FETAL COMPROMISE

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The management of the compromised fetus is a challenging task and often prompts performance of an emergency caesarean delivery. One important aspect in the care of the compromised fetus is the well-coordinated team. The team members should clearly understand the medical terms used to describe fetal status and the urgency of the necessary intervention. Fetal distress is a poorly defined term and may result in unnecessary emergency caesarean deliveries under general anesthesia.

This article reviews the pathophysiology, diagnosis, terminology and anesthetic management of the compromised fetus.

Pathophysiology of fetal asphyxia

Fetal asphyxia results from failure to maintain gas exchange. During normal labor, hypoxemia occurs transiently with uterine contractions. The healthy fetus tolerates this well.

There are four basic causes of asphyxia in the intra-partum period[1]:

1. Inadequate perfusion of the maternal side of the placenta (severe maternal hypotension, aortocaval compression).
2. Interruption of gas exchange across the placenta (placental abruption).
3. Interruption of umbilical blood flow (cord compression).
4. Inability to withstand transient, intermittent hypoxia that occurs with uterine contractions of normal labor in the compromised fetus (anemic or growth retarded fetus).

At the onset of asphyxia, the fetus reacts with a remarkable series of responses. First there is redistribution of blood flow to vital centres to limit the deleterious effects of oxygen limitation in the brain, heart and adrenal glands. A further compensatory response is that overall fetal oxygen consumption declines to values as low as 50% of the control. This level can be maintained for periods up to 45 minutes and is completely reversible on cessation of hypoxia. There is accumulation of lactate in the vascular beds with limited oxygen supply due to anaerobic glycolysis leading to gradual development of a metabolic acidosis[2].

Diagnosis of intrapartum asphyxia

The diagnosis of intrapartum asphysxia is normally made by alterations in the fetal heart rate or scalp blood gases.

Changes in fetal heart rate (FHR) characteristics and patterns[3]:

- The normal FHR is between 110 and 160bpm. Persistent **fetal tachycardia** may be associated with fetal hypoxia or be caused by fever, chorioamnionitis, administration of anticholinergic agents, beta sympathomimetic agents, or fetal anemia. Persistent **fetal bradycardia** may be associated with fetal hypoxia (most common cause), congenital heart block and administration of beta- adrenergic blocking agents.
- **Baseline variability - short-term or beat-to-beat variability** is the difference in fetal heart rate between successive beats which is measured via fetal scalp electrodes. **Long-term variability** can be detected with external or internal monitors, and appears as crude sine waves of 3-6 cycles per minute. Normal long-term variability is more than 6bpm. The presence of both long-term and short-term variability indicates normal interaction between sympathetic and parasympathetic control of FHR and an absence of cerebral hypoxia. Acute hypoxia may initially increase FHR variability. Persistent hypoxia causes loss of variability which may also occur from other factors such as maternally administered medications. These include central nervous system depressants (volatile anesthetics, barbiturates, propofol, benzodiazepines, magnesium), local anesthetics (lidocaine, chloroprocaine), narcotics, anticholinergics, and beta sympathomimetics. There is strong evidence that the presence of normal fetal heart rate variability represents normal central nervous system integrity, including adequate oxygenation[1].
- **Periodic changes.** Early, late, or variable FHR decelerations may occur.

  Early decelerations occur simultaneously with uterine contractions and usually are less than 20 bpm below baseline. The onset and offset of each deceleration coincides with the onset and offset of the uterine contraction. Early decelerations are not ominous, and in humans, thought to result from reflex vagal activity secondary to mild hypoxia.

  Late decelerations begin 10 to 30 seconds after the beginning of a uterine contraction and end 10-30 seconds after the end of the uterine contraction. Late decelerations represent a response to hypoxia, and when combined with absent or decreased FHR variability is an accurate, ominous signal of fetal distress[7].

Variable decelerations vary in depth, shape, and/or duration. Clinical studies suggest that partial or complete umbilical cord occlusion results in variable decelerations.

- **Reactivity** is acceleration in response to fetal movement. During the antepartum period, FHR accelerations in response to fetal movement signal a healthy fetus. During the intrapartum period, the presence of fetal accelerations most likely precludes significant fetal metabolic acidosis.

Fetal acid-base status

Although FHR monitoring is very accurate (99%) in predicting a healthy fetus, it suffers from lack of specificity. Abnormal FHR tracing has a poor positive predictive value for abnormal outcome. The prediction of fetal compromise has a 35-50% false positive rate. Therefore, when FHR monitoring suggests the presence of fetal compromise, fetal scalp blood pH determination may be used to confirm or exclude fetal acidosis. In general, fetal scalp blood pH of less than 7.20 is considered abnormal and delivery should be expedited. Relative contraindications to fetal scalp blood pH sampling include intact membranes and unengaged vertex, infections such as HIV or herpes simplex virus and fetal coagulopathy.

Meconium passage in utero - controversy exists over its relative importance on fetal status.

Passage of meconium into the amniotic fluid (in utero defecation) is accepted as an indicator of fetal distress. Recent experimental and clinic studies have suggested that meconium passage into the amniotic fluid (AF) alone was not necessarily a sign of fetal distress. The human fetus exchanges the amniotic volume totally by urinating, swallowing, and respiratory tract secretions every 24 to 48 hours in the last trimester. Swallowing serves to stabilize the AF volume and plays a major role in the clearance mechanism. It has been shown that fetal swallowing is suppressed under fetal distress conditions. Therefore, it is suggested that meconium-stained AF is not related to meconium passage after fetal distress; rather it reflects impaired clearance of AF, which already contains meconium caused by physiological in utero defecation[4].

Meconium-stained AF causes amniotic epithelial destruction and vascular damage resulting in further impairment of the transamniotic clearance mechanism. It also produces umbilical vein contraction[5] causing fetal hypoperfusion, thus distress. Both of these factors aggravate the impaired clearance mechanism and create a vicious cycle during intrauterine life.

The relationship between peripartum asphyxia and cerebral palsy

At the time FHR monitoring was introduced, it was felt that virtually all intrapartum deaths and neonatal cerebral palsy were due to intrapartum asphyxia, yet the prevalence of cerebral palsy has not decreased since the advent of fetal electronic monitoring.[6] A multivariate analysis of risk revealed that the leading factors associated with cerebral palsy are maternal mental retardation, birth weight less than 2000g, and fetal malformation. Other factors involved are breech presentation, severe proteinuria in the second half of pregnancy, third trimester bleeding and a gestational age of 32 weeks or less[7]. Although severe asphyxia can cause cerebral palsy, it is now clear that the proportion of cerebral palsy caused by birth asphyxia is relatively small. The incidence of cerebral palsy due to intrapartum asphyxia is of the order of 0.025% (1000 fold less than the incidence of variant FHR patterns during labor)[8].

How quickly should the baby be delivered when there is fetal compromise?

"Fetal distress" is a widely used term indicting the need for an urgent caesarean section, but it is a poorly defined, nonspecific term. It is clear that we should no longer accept the term fetal distress as a sufficient description of fetal condition.[9] This confusion of definition compounds the difficulty of making an accurate diagnosis and initiating appropriate treatment. Better terminology is:

- **Fetal asphyxia** is a non-reassuring fetal status. The term non-reassuring fetal status describes a condition arising from compromised fetal gas exchange (diminished but persisting gas exchange) and there is usually time to place a regional block.
- The term **fetal anoxia** describes a condition resulting from complete cessation of gas exchange (complete cord occlusion, sustained bradycardia, tetanic uterine contractions, uterine rupture); which can be lethal in <10 minutes. The desired time frame from the diagnosis to delivery in cases with presumed fetal anoxia is strictly as soon as possible.

Planning anaesthesia

Fetal anoxia (e.g. cord prolapse with fetal bradycardia) can be lethal in less than 10 minutes however the speed and effects of fetal asphyxia are highly variable. Some brief episodes of mild asphyxia, on the other hand, may reverse spontaneously. Therefore, **it is always advisable to check the FHR in the operating room before proceeding with the caesarean delivery.** When deciding which anesthetic technique to use, the anesthesiologist must clarify with the obstetrician the nature of the asphyxia to determine the urgency of the caesarean section.

Pregnant airway and changes in airway during labour

Major maternal risks of general anesthesia are failed intubation, failed ventilation, and pulmonary aspiration of gastric contents. A recent study indicates that the case-fatality risk ratio for regional anesthesia for obstetrics is 16.7 times less than for general anesthesia, primarily because of decreased

number of complications associated with regional anesthesia[11]. Therefore, general anesthesia should be used only when it is absolutely necessary and in most hospitals regional anesthesia is preferred during pregnancy. However in resource poor environments where drugs and equipment may be in short supply, different principles may apply.

Early assessment and communication with the anaesthetist for patients who may be at risk of requiring operative intervention allows airway and other preoperative assessment. In some units epidural analgesia is encouraged in patients thought to be at high risk for operative delivery including multiple gestation, preeclampsia, diabetes, IUGR, macrosomia, morbid obesity (caesarean delivery rate > 50%).

The result of an airway assessment in early labor, however, may no longer be valid after a period of active labor, especially after prolonged strenuous bearing down efforts. Deterioration in Malampati scores has been reported due to edema, which is explained on the basis of the intermittently raised venous pressure in the upper body.[12] Therefore, airway assessment should be repeated prior to initiation of any major obstetric anesthesia.

Intrauterine treatment for abnormal fetal heart rate patterns

As soon as fetal compromise is suspected, maternal and fetal factors that may contribute to this compromise should be identified. There are therapeutic measures that may successfully resuscitate the fetus in-utero and allow time for placement of regional anesthetic (or delivery of the fetus vaginally, if imminent). Maintaining effective uterine blood flow is a priority.

$$\text{Uterine blood flow} = \frac{\text{uterine artey pressure - venous pressure}}{\text{vascular resistance}}$$

Correctable maternal factors to improve uterine blood flow

- **Hypotension**. Normal maternal blood pressure and uterine artery pressure should be maintained by left uterine displacement to avoid aortocaval compression, intravenous fluid replacement, and giving vasopressors (ephedrine, phenylephrine) if indicated.
- **Excessive uterine activity**. Uterine contractions constrict the uterine spiral arteries and cause decreased placental perfusion and oxygen delivery. Oxytocin infusion may result in uterine tetany. Oxytocin has a plasma half-life of 1 to 6 minutes. Therefore, after stopping the infusion of oxytocin, tocolytic agents such as terbutaline and nitroglycerin may be necessary to relieve the tetanic contraction.

Supplemental Oxygen

Although there is little objective evidence that maternal supplemental oxygen improves neonatal outcome in cases of fetal distress during labor[18], most obstetricians advocate oxygen 4–6lpm by facemask to these mothers. Maternal supplemental oxygen increases fetal transcutaneous PO_2 slightly and seems unlikely to cause uterine vasoconstriction.

Correctable fetal factors to improve uterine blood flow

- **Transient umbilical cord compression** (causes variable decelerations). Oligohydraminios is a risk factor for cord compression. Changing maternal position may relieve the compression and correct fetal compromise. Amnioinfusion may also relieve the compression. This is done by infusing about 800ml lactated Ringer's solution at a rate of 10-15ml/min initially. This may be followed by repeated boluses of 250ml at a rate of 2-3ml min. Correction of abnormal FHR pattern requires 20-30 minutes. Amnio-infusion by gravity rather than by means of a pump prevents uterine over distension. Maternal infection and fatal amniotic fluid embolism have occurred during amnioinfusion.
- **Increased fetal oxygen consumption** Hyperglycemia increases fetal oxygen consumption and leads to neonatal hypoglycemia after delivery. Therefore, administration of a large bolus of glucose-containing solution is contraindicated. Maternal fever should be treated with acetaminophen, antibiotics and cooling of the mother.

Emergency cesarean sections have been divided into three categories as follows:[10]

Stable: There is stable maternal and fetal physiology. The patient requires caesarean delivery before destabilization occurs. The preferred anesthetic technique is regional anesthesia (spinal, epidural or combined spinal/epidural). Examples include chronic uteroplacental insufficiency or footling breech presentation with ruptured membranes and not in labor.

Urgent: There is unstable maternal and/or fetal physiology, but not immediately life-threatening to the mother or fetus. Regional anesthesia is preferred using the preexisting epidural catheter, single shot spinal, or combined spinal epidural. Examples include cord prolapse without fetal distress or variable FHR decelerations with prompt recovery and normal FHR variability.

Emergency: Immediate life-threatening condition exists for the mother and/or fetus. Preferred anesthetic technique is general unless epidural anesthesia can be established quickly using a pre-existing epidural catheter. Examples include prolonged fetal bradycardia or late FHR decelerations with absent FHR variability.

Regional anesthesia for emergency caesarean section

Hospitals should be able start a caesarean delivery within 30 minutes of the decision to operate.

Examples of indications that may require more rapid delivery include prolonged fetal bradycardia and late FHR decelerations with no FHR variability, cord prolapse, uterine rupture or maternal hemorrhage such as in placenta previa.

If an epidural catheter has been placed earlier for labor, and the patient is hemodynamically stable, extension of the block with 3% 2-chloroprocaine, 2% lidocaine or 0.5% bupivacaine are appropriate choices for emergency caesarean section. After an initial 2-3ml bolus of local anaesthetic (to exclude intrathecal migration of the catheter), the remaining medication, usually 15-20ml, can be given in increments of 5ml every 2-3 minutes, until an adequate level for surgery is achieved. The patient should receive an intravenous bolus of Ringer's lactate because the local anaesthetic has a fast onset of action. This rapid onset also ensures that the anesthetic level will be adequate for incision when the surgeon is ready. Chloroprocaine is rapidly metabolized by both the mother (25 seconds) and fetus (45 seconds) making it the authors' first choice. If 2-chloroprocaine is not available, 2% lidocaine with epinephrine (1 in 200,000) may be administered. The onset of anesthesia is shown to be only 2 minutes slower than 2-chloroprocaine when bicarbonate (2ml of 8.4% sodium bicarbonate per 20ml of lidocaine) and 1:200,000 epinephrine is added[13].

If an epidural catheter is not already in place, spinal anesthesia may be given safely in most urgent cases. When fetal compromise is present, the anesthetist should consider the possibility of placental abruption, concealed hemorrhage and unrecognized hypovolemia.

Fluid preloading with Ringer's solution through a large bore (18 or 16G) intravenous access should be started. A 25G pencil point spinal needle is preferred to avoid post dural puncture headache. Usually 10-12mg of hyperbaric bupivacaine (2-2.4ml of 0.5%) injected intrathecally provides rapid onset of spinal anaesthesia. Prophylactic administration of ephedrine 10-15mg IV may avoid episodes of significant hypotension. If the anaesthetist is not expert at performing spinal anaesthesia, or there are delays in establishing the block, the plan should change to general anaesthesia.

Some high-risk patients (morbidly obese, difficult airway) require a reliable and controllable regional anesthetic technique and some anaesthetists use an **intrathecal catheter** (using a spinal microcatheter) followed by incremental blousing.[14,15] For caesarean section, we use incremental doses (0.3-0.5ml) of 0.5% hyperbaric bupivacaine to achieve an adequate block. In most cases an adequate block is achieved with 1.6-2.0ml of this solution.

Characteristics of regional anesthetic techniques suitable for Caesarean section

	Spinal	Continuous Epidural	Continuous Spinal
Rapid onset	+	-	+
Controllable duration	-	+	+
Consistently effective	+	-	+
Controllable level	-	+	+
Low dose of local anesthetic	+	-	+

Source: Mallan PT, Johnson MD. Difficult airway in obstetric anesthesia J Clin Anesth. 1988;1:107

General anesthesia

General anesthesia with rapid sequence induction is required for many emergency cesarean deliveries due to fetal condition or unstable maternal condition. When there is fetal compromise anesthetists must be prepared to provide anesthesia quickly, but safely. Patients should receive normal antacid medication according to local protocols.

- **Difficult intubation**. The incidence of failed intubation in pregnancy is 1:250-1:750, which is ten times that of nonpregnant patients[16]. Difficult intubations are encountered in 5% of general anaesthetics in obstetrics[17]. A pregnant patient has not only a more difficult airway, but desaturates more rapidly (3 times faster than a nonpregnant person) due to increased oxygen consumption and decreased functional residual capacity. A difficult intubation kit with airway devices to provide emergency oxygenation such as combitube, laryngeal mask airway, percutaneous cricothyrotomy kit should be readily available in the obstetric suite.
- **Unrecognized difficult airway.** When attempts at intubation fail after the three or four attempts with repositioning of the head/neck, applying external pressure over the thyroid cartilage and using different laryngoscope blades (optimal intubation attempts), it is important to stop further manipulations and reassess the situation. Ask yourself:

  1. Is there severe fetal compromise (fetal anoxia) or a maternal obstetric emergency?
  2. Is the maternal airway okay and is there adequate gas exchange by mask?

  If there is no fetal compromise or obstetric emergency, and the patient is stable, the patient should be woken up. Continue assisted mask ventilation with 100% oxygen and cricoid pressure until awake. Once the patient is awake,

the options include awake regional anaesthesia or an awake fiberoptic intubation.

If there is severe fetal compromise or an obstetric emergency and mask ventilation with cricoid pressure is adequate, one may proceed with 100% oxygen and volatile agent such as halothane. In order to minimize passive regurgitation, the surgeon should avoid fundal pressure and uterine exteriorization.

If the patient cannot be intubated or ventilated by mask, the priority is to maintain ventilation and provide oxygen rapidly to the mother using the following rescue options. Surgery should wait until the patient is stabilized.

- LMA
- Combitube
- Percutaneous cricothyrotomy
- Surgical cricothyrotomy

Case history

35 year old, obese (130kg, 5'2"), woman (G4P0), at 34 weeks gestation is rushed to the operating room for an emergency caesarean section for "fetal distress". The obstetrician insists on general anesthesia.

She has a large tongue, and a Mallampati IV airway classification. What should the anaesthetist do?

- Ask the obstetrician the reason for concern in more specific terms.
- Check the FHR in the operating room.
- When faced with a patient who has potentially difficult airway, communicate your concerns to the obstetrician and look for therapeutic measures that may successfully resuscitate the fetus in utero and allow time for placement of regional anesthetic.
- If general anesthesia is the only choice, call for help and:
  - Optimize the position of the patient's head and neck to facilitate successful endotracheal intubation.
  - Ensure the availability of airway devices to provide O_2 rapidly such as laryngeal mask airway, combitube, percutaneous cricothyrotomy kit.
  - If intubation fails, and the mask ventilation with cricoid pressure is adequate, you may proceed with 100% oxygen and volatile agent when there is severe fetal compromise or other obstetric emergency.
  - If the patient cannot be intubated or ventilated by mask, the priority is to maintain the ventilation and oxygenate the mother. The mother's life always takes priority.

The consequences of poorly managed perinatal emergencies can be devastating. Clear communication between the anaesthetist and the obstetrician is vital in selecting the optimal anesthetic care for these patients. Most maternal deaths under anesthesia occur during emergency caesarean deliveries.

Work with your obstetrician to develop strategies to minimize the need for emergency induction of general anesthesia for caesarean delivery.

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