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Acute Fetal Distress

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Abstract

Globally, intrapartum complications are a major contributor to adverse perinatal outcomes, including stillbirth, hypoxic–ischaemic brain injury and subsequent longer term disability.

The major concern of the obstetrician is to monitor labor so as to deliver a baby who is active at birth and goes home with its mother without any interventions. Fetal distress is a state in which normal fetal function is deranged as to cause death or permanent injury in utero within a short time interval. Fetal distress is a syndrome complex of intrauterine fetal jeopardy and is a result of intrauterine fetal hypoxia. In this article, fetal distress causes, risk factors and management has been discussed. Importance of electronic fetal monitoring has been emphasized. The aim of fetal surveillance is to predict the potential adverse events by detecting warning signs and to undertake timely interventions to prevent fetal demise.

Keywords: Fetal Distress; Meconium; Fetal Surveillance; Fetal Hypoxia; Interventions.

INTRODUCTION

Globally, intrapartum complications are a major contributor to adverse perinatal outcomes, including stillbirth, hypoxic–ischaemic brain injury and subsequent longer term disability. Worldwide, of the 7.6 million deaths under 5 years of age, almost 9.4% are as a consequence of intrapartum-related complications mainly in low-income and middle-income countries. These compromised babies frequently require rapid delivery by emergency operative delivery that carries considerably more maternal risk than less urgent procedures.[1]

The goal of obstetric care for every obstetrician is to produce a healthy baby from a healthy mother. The major concern of the obstetrician is to monitor labor so as to deliver a baby who is active at birth and goes home with its mother without any interventions.[2]

Delayed cry of a neonate due to antenatal or intrapartum asphyxia, requires extensive interventions or unwanted morbidity or mortality. In recent years, Cesarean section rates have

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increased and the most common indication being fetal distress. Fetal distress may be defined as a physiological state in which there is metabolic acidosis secondary to hypoxia. Fetal distress basically occurs due to fetal hypoxia because of various reasons either maternal or fetal cause. Non-reassuring fetal status is not an adverse event per se, but rather an indication of an underlying condition resulting in temporary or permanent oxygen deprivation to the fetus which may lead to fetal hypoxia and metabolic acidosis.[3]

Labor is perhaps the most stressful period that a human being endures during its lifetime. A healthy fetus has inbuilt mechanisms to tide over these short periods of hypoxia without any consequences.[4]

During labor, the intermittent uterine contractions impede transiently the placental circulation, reducing oxygen supply to the fetus. With normal uterine contractions and normal fetoplacental circulation, the fetus can withstand repeated intermittent stress without any harm. With onset of certain complications, this stress gets accentuated to fetal distress as a result of fetal hypoxia and acidosis. The obstetrician has great responsibility to identify the problem and offer timely intervention to ensure proper quality of life for the unborn baby.

Medico-legal issues surrounding fetal asphyxia can be divided into:

- Failure to anticipate
- Failure to diagnose
- Failure to treat in time[5]

However, fetuses with borderline reserves due to chronic uteroplacental insufficiency may decompensate and show signs of overt asphyxia during labor. Intrauterine hypoxia can lead to 20-40% of preventable still births.[6]

Pathophysiology of fetal distress

A state in which normal fetal function is so deranged as to make death or permanent injury in utero a probability within short time interval.[7] Fetal distress (fetal compromise) is a syndrome complex of intrauterine fetal jeopardy and is a result of intrauterine fetal hypoxia.[9] Normal transplacental respiratory gas exchange requires an efficient interplay of circumstances that function in a coordinated manner to meet requirements of fetal growth and development.

There are inherent features in fetus which support fetal oxygenation in adverse conditions:

- The higher affinity for oxygen of fetal Hb.
- Nature of hemoglobin dissociation of fetal hemoglobin. The fetal oxyhemoglobin dissociation provides a mechanism for the release of relatively large amounts of oxygen in low oxygen tension ranges.
- The uptake of O_2 by fetal Hb in the placenta along with increased accumulation of CO_2, shifts the dissociation further to the left.
- ● In the tissues, reverse effect occurs with increased acidity; at low oxygen tensions oxyhemoglobin yields greater oxygen release, dissociation curve shifted to right.[7]

CAUSES OF FETAL DISTRESS

- **Fetal Hypoxia**
- Decreased intervillous blood flow and placental transfer
- Excessive uterine action
  - Oxytocins and prostaglandins may increase uterine tone and raise the intrauterine tone as well as the frequency and duration of contractions.
  - Abruptio placentae may increase uterine tone and raise the intervillous pressure enough to impede the intervillous circulation.
- Hypotension
- Hypovolaemia, e.g. antepartum hemorrhage
- Regional anesthesia, e.g. epidural / spinal
- Drug side-effect narcotics, tocolytics
- Supine hypotension.
- **Decreased Umbilical Blood flow**
  - Intrauterine cord compression
  - Cord entanglement
  - Positional cord compression in-utero, aggravated by oligohydramnios
  - PROM, prolonged draining, dry labour and cord compression
  - Short cord, and knots in the cord
  - Cord prolapse
- **Decreased maternal oxygenation**
  - Cyanotic Heart disease
  - Chronic pulmonary disease

- Severe anemia
- Eclampsia or epileptic seizure

➤ **Fetal anaemia**

- Severe isoimmunization
- Twin-to-twin transfusion
- Vasa praevia
- Intrauterine viral infection causing hemolysis (parvovirus, dengue)

➤ **Fetal response to hypoxia will depend on**

- Acuteness of hypoxia
- Severity of hypoxia
- Duration of insult

Acute hypoxia will cause an initial fall in FHR due to chemical receptor-mediated stimulation and later due to myocardial hypoxia leading to respiratory acidosis. If not relieved, it leads to metabolic acidosis, decrease in pH and increase in base deficit. Total sudden cessation of oxygenation will affect the pontine region and cause sudden fetal death. Graded hypoxia causes necrosis of basal ganglia and hypoxic encephalopathy. Antepartum surveillance in normal pregnancy should begin at 36 weeks. In high-risk cases, it should begin at 32 weeks. However, in cases of severe disease, starts surveillance at 28 weeks.[7]

When signs of imminent distress are noticed, oxygen is administered to the mother and she is placed in the left lateral position, intravenous fluid is given to prevent fetal compromise pending delivery. In cases of fetal distress, delivery can be by cesarean section or instrumental vaginal delivery depending on stage of labor. According to the Royal College of Obstetricians and Gynaecologists (RCOG) categorization of cesarean section, RCOG category 1 is when there is an urgent threat to the life or the health of a woman or fetus. Cesarean section for fetal distress is a grade 1 cesarean section in which surgery is expected to be done within 30 min of making the diagnosis. Fetal monitoring helps to detect warning signs of fetal distress and help prevent actual distress or compromise of the baby. Distressed fetuses will require intensive resuscitative care and monitoring at delivery. APGAR scoring is used to categorize asphyxia that may have occurred. The APGAR score at 1 min usually determines the resuscitative measure to be employed. This, by necessity, has to be carried out in the delivery room.[6]

Meconium

Meconium is the first intestinal discharge of the newborn infant. Intrauterine stress like acute or chronic hypoxia and/or Infection again result in passage of meconium in utero. The presence of meconium in the amniotic fluid is a warning sign of fetal distress.

Risk Factors

Risk factors associated with an increased risk of passage of meconium and subsequent aspiration are:

- Post term pregnancy
- Preeclampsia
- Eclampsia
- Maternal hypertension
- Maternal diabetes mellitus
- Abnormal FHR and non reassuring FHR tracing
- Intrauterine growth restriction
- Abnormal biophysical profile
- Oligohydramnios
- Maternal heavy smoking
- Chronic respiratory or cardiovascular disease in the mother
- Presence of fetal distress[8]

The meconium in the amniotic fluid is caused by relaxation of the anal muscular sphincter, induced by the failure of oxygenation of the fetal blood, causing fetal acidemia and stimulating the vagus nerve followed by intestinal peristalsis so that the anal sphincter opens. Even so, obstetricians have long realized that the presence of meconium at delivery is a problem in predicting fetal distress or asphyxia (Williams, 2014). There is still a lot of controversy regarding whether meconium amniotic fluid is a sign of fetal intestinal maturity or a risk factor for intrauterine fetal distress in the fetus. Medical personnel is advised to be able to screen and record risk factors causing fetal distress during intrauterine from an early age for planning the prevention of meconium-mixed amniotic fluid in patient.[7]

Management of Fetal Distress

➤ **Pertinent history**

- Decrease in fetal perfusion during labour can occur more often in high risk pregnancies.

- Hypertension complicating pregnancy
- Maternal diabetes
- Third trimester bleeding
- Post term pregnancies
- Preterm labor, PROM
- H/o smoking, drug abuse

➤ **Correlative fetal conditions include:**

- IUGR
- Oligohydramnios
- Disturbed Doppler umbilical blood flow
- Congenital anomalies
- Meconium in amniotic fluid
- Abnormal EFHRM patterns on NST or during Labour

➤ **Counselling**

» Patients at risk should be counselled about:

- Nature of risk factors present
- Maternal and fetal risks involved
- Risks and benefits of induced labour
- Labor augmentation
- Use of amniotomy and oxytocin

➤ **Patients should also be informed of:**

- Surveillance tools
- Resuscitation measures
- Provide information about delivery interventions.

Indications for Electronic Fetal Monitoring

➤ **Maternal**

- Hypertension
- Pregestational diabetes
- Previous cesarean section
- Induced or augmented labor
- Chorioamnionitis
- Antiphospholipid antibody syndrome
- Oligohydramnios

➤ **Fetal**

➤ Meconium stained amniotic fluid

➤ Fetal growth restriction

- Multiple pregnancy
- Prematurity
- Postterm
- Previous intrapartum asphyxia/death

➤ **Intermittent auscultation**

- Baseline fetal heart rate
- Normal: 110-160 bpm
- Accelerations - Abrupt rise >15bpm above baseline lasting 15-60 seconds - Reassuring sign
- Decelerations - Abrupt drop below baseline Lasting 15-60 seconds - Cannot diagnose etiology
- Tachycardia - Fetal heart rate above 160 bpm for >10 minutes
- Bradycardia - Fetal heart rate below 110 bpm for >10 minutes.

The Causes for Fetal Tachycardia and Bradycardia

➤ **Tachycardia (heart rate of >160 bpm)**

➤ **Causes of tachycardia**

- Maternal fever
- Chorioamnionitis
- B-Sympathomimetics
- Fetal compromise
- Bradycardia (heart rate of <110bpm)

➤ **Causes of bradycardia**

- Head compression
- Fetal compromise.[5]

Fetal Heart Rate Monitoring by Auscultation

In a poor resource country like ours, most of the labor is managed by intermittent fetal heart rate monitoring by stethoscope or fetoscope. (We follow the guidelines laid down by ACOG 1005)

Time interval for intermittent auscultation in labour

Intermittent auscultation in labor	Time interval in low risk pregnancy	Time interval in high risk pregnancy
Active phase of		
1st stage of labor	30 minutes	15 minutes
2nd stage of labor	15 minutes	5 minutes

Note: Every time the fetal heart rate should be auscultated for a complete 1 minute starting immediately after the uterine contraction. For

optimal results of intermittent auscultation 1:l ratio of midwife/doctor and patient should be there.[7]

The increased citation of fetal distress as an indication of cesarean section during the last two decades raises the suspicion that electronic fetal monitoring interpretation has become more reflective of the legal climate than of the fetal condition. This limitation of CTG in predicting adverse neonatal outcomes has led to the development of newer technologies like ECG to improve the predictive value of fetal monitoring. Currently, the use of ST analysis of the fetal ECG in conjunction with conventional CTG has been shown to reduce both the rates of operative deliveries for fetal distress and metabolic acidosis during birth. The use of other modalities like fetal ECG as an adjunct to CTG may help in improving the predictive value of fetal monitoring. In addition an overall assessment of the patient's details may help to differentiate between fetuses that require prompt delivery and the fetus not actually in acute distress.[5]

Interpretation of an Electronic Fetal Monitoring Graph

Interpretation of an electronic fetal monitoring graph requires mention of the following:

- Baseline fetal heart rate (in bpm)
- Baseline variability (normal, decreased, absent)
- Presence of accelerations (duration and elevation above baseline)
- Presence of decelerations (duration, decrease below baseline, and relation to contraction)
  - **Early**
  - **Late**
  - **Variable**[5]

Advantages of external electronic monitoring are:

- The membrane remains intact and does not involve fetal head clips.

Disadvantages include:

- Difficulty in assessment of beat to beat variability
- Quality of tracing depends on patient being relatively immobile
- Movement can give artifact.[6]

Means of increasing the precision of fetal heart rate monitoring in detecting fetal distress is more frequent but sensible usage of fetal blood sampling.

Routine umbilical cord blood analysis should be encouraged as a simple means of auditing the incidence of severe acidosis at birth and the value of clinical management in detecting or preventing it. There are obvious problems with both selective and universal policies for continuous fetal heart rate monitoring. It is the responsibility of individual obstetric units to formulate their own policies as governed by their own resources and populations and to audit the value of their own obstetric management.[9]

CONCLUSION

The aim of obstetricians is to monitor labour so as to deliver a healthy baby. The aim of fetal surveillance is to predict the potential adverse events by detecting warning signs and to undertake timely interventions to prevent fetal demise. Identification of high risk pregnancies that can cause utero-placental insufficiency should be the primary aim for prevention strategy.

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