CHAPTER 11
FETAL HEALTH SURVEILLANCE IN LABOUR

Learning Objectives
By the end of this chapter, the participant will:

1. Define and recognize normal (reassuring), atypical and abnormal (non-reassuring) fetal health status findings.
2. List the possible causes of fetal hypoxia.
3. Describe appropriate monitoring of fetal well being with intermittent auscultation.
4. Describe appropriate intrauterine resuscitation measures

Introduction

Labour is a stress for all fetuses. Uterine contractions decrease uteroplacental blood flow, which may have an impact on oxygen delivery to the fetus. Most fetuses tolerate this reduction in oxygen flow, and therefore experience no adverse effects. Fetal monitoring techniques have been developed to identify fetuses who may not be responding well to the changes in the availability of oxygen.

Definitions

Accurate, non-subjective terms should be used when discussing fetal health

- Hypoxemia - decreased oxygen content in blood
- Hypoxia - decreased oxygen content in tissues
- Acidemia - increased H+ content in blood
- Acidosis - increased H+ content in tissues
- Asphyxia - hypoxia with metabolic acidosis

Unfortunately, the term ”fetal distress” to imply hypoxia or asphyxia has been used inappropriately when fetal heart rate (FHR) monitoring is found to be atypical or “non-reassuring.” The predictive value of FHR monitoring is very low, especially in the low-risk fetus. Some studies showed that up to 80% of all labours have at some point or another atypical (non-reassuring) FHR (Umstad et al, 1994). For this reason, most fetuses that have “abnormal” FHR are actually not experiencing hypoxia or asphyxia. The diagnosis of asphyxia can only be made in retrospect or by using fetal blood scalp sampling. Inappropriate use of the term “fetal distress” may lead to false conclusions that there was a significant intrapartum hypoxic event. This misperception may lead to medico-legal action if the child is subsequently compromised in any way.

Physiology

Fetal oxygenation

Although placental permeability to oxygen is high, fetal oxygen concentration (PO_2) is markedly low compared with maternal (40 mm Hg in umbilical vein vs. 95 mm Hg in maternal artery). However, oxygen saturation and content in the umbilical vein are almost identical to maternal arterial blood. This could be explained by higher hemoglobin concentration and higher affinity for oxygen of fetal blood. The fetal oxygen dissociation curve is shifted to the left and is steeper compared with the maternal curve. This allows the fetus to have a higher oxygen saturation and content at a low PO_2 value, and produces a larger fall in oxygen saturation (releases oxygen to the tissues). Another important compensatory mechanism for the low fetal PO_2 is increased tissue oxygen extraction and a high organ blood flow secondary to high cardiac output (Rurak, 1994).

Uterine contractions during labour decrease uteroplacental blood flow. This results in reduced oxygen delivery to the placenta, and from there to the fetus. Most fetuses tolerate this reduction in oxygen flow and experience no adverse effects. The distribution of oxygen to the fetus depends on the delivery of oxygen from maternal lungs to the uterus and placenta, diffusion from the placenta to fetal blood, and distribution of fetal blood to various fetal tissues through fetal cardiovascular activities. Disturbances in any of these three will reduce availability of oxygen to the fetus.

Maternal factors

Decreased maternal arterial oxygen tension
- Respiratory disease
- Hypoventilation
- Seizures
- Trauma
- Tobacco smoking, including chronic exposure to second-hand smoke

Decreased maternal oxygen carrying capability
- Significant anemia (e.g. iron deficiency, hemoglobinopathies, parasites like malaria)
- Carboxihemoglobin (smokers), poisoning, intoxication, drugs

Decreased uterine blood flow
- Hypovolemia due to blood loss
- Hypotension due to blood loss, sepsis etc.
- Conduction anesthesia such as epidural
- Maternal positioning including dorsal or lithotomy positions

Chronic maternal conditions
- Vasculopathies (e.g., systemic lupus erythematosis (SLE), type I diabetes, chronic hypertension)
- Antiphophoslipid syndrome

Uteroplacental factors
- Excessive uterine activity or uterine hypertonus
- Hyperstimulation secondary to oxytocin, prostaglandins E_2 or normal labour
- Placental abruption
- Uteroplacental dysfunction
- Placental infarction dysfunction, marked by intrauterine growth restriction, oligohydramnios, or abnormal Doppler studies
- Chorioamnionitis
- Anomalies of the uterus (fibroids, septa)
- Abnormal implantation of the placenta (i.e. placenta previa) or cord (velamentous insertion)

Fetal factors

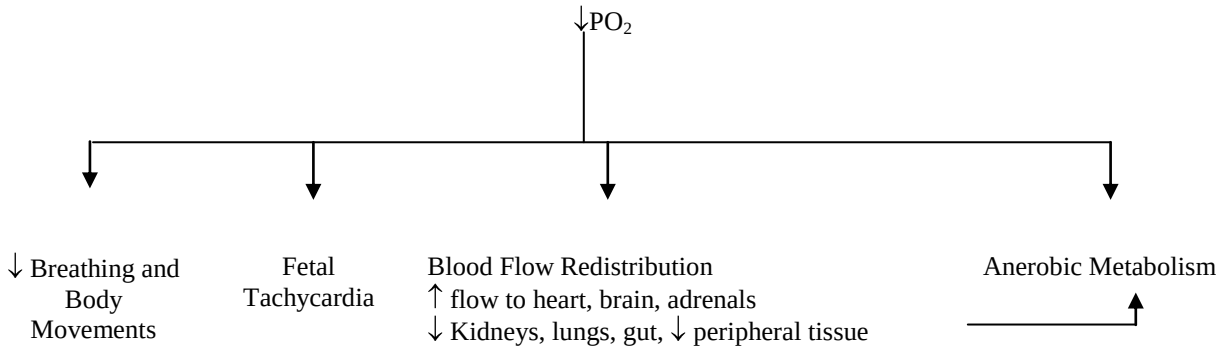
Cord compression
- Oligohydramnios
- Cord prolapse or entanglement

Decreased fetal oxygen carrying capability
- Significant anemia (e.g., isoimmunization, fetomaternal bleed)
- Carboxyhemoglobin (smokers)

Fetal responses to hypoxia

The progression from normal oxygenation to asphyxia is a continuum with progressive changes in vital signs and end-organ effects. The **compensatory responses** of the fetus that is developing asphyxia include:

1. Fetal tachycardia to increase the fetal cardiac output
2. Decreased oxygen consumption through decreased movement, tone, and breathing
3. Redistribution of fetal blood flow
   - Increased flow to the brain, heart, and adrenals
   - Decreased flow to the kidneys, lungs, gut, and liver



Thus, any injury to the fetal brain as a result of intrapartum hypoxia is associated with injury to other organ systems first, except in rare circumstances. Severe metabolic or mixed metabolic acidosis, indicating decompensation with damage to target organs such as lungs, heart, and kidneys, is required to diagnose fetal asphyxia.

If brain injury is to be attributed to intrapartum asphyxia, the following would therefore be expected:

- Depressed neonatal vital signs (Apgar ≤3 at 5 minutes)
- Neonatal neurological sequelae (neonatal encephalopathy) hypotonia, irritability, seizures
- Neonatal multi-organ dysfunction
  - Renal – oliguria, anuria, azotemia
  - Lung – respiratory distress syndrome, pulmonary hypertension
  - Gut – necrotizing enterocolitis
  - Liver – hypoglycemia, elevated liver enzymes, coagulopathy
  - Hematologic – thrombocytopenia, leukopenia
  - Cardiac – cardiomyopathy, patent ductus arteriosus
- Biochemical evidence of severe metabolic acidosis
  - Umbilical artery pH <7.0
  - Umbilical artery base deficit <16 mmol/L

Accordingly, evidence of damage to several end organs, as well as metabolic acidosis, is required before a diagnosis of fetal asphyxia can be assumed.

Neurobiology

Neonatal encephalopathy and its subset of hypoxic-ischemic encephalopathy (HIE) are conditions defined to term infants (>37 completed weeks of gestation) and near-term infants (>34 completed weeks of gestation). The combined incidence of neonatal encephalopathy and HIE is 1.9–3.8 per 1,000. The overall incidence of neonatal encephalopathy attributable to intrapartum abnormalities is approximately 1.6 per 10,000. Although term and near-term infants are at relatively low risk for cerebral palsy (CP) compared with very preterm infants, they still make up at least one-half of all cases of CP. Infants <1,500 grams at birth account for approximately one-quarter of the cases of cerebral palsy. Neonatal encephalopathy can result from many conditions, 90% of which are secondary to events

arising before the onset of labour; prenatal stroke, infection, cerebral malformation, genetic disorders, and other unknown factors. Only 10% of cases of neonatal encephalopathy met what were very nonstringent criteria for intrapartum hypoxia, with another 10% experiencing a significant intrapartum event that may have been associated with intrapartum hypoxia. The pathway from intrapartum HIE to subsequent CP must progress through neonatal encephalopathy (ACOG, 2003).

Ultimately, sustained hypoxia leading to severe metabolic acidosis and cardiovascular decompensation with systemic hypotension may lead to cerebral ischemia and brain damage. Asphyxia may occur at any point in the infant's antepartum, intrapartum, or postpartum life.

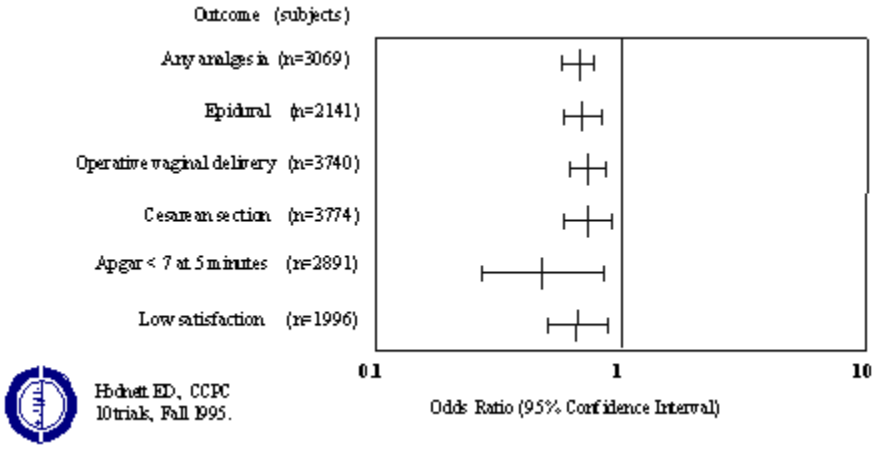
- The type of cerebral injury caused by HIE depends on the nature of the insult, the maturation of the brain, and its vascular development.
  - The term fetus principally sustains injury to the subcortical white matter and cerebral cortex. These "watershed" areas between the end branches of the major cerebral vessels are the regions of the brain at highest risk. Often, this injury involves the motor cortex, especially the proximal extremities and upper extremities. The most frequent consequence of this injury is spastic quadriplegia. Deeper brain substance injury may occur with severe hypoxic or hypotensive insult.
  - The preterm fetus is more susceptible to decreases in cerebral perfusion affecting the periventricular white matter. This region involves descending fibers from the motor cortex. The lesion is called periventricular leukomalacia, and is visible on cranial ultrasound 3 weeks later. Moderate injury is more likely to affect the lower limbs, but severe lesions often involve both extremities. Long-term manifestations include spastic diplegia, spastic quadriplegia, and other visual and cognitive deficits.

Support from Labour Companions

Every woman should have a caring companion with her in labour. Evidence shows that this decreases the duration of labour with benefits to both mother and fetus. A companion also decreases stress and anxiety. This has a potentially positive effect on the baby's compensatory mechanisms. Companionship during labour should be discussed with the woman in the prenatal period. Women should be encouraged and supported by health care facility policy to have their spouse or partner and/or other health companions present. Women without family or social support should be encouraged to seek out a labour companion. Health care providers together with health care facilities should develop and maintain a list of community resources.

Support during childbirth from caregivers has been shown to have a positive impact on labour and delivery, as shown in Figure 1.

Figure 1 – Outcomes related to support from caregivers during labour



Cerebral Palsy

Definition

Cerebral palsy is a chronic motor disorder of cerebral origin characterized by the early onset of abnormal movement or posture not attributable to recognized progressive disease.

"RESEARCH SUPPORTS THAT SPASTIC QUADRIPLEGIA, ESPECIALLY WITH AN ASSOCIATED MOVEMENT DISORDERS, IS THE ONLY TYPE OF CEREBRAL PALSY ASSOCIATED WITH ACUTE INTERRUPTION OF BLOOD SUPPLY. PURELY DYSKINETIC OR ATAXIC CEREBRAL PALSY, ESPECIALLY WHEN THERE IS AN ASSOCIATED LEARNING DIFFICULTY, COMMONLY HAS A GENETIC ORIGIN AND IS NOT CAUSED BY INTRAPARTUM OR PERIPARTUM ASPHYXIA." *(NELSON, 1998)*

Incidence

The incidence of CP at full term is 2–3/1,000 live births and has not changed in the past three or four decades.

Advances in neonatal care have increased survival of extremely premature neonates. This has resulted in an increase in the incidence of CP in the very low-birth weight survivors. However, these infants are of such a small number relative to the overall population that the effect on the total incidence of CP is not significant.

Factors associated with cerebral palsy

- Prematurity
- Intrauterine growth restriction
- Developmental abnormalities
- Metabolic abnormalities
- Autoimmune conditions
- Coagulation disorders
- Infections (clinical chorioamnionitis and severe, not mild, histological chorioamnionitis)
  - In utero infection (primary infection: viral or parasitosis, such as toxoplasmosis)
- Multiple gestation
- Trauma
- Asphyxia – antepartum, intrapartum or neonatal
  - Substance abuse and/or smoking
  - Maternal medical conditions (e.g. hypertension)
  - Placental abnormalities

Criteria to define an acute intrapartum hypoxic event as sufficient to cause cerebral palsy

The Society of Obstetricians and Gynaecologists of Canada criteria

The Society of Obstetricianss and Gynaecologists of Canada (SOGC) clinical practice guideline on fetal health surveillance in labour (Liston et al, 2002a and Liston et al, 2002b) states the following:

Essential criteria of the newborn response to asphyxia of such a degree as to be likely to cause harm are:

- Apgar score 0 to 3 for ≥5 minutes
- Neonatal neurologic sequelae (e.g. hypotonia, seizures, coma)
- Evidence of multi-organ system dysfunction in the immediate neonatal period
- Umbilical cord arterial pH <7.0
- Umbilical cord arterial base deficit <-16 mmol/L

All of these conditions must be present. In cases where such evidence is lacking, one cannot conclude that hypoxic acidemia existed or had the potential to cause neurologic deficits.

The American College of Obstetricians and Gynecologists criteria

The American College of Obstetricians and Gynecologists (ACOG) criteria are similar to those of the SOGC but differ in the base deficit threshold and address criteria that would suggest the intrapartum timing of asphyxia.

Essential criteria (must meet all four)
- Evidence of a metabolic acidosis in fetal umbilical cord arterial blood obtained at delivery (pH <7.0 and base deficit ≤-12 mmol/L)
- Early onset of severe or moderate neonatal encephalopathy in infants born at 34 or more weeks of gestation
- Cerebral palsy of the spastic quadriplegic or dyskinetic type
- Exclusion of other identifiable etiologies such as trauma, coagulation disorders, infectious conditions, or genetic disorders

Criteria that collectively suggest intrapartum timing (within close proximity to labour and delivery, e.g. 0 to 48 hours) but are non-specific to asphyxial insults
- A sentinel (signal) hypoxic event occurring immediately before or during labour (ex.: abruptio)
- A sudden and sustained fetal bradycardia or the absence of FHR variability in the presence of persistent, late or variable decelerations, usually after a hypoxic sentinel event when the pattern was previously normal
- Apgar scores of 0 to 3 beyond 5 minutes
- Onset of multi-system involvement within 72 hours of birth
- Early imaging study showing evidence of acute nonfocal cerebral abnormality

Although it is unlikely that neurological deficit could be correlated with acidemia, indicated by a base deficit of ≤-6 mmol/L, the ACOG Task Force chose a base deficit of ≤-12 mmol/L as a reasonable criterion because of occasional cases where the base deficit was in the range of -12 to -15. Many colleges of obstetrics and gynaecology, general practice, and midwifery around the world endorsed this consensus.

Fetal Heart Rate Monitoring in Labour

The goal of intrapartum fetal monitoring is to detect potential fetal decompensation, and to allow timely and effective intervention to prevent damage or death. The fetal brain is the primary organ of interest but at present it is not clinically feasible to assess its function. However, the FHR can be assessed. The fact that changes in FHR precede brain injury is the rationale for FHR monitoring. Timely response to abnormal FHR patterns might be effective in preventing brain injury.

During the contractions of normal labour, there is a decrease in uteroplacental blood flow. In the fetus with normal uteroplacental function, this results in an increase in PCO_2 and a decrease in PO_2 and pH. These values do not fall outside of normal values, and the fetus does not display any changes in heart rate. In the fetus with diminished uteroplacental function, the decreased blood flow results in an increase in PCO_2 and a decrease in PO_2 and pH that exceed some critical thresholds, and the fetus may show changes in heart rate.

During the course of normal labour, there is a gradual increase in PCO_2 and a slight decrease in PO_2 and pH. These changes are normal and generally well tolerated. However, even a fetus with normal uteroplacental function may be challenged by a long labour or by excessive uterine contractions.

Two methods of FHR monitoring exist:
- Intermittent auscultation (IA)
- Electronic fetal monitoring (EFM)

Assessment of the FHR by IA in the time period immediately after a contraction will allow for the detection of significant changes in the FHR. The FHR does not need to be sought during a contraction. Repetitive decelerations detected by IA may be further clarified by the changing to EFM, if available. Assessment of fetal well-being also requires consideration of the "whole picture," including antepartum concerns or intrapartum events that may have impact on the status of this fetus in labour.

Fetal monitoring is a screening tool only. When atypical (non-reassuring) FHRs are heard, further diagnostic tests such fetal scalp sampling should be used to help with management, if available. Other intrapartum interventions may also be required.

With either method, it is important that health care providers receive continuing education about and mentoring in both the FHR monitoring techniques and their interpretation. With IA, palpation of contractions and auditory recognition of FHR changes is required. With EFM, health care providers must be able to interpret the EFM findings. Health care facilities should have an institutional policy describing the technique and frequency of monitoring, with defined interventions for atypical (non-reassuring) findings.

Intermittent auscultation

Effective IA requires the presence of 1:1 caregiver attention when auscultation is required every 15 minutes or less.

Frequency of intermittent auscultation

There is no research to demonstrate optimal time intervals for IA. The Society of Obstetricians and Gynaecologists of Canada (SOGC) guidelines for IA are the following:

- Every 30 minutes in the latent phase if the woman has been admitted to a health care facility for medical indication. Most women pass through the latent phase of labour with family support at home; fetal monitoring can be accomplished by assessing fetal movements as perceived by the mother.
- Every 15 to 30 minutes in the active phase
- Every 15 minutes in the second stage before pushing
- Every 5 minutes in the second stage when pushing

Method of intermittent auscultation:

- Palpate the maternal abdomen to identify fetal presentation and position.
- Place the Pinard, fetoscope or Doppler over the area of maximum intensity of the fetal heart sounds (usually over the fetal back).
- Place a finger on the mother's radial pulse to differentiate maternal from FHR.
- To determine a baseline rate auscultate for 1 full minute between contractions. A baseline rate determined by IA is written as a single number, not a range.

Auscultate immediately after a contraction for 1 full minute according to the frequency listed above. However, variations in the frequency and duration of IA monitoring have not been assessed in relation to outcome measurements (ACOG, 2005).

Intermittent auscultation assessment

Assessment includes baseline FHR, rhythm (regular or irregular), and the presence or absence of accelerations or decelerations. The technique of IA cannot assess baseline variability or classify decelerations if present.

Intermittent auscultation and electronic fetal monitoring

EFM was introduced as an attempt to use the correlation between FHR patterns and fetal well-being as a predictor for outcome. However, the power of EFM to predict adverse outcomes, especially in low-risk pregnancies, is poor. When EFM was evaluated against IA, it was found that the fetal outcome was not improved, but the rate of operative delivery including cesarean section was significantly increased. A meta-analysis of the trials comparing IA to EFM is shown in the Figures 2 and 3.

Figure 2 – Effect of electronic fetal monitoring (no scalp sampling) versus intermittent auscultation in labour

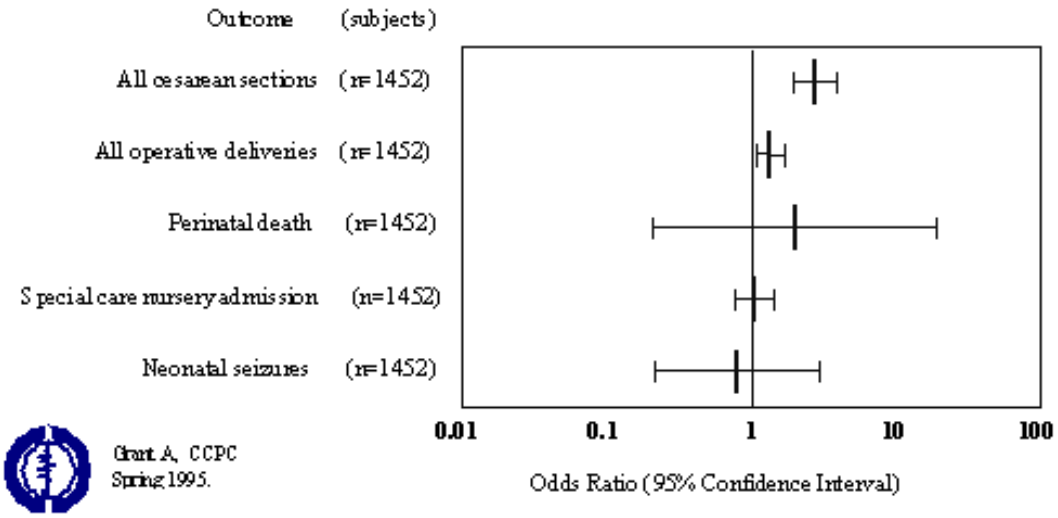
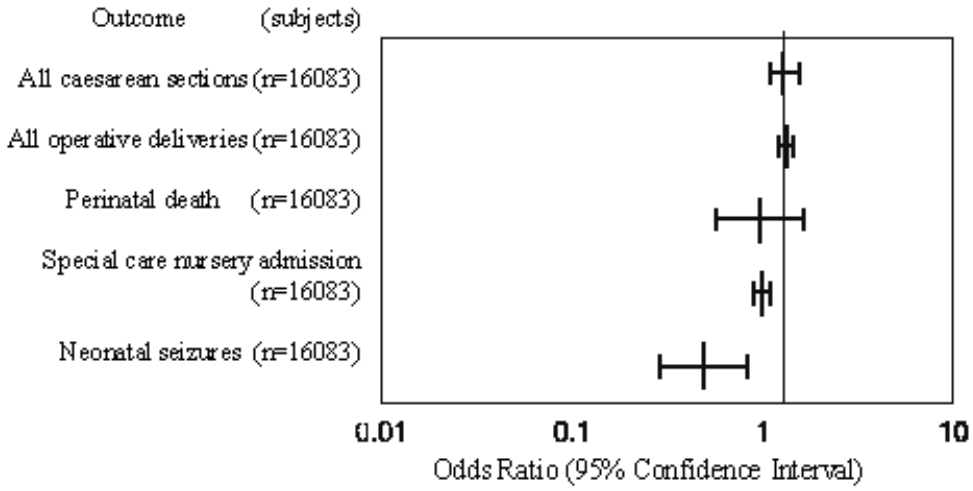


Figure 3 - Effect of electronic fetal monitoring (with scalp sampling) versus intermittent auscultation in labour



IA has been accepted as the standard for evaluating fetal well-being. Neither EFM nor IA has ever been tested against a "placebo," i.e. no intrapartum assessment of the fetus. IA, like EFM, may not be interpreted reliably in all instances.

Every maternity unit or ward must have a written policy on IA and EFM. Both must be used according to an acceptable method with well-defined criteria for documentation and intervention in the case of atypical or abnormal findings.

An important consequence of the surveillance of fetal well-being by auscultation is that a health care provider must be present at the bedside almost continuously. There have been several studies showing the benefits of such a presence.

EFM has not been efficacious in reducing the number of infants with deficits, because most of these deficits are not due to intrapartum events. Even when there is evidence of intrapartum asphyxia, most surviving fetuses experience no long-term deficits. Brain injury, even after an intrapartum asphyxial insult, is frequently due to events that preceded or followed labour. Fetuses with pre-existing brain injury may show atypical (non-reassuring) FHR patterns because of their underlying neuropathology.

If EFM is used, it must be evaluated like any other test for reliability, validity, and causal relationship. Reliability, expressed by intra- and inter-observer agreement of recognition and interpretation of patterns, is very poor. Computer-assisted interpretation produces greater consistency in interpretation, but little improvement in the predictive power of EFM. The Dublin trial illustrated the limitations of validity and causal relationships of both surveillance methods to outcomes (MacDonald, 1985)**.** Initial results indicated a reduction of seizures in the neonates whose labour had been monitored electronically. However, later follow-up found no difference in the incidence of CP among the groups. In addition, most of the children with CP had normal surveillance and were apparently normal neonates. This and other data suggest that the causal association between EFM abnormalities and outcome is far from established

The SOGC and other health care provider associations have concluded that EFM should be replaced by IA in low-risk pregnancies. This conclusion has been reached because the neonatal outcome is the same and the frequency of intervention is decreased with IA compared to EFM. The use of IA requires the frequent or preferably continuous presence of a health care provider during labour. The provision of continuous care will require increased staffing in many maternity wards or units. One-to-one care involves the continuous presence of a health care provider competent in IA.

Health care providers need to develop, evaluate, and maintain their ability to recognize atypical (non-reassuring) FHR patterns. Health care facilities must provide continuing education programs to support the development of these skills.

Electronic fetal monitoring

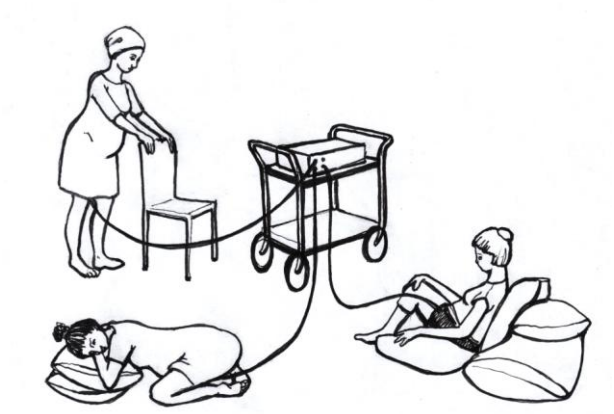


Figure 4 - Electronic fetal heart rate monitoring in different maternal positions

Indications for electronic fetal monitoring in labour

Antepartum

- Maternal
  - Hypertensive disorders of pregnancy
  - Pre-existing diabetes mellitus and/or gestational diabetes
  - Antepartum hemorrhage
  - Maternal medical disease such cardiac, anemia, hyperthyroidism, vascular disease and renal disease
  - Maternal manual vacuum aspiration or other trauma

- Fetal
  - Intrauterine growth restriction
  - Prematurity
  - Oligohydramnios
  - Isoimmunization
  - Multiple pregnancy
  - Breech presentation

Intrapartum

- Maternal
  - Vaginal bleeding in labour
  - Intrauterine infection or chorioamnionitis
  - Previous cesarean section
  - Prolonged rupture of membranes >24 hours at term
  - Preterm labour
  - Induction or augmentation of labour with oxytocin
  - Abnormal FHR detected by auscultation
  - Prolonged labour
  - Post-term pregnancy (>42 weeks)
- Fetal
  - Meconium staining of the amniotic fluid
  - Abnormal FHR on auscultation

There is no evidence to support the performance of routine admission FHR tracings in healthy, term women in labour.

When a normal (reassuring) FHR tracing is identified, EFM may be interrupted of short periods of time to facilitate ambulation, bathing, showering, or position changes providing that (1) the maternal and fetal conditions are stable, and (2) if oxytocin is being administered, the infusion rate is not increased during the period of time when EFM is interrupted.

Systematic interpretation of electronic fetal monitoring

A National Institute of Child Health workshop was convened to develop standardized and unambiguous definitions for FHR (NICH, 1997). Adequate tracing of the FHR and uterine contractions is required. A full description of EFM requires an assessment of maternal risk factors and a qualitative and quantitative description of:

- Uterine activity characteristics (frequency, duration, intensity of contractions and resting tone)
- Baseline FHR
- Baseline FHR variability
- Presence of accelerations
- Periodic or episodic decelerations
- Changes or trends of FHR patterns over time
- Overall assessment of the surveillance, i.e., normal (reassuring), or atypical or abnormal (non-reassuring)

The EFM should be reviewed and documented with the same frequency as described for IA.

Baseline fetal heart rate

Baseline FHR is the approximate mean FHR rounded to increments of 5 beats per minute (bpm) during a 10-minute segment of the tracing, excluding periodic changes and periods of marked variability (>25 bpm). If the baseline FHR is <110 bpm, it is termed bradycardia. If a baseline FHR is >160 bpm, it is termed tachycardia. The presence of either of these findings is atypical (non-reassuring) and requires further assessment.

Fetal heart rate variability

Variability refers to the fluctuations in the baseline FHR. It is determined by choosing 1 minute of a 10-minute section of the FHR tracing with at least 2 cycles/minute (normal is 2 to 4 cycles/minute) that is free from accelerations and decelerations, and measuring the difference between the lowest and highest rate. The difference is the range and amplitude of variability.

- **Absent** variability is undetectable.
- **Minimal** variability is detectable but ≤5.
- **Moderate** or **average** variability is 6–25.
- **Marked** variability is >25 bpm.

These terms are preferred, as opposed to the terms good or poor variability.

Physiologic variability is a normal characteristic of the FHR. Variability of the FHR is largely controlled by the effect of the vagus nerve on the heart. Persistent hypoxia causing acidosis leads to a decrease in FHR rate variability. Other conditions can lead to decreased or absent FHR variability. These conditions include:

- Fetal sleep
- Medications: narcotics, sedatives, β-blockers, betamethasone
- Preterm infant
- Fetal tachycardia
- Congenital anomalies

Moderate FHR variability suggests that the fetus acid–base status is acceptable. Such variability can be appreciated only with EFM and not with auscultation or external Doppler monitoring. Only internal fetal monitoring is capable of accurately recording short-term variability. FHR variability is episodic due to fetal sleep cycles. Variability will therefore be absent intermittently even in the healthy fetus. The appropriate management in such cases is to extend the observation time. Given that pathological loss of variability reflects fetal acidosis, an absence of variability **without** hypoxic decelerations is unlikely to indicate ongoing fetal hypoxia.

Persistent loss of variability requires further assessment. Such variability is best appreciated with internal electronic monitoring.

Fetal heart rate acceleration

An acceleration is an **abrupt** increase (defined as onset to peak of <30 seconds) in FHR 15 bpm above the baseline for 15 seconds (10 bpm for 10 seconds for gestations <32 weeks). A prolonged acceleration is an increase ≥2 minutes, and if the increase is sustained for ≥ 10 minutes it is a change in baseline rate. The presence of accelerations is a normal (reassuring) finding.

Fetal heart rate decelerations

Early decelerations

Early decelerations are defined as a **gradual** decrease in the FHR (defined as onset of deceleration to nadir ≥30 seconds) and return to baseline associated with uterine contraction. The onset, nadir, and recovery of the decelerations are coincident with the beginning, peak, and ending of the contraction, respectively. Early decelerations are felt to be secondary to fetal head compression and are benign.

Variable decelerations

Variable decelerations are defined as an **abrupt** decrease in the FHR with the onset of the deceleration to the nadir usually of <30 seconds. The deceleration should be at least 15 beats below the baseline, lasting for at least 15 seconds, but <2 minutes in duration. Variable decelerations are felt to be a response of the FHR to cord compression and are the most common decelerations seen in labour. Variable decelerations may be further divided into normal (reassuring) decelerations and atypical (non-reassuring) variable decelerations.

Atypical (non-reassuring) features of variable decelerations include:

- Deceleration to <70 bpm lasting >60 seconds
- Loss of variability in the baseline FHR and in the trough of deceleration
- Biphasic deceleration
- Prolonged secondary acceleration (post-deceleration smooth overshoot of >20 bpm increase and/or lasting >20 seconds)
- Slow return to baseline
- Continuation of a baseline at a lower level than prior to the deceleration
- Presence of fetal tachycardia

Late decelerations

Late decelerations are defined as a **gradual, repetitive** decrease in the FHR and return to baseline with the onset of the deceleration to the nadir usually >30 seconds. The onset, nadir, and recovery of the decelerations occur after the beginning, peak, and ending of the contraction, respectively. Late decelerations are atypical (non-reassuring) and sometimes caused by fetal hypoxia and therefore need to be investigated and acted upon.

Prolonged decelerations

A prolonged deceleration is defined as having duration of ≥2 minutes but <10 minutes. If the prolonged deceleration is ≥10 minutes, it is a baseline change.

Documentation

Documentation may consist of narrative notes or the use of comprehensive flow charts such as the World Health Organization partograph. Documentation should provide details of periodic assessments of the woman and her fetus. The following information should be included:

Intermittent auscultation

1. Uterine activity characteristics obtained by palpation
   - Frequency
   - Duration
   - Intensity
   - Relaxation between contractions

2. Fetal heart rate data
   - Numerical baseline rate (in bpm)
   - Rhythm (regular or irregular)
   - Nature of the changes (gradual or abrupt acceleration or deceleration)

3. Interpretation of findings as normal or abnormal and specific actions taken when changes in FHR occur
4. Other maternal observations and assessments
5. Maternal and fetal responses to interventions

Electronic fetal monitoring

1. The indication for initiating EFM
2. Baseline rate, variability, the presence or absence of accelerations, the presence and type of deceleration, and the uterine activity
3. Classification of the tracing as normal, atypical, or abnormal, and specific actions taken when the tracing is atypical or abnormal, including documentation of communication with the primary care provider.
4. Other maternal observations and assessments
5. Maternal and fetal responses to interventions

Results from EFM tracings or findings should be documented in the woman's chart at least hourly in latent labour, q 15 minutes if oxytocin infusing, q 15–30 minutes in active labour, and q 5 minutes in second stage of labour.

Each individual EFM tracing should have maternal data (i.e. name, date of birth, and facility-based patient identification information) noted on the tracing. Each tracing should be identified. If maternal data (e.g. pulse, blood pressure temperature, etc.) is charted on the tracing, it is important that any related notes in the woman's chart have the same details, and that the times recorded are identical. All tracings must be saved as a legal component of the woman's medical record. They should be stored with the woman's chart. They need be stored so that they are readily available if needed in the future.

Reducing Unnecessary Interventions as a Result of Electronic Fetal Monitoring

Efforts to reduce intervention due to false positive EFM should include:
- Limited use of EFM in low-risk pregnancies because of the low prevalence of true fetal compromise
- Use of fetal scalp pH to clarify any atypical (non-reassuring) pattern
- If a scalp pH is not feasible or unavailable, try intrapartum scalp stimulation. FHR acceleration in response to stimulation is a normal (reassuring) finding
- Attention to all aspects of the EFM record, including baseline variability. Good FHR variability means that fetal acidosis is unlikely to be present
- Intrauterine resuscitation in all instances of atypical (non-reassuring) monitoring

Responses to atypical (non-reassuring) fetal surveillance

Atypical (Non-reassuring) variable or late decelerations
- Perform intrauterine resuscitation.
- Confirm fetal well-being.
- Expedite delivery if fetal well-being has not been confirmed.

Bradycardia
- Check maternal pulse, differentiate from FHR.
- Perform a vaginal examination to rule out cord prolapse or to determine if the baby's head is deep in the pelvis ready to be delivered.
- Perform intrauterine resuscitation.
- If cause not obvious or correctable, consider intrapartum ultrasound to evaluate arrhythmia.
- If persistently severe (<100 bpm), expedite delivery.

Tachycardia
- Assess maternal temperature.
- Assess risk of fetal infection (group B streptococcus status, duration of membrane rupture).
- Consider impact of medications.
- Perform intrauterine resuscitation.
- If cause not obvious or correctable, consider intrapartum ultrasound to evaluate arrhythmia.
- If persistently severe and cannot confirm fetal well-being, expedite delivery.

Intrauterine resuscitation

In cases of suspected intrauterine hypoxia-acidosis, intrauterine resuscitation should be undertaken immediately. Simultaneously and with the help of labour companions and other health care providers:
- Call for help.
- Explain the situation to the woman and her family.
- Ask or help the woman to move to the lateral position. Maternal repositioning will often improve uterine blood flow and may relieve cord compression.
- Increase fluid load to correct hypotension or hypovolemia.

- Increase maternal oxygen saturation:
  - Give oxygen, if available. However, there is little evidence to evaluate its effectiveness.
  - Modify breathing techniques to improve oxygen saturation.
  - Modify pushing techniques i.e. encourage woman to breathe through contractions rather than hold her breath for prolonged periods.
- Stop oxytocin administration.
- Perform vaginal examination:
  - Exclude cord prolapse
  - Assess cervical dilatation:
    - Estimate duration of remaining labour to assist in determination of best course of action.
    - If cervix fully dilated, perform operative vaginal delivery, if safely feasible.

Digital fetal scalp stimulation is recommended in the presence of atypical FHR patterns. In the absence of a positive acceleratory response with digital fetal scalp stimulation, fetal scalp blood sampling is recommended, when available. If fetal scalp blood sampling is not available, prompt delivery should be considered depending on the overall clinical condition.

Fetal scalp blood sampling

Indications
Atypical (non-reassuring) IA findings
Atypical (non-reassuring) EFM patterns:
- Unexplained absent baseline variability
- Repetitive late decelerations
- Complex variable decelerations
- Fetal cardiac arrhythmias

Interpretation of fetal scalp pH
Fetal scalp sampling should be performed when indicated in the presence of atypical (non-reassuring) FHR patterns that are not responsive to intrauterine resuscitation. The following thresholds should be used when interpreting fetal scalp pH.
- pH ≥7.25 FSS should be repeated if the FHR abnormality persists.
- pH 7.21– 7.24 Repeat within 30 minutes or consider delivery if rapid fall since last sample.
- pH ≤7.20 Expedite delivery.

Limitations
- Provides only instantaneous, and not continuous, information; repeat sampling may be necessary.
- Technical limitations: operator skill, maternal discomfort, requires cervix to be at least 2 cm dilated, sample contamination with amniotic fluid.
- May be normal in early stages of metabolic acidosis because it takes time before hydrogen ions produced in peripheral tissue to cross to the blood stream.

Contraindications
- Known or suspected fetal blood dyscrasia (hemophilia, von Willebrand's).
- Active maternal infection (HIV, genital herpes).

Umbilical cord blood analysis

The SOGC recommends routine cord gases be taken after all deliveries. This facilitates effective newborn care and quality assurance. The availability of cord gas analysis may reduce the incidence of successful litigation in the event of a poor outcome, particularly when the outcome is delayed (not apparent in the neonate). There are varying recommendations from international organizations about the use of umbilical artery versus the artery and vein sample.

Dr. Virginia Apgar developed the Apgar score as a rapid tool to assess the immediate status of the newborn, not for assessing the degree of asphyxia. The Apgar score alone cannot link birth events to neurological sequelae. A low Apgar score can be associated with various maternal–fetal conditions, e.g. fetal malformation, infection, meconium aspiration, vigorous manipulation of upper airways, and immaturity.

Umbilical cord blood acid–base analysis provides an objective method to evaluate the newborn condition.

Rationale for routine cord gases

The SOGC recommends measuring both umbilical arterial and venous cord blood gases after all deliveries for quality assurance and improvement purposes. It further advises that if only one sample is possible, it should be an arterial sample. The ACOG recommends selective measurement of cord gases and sampling the artery alone (ACOG, 1995). The (British) Royal College of Obstetricians and Gynaecologists recommends selective measurement of acid–base status in the umbilical artery as a minimum (RCOG, 2001).

The objectives of measuring cord pH and acid–base status are to quantify perinatal asphyxia. The use of pH alone will not differentiate between respiratory and metabolic acidosis. In the perinatal period, both respiratory and metabolic acidosis occur in parallel and it is important to identify them separately.

Important Points

1. Delayed clamping of umbilical cord after delivery will affect PCO_2, causing a false increase in base deficit. In one study, 12.9% of all cord pH <7.10 cases had late clamping of the cord, causing a false high base deficit (Westgate et al, 1994).
2. The objectives to sample umbilical artery and umbilical vein are:
   a) Umbilical vein is required for quality control. Without an umbilical vein sample, there is no way to identify sample error. In one study, the rate of sample error was 15% (Westgate et al, 1994). Suggested normal range of difference between vessels is 0.03 in pH and 8 mm Hg in PCO_2.
   b) Identify the type and severity of fetal acidosis. The following example of two cases with similar arterial but not venous values had dissimilar neonatal outcomes. Case A required resuscitation at birth, ventilated for 48 hours and developed CP at 1 year of age. Case B had 5 minutes Apgar score of 8 with no neonatal problems.

	Case A		Case B	
	Artery	Vein	Artery	Vein
pH	7.03	7.10	7.04	7.32
PCO_2 (mm Hg)	63	50	67	38
PO_2 (mm Hg)	6.8	20	13.5	34
BE (mmol/L)	-12.5	-12.6	-11.2	-5.5

Source: Westgate J et al. *Br J Obstet Gynaecol* 1994;101(12):1054-63.

   c) Use as objective measure for audit tools.
3. In the presence of high PCO_2, metabolic acidosis may be falsely reported when base deficit is calculated using blood. In the perinatal period, calculating base deficit using extra-cellular fluid will prevent the influence of PCO_2 on metabolic acidosis. Most hospitals do not recognize this fact and proceed to analyze acid–base using blood.

Interpretation of Results

Respiratory and metabolic acidosis have different pathogenesis and clinical significance. Respiratory acidosis develops rapidly and disappears rapidly following the first neonatal breaths. It is considered a part of normal delivery. Respiratory acidosis occurs in the blood vessels and develops when interruption of blood flow occurs, for example with cord compression, causing a decrease in CO_2 transport from the fetus to the placenta. Carbon dioxide accumulates and after reacting with water produces hydrogen ions and bicarbonate. When the hydrogen ions exceed the buffer capacity of the blood, they accumulate in the vessel causing decrease in pH.

Metabolic acidosis, on the other hand, develops as a result of fetal hypoxia that causes the fetus to shift to anaerobic metabolism to maintain positive energy balance. Lactic acid is produced in the tissue and is dissociated to lactate and hydrogen ions. Some of the latter find their way to blood vessels, reducing the pH value. Metabolic acidosis is generated in hypoxic tissues, takes longer to develop and disappear, and has the potential to cause significant fetal damage.

Table 1 - Types of acidosis (decreased pH)

Respiratory:	Increased PCO_2 and normal base excess/deficit
Metabolic:	Normal PCO_2 increased base deficit/decreased base excess
Mixed:	Increased PCO_2 and increased base deficit/decreased base excess

Terminology

AVOID THE FOLLOWING:
- "Fetal distress"
- "Asphyxia" without hard evidence
- Qualifiers such as "significant," "severe," etc.

INSTEAD, USE:
- "Atypical FHR pattern"
- "Asphyxia" only with biochemical evidence

This is consistent with the recommendations of the SOGC Task Force Report on Cerebral Palsy and Asphyxia (SOGC, 1996).

Meconium

Approximately 20% of fetuses pass meconium prior to delivery. One stimulus to the passage of meconium is fetal hypoxia, which may cause hyperperistalsis of the bowel and relaxation of the anal sphincter. However, in most cases of meconium staining in labour, the fetus is not hypoxic, or else the hypoxic episode was transient. On the other hand, fetal hypoxia may exist without the passage of meconium, especially in the premature (<34 weeks' gestation) fetus, due to immaturity of the digestive tract. As the fetus in breech presentation descends in the pelvis, meconium is often passed without signifying hypoxia because of compression of the fetal abdomen during the baby's passage through the birth canal.

In general, old, thin meconium staining of the amniotic fluid does not have a strong association with hypoxia, although it is a warning to be heeded. Fresh, thick, green meconium is frequently ominous, and should be considered indicative of fetal hypoxia until proved otherwise. The passage of meconium during labour should be taken as an indication for careful FHR monitoring.

Summary
- All women in labour should have a skilled care provider.
- IA is the preferred method of surveillance of fetal well-being in labour, particularly for labour that is progressing normally.
- IA and EFM must be used according to an acceptable method with well-defined criteria for documentation and intervention in the case of atypical (non-reassuring) findings. Health care facilities are responsible for ensuring adequate ongoing education programs.
- Fetal scalp sampling can be used when indicated in the presence of atypical (non-reassuring) FHR findings not responsive to intrauterine resuscitation.
- Cord blood gas sampling, if available, should be done after every delivery.

- All available tests of fetal well-being are NOT highly predictive of adverse outcomes.

- Most cases of CP occur in uncomplicated term deliveries. Despite improved technology and improved neonatal care, the rates of CP are still 2–2.5/1000 live births.
- The preferred term for abnormal results of FHR monitoring is "atypical FHR pattern."

These guidelines, properly applied, should help to prevent perinatal deaths and that portion of morbidity or adverse outcome due to intrapartum events. These guidelines can help to make labour safe for all fetuses, and make the birth experience both safe and satisfying for mothers and families.

Key Messages

1. No fetal health surveillance method can diagnose fetal hypoxia.
2. IA is the preferred method of monitoring fetal well-being. It is important to learn how to perform it correctly.
3. EFM requires specialized training for use and interpretation. Continuing professional education is required.
4. If fetal compromise is suspected, consider intrauterine resuscitation or immediate delivery.

Suggestion for Applying the Sexual and Reproductive Rights Approach to this Chapter

During prenatal care women should be offered information about the benefits, limitations, indications, and risks of IA and EFM use during labour, including the available options in the probable location of delivery. If specific indications for EFM are present or anticipated, this needs to be communicated to the woman as soon as they are identified.

If an atypical FHR is found during labour, there is usually time to discuss the possibility of interventions that may become necessary. Describe the proposed intervention in a way that is non-alarming. Include the woman, her spouse or birth companion in the discussion. In this way, if an operative delivery or cesarean section becomes necessary, then it is not a complete surprise. The woman and her family will have some time to mentally prepare themselves.

APPENDIX 1

UMBILICAL CORD PROLAPSE

Definition

Umbilical cord prolapse is defined as the presentation of the umbilical cord below or adjacent to the fetal presenting part. Several types of umbilical cord prolapse have been described:

1) **Overt umbilical cord prolapse:** Loops of cord palpable on pelvic exam or protruding through the introitus.
2) **Occult umbilical cord prolapse:** Rarely palpated; umbilical cord is beside the presenting part in the birth canal detected by fetal heart rate changes associated with cord compression.
3) **Funic umbilical cord prolapse** (also know as cord presentation)**:** Prolapse of the umbilical cord below presenting part diagnosed prior to rupture of membranes.

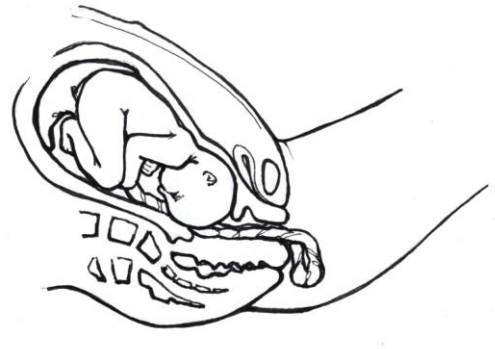


Figure A1.1 - Overt prolapse

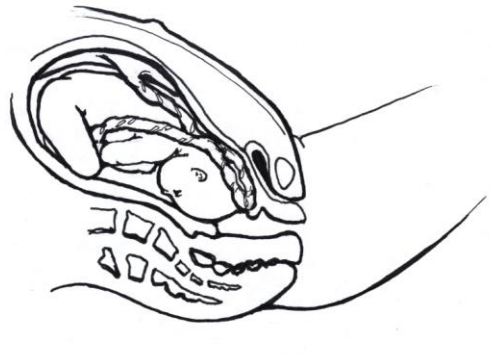


Figure A1.2 - Occult prolapse

Incidence

Based on retrospective reviews of large samples, the incidence of cord prolapse has been reported to be from 0.17% to 0.4% of births. Incidence of overt cord prolapse varies with fetal presentation with lowest occurrence in cephalic presentation and highest in transverse lie.

Risk Factors

The following factors are associated in the literature with increased occurrence of cord prolapse:

- Malpresentation
- Hydramnios
- Prematurity
- Grand multiparity (i.e. parity of five or greater)
- Male gender
- Pelvic tumours
- Placenta previa and low-lying placenta*
- Cephalopelvic disproportion
- Multiple gestations
- Premature rupture of membranes

* Although placenta previa increases the risk of malpresentation and therefore cord prolapse, a complete previa would prevent overt cord prolapse by obstructing the uterine outlet to the vagina. Occult cord prolapse could still occur.

Rupture of the membranes is a prerequisite for overt umbilical cord prolapse.

A review of 87 occurrences of cord prolapse showed that umbilical cord prolapse is associated with obstetrical intervention in 47% of occurrences. These interventions included:

- Amniotomy
- Attempted external cephalic version
- Manual rotation of fetal head
- Expectant management of preterm premature rupture of membranes,
- Scalp electrode application
- Intrauterine pressure catheter insertion
- Amnioreduction

Prevention

Education for women with risk factors for cord prolapse is important. Women need to be aware of the potential for prolapse, the need to call for help urgently, positions that would be helpful for delivery, and the intervention that would occur in the event of cord prolapse. This is true for the woman at home or in hospital.

Because the majority of cord prolapses occur during labour, evaluation of the risks of prolapse and thus the need for urgent fetal surveillance at the time of membrane rupture is indicated. Interventions, such as amniotomy, should be carefully timed, and careful consideration given to their indications and the risks and benefits of the intervention. Care should be taken to ensure good application of the presenting part to the cervix.

Morbidity and Mortality

There is significant morbidity associated with umbilical cord prolapse even with appropriate treatment. Markers of possible morbidity are demonstrated by low Apgar scores and low cord pH. Other markers of morbidity are not significantly higher. Perinatal mortality is quoted from 0.02% to 12.6%.

Diagnosis

Overt umbilical cord prolapse is diagnosed most commonly by either visualizing the cord through the introitus or palpation of the cord in the vagina. A sudden fetal heart rate deceleration in women with rupture of membranes is often the first indication of a cord prolapse. This prompts vaginal examination as part of intrauterine resuscitation for a non-reassuring fetal surveillance.

Funic cord prolapse is diagnosed either by palpation of the cord through the membranes or as an incidental finding on ultrasound.

Occult cord prolapse must be suspected in all situations where decelerations are present, whether heard on auscultation or by electronic fetal monitoring. Fetal heat monitoring may reveal variable decelerations with contractions and prompt return to baseline with occult prolapse. Sometimes movement of the mother will resolve the occult cord prolapse; when the mother moves from side to side or moves to a knee chest position, the cord may change position. Asking the woman to change position is part of intrauterine resuscitation in the case of cord prolapse.

Management

Overt prolapse is an emergency situation requiring immediate and life-saving interventions. The management of overt cord prolapse includes:

- Calling for help from all available personnel
- Perform a pelvic exam to determine cervical effacement and dilatation, station of the presenting part, and the strength and frequency of pulsations within the cord vessels.

- If cord pulsations are present:
  - Leave your examining hand in place, and elevate or push up on the presenting part. Hold it above the brim and pelvis until delivery by cesarean section; this may require insertion of your entire hand into the woman's vagina.
  - Talk with the woman about the emergency and your management plan.
  - Instruct an assistant (family or staff) to help position the mother in knee–chest or Trendelenburg's position. (Note: It may be acceptable to elevate the woman's hips versus placing the bed in Trendelenburg's position, especially in some beds which restrict the utility of this position or where such beds are not available.)

Figure A1.3 - Knee–chest position

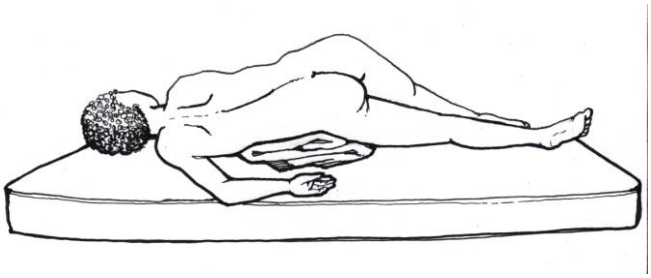


Figure A1.4 - Elevating the hips

  - Do not attempt to replace the cord; keep it warm, and avoid manipulation of the cord (e.g. warm, saline-soaked cloth).
  - Give the woman oxygen.
  - Prepare her for a cesarean section, including obtaining and documenting her informed consent.
  - Promptly perform a cesarean section.
  - Prepare for resuscitation of a potentially depressed infant by calling for skilled care providers.

- If in an out-of-hospital setting, prepare for transfer:
  - Arrange transportation.
  - Have an assistant (family or staff) prepare referral notes.
  - Prepare an emergency delivery pack.
  - Contact the receiving health facility.
  - Talk with the woman about the management plan.

- If transfer is unavailable, allow the labour to progress; talk to the woman about the probable death of her baby.

- If vaginal delivery is imminent and immediately feasible, then it is acceptable to proceed with vaginal delivery while preparing for transfer or organizing a cesarean section:
  - Call for additional help.
  - Prepare for neonatal resuscitation.
  - Ask the woman to assume an upright or squatting position to help progress; instruct assistants (family, staff) to help her maintain this position.

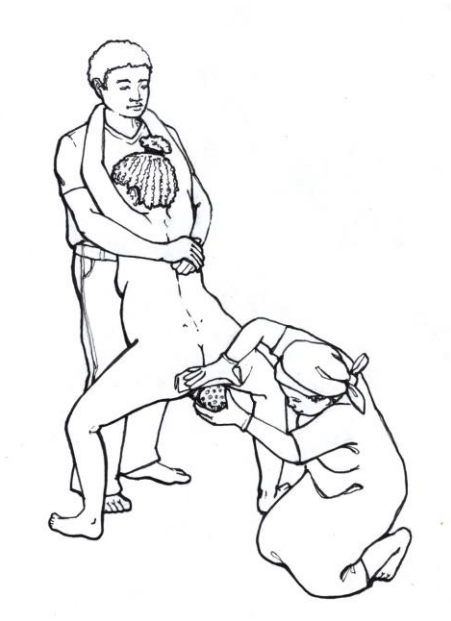


Figure A.1.4 - Supported squat for facilitating rapid delivery

- Expedite delivery by encouraging the woman to push with each contraction.
- Explain to the woman that her baby may need resuscitation, and/or may not survive.

- In the absence of immediate cesarean section capability, where the cervix is fully dilated and the head is engaged, assisted vaginal delivery with vacuum or forceps may be appropriate

- If prolonged time to cesarean section or to transport to another centre, consider:
  - Filling bladder with 500–700cc normal saline (this must be drained prior to cesarean section)
  - Tocolysis

- If cord pulsations are NOT present:
  - Explain to the woman that her baby has died.
  - Confirm absence of fetal heart tones with fetoscope, Doppler, or ultrasound, depending on available technology.
  - Discuss options for management with her including:
    - Waiting for labour to begin or progress
    - Induction or augmentation as needed
    - Transfer to a higher-level facility for these or other procedures, if indicated
    - Provide emotional and other support as needed.

Funic cord prolapse is managed by elective cesarean section prior to rupture of membranes. For women who have had ultrasound identification of cord presentation in the third trimester, repeat ultrasound (or intrapartum ultrasound if in labour) are indicated. However, in a retrospective review, only two of 42 women with cord prolapse had an ultrasound demonstrating cord presentation.

In viable premature infants with a funic cord presentation, bed rest with the woman in Trendelenburg until the cord moves or the woman is safe to deliver is suggested by some. If bed rest is maintained for more than a few days, preventive measures for deep vein thrombosis should be considered including passive exercise or antithrombotic stockings.

Resources:

- American College of Obsetricians and Gynecologists. Intrapartum fetal heart rate monitoring. [ACOG Practice Bulletin no 70]. Washington: American College of Obstetricians and Gynecologists; 2005.
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