CASE FOUR

Short case number: 3_13_4
Category: Reproductive
Discipline: Obstetrics and Gynaecology
Setting: Hospital-labour ward
Topic: Prematurity and neonatal resuscitation

Case

Kate Sobel, aged 31 years, delivers a male infant at 30 weeks gestation. Kate had presented in preterm labour 72 hours earlier and was treated with nifedipine and corticosteroids, labour then progressed to SVD.

Questions

1. Describe the diagnosis of preterm labour and in a flow chart summarise the management of preterm labour.
2. Why did Kate administered nifedipine and corticosteroids?
3. List the risk factors for preterm birth
4. What are the main problems of prematurity?
5. Summarise the foetal adaptation to birth.
6. The newborn baby has an Apgar score of 3 and 6 at 1 and 5 minutes respectively. What is an Apgar score and summarise the principles of resuscitation of the newborn.

Suggested Reading

- Abbott, J., Bowyer, L., & Finn, M. (2014). *Obstetrics and Gynaecology: an evidence-based guide (2nd ed).* Australia, Elsevier.
- Edmonds K, editor. Dewhurst's Textbook of Obstetrics and Gynaecology. 8th Edition. Wiley-Blackwell; 2012.

Students should view the neonatal eHandbook available at:
https://www2.health.vic.gov.au/hospitals-and-health-services/patient-care/perinatal-reproductive/neonatal-ehandbook/procedures/resuscitation (accessed Oct 2018)

ANSWERS

1. Management of preterm labour

Preterm labour is diagnosed when painful contractions + cervical dilation/effacement persist prior to 37 weeks gestation.

Foetal Fibronectin test — a glycoprotein found in liquor and placental tissue. The test is more useful for negative predictive value of 99% in predicting labour in next 7-10 days.

2.

Labour: Threatened Preterm Clinical Practice Guideline

Definition

Uterine contractions present (> 20 weeks and <37 weeks gestation)

Assessment

Consider:

- Evidence of APH (Placental abruption or praevia)
- Are the membranes intact (Refer to CPG PPROM)
- Evidence of chorioamnionitis
- Evidence of maternal systemic infection e.g. UTI
- Evidence of maternal systemic illness
- Evidence of uterine anomaly
- Evidence of polyhydramnios
- Past obstetric and gynaecological history
- Evidence of IUGR

Examination

- Uterine tenderness, confirm contractions (frequency), FHR –CTG
- Speculum examination: SRM, Cervical dilatation, fFN, MC&S

Investigations

Initial investigations:

- Foetal Fibrinectin (fFN), FBE, Cervical, high vaginal swabs,
- U/S scan, Presentation, Biometry, Anomaly, AFI, Doppler studies of umbilical artery, Cervical morphology (status)

Management

Consider transfer to tertiary unit (where NICU facilities) if fFN positive or preterm.

In peripheral areas consideration is given to difficulties of transporting patient and costs and delivery during transfer. All cases are discussed with referral centre on-call obstetrician.

If labour obvious, if delivery not imminent, transport under tocolysis possible in some areas otherwise NETS retrieval.

If foetal distress or any evidence of foetal compromise allow labour to continue or LSCS NETS retrieval.

- Hourly maternal observations
- IV access
- Commence continuous Electronic Foetal Monitoring (EFM) if gestational age > 25 weeks

If fFN negative

- Withhold tocolytics unless uterine activity increases in frequency / severity over the following two hours or documented changes in cervix.
- If tocolytics required commence Nifedipine (Refer to CPG Tocolytics) continue whilst steroid cover administered
- If steroids are required to promote foetal lung maturity; administer betamethasone 11.4mg (IM) Daily, X 2 injections, 12-24 hours apart
- Antibiotics not indicated in the presence of intact membranes and absence of evidence of infection

Subsequent management

- If symptoms of PTL persist for four hours following initial presentation re-evaluate the patient including cervical assessment
- If established in labour – management re preterm labour, avoid narcotics which can depress infant respiration.

Clinical Practice Guidelines (CPGs) are intended to provide guidance to health care professionals, based on a thorough evaluation of research evidence, on the practical assessment and management of specific clinical issues or situations. The guidelines allow some flexibility on the part of the health care professional based on the needs of the specific patient for whom they are caring.

3. Nifidipine and steroids

Tocolysis — medication to stop uterine contractions is given while course of steroids is administered.
Nefidipine (calcium channel blocker) lower side effects, ease administration.
Also available betamimetics (salbutamol, given IV, terbutaline), nitric oxide donors (nitroglycerine), oxytocin receptor antagonists (ritodrine).

Steroids — mature foetal lung structure and increase production of surfactant and prevent neonatal mortality, respiratory distress syndrome and reduce risk of intraventricular haemorrhage when given 26-34+6 weeks gestation.
Betamethasone 11.4mg, 2 injections 12-24 hours apart.

Risk factors for preterm birth.

- Elective — IUGR. Foetal compromise. APH, continuing haemorrhage with placenta praevia. Maternal medical conditions; renal and heart disease.

- Spontaneous — previous preterm delivery, association with poverty, smoking, poor antenatal care, young maternal age and skin colour black.

- Previous cervical surgery, Uterine abnormality, Chorioamnionitis, abruption.

- Polyhydramnios and multiple pregnancies. 40% preceded by PPROM

4. Main problems of prematurity

- Survival rate proportional to maturity
- Risk life altering disability – risk of CP, developmental delays
- Respiratory distress syndrome, Chronic Lung Disease (sec to ventilation)
- Hypothermia
- Hypoglycaemia
- Jaundice
- Patent ductus arteriosis persists
- Necrotising enterocolitis
- Retinopathy of prematurity
- Intraventricular haemorrhage
- Sepsis / Infection
- Feed intolerance (GORD)
- Need resuscitation at birth
- Prolong hospital stay (separation from family - Psychosocial)
- Apnoea of prematurity
- Anaemia

5. Foetal adaptation at birth.

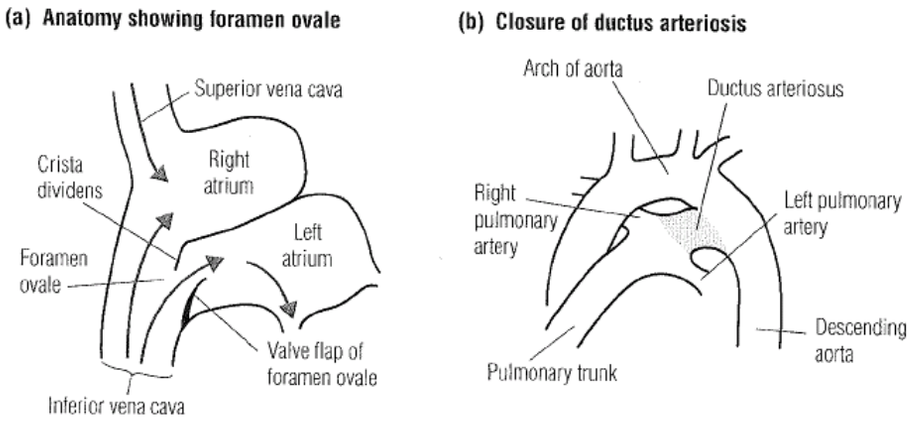
The newborn infant

Adaptation at birth

At birth the fetus undergoes a transition from an environment in which the gas exchanges, nutrition and thermoregulation are provided by the placenta to one in which it is self-sufficient for these functions. The physiological processes which permit this transition begin well before birth and include reabsorption of lung liquid, synthesis and release of pulmonary surfactant, and dramatic changes in hormonal milieu which alter the function of nearly every fetal tissue and organ. The changes that occur at birth are sudden and dramatic, and include:

- The onset of respiration when the lungs become filled with air and lung fluid is absorbed
- The falling of the right ventricular pressure as the lungs expand and the pulmonary arterial pressure is reduced
- An increase in the systemic vascular resistance and left ventricular pressure when the placental circulation is removed and surface cooling occurs
- Flow of blood now from left to right through the foramen ovale—this will cause its closure (Figure 8.1)
- Decrease in the blood flow through the ductus arteriosus as the pressures on the left and right sides of the heart become equal
- Closure of the ductus arteriosus as a result of constriction of the ductal smooth muscle (Figure 8.1)

FIGURE 8.1 Changes in the heart at birth. With the first breath the lungs expand and the pulmonary vessels increase in size, the ductus arteriosus closes by muscular spasm and the amount of blood flowing through the lung vessels rapidly increases. The pressure in the left atrium rises, and the pressure in the right atrium decreases. The septum primum is then apposed to the septum secundum and the ovale foramen closes functionally.



- Increase in glycogenolysis
- Onset of thermoregulation
- Activation of gut motility.

Maladaptation of this process has important implications and may result in a variety of clinical conditions, including:

- neonatal depression/perinatal asphyxia
- meconium aspiration syndrome (MAS)
- transient tachypnoea of newborn (TTN)
- respiratory distress syndrome (RDS)
- persistent pulmonary hypertension of newborn (PPHN)
- patent ductus arteriosus (PDA)
- polycythaemia/hypervolaemia.

6. What is an Apgar score and summarise the principles of resuscitation of the newborn (continued ...)

TABLE 8.1 The Apgar score

Sign	0	1	2
Heart rate	Absent	< 100/min	> 100/min
Respiratory effort	Absent	Weak cry	Strong cry
Muscle tone	Limp	Some motion	Cry
Reflex irritability (suctioning pharynx)	No response	Some motion	Cry
Colour	Pale Overall cyanosis	Centrally pink Otherwise blue	Pink

cycles of reassessment and decision making. The key to successful neonatal resuscitation is establishment of adequate ventilation. Reversal of hypoxia, acidosis and bradycardia depends on adequate inflation of the fluid-filled lungs with air or oxygen.

1 Prepare for the delivery
- Turn on the radiant warmer on the neonatal resuscitation trolley.
- Ensure that all equipment is present, clean and in working order.
- If advanced resuscitation is anticipated, start the oxygen and low suction flow and ensure that the equipment for suction and intubation is in immediate reach.
- Call for assistance if needed.

2 Receive the baby
- Transfer the baby to the preheated neonatal resuscitation trolley, note the time and turn the timer clock on.
- Dry the baby and remove wet blankets.
- Place the baby on dry warm blankets to prevent heat loss.

3 Assess the baby's condition
- Apgar score—see Table 8.1.

4 Airway (A)
- Position the baby flat on his/her back with the head slightly extended in the sniffing position to ensure a patent airway.
- Suction the airway for secretions as required.
- Provide tactile stimulation by rubbing the soles and slapping the heels of the feet or rubbing the infant's back.
- Administer facial oxygen at the same time.

5 Breathing (B)
- If the baby is cyanosed but breathing, suction the airway, oropharynx then nasopharynx (15–20 kPa) with a No 8 suction catheter to 5 cm.
- Administer facial oxygen (5 lpm) through the corrugated end of the Laerdal bagging system or oxygen tubing with a cupped hand.

- If there are no respirations, suction the airway as above and position the baby in the sniffing position. Create a tight seal over the nose and mouth with the face mask. Initiate positive pressure ventilation via a bag and mask at a rate of 40–60 bpm. Oxygen should be administered at 10 lpm with a Laerdal bag or 5 lpm with an anaesthetic bag.
- If there is not adequate chest wall movement, no air entry and no improvement in colour with ventilation, check the mask fit, airway patency and head position. Consider inserting a pharyngeal (Guedel's) airway and recommence bag and mask ventilation. On arrival of a neonatal registrar/consultant or other team member skilled at intubation, endotracheal intubation and intermittent positive pressure ventilation will be commenced if necessary.

6 Circulation (C)

After 15–30 seconds of effective mask ventilation:

- Assess brachial/umbilical or apical heart rate (6 seconds) to determine the need for external cardiac massage.
- If the heart rate is greater than 80–100 bpm continue appropriate respiratory support.
- If heart rate is less than 60 bpm and not increasing with adequate ventilation, initiate external cardiac massage at a rate of 90 compressions per minute. Find the position for compression by drawing an imaginary line across the nipples—just below that line is the lower sternum. Either the two-finger or thumb technique may be used (Figure 8.2). The sternum should be compressed to a depth of 1–2 cm. The compression-to-ventilation ratio is approximately 1 breath to 3 compressions (120 events per minute) with two operators.

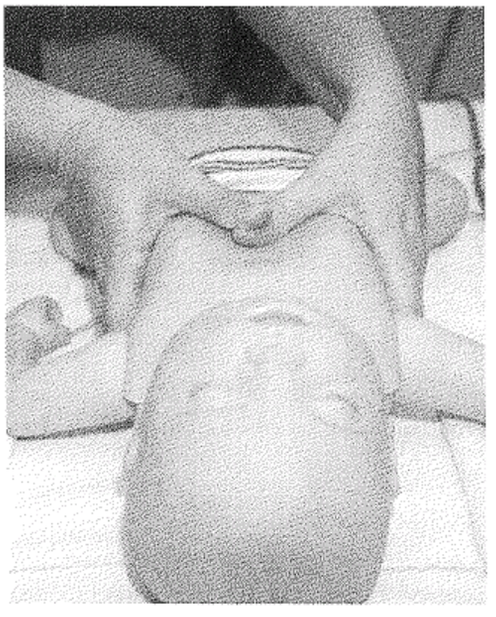


FIGURE 8.2 External cardiac massage, two-thumb technique: the sternum should be compressed by about 1–2 cm in a term baby at a rate of about two compressions per second, and the lungs should be reinflated with oxygen after every three compressions.

- Assess the heart rate at 30 seconds and if greater than 60–100 bpm and increasing with a good pulse and improving perfusion, cease external cardiac massage; otherwise continue the resuscitation.

7 Continued assessment
- Assess brachial/apical pulse at 30 seconds and then at 1-minute intervals during cardiopulmonary resuscitation to ascertain effectiveness of external cardiac compression.
- Do not interrupt the cardiopulmonary resuscitation cycle for more than 10 seconds, to ensure maintenance of oxygenation and circulation.

Meconium aspiration syndrome

Meconium aspiration syndrome (MAS) is a serious and potentially preventable cause of respiratory distress in the newborn. Meconium staining of the amniotic fluid occurs in about 13 per cent of deliveries and might indicate fetal distress, breech presentation or post-term delivery. The possibility of aspiration into the lungs must be taken seriously whether or not it is a sign of fetal distress. Meconium aspiration may occur during labour or at the onset of neonatal respiration. The response of the infant to intrapartum asphyxia is to gasp, and if meconium is present it will be aspirated deep into the bronchi. Once respiration begins, distal migration of the meconium into small airways occurs (Figure 8.3).

Resuscitation (NETS)

Summary

- newborn resuscitation is a critical skill that requires constant practice
- effective ventilation is the key to successful resuscitation
- evaluation and resuscitation interventions are ongoing, continuous and simultaneous processes
- preparation for resuscitation and organization of personnel, particularly assignment of roles is critical

Introduction

Approximately 1 - 10% of in hospital delivered newborns require resuscitation.

The aim of resuscitation is to prevent neonatal death and adverse long term neurodevelopmental sequelae associated with perinatal asphyxia.

Substantial physiologic changes occur in the transition from foetal to extrauterine life including

- the role of the placenta in gas exchange is taken over by the lungs
- changes from fluid-filled to air filled lungs
- dramatic increase in blood flow to the lungs with reversal, then closure of intra and extra cardiac shunts

Failure or disruption of these changes may result in further difficulties with resuscitation in the newborn infant. For example, failure to increase alveolar oxygen and reduce pulmonary vascular resistance may lead to persistence of foetal circulation or pulmonary hypertension.

The phases of asphyxia are well described from experimental evidence in animal models.

- After a few shallow breaths the asphyxiated infant stops breathing. This phase of **primary apnoea** may last for as long as 10 minutes. Most infants with primary apnoea respond to stimulation alone. During this phase heart rate and pH are maintained.
- Following this period, the infant begins to gasp. The period between the last gasp and cardiac arrest is **secondary apnoea**. In the phase of secondary or terminal apnoea the newborn has a mixed acidosis and active intervention is required to stimulate respiration.

It is not possible to clinically distinguish primary from secondary apnoea and for this reason it is important to assume the apnoeic infant is in secondary apnoea. If there is no response to simple interventions the infant requires the immediate commencement of active resuscitation.

Preparation

- **Personnel**
    - at least two trained people are required for adequate resuscitation involving ventilation and cardiac compressions. Therefore, always call for help
    - the most senior person available needs to co-ordinate resuscitation – assign role
    - each person must have a dedicated job, for example with three people, one should be solely responsible for airway, one solely responsible for chest compressions and the third person should co-ordinate the resuscitation and administer medication as necessary. If possible have another person record events including time of administration of drugs, HR response.

- **Check equipment**
    - resuscitation equipment should be checked at least daily and after each usage
    - when use is anticipated at a birth recheck equipment including medical air and oxygen supply, suction, resuscitation equipment, laryngoscope, and endotracheal tubes. If an infant is expected to be in poor condition have medication readily available

- **Communication** is vital to smooth resuscitation
    - with anaesthetic and obstetric staff regarding maternal condition, Foetal condition, maternal therapies
    - if time permits, meet the family before delivery

- **Environment**
    - prevention of heat loss is important
    - where possible deliver infant into a warm draft free environment
    - dry infant, remove wet towelling and replace with dry, warm towels

Assessment

The steps of evaluation and intervention are often simultaneous processes. Evaluation begins immediately after birth and continues throughout the resuscitation process until vital signs have normalized. Key features to evaluate are

- **Respiration**
  The newly born infant should establish regular respirations in order to maintain HR > 100 bpm

- **Heart Rate**
  Determined from direct palpation of cord or with stethoscope. Peripheral pulses are often difficult to feel
  If no pulsation is felt on palpation of the cord do not assume there is no heart beat but auscultate the chest. The HR should be > 100 bpm in a well newly born infant

- **Colour**
  The well newly born infant should be able to maintain a central pink colour in room air; consider acrocyanosis

Management

- **Temperature Control**

A warm draft free environment should be available. Drying the infant with pre-warmed towels will help minimise heat loss in addition to use of a radiant warmer.

Infants less than 28 weeks gestation should be placed immediately after birth in a polyethylene wrap and the body completely covered (appropriate size, food grade, heat resistant).

- **Stimulation**
  Drying with a soft towel will stimulate most newborns to breath & discard the wet towel

If meconium is present in a non-vigorous infant suction under direct vision. Delay tactile stimulation to avoid gasping in the infant with an oropharynx full of particulate meconium.

- **Airway**
  The head should be in a neutral or slightly extended 'sniffing' position.

Suction should not exceed -100 mmHg. It should be limited in depth to 5 cm below the lips.

- **Breathing**
  Attend to adequate inflation and ventilation before oxygenation

The rate for assisted ventilation is 60 bpm
Tidal volume is assessed clinically, that is adequate chest excursion with each breath

Few infants require immediate intubation. The majority of infants can be managed with bag and mask ventilation.

See intubation section for technical details

- **Circulation**

In the majority of infants establishment of adequate ventilation will restore circulation.

Begin chest compressions for either

- absent HR or
- HR < 60 for 30 seconds.

Aim for approximately a ratio of 90 chest compressions to 30 breaths per minute (3:1). (120 events per minute) - count one-and-two-and-three-and-breath etc.

The "two thumb" technique is preferred. Both thumbs meet over the sternum with fingers around the chest wall. The sternum should be compressed to one third of the antero-posterior chest dimension.

- **Medications**

Route of Delivery

- umbilical venous catheter
- ET - for either adrenaline
- peripheral intravenous line - difficult to cannulate in the collapsed infant
- umbilical arterial catheter **should not** be used for drug administration during resuscitation

Adrenaline
For HR < 60 for > 30 sec despite compressions
Dosage: 0.1 -0.3 ml/kg 1 in 10,000 as a quick push IV repeated at 3-5 minutely intervals. It should be followed by a small saline flush. 0.3 - 1.0ml/kg 1in 10,000 via ET.

Volume (preload)
10 - 15 ml/kg normal saline repeated 2 or 3 times

Naloxone
Naloxone does not form part of the initial resuscitation of newborns with respiratory depression in the delivery room.

Dosage - 0.1mg/kg of 0.4mg/ml solution
Contra-indication - infants of narcotic dependent mothers, may result in rapid withdrawal with seizures.

Any infant treated with naloxone should be carefully monitored for several hours as retreatment may be required.

Bicarbonate
Currently there is insufficient evidence for routine use

Argument for correction of acidosis includes theoretical concerns about hypoxia and elevated pulmonary vascular bed pressure and poor cardiac contractility with acidosis.
Argument against correction includes concerns regarding hyperosmolarity and CO_2 generation with intracellular acidosis from alkali infusion.

Stopping Resuscitation

- it is difficult to accurately define a time beyond which active support worsens brain injury
- it is reasonable to consider stopping treatment if the infant has not responded with a spontaneous circulation by 15 minutes of age
- it is helpful to be able to review events during resuscitation and this is made easier when events are recorded during resuscitation

Areas of Uncertainty in Clinical Practice

- Recent animal studies suggest that cerebral hypothermia may be beneficial to the asphyxiated infant. There is not enough current evidence to recommend this practice for routine care. This should only be undertaken in the context of a properly controlled trial

- The question of whether air or oxygen should be used is not fully resolved. Published studies are of variable quality. **If a supply of medical air is not available, oxygen should be used.** Current Australian Resuscitation Guidelines recommend that air should be used initially, with supplemental oxygen reserved for infants whose condition does not improve during the first minutes of life.

- Theoretically, by its effect on lung volume PEEP preserves surfactant function. PEEP sets up FRC and is therefore important in ventilation and oxygenation. It is possible to provide PEEP during the acute by use of either an anaesthetic bag or mask (considerable practice is required to develop competence with this technique) or the Neopuff (this technique can be easily applied but the device will require a flow of gas to operate).

References

Neonatal Resuscitation Flowchart Australian Resuscitation Council December 2010

http://www.neoresus.org.au [Accessed January 2016]

https://resus.org.au/guidelines/flowcharts-3/

Rapid correction of early metabolic acidosis versus placebo, no intervention or slow correction in LBW infants. Kecskes Z, Davies MW Cochrane Database of Systematic Reviews. Issue 1, 2001 http://onlinelibrary.wiley.com/doi/10.1002/14651858.CD002976/abstract [Accessed December 2015]