15-40% recto-vaginal carrier rate

Risk Factors (for neonatal disease)
- maternal intrapartum GBS colonization during current pregnancy
- GBS bacteria at any time during the current pregnancy
- previous infant with invasive GBS disease
- prolonged rupture of membranes ≥18 h
- maternal fever (temperature ≥38°C)

Clinical Features
- not harmful to mother
- risk of vertical transmission (neonatal sepsis, meningitis or pneumonia, and death)

Investigations
- offer screening to all women at 35-37 wk with vaginal and anorectal swabs for GBS culture

Treatment
- prophylactic treatment of maternal GBS at delivery decreases neonatal morbidity and mortality
- indications for antibiotic prophylaxis: positive GBS screen, GBS in urine, previous infant with GBS disease, or GBS status unknown + one of the other risk factors
- antibiotics for GBS prophylaxis (should be given 4 h prior to delivery to be considered adequate)
  - penicillin G, 5 million IU IV, then 2.5 million IU IV q4h until delivery
  - penicillin allergic but not at risk for anaphylaxis: cefazolin 2 g IV then 1 g q8h
  - penicillin allergic and at risk of anaphylaxis: vancomycin 1 g IV q12h until delivery (vancomycin and clindamycin levels in amniotic fluid do not reach therapeutic levels, all babies should be screened for GBS despite treatment)
- if maternal fever, broad spectrum antibiotic coverage is advised

Indications for Intrapartum Antibiotic GBS Prophylaxis
Centres for Disease Control and Prevention. Prevention of Perinatal Group B Streptococcal Disease. MMWR 2010;59(RR-10):14
- Previous infant with invasive GBS disease
- GBS bacteriuria during any trimester of the current pregnancy
- Positive GBS vaginal-rectal screening culture in late gestation during current pregnancy
- Unknown GBS status at the onset of labour (culture not done, incomplete, or results unknown) and any of the following:
  - Delivery at <37 wk gestation
  - Amniotic membrane rupture ≥18 h
  - Intrapartum temperature ≥38.0 °C (≥100.4 °F)
  - Intrapartum nucleic-acid amplification test positive for GBS

Urinary Tract Infection

Epidemiology
- most common medical complication of pregnancy
- asymptomatic bacteriuria in 2-7% of pregnant women, more frequently in multiparous women
- note: asymptomatic bacteriuria should be treated in pregnancy due to increased risk of pyelonephritis and preterm labour

Treat asymptomatic bacteriuria in pregnancy because of increased risk of progression to cystitis, pyelonephritis, and probable increased risk of preterm labour

Etiology
- increased urinary stasis from mechanical and hormonal (progesterone) factors
- organisms include GBS as well as those that occur in non-pregnant women

Clinical Features
- may be asymptomatic
- dysuria, urgency, and frequency in cystitis
- fever, flank pain, and costovertebral angle tenderness in pyelonephritis

Investigations
- urinalysis, urine C&S
- cystoscopy and renal function tests in recurrent infections

Management
- uncomplicated UTI
  - first line: amoxicillin (250-500 mg PO q8h x 7 d)
  - alternatives: nitrofurantoin (100 mg PO bid x 7 d) or cephalosporins
  - follow with monthly urine cultures
- pyelonephritis
  - hospitalization and IV antibiotics

Prognosis
- complications if untreated: acute cystitis, pyelonephritis, and possible preterm labour
- recurrence is common

Infections During Pregnancy

Table 15. Infections During Pregnancy

Infection	Agent	Source of Transmission	Greatest Transmission Risk to Fetus	Effects on Fetus	Effects on Mother	Diagnosis	Management
Chicken Pox	Varicella zoster virus (herpes family)	To mother: direct, respiratory To baby: transplacental	13-30 wk GA, and 5 d pre- to 2 d post-delivery	Congenital varicella syndrome (limb aplasia, chorioretinitis, cataracts, cutaneous scars, cortical atrophy, IUGR, hydrops), preterm labour	Fever, malaise, vesicular pruritic lesions	Clinical, ± vesicle fluid culture, ± serology	Varicella-zoster immune globulin for mother if exposed, decreases congenital varicella syndrome Note: do not administer vaccine during pregnancy (live attenuated vaccine)
***Cytomegalovirus**	DNA virus (herpes family)	To mother: blood/ organ transfusion, sexual contact To baby: transplacental, during delivery, breast milk	T1-T3	5-10% develop CNS involvement (mental retardation, cerebral calcification, hydrocephalus, microcephaly, deafness, chorioretinitis)	Asymptomatic or flu-like	Serologic screen; isolate virus from urine or secretion culture	No specific treatment; maintain good hygiene and avoid high risk situations
Erythema Infectiosum (Fifth Disease)	Parvovirus B19	To mother: respiratory, infected blood products To baby: transplacental	10-20 wk GA	Spontaneous abortion (SA), stillbirth, hydrops *in utero*	Flu-like, rash, arthritis; often asymptomatic	Serology, viral PCR, maternal AFP; if IgM present, follow fetus with U/S for hydrops	If hydrops occurs, consider fetal transfusion
Hepatitis B	DNA virus	To mother: blood, saliva, semen, vaginal secretions To baby: transplacental, breast milk	T3 10% vertical transmission if asymptomatic and HBsAg +ve; 85-90% if HBsAg and HBeAg +ve	Prematurity, low birth weight, neonatal death	Fever, N/V, fatigue, jaundice, elevated liver enzymes	Serologic screening for all pregnancies	Rx neonate with HBIG and vaccine (at birth, 1, 6 mo); 90% effective
***Herpes Simplex Virus**	DNA virus	To mother: intimate mucocutaneous contact To baby: transplacental, during delivery	Delivery (if genital lesions present); less commonly *in utero*	Disseminated herpes (20%); CNS sequelae (35%); self-limited infection	Painful vesicular lesions	Clinical diagnosis	Acyclovir for symptomatic women, suppressive therapy at 36 wk controversial Suggested C/S if active genital lesions, even if remote from vulva

Table 15. Infections During Pregnancy (continued)

Infection	Agent	Source of Transmission	Greatest Transmission Risk to Fetus	Effects on Fetus	Effects on Mother	Diagnosis	Management
HIV	RNA retrovirus	To mother: blood, semen, vaginal secretions To baby: in utero, during delivery, breast milk	1/3 *in utero*, 1/3 at delivery, 1/3 breastfeeding	IUGR, preterm labour, PROM	See Infectious Diseases, ID25	Serology, viral PCR All pregnant women are offered screening	Triple anti-retroviral therapy decreases transmission to <1% Elective C/S: no previous antiviral Rx or monotherapy only, viral load unknown or >500 RNA copies/mL, unknown prenatal care, patient request
***Rubella**	ssRNA togavirus	To mother: respiratory droplets (highly contagious) To baby: transplacental	T1	SA or congenital rubella syndrome (hearing loss, cataracts, CV lesions, mitral regurgitation, IUGR, hepatitis, CNS defects, osseous changes)	Rash (50%), fever, posterior auricular or occipital lymphadenopathy, arthralgia	Serologic testing; all pregnant women screened (immune if titre >1:16); infection if IgM present or >4x increase in IgG	No specific treatment; offer vaccine following pregnancy Do not administer during pregnancy (live attenuated)
Syphilis	Spirochete (*Treponema pallidum*)	To mother: sexual contact To baby: transplacental	T1-T3	Risk of preterm labour, multisystem involvement, fetal death	See Infectious Diseases, ID23	VDRL screening for all pregnancies; if positive, requires confirmatory testing	Penicillin G 2.4 million IU IM x 1 dose if early syphilis, 3 doses if late syphilis, monitor VDRL monthly If penicillin G allergic: clindamycin 900 mg IV q8h
***Toxoplasmosis**	Protozoa (*Toxoplasma gondii*)	To mother: raw meat, unpasteurized goat's milk, cat feces/urine To baby: transplacental	T3 (but most severe if infected in T1); only concern if primary infection during pregnancy	Congenital toxoplasmosis (chorioretinitis, hydrocephaly, intracranial calcification, mitral regurgitation, microcephaly) NB: 75% initially asymptomatic at birth	Majority subclinical; may have flu-like symptoms	IgM and IgG serology; PCR of amniotic fluid	Self-limiting in mother; spiramycin decreases fetal morbidity but not rate of transmission

* Indicates TORCH infection

Venous Thromboembolism

Epidemiology

- incidence of 12.1/10,000 (DVT), and 5.4/10,000 (PE)
- increased risk of VTE throughout pregnancy with highest risk of DVT in T3 and post-partum period; highest risk of PE post-partum (first 6 wk)

Risk Factors

- previous VTE, age >35, obesity, infection, bedrest/immobility, shock/dehydration, and thrombophilias (see Hematology, H35)

Table 16. Risk Factors for VTE Specific to Pregnancy

Hypercoagulability	Stasis	Endothelial
Increased Factors: II, V, VII, VIII, IX, X, XII, fibrinogen Increased platelet aggregation Decreased protein S, tPA, factors XI, XIII Increased resistance to activated protein C Antithrombin can be normal or reduced	Increased venous distensibility Decreased venous tone 50% decrease in venous flow in lower extremity by T3 Uterus is mechanical impediment to venous return	Vascular damage at delivery (C/S or SVD) Uterine instrumentation Peripartum pelvic surgery

Virchow's Triad for VTE
- Hypercoagulable state
- Stasis
- Endothelial damage

Clinical Features

- most DVTs occur in the iliofemoral or calf veins with a predilection for the left leg
- signs of a pulmonary embolism are non-specific

Investigations

- duplex venous Doppler sonography for DVT
- CXR and V/Q scan or spiral CT for PE

Management

- before initiating treatment, obtain a baseline CBC including platelets and aPTT
- treatment with LMWH preferred
  - dosing varies depending on specific LMWH used
  - should be discontinued at least 24 h prior to delivery

- unfractionated heparin
  - bolus of 5000 IU followed by an infusion of ~30,000 IU/24h
  - measure aPTT 6 h after the bolus
  - maintain aPTT at a therapeutic level (1.5-2x normal)
  - repeat q24h once therapeutic
  - heparin-induced thrombocytopenia (HIT) uncommon (3%), but serious complication
- warfarin is contraindicated during pregnancy due to its potential teratogenic effects
- compression stockings
- poor evidence to support a recommendation for or against avoidance of prolonged sitting
- VTE prophylaxis
  - women on long-term anticoagulation: full therapeutic anticoagulation throughout pregnancy and for 6-12 wk postpartum
  - women with a non-active PMHx of VTE: unfractionated heparin regimens suggested
  - insufficient evidence in pregnancy to recommend routine use of LMWH for all patients
  - current prophylaxis regimens for acquired thrombophilias (e.g. APS) include low dose ASA in conjunction with prophylactic heparin

Normal Labour and Delivery

Definition of Labour

- true labour: regular, painful contractions of increasing intensity associated with progressive dilatation and effacement of cervix and descent of presenting part, or progression of station
  - preterm (≥20 to ≤36+6 wk GA)
  - term (37-41+6 wk GA)
  - postterm (≥42 wk GA)
- false labour (Braxton-Hicks contractions): irregular contractions, with unchanged intensity and long intervals, occur throughout pregnancy and not associated with any cervical dilatation, effacement, or descent
  - often relieved by rest or sedation

The Cervix

- see Bishop Score (see Table 21, OB37)
  - dilatation: latent phase (0-4 cm, variable time); active phase (4-10 cm)
  - effacement: thinning of the cervix by percentage or length of cervix (cm)
  - consistency: firm, medium, or soft
  - position: posterior, mid, or anterior
- other consideration:
  - application: contact between the cervix and presenting part (i.e. well or poorly applied)

The Fetus

- **fetal lie**: orientation of the long axis of the fetus with respect to the long axis of the uterus (longitudinal, transverse, and oblique)
- **fetal presentation**: fetal body part closest to the birth canal
  - breech (complete, frank, and incomplete) (see Figure 5, OB23)
  - cephalic (vertex/occiput, face, or brow)
  - transverse (shoulder)
  - compound (fetal extremity prolapses along with presenting part)
  - all except vertex are considered malpresentations (see *Obstetrical Complications*, OB16)
- **fetal position**: position of presenting part of the fetus relative to the maternal pelvis
  - OA: most common presentation ("normal") – left OA most common
  - OP: most rotate spontaneously to OA; may cause prolonged second stage of labour
  - OT: leads to arrest of dilatation
    - normally, fetal head enters maternal pelvis and engages in OT position
    - subsequently rotates to OA position (or OP in a small percentage of cases)
- **attitude**: flexion/extension of fetal head relative to shoulders
  - brow presentation: head partially extended (requires C/S)
  - face presentation: head fully extended
    - mentum posterior always requires C/S, mentum anterior can deliver vaginally
- **station**: position of presenting bony part relative to ischial spines – determined by vaginal exam
  - at ischial spines = station 0 = engaged
  - –5 to –1 cm above ischial spines
  - +1 to +5 cm below ischial spines
- **asynclitism**: alignment of the sagittal suture relative to the axis of the birth canal
  - lateral tilt seen with either anterior or posterior asynclitism and may impact descent

Maternal Triage Assessment
ID: Age, GPA, EDD, GA, GBS, Rh, Serology
CC
HPI: 4 key questions:
- Contractions: Since when, how close (q x min), how long (x s), how painful
- Bleeding: Since when, how much (pads), colour (pinky vs. brownish vs. bright red), pain, last U/S, trauma/intercourse
- Fluid (ROM): Since when, large gush vs. trickle, soaked pants, clear vs. green vs. red, continuous
- FM: As much as usual?, When last movement?, Kick counts (lie still for 1-2 h, cold juice, feel FM – should have 6 movements in 2 h)

PregHx: Any complications (HTN, GDM, infections), IPS/FTS screening, last U/S (BPP score, growth/estimated fetal weight, position), last vaginal exam
POBHx: Every previous pregnancy and outcome: year, SVD/C-section/miscarriage/abortion, baby size, length of labour, use of vacuum or forceps, complications
PMHx, Meds, Allergies, SHx
O/E: Maternal vitals, fetal heart tracing (baseline, variability, presence of accelerations/decelerations), Leopold's, vaginal exam, U/S

Reference Point for Describing Fetal Position
- Occiput for cephalic presentation
- Sacrum for breech presentation
- Mentum for face presentation

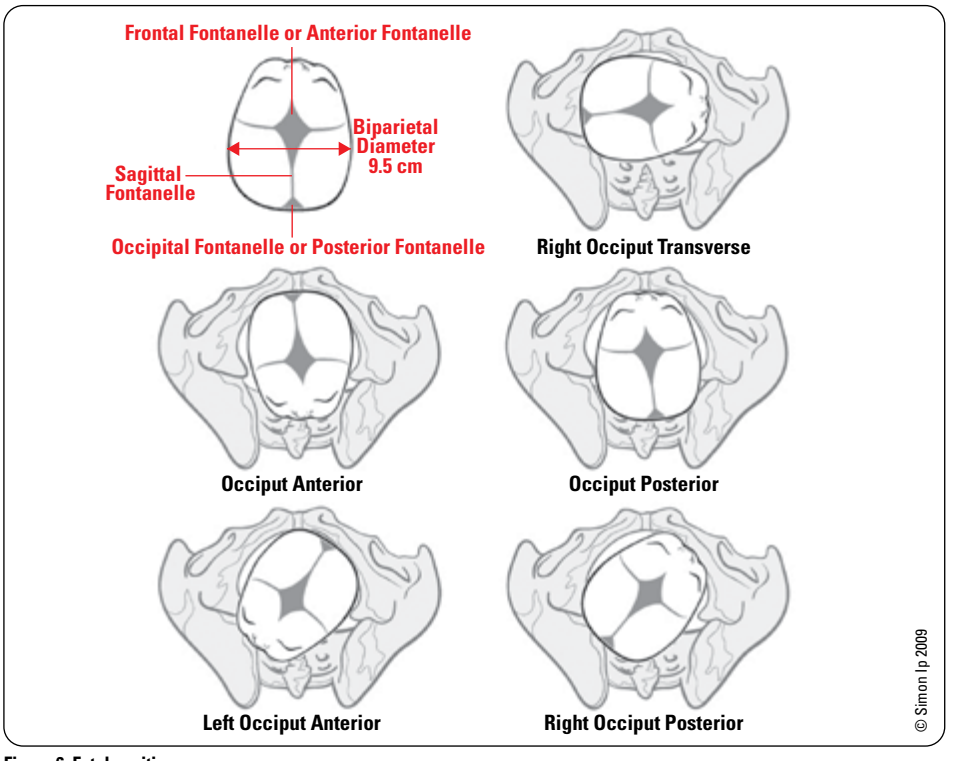


Figure 6. Fetal positions

Four Stages of Labour

First Stage of Labour (0 – 10 cm cervical dilation)

- latent phase
  - uterine contractions typically infrequent and irregular
  - slow cervical dilatation (usually to 4 cm) and effacement
- active phase
  - rapid cervical dilatation to full dilatation (nulliparous ≥1.0 cm/h, multiparous ≥1.2 cm/h)
  - phase of maximum slope on cervical dilatation curve
  - painful, regular contractions q2-3min, lasting 45-60 s
  - contractions strongest at fundus

Second Stage of Labour (10 cm dilation – delivery of the baby)

- from full dilatation to delivery of the baby; duration varies based on parity, contraction quality, and type of analgesia
- mother feels a desire to bear down and push with each contraction
- women may choose a comfortable position that enhances pushing efforts and delivery
  - upright (semi-sitting, squatting) and LLDP are supported in the literature
- progress measured by descent

Third Stage of Labour (delivery of the baby – delivery of the placenta)

- from baby's birth to separation and expulsion of the placenta
- can last up to 30 min before intervention is indicated
- demonstrated by gush of fresh blood, umbilical cord lengthening, uterine fundus changing shape (firm and globular), and rising upward
- active management: start oxytocin IV drip, or give 10 IU IM or 5 mg IV push, after delivery of anterior shoulder in anticipation of placental delivery, otherwise give after delivery of placenta
- routine oxytocin administration in third stage of labour can reduce the risk of PPH by >40%

Fourth Stage of Labour

- first postpartum hour
- monitor vital signs and bleeding, repair lacerations
- ensure uterus is contracted (palpate uterus and monitor uterine bleeding)
- inspect placenta for completeness and umbilical cord for presence of 2 arteries and 1 vein
- 3rd and 4th stages of labour most dangerous to the mother (i.e. hemorrhage)

Course of Normal Labour*

Stage	Nulliparous	Multiparous
First	6-18 h	2-10 h
Second	30 min-3 h	5-30 min
Third	5-30 min	5-30 min

*without epidural

Signs of Placental Separation

- Gush of blood
- Lengthening of cord
- Uterus becomes globular
- Fundus rises

Continuous Support for Women During Childbirth
Cochrane DB Syst Rev 2011;16:CD003766
Study: Systematic review of 21 RCTs from 11 countries, 15,061 women in labour.
Intervention: Continuous support during labour vs. usual care.
Outcome: Effects on mothers and their babies.
Results: Continuous intrapartum support increased likelihood of shorter labour, spontaneous vaginal birth, decrease in analgesia use, and a decrease in dissatisfaction with childbirth experience. Greatest benefit when provider is not a health care professional. Continuous support was also associated with decreased likelihood to have a Cesarean or instrumental vaginal birth, regional analgesia, or a baby with a low 5 min APGAR score.

The Cardinal Movements of the Fetus During Delivery

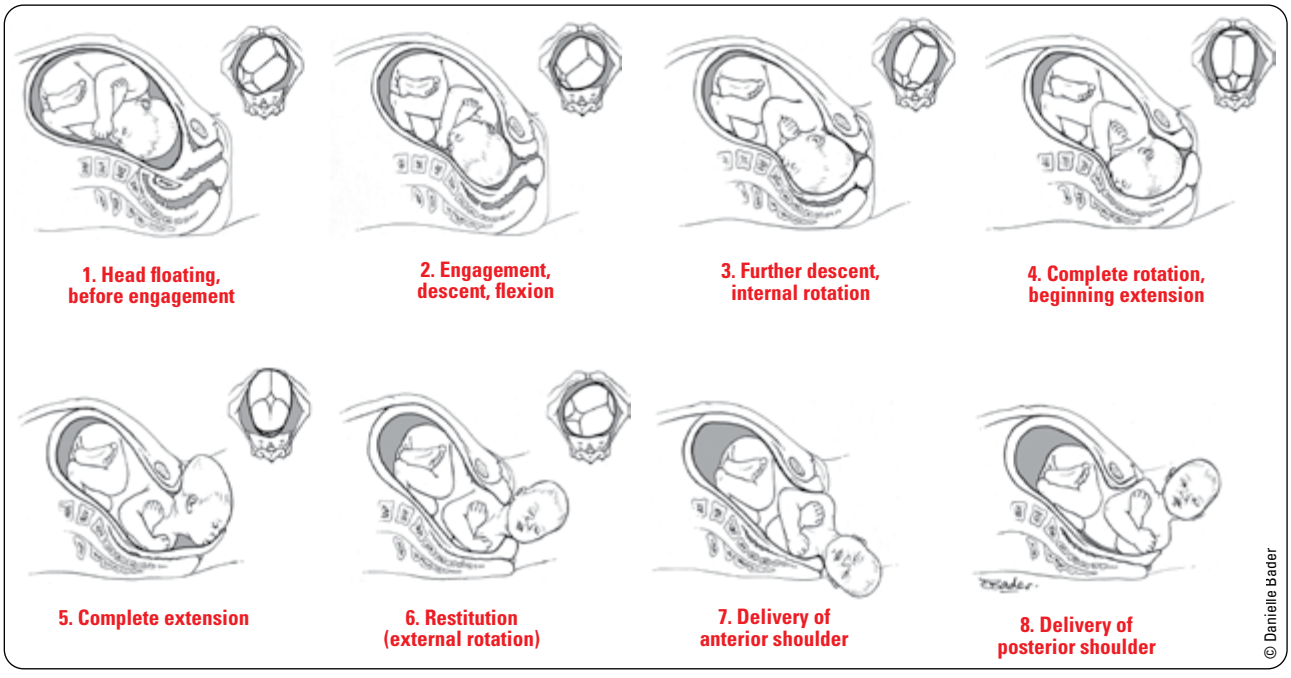


Figure 7. Cardinal movements of fetus during delivery
Adapted from illustration in Williams Obstetrics, 19th ed

Analgesic and Anesthetic Techniques in Labour and Birth

- pain or anxiety leads to high endogenous catecholamines, which produce a direct inhibitory effect on uterine contractility

Non-Pharmacologic Pain Relief Techniques

- reduction of painful stimuli
  - maternal movement, position change, counter-pressure, and abdominal compression
- activation of peripheral sensory receptors
  - superficial heat and cold
  - immersion in water during labour
  - touch and massage, acupuncture, and acupressure
  - TENS
  - intradermal injection of sterile water
  - aromatherapy
- enhancement of descending inhibitory pathways
  - attention focusing and distraction
  - hypnosis
  - music and audio analgesia
  - biofeedback

Pharmacologic Methods (see Anesthesia and Perioperative Medicine, A27)

- nitrous oxide (e.g. self-administered Entonox®)
- narcotics (usually combined with anti-emetic)
- pudendal nerve block
- perineal infiltration with local anesthetic
- regional anesthesia (epidural block, combined spinal-epidural, and spinal)

Fetal Monitoring in Labour


- see online **Fetal Heart Rate Tutorial**

Vaginal Exam

- membrane status, as indicated by amniotic fluid (clear, pink, bloody, and meconium)
- cervical effacement (thinning), dilatation, consistency, position, and application
- fetal presenting part, position, and station
- bony pelvis size and shape
- monitor progress of labour at regular intervals and document in a partogram

Intrapartum Fetal Monitoring

- intermittent fetal auscultation with Doppler device q15-30min for 1 min in first stage active phase following a contraction, q5min during second stage when pushing has begun
- continuous electronic FHR monitoring reserved for abnormal auscultation, prolonged labour, labour which is induced or augmented, meconium present, multiple gestation/fetal complication, and concerns about the fetus tolerating labour
  - use of continuous electronic monitoring shown to lead to higher intervention rates and no improvement in outcome for the neonate when used routinely in all patients (i.e. no risk factors)
  - techniques for continuous monitoring include external (Doppler) vs. internal (fetal scalp electrode) monitoring
- fetal scalp sampling should be used in conjunction with electronic FHR monitoring and contraction monitoring (CTG) to resolve the interpretation of abnormal or atypical patterns

Electronic FHR Monitoring

- FHR measured by Doppler; contractions measured by tocometer
- described in terms of baseline FHR, variability (short-term, long-term), and periodicity (accelerations, decelerations)
- **Baseline FHR**
  - normal range is 110-160 bpm
  - parameter of fetal well-being vs. distress
- **Variability**
  - physiologic variability is a normal characteristic of FHR
  - variability is measured over a 15 min period and is described as: absent, minimal (<6 bpm), moderate (6-25 bpm), or marked (>25 bpm)
  - normal variability indicates fetal acid-base status is acceptable
  - can only be assessed by electronic contraction monitoring (CTG)
  - variability decreases intermittently even in healthy fetus
  - see Table 19, OB35
- **Periodicity**
  - accelerations: increase of ≥15 bpm for ≥15 s, (or ≥10 bpm for ≥10 s if <32 wk GA)
  - decelerations: 3 types, described in terms of shape, onset, depth, duration recovery, occurrence, and impact on baseline FHR and variability

Table 17. Factors Affecting Fetal Heart Rate

	Fetal Tachycardia (FHR >160 bpm)	Fetal Bradycardia (FHR <110 bpm)	Decreased Variability
Maternal Factors	Fever, hyperthyroidism, anemia, dehydration	Hypothermia, hypotension, hypoglycemia, position, umbilical cord occlusion	Infection Dehydration
Fetal Factors	Arrhythmia, anemia, infection, prolonged activity, chronic hypoxemia, congenital anomalies	Rapid descent, dysrhythmia, heart block, hyopoxia, vagal stimulation (head compression), hypothermia, acidosis	CNS anomalies Dysrhythmia Inactivity/sleep cycle, preterm fetus
Drugs	Sympathomimetics	β-blockers Anesthetics	Narcotics, sedatives Magnesium sulphate, β-blockers
Uteroplacental	Early hypoxia (abruption, HTN) Chorioamnionitis	Late hypoxia (abruption, HTN) Acute cord prolapse Hypercontractility	Hypoxia

Approach to the Management of Abnormal FHR

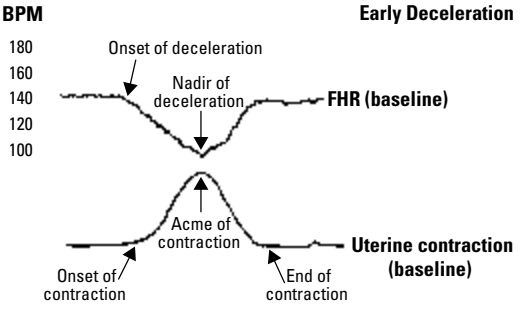
POISON – ER
Position (LLDP)
O$_2$ (100% by mask)
IV fluids (corrects maternal hypotension)
Fetal **s**calp stimulation
Fetal **s**calp electrode
Fetal **s**calp pH
Stop **o**xytocin
Notify MD
Vaginal **e**xam to rule out cord prolapse
Rule out fever, dehydration, drug effects, prematurity
- If above fails, consider C/S

Continuous CTG as a Form of EFM for Fetal Assessment During Labour
Cochrane DB Syst Rev 2013;5:CD006066
Purpose: To examine the effectiveness of continuous electronic fetal monitoring or cardiotocography during labour.
Selection Criteria: Randomized and quasi-randomized controlled trials comparing continuous CTG (with and without fetal blood sampling) to a) no fetal monitoring, b) intermittent auscultation, or c) intermittent CTG.
Results: 13 trials, 37,000 women. Continuous CTG compared with intermittent auscultation showed no difference in overall perinatal death rate or cerebral palsy rates. Nonetheless, neonatal seizures were halved (RR 0.50, 95% CI 0.31-0.80) and there was a significant increase in C/S (RR 1.63, 95% CI 1.29-2.07) and instrumental vaginal birth (RR 1.15, 95% CI 1.01-1.33) with CTG.
Conclusion: Continuous CTG may reduce the incidence of neonatal seizures, but has no effect on cerebral palsy rates, infant mortality, or other measures of neonatal well-being. Continuous CTG was also associated with an increase in C/S and instrumental deliveries.

Table 18. Comparison of Decelerations

Early Decelerations

- Uniform shape with onset early in contraction, returns to baseline by end of contraction, mirrors contraction (nadir occurs at peak of contraction)
- Gradual deceleration and return to baseline
- Often repetitive; no effect on baseline FHR or variability
- Benign, due to vagal response to head compression

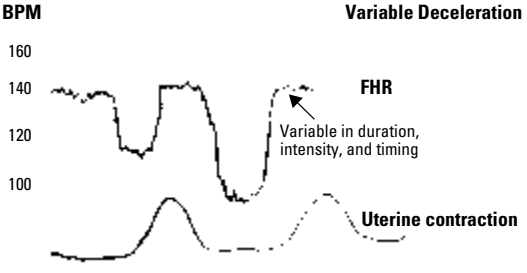


Variable Decelerations

- Variable in shape, onset, and duration
- Most common type of periodicity seen during labour
- Often with abrupt drop in FHR >15 bpm below baseline (>15 s, <2 min); usually no effect on baseline FHR or variability
- Due to cord compression or, in second stage, forceful pushing with contractions

Complicated Variable Decelerations

- FHR drop <70 bpm for >60 s
- Loss of variability or decrease in baseline after deceleration
- Biphasic deceleration
- Slow return to baseline
- Baseline tachycardia or bradycardia
- May be associated with fetal acidemia



Rule of 60s Suggesting Severe Variable Decelerations
Deceleration to <60 bpm
>60 bpm below baseline
>60 s in duration with slow return to baseline

Late Decelerations

- Uniform shape with onset, nadir, and recovery occurring after peak of contraction, slow return to baseline
- May cause decreased variability and change in baseline FHR
- Due to fetal hypoxia and acidemia, maternal hypotension, or uterine hypertonus
- Usually a sign of uteroplacental insufficiency (an ominous sign)

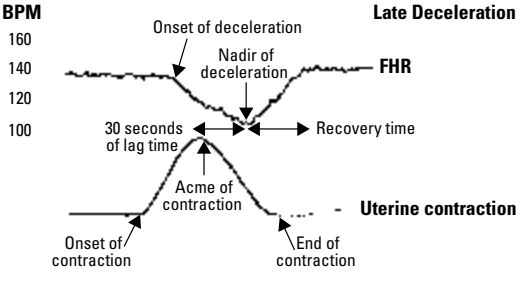


Table 19. Classification of Intrapartum EFM Tracings

	Normal Tracing (Category 1)	Atypical Tracing* (Category 2)	Abnormal Tracing* (Category 3)
Baseline	110-160 bpm	Bradycardia 100-110 bpm Tachycardia >160 for 30-80 min Rising baseline	Bradycardia <100 bpm Tachycardia >160 bpm for >80 min Erratic baseline
Variability	6-25 bpm ≤5 bpm for <40 min	≤5 bpm for 40-80 min	<5 bpm for >80 min ≥25 bpm for >10 min
Decelerations	None Early decelerations Occasional uncomplicated variable decelerations	Repetitive (≥3) uncomplicated variable decelerations Occasional late decelerations Any prolonged deceleration (2-3 min)	Repetitive (≥3) complicated variable decelerations Repetitive late decelerations Any prolonged deceleration (≥3 min)
Accelerations	Accelerations spontaneous or during scalp stimulation	Absent with scalp stimulation	Nearly absent
Action	EFM may be interrupted for ≤30 min if mother/fetus stable	Further assessment required	Action required: review clinical situation, obtain scalp pH, prepare for possible delivery

Adapted from SOGC Guidelines, September 2008
*Previous classification was "reassuring" vs. "non-reassuring", but distinction is now made between tracings that have some concerning changes but do not require immediate action (atypical) versus those with major concerns requiring immediate intervention (abnormal)

Fetal Scalp Blood Sampling

- cervix must be adequately dilated
- indicated when atypical or abnormal fetal heart rate is suggested by clinical parameters including heavy meconium or moderately to severely abnormal FHR patterns (including unexplained low variability, repetitive late decelerations, complex variable decelerations, and fetal cardiac arrhythmias)

- done by measuring pH or more recently fetal lactate
  - pH ≥7.25, lactate <4.2 mmol/L: normal, repeat if abnormal FHR persists
  - pH 7.21-7.24, lactate 4.2-4.8 mmol/L: repeat assessment in 30 min or consider delivery if rapid fall since last sample
  - pH ≤7.20, lactate >4.8 mmol/L indicates fetal acidosis, delivery is indicated
- contraindications
  - known or suspected fetal blood dyscrasia (hemophilia, vWD)
  - active maternal infection (HIV, genital herpes)

Fetal Oxygenation

- uterine contractions during labour decrease uteroplacental blood flow, which results in reduced oxygen delivery to the fetus
- most fetuses tolerate this reduction in flow and have no adverse effects
- distribution of oxygen to the fetus depends on maternal, uteroplacental, and fetal factors
- fetal response to hypoxia/asphyxia:
  - decreased movement, tone, and breathing activities
  - anaerobic metabolism (decreased pH)
  - transient fetal bradycardia followed by fetal tachycardia
  - redistribution of fetal blood flow
- increased flow to brain, heart, and adrenals
- decreased flow to kidneys, lungs, gut, liver, and peripheral tissues
- increase in blood pressure

Table 20. Factors Affecting Fetal Oxygenation

Factor	Mechanism	Example
Maternal	Decreased maternal oxygen carrying capacity	Significant anemia (iron deficiency, hemoglobinopathies), carboxyhemoglobin (smokers)
	Decreased uterine blood flow	Hypotension (blood loss, sepsis), regional anesthesia, maternal positioning
	Chronic maternal conditions	Vasculopathies (SLE, type 1 DM, chronic HTN), APS, cyanotic heart disease, COPD
Uteroplacental	Uterine hypertonus	Placental abruption, hyperstimulation secondary to oxytocin, prostaglandins, or normal labour
	Uteroplacental dysfunction	Placental abruption, placental infarction (dysfunction marked by IUGR, oligohydramnios, abnormal Doppler studies), chorioamnionitis, placental edema (DM, hydrops), placental senescence (post-dates)
Fetal	Cord compression	Oligohydramnios, cord prolapse, or entanglement
	Decreased fetal oxygen carrying capacity	Significant anemia (isoimmunization, feto-maternal bleed), carboxyhemoglobin (exposure to smokers)

Induction of Labour

Definition

- artificial initiation of labour in a pregnant woman prior to spontaneous initiation to deliver the fetus and placenta

Prerequisites for Labour Induction

- capability for C/S if necessary
- maternal
  - inducible/ripe cervix: short, thin, soft, anterior cervix with open os
  - if cervix is not ripe, use prostaglandin vaginal insert (Cervidil®), prostaglandin gel (Prepidil®), misoprostol (Cytotec®), or Foley catheter
- fetal
  - normal fetal heart tracing
  - cephalic presentation
  - adequate fetal monitoring available
- likelihood of success determined by Bishop score
  - cervix considered unfavourable if <6
  - cervix favourable if ≥6
  - score of 9-13 associated with high likelihood of vaginal delivery

Induction is indicated when the risk of continuing pregnancy exceeds the risks associated with induced labour and delivery

Table 21. Bishop Score

Cervical Characteristic	0	1	2	3
Position	Posterior	Mid	Anterior	–
Consistency	Firm	Medium	Soft	–
Effacement (%)	0-30	40-50	60-70	≥80
Dilatation (cm)	0	1-2	3-4	≥5
Station of Fetal Head	-3	-2	-1, 0	+1, +2, +3

Induction vs. Augmentation
Induction is the artificial initiation of labour
Augmentation promotes contractions when spontaneous contractions are inadequate

Indications
- post-dates pregnancy (generally >41 wk) = most common reason for induction
- maternal factors
  - DM = second most common reason for induction
  - gestational HTN ≥37 wk
  - preeclampsia
  - other maternal medical problems, e.g. renal or lung disease, chronic hypertension, and cholestasis
  - maternal age over 40
- maternal-fetal factors
  - isoimmunization, PROM, chorioamnionitis
- fetal factors
  - suspected fetal jeopardy as evidenced by biochemical or biophysical indications
  - macrosomia, fetal demise, IUGR, oligo/polyhydramnios, anomalies requiring surgical intervention, and twins
  - previous stillbirth or low PAPP-A

Consider the Following Before Induction
- Indication for induction
- Contraindications
- GA
- Cervical favourability
- Fetal presentation
- Potential for CPD
- Fetal well-being/FHR
- Membrane status

Risks
- failure to achieve labour and/or vaginal birth
- uterine hyperstimulation with fetal compromise or uterine rupture
- maternal side effects to medications
- uterine atony and PPH

Contraindications
- maternal
  - prior classical or inverted T-incision C/S or uterine surgery (e.g. myomectomy)
  - unstable maternal condition
  - active maternal genital herpes
  - invasive cervical carcinoma
  - pelvic structure deformities
- maternal-fetal
  - placenta previa or vasa previa
  - cord presentation
- fetal
  - fetal distress, malpresentation/abnormal lie, or preterm fetus without lung maturity

Induction Methods

CERVICAL RIPENING

Definition
- use of medications or other means to soften, efface, and dilate the cervix; increases likelihood of successful induction
- ripening of an unfavourable cervix (Bishop score <6) is warranted prior to induction of labour

Methods
- intravaginal prostaglandin PGE2 gel (Prostin® gel): long and closed cervix
  - recommended dosing interval of prostaglandin gel is every 6-12 h up to 3 doses
- intravaginal PGE2 (Cervidil®): long and closed cervix, may use if ROM
  - continuous release, can be removed if needed
  - controlled release PGE2
- intracervical PGE2 (Prepidil®)
- intravaginal PGE1 misoprostol (Cytotec®): long and closed cervix
  - inexpensive, stored at room temperature
  - more effective than PGE2 for achieving vaginal delivery and less epidural use
- Foley catheter placement to mechanically dilate the cervix

Evidence for Cervical Ripening Methods (SOGC Guidelines)
- Meta-analysis of five trials has concluded that the use of oxytocin to ripen the cervix is not effective
- Since the best dose and route of misoprostol for labour induction with a live fetus are not known and there are concerns regarding hyperstimulation, the use of misoprostol for induction of labour should be within clinical trials only (Level Ib evidence) or in cases of intrauterine fetal death to initiate labour

Intravaginal PGE2 (Cervidil®) Compared to Intravaginal Prostaglandin Gel
4 RCTs have compared the two with varying results, depending on the dosing regime of gel used.
Theoretical advantages of Cervidil®:
- Slow, continuous release
- Only one dose required
- Ability to use oxytocin 30 min after removal vs. 6 hours for gel
- Ability to remove insert if required (i.e. excessive uterine activity)

INDUCTION OF LABOUR

Amniotomy

- artificial ROM (amniotomy) to stimulate prostaglandin synthesis and secretion; may try this as initial measure if cervix is open and soft, the membranes can be felt, and if the head is present at the cervix
- few studies address the value of amniotomy alone for induction of labour
- amniotomy plus intravenous oxytocin: more women delivered vaginally at 24 h than amniotomy alone (relative risk = 0.03) and had fewer instrumental vaginal deliveries (relative risk = 5.5)

Oxytocin

- oxytocin (Pitocin®): 10 U in 1L NS, run at 0.5-2 mU/min IV increasing by 1-2 mU/min q20-60min
- reduces rate of unsuccessful vaginal deliveries within 24 h when used alone (8.3% vs. 54%, RR 0.16)
- ideal dosing regimen of oxytocin is not known
- current recommendations: use the minimum dose to achieve active labour and increase q30min as needed
- reassessment should occur once a dose of 20 mU/min is reached
- potential complications
  - hyperstimulation/tetanic contraction (may cause fetal distress or uterine rupture)
  - uterine muscle fatigue, uterine atony (may result in PPH)
  - vasopressin-like action causing anti-diuresis

Oxytocin t1/2 = 3-5 min

Augmentation of Labour

- augmentation of labour with oxytocin is used to promote adequate contractions when spontaneous contractions are inadequate and cervical dilatation or descent of fetus fails to occur

Provided there are no contraindications, oxytocin is utilized to improve uterine contraction strength and/or frequency

Abnormalities and Complications of Labour and Delivery

Abnormal Progression of Labour (Dystocia)

Definition

- expected patterns of descent of the presenting part and cervical dilatation fail to occur in the appropriate time frame; can occur in all stages of labour
- during active phase: >4 h of <0.5 cm/h
- during 2nd stage: >1 h with no descent during active pushing

Etiology

- Power (leading cause): contractions (hypotonic, uncoordinated), inadequate maternal expulsive efforts
- Passenger: fetal position, attitude, size, anomalies (hydrocephalus)
- Passage: pelvic structure (CPD), maternal soft tissue factors (tumours, full bladder or rectum, vaginal septum)
- Psyche: hormones released in response to stress may contribute to dystocia; psychological and physiological stress should be evaluated as part of the management once dystocia has been diagnosed

The 4 Ps of Dystocia
Power
Passenger
Passage
Psyche

Management

- confirm diagnosis of labour (rule out false labour)
- search for factors of CPD
- concern for dystocia if adequate contractions measured by intrauterine pressure catheter (IUPC) with no descent/dilatation for >2 h
- management: if CPD ruled out, IV oxytocin augmentation ± amniotomy

Risks of Dystocia

- inadequate progression of labour is associated with an increased incidence of:
  - maternal stress
  - maternal infection
  - PPH
  - need for neonatal resuscitation
  - fetal compromise (from uterine hyperstimulation)
  - uterine rupture
  - hypotension

Shoulder Dystocia

Definition
- fetal anterior shoulder impacted above pubic symphysis after fetal head has been delivered
- life threatening emergency

Etiology/Epidemiology
- incidence 0.15-1.4% of deliveries
- occurs when breadth of shoulders is greater than biparietal diameter of the head

Risk Factors
- maternal: obesity, DM, multiparity, and previous shoulder dystocia
- fetal: prolonged gestation or macrosomia (especially if associated with GDM)
- labour
  - prolonged 2nd stage
  - instrumental midpelvic delivery

Presentation
- "turtle sign": head delivered but retracts against inferior portion of pubic symphysis
- complications
  - fetal
    - hypoxic ischemic encephalopathy (chest compression by vagina or cord compression by pelvis can lead to hypoxia)
    - brachial plexus injury (Erb's palsy: C5-C7; Klumpke's palsy: C8-T1), 90% resolve within 6 mo
    - fracture (clavicle, humerus, and cervical spine)
    - death
  - maternal
    - perineal injury
    - PPH (uterine atony or lacerations)
    - uterine rupture

Treatment
- goal: to displace anterior shoulder from behind symphysis pubis; follow a stepwise approach of maneuvers until goal achieved (see sidebar)
- other options
  - cleidotomy (deliberate fracture of neonatal clavicle)
  - Zavanelli maneuver: replacement of fetus into uterine cavity and emergent C/S
  - symphysiotomy

Prognosis
- 1% risk of long-term disability for infant

Approach to the Management of Shoulder Dystocia

ALARMER
Apply suprapubic pressure and ask for help
Legs in full flexion (McRobert's maneuver)
Anterior shoulder disimpaction (suprapubic pressure)
Release posterior shoulder by rotating it anteriorly with hand in the vagina under adequate anesthesia
Manual corkscrew i.e. rotate the fetus by the posterior shoulder until the anterior shoulder emerges from behind the maternal symphysis
Episiotomy
Rollover (on hands and knees)
*Note that suprapubic pressure and McRobert's maneuver together will resolve 90% of cases

Umbilical Cord Prolapse

Definition
- descent of the cord to a level adjacent to or below the presenting part, causing cord compression between presenting part and pelvis

Etiology/Epidemiology
- increased incidence with prematurity/PROM, fetal malpresentation (~50% of cases), low-lying placenta, polyhydramnios, multiple gestation, and CPD
- incidence: 1/200-1/400 deliveries

Presentation
- visible or palpable cord
- FHR changes (variable decelerations, bradycardia, or both)

Treatment
- emergency C/S if not fully dilated and vaginal delivery not imminent
- O_2 to mother, monitor fetal heart
- alleviate pressure of the presenting part on the cord by elevating fetal head with a pelvic exam (maintain this position until C/S)
- keep cord warm and moist by replacing it into the vagina ± applying warm saline soaks
- roll mother onto all fours or position mother in Trendelenburg or knee-to-chest position
- if fetal demise or too premature (<22 wk), allow labour and delivery

Umbilical Cord Accident Causes
- Nuchal cord
  - Type A (looped)
  - Type B (hitched)
- Body loop
- Single artery
- True knot
- Torsion
- Velamentous
- Short cord <35 cm
- Long cord >80 cm

- 1/3 of protraction disorders develop into 2° arrest of dilatation due to CPD
- 2/3 of protraction disorders progress through labour to vaginal delivery

Uterine Rupture

Definition
- associated with previous uterine scar (in 40% of cases), hyperstimulation with oxytocin, grand multiparity, and previous intrauterine manipulation
- generally occurs during labour, but can occur earlier with a classical incision
- 0.5-0.8% incidence, up to 12% with classical incision

Presentation
- prolonged fetal bradycardia – most common presentation
- acute onset of constant lower abdominal pain, may not have pain if receiving epidural analgesia
- hyper/hypotonic uterine contractions
- abnormal progress in labour
- vaginal bleeding
- intra-abdominal hemorrhage
- loss of station of the presenting fetal part
- maternal tachycardia, hypotension, or shock

Risk Factors
- uterine scarring (i.e. previous uterine surgeries including C/S (especially classical incision), perforation with D&C, and myomectomy)
- excessive uterine stimulation (i.e. protracted labour, oxytocin, prostaglandins)
- uterine trauma (i.e. operative equipment, ECV)
- multiparity
- uterine abnormalities
- malpresentation
- placenta accreta

Treatment
- rule out placental abruption
- maternal stabilization (may require hysterectomy), treat hypovolemia
- immediate delivery for fetal survival

Complications
- maternal mortality 1-10%
- maternal hemorrhage, shock, DIC
- amniotic fluid embolus
- hysterectomy if uncontrollable hemorrhage
- fetal distress, associated with infant mortality as high as 15%

Maternal Mortality Causes
- Thromboembolism
- Cardiac event
- Suicide
- Sepsis
- Ectopic pregnancy
- HTN
- Amniotic fluid embolism
- Hemorrhage

* In Canada (2013), lifetime risk of maternal death is 1 in 5200

Amniotic Fluid Embolus

Definition
- amniotic fluid debris in maternal circulation triggering an anaphylactoid immunologic response

Etiology/Epidemiology
- rare intrapartum or immediate postpartum complication
- 13-30% maternal mortality rate
- leading cause of maternal death in induced abortions and miscarriages
- 1/8000-1/80,000 births

Risk Factors
- placental abruption
- rapid labour
- multiparity
- uterine rupture
- uterine manipulation
- induction medication and procedures

Differential Diagnosis
- pulmonary embolus, drug-induced anaphylaxis, septic shock, eclampsia, HELLP syndrome, abruption, and chronic coagulopathy

Presentation
- sudden onset of respiratory distress, cardiovascular collapse (hypotension, hypoxia), and coagulopathy
- seizure in 10%
- ARDS and left ventricular dysfunction seen in survivors

Management
- should be managed in the ICU by a multidisciplinary team
- supportive measures (high flow O_2, ventilation support, fluid resuscitation, inotropic support, ± intubation) and coagulopathy correction

Chorioamnionitis

Definition
- infection of the chorion, amnion, and amniotic fluid

Etiology/Epidemiology
- incidence 1-5% of term pregnancies and up to 25% in preterm deliveries
- ascending infection (microorganisms from vagina)
- predominant microorganisms include: GBS, *Bacteroides* and *Prevotella* species, *E. coli*, and anaerobic *Streptococcus*

Clinical Features of Chorioamnionitis
- Temperature
- Tachycardia (maternal or fetal)
- Tenderness (uterine)
- Foul discharge

Risk Factors
- low parity, prolonged ROM, long labour, multiple vaginal exams during labour, and internal monitoring
- bacterial vaginosis and other vaginal infections

Clinical Features
- maternal fever ≥38° C, maternal or fetal tachycardia, uterine tenderness, and foul and purulent cervical discharge

Investigations
- CBC: leukocytosis
- amniotic fluid: Gram stain, glucose, or culture results consistent with infection

Treatment
- IV antibiotics
  - ampicillin 2 g IV q6h + gentamicin 2 mg/kg load, then 1.5 mg/kg IV q8h
  - anaerobic coverage (i.e. clindamycin 900 mg IV q8h)
  - if at risk for endometritis, continue treatment post-partum especially if C/S delivery
- antipyretics
- proper labour progression (not an indication for immediate delivery or C/S)

Complications
- bacteremia of mother or fetus, wound infection if C/S, pelvic abscess, neonatal meningitis, neonatal sepsis, and neonatal death
- long-term infant complications: cerebral palsy and bronchopulmonary dysplasia

Meconium

Epidemiology
- present early in labour in 10% of pregnancies, more common in postdate pregnancies
- in general, meconium may be present in up to 25% of all labours; usually NOT associated with poor outcome
- concern if fluid changes from clear to meconium-stained
- always abnormal if seen in preterm fetus

Particulate (thickened) meconium is associated with lower APGARs, an increased risk of meconium aspiration, and perinatal death. Particulate meconium generally has a darker green or black colour, whereas thin meconium is usually yellow to light green

Etiology
- likely cord compression ± uterine hypertonia
- may indicate undiagnosed breech
- increasing meconium during labour may be a sign of fetal distress

Features
- may be watery or thicker (particulate)
- light yellow/green or dark green-black in colour

Treatment
- call respiratory therapy, neonatology, or pediatrics to delivery room
- closely monitor FHR for signs of fetal distress

Operative Obstetrics

Operative Vaginal Delivery

Definition
- forceps or vacuum extraction

Indications
- fetal
  - atypical or abnormal fetal heart rate tracing, evidence of fetal compromise
  - consider if second stage is prolonged, as this may be due to poor contractions or failure of fetal head to rotate
- maternal
  - need to avoid voluntary expulsive effort (e.g. cardiac/cerebrovascular disease)
  - exhaustion, lack of cooperation, and excessive analgesia may impair pushing effort

Contraindications
- unknown fetal head presentation
- unengaged head
- fetal bone demineralization disorder (e.g. osteogenesis imperfecta)
- fetal bleeding disorder (e.g. hemophilia or vWD)

Prerequisites for Operative Vaginal Delivery

ABCDEFGHIJK
Anesthesia (adequate)
Bladder empty
Cervix fully dilated and effaced with ROM
Determine position of fetal head
Equipment ready (including facilities for emergent C/S)
Fontanelle (posterior fontanelle midway between thighs)
Gentle traction
Handle elevated
Incision (episiotomy)
Once **j**aw visible remove forceps
Knowledgeable operator

Forceps

Outlet Forceps Position
- head visible between labia in between contractions
- sagittal suture in or close to AP diameter
- rotation cannot exceed 45°

Low Forceps Position
- presenting part at station +2 or greater
- subdivided based on whether rotation less than or greater than 45°

Mid Forceps Position
- presenting part below spines but above station +2

Types of Forceps
- Simpson or Tucker-McLane forceps for OA presentations
- Kielland (rotational) forceps when rotation of head or correction of asynclitism is required
- Piper forceps for after-coming head in breech delivery
- Wrigley's for preterm babies

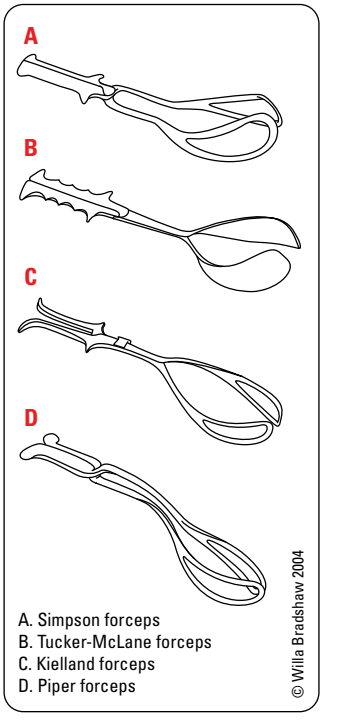


Figure 8. Types of forceps

Vacuum Extraction

- traction instrument used as alternative to forceps delivery; aids maternal pushing
- contraindications: <34 wk GA (<2500 g), fetal head deflexed, fetus requires rotation, fetal condition (e.g. bleeding disorder)

Limits for Trial of Vacuum
- After 3 pulls over 3 contractions with no progress
- After 3 pop-offs with no obvious cause
- 20 min and delivery is not imminent

Table 22. Advantages and Disadvantages of Forceps vs. Vacuum Extraction

	Forceps	Vacuum Extraction
Advantages	Higher overall success rate for vaginal delivery Decreased incidence of fetal morbidity	Easier to apply Less anesthesia required Less maternal soft-tissue injury compared to forceps
Disadvantages	Greater incidence of maternal injury	Suitable only for vertex presentations Maternal pushing required Contraindicated in preterm delivery
Complications	Maternal: anesthesia risk, lacerations, injury to bladder, uterus, or bone, pelvic nerve damage, PPH, and infections Fetal: fractures, facial nerve palsy, trauma to face/scalp, intracerebral hemorrhage, cephalohematoma, and cord compression	Increased incidence of cephalohematoma and retinal hemorrhages, and jaundice compared to forceps Subgaleal hemorrhage Subaponeurotic hemorrhage Soft tissue trauma

Lacerations

- first degree: involves skin and vaginal mucosa but not underlying fascia and muscle
- second degree: involves fascia and muscles of the perineal body but not the anal sphincter
- third degree: involves the anal sphincter (partial IIIa or complete IIIb)
- fourth degree: extends through the anal sphincter into the rectal mucosa
- for third and fourth degree tears, a single prophylactic dose of IV antibiotics (2nd generation cephalosporin, e.g. cefoxitin or cefotetan) should be administered to reduce perineal wound complications; laxatives should also be prescribed and constipation should be avoided

Episiotomy

Definition

- incision in the perineal body at the time of delivery
- essentially a controlled second degree laceration
- midline: incision through central tendinous portion of perineal body and insertions of superficial transverse perineal and bulbocavernosus muscles
  - heals better, but increases risk of 3rd/4th degree tears
- mediolateral: incision through bulbocavernosus, superficial transverse perineal muscle, and levator ani
  - reduced risk of extensive tear but more painful

Indications

- to relieve obstruction of the unyielding perineum
- to expedite delivery (e.g. abnormal FHR pattern)
- instrumental delivery
- controversial between practitioners as to whether it is preferable to make a cut or let the perineum tear as needed
- current evidence suggests letting perineum tear and then repair as needed (restricted use)

Complications

- infection, hematoma, extension into anal musculature or rectal mucosa, fistula formation, and incontinence

Risk Factors for the Development of Obstetric Anal Sphincter Injuries in Modern Obstetric Practice
Obstet Gynecol 2018;131(2):290-96
Objective: To characterize the rate of obstetric anal sphincter injuries and identify key risk factors of obstetric anal sphincter injuries, including duration of the second stage of labour.
Methods: Retrospective cohort study including all singleton, term, cephalic vaginal deliveries from 2013 to 2014.
Results: The overall incidence rate of obstetric anal sphincter injuries was 4.9% (3.6% of women who delivered spontaneously vs 24.0% of women who had a vacuum-assisted vaginal delivery, P<.001, CI 18.1–22.6%). In bivariate and multivariate analyses, obstetric anal sphincter injury incidence was higher among women with second stage of labour longer than 2 hours, Asian race, nulliparity, vaginal birth after cesarean delivery, episiotomy, and vacuum delivery. Women with a vacuum-assisted vaginal delivery had four times the odds of obstetric anal sphincter injury (adjusted odds ratio [OR] 4.23, 95% CI 3.59-4.98) and those whose second stage of labour lasted at least 180 minutes vs less than 60 minutes had three times the odds of incurring obstetric anal sphincter injury (adjusted OR 3.20, 95% CI 2.62-3.89).

Cesarean Delivery

Epidemiology

- overall 28% rate in Canada (range 18.5-35.3% by province/territory)

Indications

- maternal: obstruction, active herpetic lesion on vulva, invasive cervical cancer, previous uterine surgery (past C/S is most common), and underlying maternal illness (eclampsia, HELLP syndrome, heart disease)
- maternal-fetal: failure to progress, placental abruption or previa, and vasa previa
- fetal: abnormal fetal heart tracing, malpresentation, cord prolapse, and certain congenital anomalies

Types of Cesarean Incisions

- skin
  - transverse (Pfannenstiel)
  - decreased exposure and slower entry
  - improved strength and cosmesis
  - vertical midline
  - rapid peritoneal entry and increased exposure
  - increased dehiscence
- uterine
  - low transverse (most common): in non-contractile lower segment
- decreased chance for rupture in subsequent pregnancies
  - low vertical
  - used for very preterm infants or poorly developed maternal lower uterine segment
  - classical (rare): in thick, contractile segment
  - used for transverse lie, preterm breech, fetal anomaly, >2 fetuses, lower segment adhesions, obstructing fibroid, and inaccessible lower uterine segment (e.g. morbid obesity)

Risks/Complications

- anaesthetic complications (e.g. aspiration)
- hemorrhage (average blood loss ~1000 cc)
- infection (UTI, wound, and endometritis)
  - single dose prophylactic antibiotic should be used (e.g. cefazolin 1-2 g)
- injury to surrounding structures (bowel, bladder, ureter, and uterus)
- thromboembolism (DVT, PE)
- increased recovery time/hospital stay
- maternal mortality (<0.1%)

Common OR Questions

7 Layers to Dissect
Skin, fatty layer, fascia, muscle separation (rectus abdominis), peritoneum, bladder flap, uterus

Layers of the Rectus Sheath
Above the arcuate line: external oblique, external internal oblique, internal oblique, rectus abdominis, internal internal oblique, transversus abdominis
Below the arcuate line: external oblique, internal oblique, transversus abdominis, rectus abdominis

Name of the Obliterated Umbilical Ligament Urachus

Most C/S performed with regional analgesia

Trial of Labour after Cesarean Section (TOLAC)

- should be recommended if no contraindications after previous low transverse incision
- success rate varies with indication for previous C/S (generally 60-80%)
- risk of uterine rupture (<1% with low transverse incision), increased by interval <18 mo and one layer closure

Contraindications
- previous classical, inverted T, or unknown uterine incision, or complete transection of uterus (6% risk of rupture)
- history of uterine surgery (e.g. myomectomy) or previous uterine rupture
- multiple gestation
- non-vertex presentation or placenta previa
- inadequate facilities or personnel for emergency C/S

VBAC*
- Rate of successful VBAC ranges from 60-82%
- No significant difference in maternal deaths or hysterectomies between VBAC or C/S
- Uterine rupture more common in VBAC group
- Evidence regarding fetal outcome is lacking

*Safety of vaginal birth after Cesarean section: A systematic review. *Obstet Gynecol* 2004;103:420-9

Puerperal Complications

- puerperium: 6 wk period of adjustment after pregnancy when pregnancy-induced anatomic and physiologic changes are reversed

Postpartum Hemorrhage

Definition
- loss of >1000 ml of blood or bleeding associated with signs/symptoms of hypovolemia within 24 hours of birthing process regardless of mode of delivery
- primary – within first 24 h postpartum
- secondary – after 24 h but within first 12 wk

Epidemiology
- incidence 5-15%

Etiology (4 Ts)
1. Tone (uterine atony)
- most common cause of PPH (70-80%)
- avoid with active management of 3rd stage of labour with 1) oxytocin administration 2) uterine massage 3) umbilical cord traction
- due to:
  - overdistended uterus (polyhydramnios, multiple gestations, and macrosomia)
  - uterine muscle exhaustion (prolonged or rapid labour, grand multiparity, oxytocin use, and general anesthetic)
  - uterine distortion (fibroids)
  - intra-amniotic infection (fever or prolonged ROM)
  - bladder distension (preventing uterine contraction)

2. Tissue
- retained placental products (membranes, cotyledon, or succenturiate lobe)
- retained blood clots in an atonic uterus
- gestational trophoblastic neoplasia
- abnormal placentation (e.g. placenta previa or placental abruption)

3. Trauma
- laceration (vagina, cervix, or uterus), episiotomy, hematoma (vaginal, vulvar, or retroperitoneal), uterine rupture, and uterine inversion

4. Thrombin
- coagulopathy (pre-existing or acquired)
  - most identified prior to delivery (low platelets increases risk)
  - includes hemophilia, DIC, ITP, TTP, and vWD (most common)
  - therapeutic anti-coagulation

Uterine atony is the most common cause of PPH

DDx of Early PPH – 4 Ts
Tone (atony)
Tissue (retained placenta, clots)
Trauma (laceration, inversion)
Thrombin (coagulopathy)

DDx of Late PPH
Retained products
± endometritis
Sub-involution of uterus

Investigations
- assess degree of blood loss and shock by clinical exam
- explore uterus and lower genital tract for evidence of atony, retained tissue, or trauma
- may be helpful to observe red-topped tube of blood – no clot in 7-10 min indicates coagulation problem

Management
- ABCs, call for help
- 2 large bore IVs, run crystalloids wide open
- CBC, coagulation profile, cross and type pRBCs
- treat underlying cause
- Foley catheter to empty bladder and monitor urine output

Medical Therapy
- oxytocin 10 IU IM is preferred in low-risk vaginal deliveries, oxytocin IV infusion (20-40 IU in 1000 mL crystalloid at 150 mL/h) is an acceptable alternative. Oxytocin 5-10 IU IV bolus (20-40 IU in 250 mL crystalloid) can be used after vaginal birth, but not with elective C/S
- carbetocin, a long-acting oxytocin, 100 μg IV bolus over 1 min for elective C/S or 100 ug IM for vaginal deliveries with 1 risk factor for PPH (instead of a continuous oxytocin infusion)
- methylergonavine maleate (ergotamine) 0.25 mg IM/IMM q15min up to 1.25 mg; can be given as IV bolus of 0.125 mg (may exacerbate HTN)
- carboprost (Hemabate®), a synthetic PGF-1α analog, 250 μg IM/IMM q15min to max 2 mg (major prostaglandin side effects and contraindicated in cardiovascular, pulmonary, renal, and hepatic dysfunction)
- misoprostol 600-800 μg PO/SL (faster) or PR/PV (side effect: pyrexia if >600 μg)
- tranexamic acid (Cyklokapron®), an antifibrinolytic, 1 g IV

Local Control
- bimanual massage: elevate the uterus and massage through patient's abdomen
- uterine packing (mesh with antibiotic treatment)
- Bakri Balloon for tamponade: may slow hemorrhage enough to allow time for correction of coagulopathy or for preparation of an OR

Surgical Therapy (Intractable PPH)
- D&C (beware of vigorous scraping, which can lead to Asherman's syndrome)
- embolization of uterine artery or internal iliac artery by interventional radiologist
- laparotomy with bilateral ligation of uterine artery (may be effective), ovarian artery, or hypogastric artery, compression sutures (B-Lynch or Cho sutures)
- hysterectomy last option, with angiographic embolization if post-hysterectomy bleeding

Retained Placenta

Definition
- placenta undelivered after 30 min postpartum

Etiology
- placenta separated but not delivered
- abnormal placental implantation (placenta accreta, placenta increta, and placenta percreta)

Risk Factors
- placenta previa, prior C/S, post-pregnancy curettage, prior manual placental removal, and uterine infection

Clinical Features
- risk of PPH and infection

Investigations
- explore uterus
- assess degree of blood loss

Management
- 2 large bore IVs, type and screen
- Brandt maneuver (firm traction on umbilical cord with one hand applying suprapubic pressure cephalad to avoid uterine inversion by holding uterus in place)
- oxytocin 10 IU in 20 mL NS into umbilical vein
- manual removal if above fails
- D&C if required (U/S guidance if available)
- cefazolin 2 g IV if manual removal or D&C

Uterine Inversion

Definition
- inversion of the uterus through cervix ± vaginal introitus

Etiology/Epidemiology
- often iatrogenic (excess cord traction with fundal placenta)
- excessive use of uterine tocolytics
- more common in grand multiparous women (lax uterine ligaments)
- 1/1500-1/2000 deliveries

Clinical Features
- can cause profound vasovagal response with bradycardia, vasodilation, and hypovolemic shock
- shock may be disproportionate to maternal blood loss

Management
- urgent management essential, call anesthesia
- ABCs: initiate IV crystalloids
- can use tocolytic drug (see *Preterm Labour*, OB16) or nitroglycerin IV to relax uterus and aid replacement
- replace uterus without removing placenta
- remove placenta manually and withdraw slowly
- IV oxytocin infusion (only after uterus replaced)
- re-explore uterus
- may require general anesthetic ± laparotomy

Postpartum Pyrexia

Definition
- fever >38°C on any 2 of the first 10 d postpartum, except the 1st day

Etiology
- endometritis
- wound infection (check C/S and episiotomy sites)
- mastitis/engorgement
- UTI
- atelectasis
- pneumonia
- DVT or pelvic thrombophlebitis

Etiology of Postpartum Pyrexia

B-5W
Breast: engorgement, mastitis
Wind: atelectasis, pneumonia
Water: UTI
Wound: episiotomy, C/S site infection
Walking: DVT, thrombophlebitis
Womb: endometritis

Investigations
- detailed history and physical exam, relevant cultures
- for endometritis: blood and genital cultures
- serum lactic acid for early detection of sepsis

Treatment
- depends on etiology
  - infection: empiric antibiotics, adjust when sensitivities available
- endometritis: clindamycin + gentamicin IV
- mastitis: cloxacillin or cephalexin
- wound infection: cephalexin + frequent sitz baths for episiotomy site infection
  - DVT: anticoagulants
- prophylaxis against post-C/S endometritis: administer 2 g cefazolin IV 30 min prior to skin incision

Risk Factors for Endometritis
C/S, intrapartum chorioamnionitis, prolonged labour, prolonged ROM, multiple vaginal examinations

ENDOMETRITIS
- definition: inflammation of the endometrium most commonly due to infection
- clinical features: fever, chills, abdominal pain, uterine tenderness, foul-smelling vaginal discharge, or lochia
- treatment: depends on infection severity; oral antibiotics if well, IV antibiotics with hospitalization in moderate to severe cases

VENOUS THROMBOEMBOLISM
- see *Venous Thromboembolism*, OB30

Mastitis

- **definition**: inflammation of mammary glands
- must rule out inflammatory carcinoma, as indicated
- differentiate from mammary duct ectasia: mammary duct(s) beneath nipple clogged and dilated ± ductal inflammation ± nipple discharge (thick, grey to green), often postmenopausal women

Table 23. Lactational vs. Non-Lactational Mastitis

	Lactational	Non-Lactational
Epidemiology	More common than non-lactational Often 2-3 wk postpartum	Periductal mastitis most common Mean age 32 yr
Etiology	*S. aureus*	May be sterile May be infected with *S. aureus* or other anaerobes Smoking is risk factor May be associated with mammary duct ectasia
Symptoms	Unilateral localized pain Tenderness Erythema	Subareolar pain May have subareolar mass Discharge (variable colour) Nipple inversion
Treatment	Heat or ice packs Continued nursing/pumping Antibiotics (cloxacillin/cephalexin) (erythromycin if penicillin-allergic)	Broad-spectrum antibiotics and I&D Total duct excision (definitive)
Abscess	Fluctuant mass Purulent nipple discharge Fever, leukocytosis Discontinue nursing, IV antibiotics (nafcillin/oxacillin), I&D usually required	If mass does not resolve, fine-needle aspiration to exclude cancer and U/S to assess presence of abscess Treatment includes antibiotics, aspiration, or I&D (tends to recur) May develop mammary duct fistula A minority of non-lactational abscesses may occur peripherally in breast with no associated periductal mastitis (usually *S. aureus*)

Postpartum Mood Alterations

POSTPARTUM BLUES

- 40-80% of new mothers, onset day 3-10; extension of the "normal" hormonal changes and adjustment to a new baby
- self-limited, should resolve by 2 wk
- manifested by mood lability, depressed affect, increased sensitivity to criticism, tearfulness, fatigue, irritability, poor concentration/despondency, anxiety, and insomnia

POSTPARTUM DEPRESSION

- **definition**: major depression occurring in a woman within 6 mo of childbirth (see Psychiatry, PS12)
- **epidemiology**: 10-15%, risk of recurrence 50%
- **risk factors**
  - personal or family history of depression (including PPD)
  - prenatal depression or anxiety
  - stressful life situation
  - poor support system
  - unwanted pregnancy
  - colicky or sick infant
- **clinical features**: suspect if the "blues" last beyond 2 wk, or if the symptoms in the first 2 wk are severe (e.g. extreme disinterest in the baby, suicidal or homicidal/infanticidal ideation)
- **assessment**: Edinburgh Postnatal Depression Scale or others
- **treatment**: antidepressants, psychotherapy, supportive care, and ECT if refractory
- **prognosis**: interferes with bonding and attachment between mother and baby, so it can have long-term effects

POSTPARTUM PSYCHOSIS

- **definition**: onset of psychotic symptoms over 24-72 h within first month postpartum, can present in the context of depression
- **epidemiology**: rare (0.2%)

Postpartum Care

Postpartum Office Visit at 6 Weeks

Care of Mother (The 10 Bs)

- **B**e careful: do not use douches or tampons for 4-6 wk post-delivery
- **B**e fit: encourage gradual increases in walking, Kegel exercises
- **B**irth control: assess for use of contraceptives
- **B**reastfeeding is NOT an effective method of birth control (see Gynecology, GY15, for more detail about different contraceptive options postpartum)
- **B**ladder: assess for urinary incontinence, maintain high fluid intake
- **B**lood pressure: especially if gestational HTN
- **B**lood tests: CBC (for anemia if had PPH)
- **B**lues: (see *Postpartum Mood Alterations*, OB47)
- **B**owel: fluids and high-fibre foods, bulk laxatives; for hemorrhoids/perineal tenderness: pain meds, doughnut cushion, sitz baths, and ice compresses
- **B**reast and pelvic exam: watch for Staphylococcal or Streptococcal mastitis/abscess, ± Pap smear at 6 wk if due for screening

The acronym "**BUBBLES**" for what to ask about when rounding on postpartum care. Modify this for C/S or vaginal delivery

Baby care and breastfeeding-- Latch? Amount?
Uterus – firm or boggy?
Bladder function – Voiding well? Dysuria?
Bowel function – Passing gas or stool? Constipated?
Lochia or discharge – Any blood?
Episiotomy/laceration/incision – Pain controlled?
Symptoms of VTE – Dyspnea? Calf pain?

Physiological Changes Postpartum

- uterus weight rapidly diminishes through catabolism, cervix loses its elasticity and regains firmness
  - should involute ~1 cm below umbilicus per day in first 4-5 d, reaches non-pregnant state in 4-6 wk postpartum
- ovulation resumes in ~45 d after giving birth, non-lactating women usually ovulate sooner than lactating women
- lochia: normal vaginal discharge postpartum, uterine decidual tissue sloughing
  - decreases and changes in colour from red (lochia rubra; presence of erythrocytes, 3-4 d) → pale (lochia serosa) → white/yellow (lochia alba; residual leukorrhea) over 3-6 wk
- foul-smelling lochia suggests endometritis

Breastfeeding Problems

- inadequate milk: consider domperidone
- breast engorgement: cool compress, manual expression/pumping
- nipple pain: clean milk off nipple after feeds, moisturizer, topical steroid if needed
- mastitis: treat promptly (see *Postpartum Pyrexia*, OB46)
- inverted nipples: makes feeding difficult
- maternal medications: may require pediatric consultation (see *Breastfeeding and Drugs*, OB48)

Bladder Dysfunction

- pelvic floor prolapse can occur after vaginal delivery
- stress or urge urinary incontinence common
- increased risk with instrumental delivery or prolonged second stage
- conservative management for stress and urge incontinence: pelvic floor retraining with Kegel exercises/pelvic physiotherapy, vaginal cones or pessaries, and lifestyle modifications (e.g. limit fluid, caffeine intake)
- surgical management for stress incontinence: midurethral slings including retropubic tension free vaginal tape (TVT) or transobturator tape (TOT), retropubic urethropexy (Burch), urethral bulking

Puerperal Pain

- "after pains" common in first 3 d due to uterine contractions; encourage simple analgesia
- ice packs can be used on perineum if painful
- encourage regular analgesia and stool softener

Breastfeeding and Drugs

Table 24. Drug Safety During Breastfeeding

Safe During Breastfeeding	Contraindicated When Breastfeeding
Analgesics (e.g. acetaminophen, NSAIDs)	Chloramphenicol (bone marrow suppression)
Anticoagulants (e.g. heparin)	Cyclophosphamide (immune system suppression)
Antidepressants (e.g. sertraline, fluoxetine, tricyclic antidepressants)	Sulphonamides (in G6PD deficiency, can lead to hemolysis)
Antiepileptics (e.g. phenytoin, carbamazepine, valproic acid)	Nitrofurantoin (in G6PD deficiency, can lead to hemolysis)
Antihistamines	Tetracycline
Antimicrobials (e.g. penicillins, aminoglycosides, cephalosporins)	Lithium
β-adrenergics (e.g. propanolol, labetalol)	Phenindione
Insulin	Bromocriptine
Steroids	Anti-neoplastics and immunosuppresants
OCP (low dose) – although may decrease breast milk production	Psychotropic drugs (relative contraindication)

Common Medications

Table 25. Common Medications

Drug Name (Brand Name)	Dosing Schedule	Indications/Comments
betamethasone valerate (Celestone®)	12 mg IM q24h x 2 doses	Enhancement of fetal pulmonary maturity for PTL
carboprost (Hemabate®)	0.25 mg IM/IMM q15min Max 2 mg	Treatment of uterine atony
cefazolin	2 g IV then 1 g q8h	GBS prophylaxis (penicillin allergic and not at risk for anaphylaxis)
clindamycin	900 mg IV q8h	Used in endometritis
dexamethasone	6 mg IM q12h x 4 doses	Enhancement of fetal pulmonary maturity for PTL
dinoprostone (Cervidil®: PGE2 impregnated thread)	10 mg PV (remove after 12 h) Max 3 doses	Induction of labour Advantage: can remove if uterine hyperstimulation
doxylamine succinate (Diclectin®)	2 tabs qhs + 1 tab qam + 1 tab qpm Max 8 tabs/d	Each tablet contains 10 mg doxylamine succinate with vitamin B6 Used first-line for N/V in pregnancy, including hyperemesis gravidarum
erythromycin	500 mg IV q6h	GBS prophylaxis (penicillin allergic and at risk for anaphylaxis)
folic acid	0.4-1 mg PO OD x 1-3 mo preconception and T1 5 mg PO OD with past Hx of NTD/risks for NTD	Prevention of ONTD
methotrexate	50 mg/mL IM or 50 mg PO x 1 dose	For ectopic pregnancy or medical abortion
methylergonavine maleate (Ergotamine®)	0.25 mg IM/IMM q15min up to 1.25 mg or IV bolus 0.125 mg	Treatment of uterine atony
misoprostol (Cytotec®)	600-1000 µg PR x 1 dose 400 µg PO/SL x 1 dose or 800 µg PV x 1 dose 3-7 d after methotrexate	For treatment of PPH For medical abortion/retained products of conception
oxytocin (Pitocin®)	0.5-2.0 mU/min IV or 10 U/L NS increase by 1-2 mU/min q20-60min Max 36-48 mU/min 10 IU IM at delivery of anterior shoulder and of placenta 20 IU/L NS or RL IV continuous infusion	Augmentation of labour (also induction of labour) Prevention of uterine atony Treatment of uterine atony
Penicillin G	5 million IU IV, then 2.5 million IU IV q4h until delivery	GBS prophylaxis
PGE2 gel (Prostin® gel)	0.5 mg PV q6-12h; max 3 doses	Induction of labour
Rh IgG (Rhogam®)	300 µg IM x 1 dose	Given to Rh-negative women Routinely at 28 wk GA Within 72 h of birth of Rh+ fetus Positive Kleihauer-Betke test With any invasive procedure in pregnancy Ectopic pregnancy Antepartum hemorrhage Miscarriage or therapeutic abortion (dose: 50 µg IM only)

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