The introduction over 40 years ago of electronic fetal monitoring (EFM) for the management of labour and delivery was expected to greatly reduce the incidence of cerebral palsy and adverse neonatal outcomes. It was developed to detect developing fetal hypoxia, with the goal of preventing subsequent acidemia and cell damage, and thus reduce hypoxic intrapartum perinatal mortality. At first, it was utilised for monitoring “high risk” pregnancies, but by the end of the 1970s, it was used in more than 60% of all pregnancies, reaching 90% of use in the mid-1990s in the US. Thus the obstetric anaesthesiologist needs to understand what factors affect fetal heart rate (FHR) tracings, and in what way analgesia and anaesthesia may impact on fetal well-being and neonatal outcome.

The limitations of EFM include poor specificity and wide inter-observer variation in interpretation, which can lead to an increase in caesarean and operative vaginal deliveries (1). Thus some have suggested limiting the routine use of continuous electronic heart rate monitoring (2). In an attempt to reduce the false-positive rate, other monitoring tools, such as fetal scalp pH measures (3), fetal pulse oximetry (4) and fetal ECG (ST analysis) (5,6) have been developed.

**Basic fetal physiology**

The fetal circulation a high output, low pressure system. Oxygenated blood from the placenta is transported to the fetus via the umbilical vein, and with a normal blood flow, most of this oxygenated blood enters the left atrium via the foramen ovale. This allows oxygenated blood to circulate to the heart and the brain. Blood with a low oxygen content is transported back from the right heart to the aorta via the ductus arteriosus, and thus to the placenta via the umbilical arteries.

The normal fetal metabolism is predominantly aerobic, oxygen-dependent metabolism of glucose. Some cellular glucose is stored as glycogen, reserves which appear mainly during the 3rd trimester. The energy produced during aerobic metabolism is utilised for fetal activity and for growth. During hypoxia, the fetus compensates with an anaerobic non-oxygen-dependent metabolism, involving glucose in the blood and the glycogen stores, but producing lactic acid.

**Oxygenation**

**Definitions**

- Hypoxemia = reduction in oxygen content of the arterial blood
- Hypoxia = reduction in oxygen content of peripheral organs
- Asphyxia = lack of oxygen in central organs
- « Fetal distress » = Non-reassuring fetal status
  Progressive asphyxia, if uncorrected, will lead to decompensation of physiological responses and permanent central nervous damage and death

**The fetal response to hypoxemia**

Hypoxemia is the initial phase of a lack of oxygen supply. The arterial oxygen saturation is reduced, but cellular and organ functions remain intact. The fetus compensates by increasing oxygen extraction. Fetal activity may decrease, and during prolonged hypoxemia (several weeks), intra-uterine growth will be restricted.

**The fetal response to hypoxia**

If a reduction in oxygen supply persists, the above mechanisms will be inadequate. Stress hormones are released initially and the peripheral blood supply reduced, in order to redistribute blood to the heart and brain. The release of epinephrine stimulates the β-adrenergic receptors, activating glycogenolysis (to transform glycogen into glucose), and resulting in an anaerobic metabolism. As long as hypoxia is limited to peripheral tissues, no organ damage will occur. The fetus can endure several hours of hypoxia before central organ damage.
The fetal response to asphyxia

When metabolic requirements exceed cellular energy production, the fetus continues to release stress hormones, and anaerobic metabolism starts in central organs. The fetal brain depends on the cardiac and hepatic supply of glucose through glycogenolysis, because the cerebral glycogen reserves are extremely limited. After several minutes of asphyxia, cardiac failure occurs in the form of a terminal bradycardia.

Fetal heart rate

FHR regulation responds to input from the autonomic nervous system, an independent pathway of the central nervous system, and from baro- and chemoreceptors.

Parasympathetic activation

The main goal of parasympathetic activation is to allow a rapid response of the cardiovascular system to internal and external events, such as the sudden increase in blood pressure following cord compression, or in response to pressure on the eye (oculocardiac reflex). Parasympathetic activation is mediated via the vagus nerve, and results in fetal bradycardia. This pathway becomes effective at about 28-32 weeks.

Sympathetic activation

Sympathetic activation releases adrenal stress hormones, resulting in fetal tachycardia. It has a slower onset than parasympathetic activation. Catecholamines maintain fetal heart rate during hypoxia, which would otherwise cause bradycardia and cerebral depression. The net effect of these two inputs is beat to beat variability (short term variability).

Etiology of fetal heart rate changes

Normal changes due to reduced fetal activity

Sensitivity to the autonomous nervous system is reduced during fetal sleep, as is FHR variability. However, fetal activity may increase, with an increase in FHR accelerations and variability, during paradoxical (REM) sleep.

Variations in placental blood flow

Umbilical cord compression during contractions can be considered as a “stress test” because of the reduction in blood flow to the fetus, and the various reactions such as cardiac and large vessel baroreceptor activation that it mediates. To compensate for the sudden increase in umbilical cord pressure, bradycardia reduces cardiac output. When the cord compression is released, blood flow is rapidly restored and a compensatory acceleration results. However, with a prolonged contraction, baroreceptor activation is followed by vagal stimulation, prolonging the bradycardia.

Adaptations to hypoxia

Acute hypoxia activates chemoreceptors, resulting in sympathetic and parasympathetic stimulation. The initial FHR response is slowing, although FHR increases with progressive hypoxia. Reduced placental blood flow during uterine contractions can result in hypoxia and activation of chemoreceptors. This may lead to repeated late decelerations, with fetal tachycardia each time blood flow and oxygenation are restored.

External stimuli

During contractions, compression of the head causes transient intracranial hypertension, causing simultaneous FHR decelerations. During the final phase of labour, pressure on the eye will often cause profound bradycardia via the oculocardiac reflex.

Temperature

Increased maternal temperature, as observed during labour with or without neuraxial analgesia, increases fetal metabolism, blood flow and oxygen consumption. This may cause fetal tachycardia, and a decreased capacity to sustain hypoxemia. Increased maternal temperature therefore requires treatment, such as hydration and paracetamol. With chorioamnionitis, the fetus’ ability to tolerate asphyxia is greatly impaired.
MEDICATION

- Oxytocin and prostaglandins increase uterine activity and tone, and may result in hypoxia and fetal bradycardia.
- β-adrenergic blockade may decrease the ability to react to hypoxia, decreasing short term variability, and may affect cerebral function.
- β-adrenergic stimulation (tocolysis, ephedrine) increases fetal metabolism, uses up glycogen stores, causing fetal tachycardia and possibly acidosis.
- Ephedrine increases fetal acidosis (7-10).
- Systemic opioids (pethidine and the long acting metabolite, norpethidine (11)), sedatives (and general anaesthesia (12)) and magnesium sulfate (13) cross the placental barrier and reduce short-term variability.
- Epidural analgesia with local anesthetics may decrease uteroplacental blood flow and cause fetal hypoxia by reducing maternal blood pressure. Avoiding aortocaval compression with uterine displacement via lateral tilt may prevent hypotension after sympathetic blockade during neuraxial analgesia.
- Effective analgesia via epidurals and combined-spinal epidurals facilitate the avoidance of maternal hyperventilation, improving fetal acid-base balance (14).
- Intrathecal opioids increase uterine tone, and may cause “uterine tetany” resulting in fetal bradycardia, which will require tocolysis (15).

WHAT TO DO WITH AN ABNORMAL TRACING?

- The quality of the tracing needs to be assessed, and artifacts caused by the maternal ECG need to be ruled out. It is extremely important to interpret the FHR tracing in conjunction with the tocogram, and assess whether the uterus is hypertonic, which would require cessation of oxytocin infusion or prostaglandins (if applicable), and consideration of tocolysis. If the mother is tachycardic, treat maternal fever with paracetamol and give iv fluids.
- If the mother is hypotensive, consider aortocaval compression and instigate lateral tilt, iv fluids and phenylephrine (ephedrine?!).
- If the mother is receiving drugs, expect fetal tachycardia with ephedrine, and loss of variability with magnesium sulfate and systemic opioids or sedation.

INTERPRETATION OF CTG

THE CTG SHOULD BE MONITORED FOR AT LEAST 20 MINUTES FOR AN APPROPRIATE INTERPRETATION TO AVOID THE INFLUENCE OF UTERINE ACTIVITY AND DECREASED ACTIVITY DURING SLEEP. GUIDELINES FOR CTG INTERPRETATION HAVE BEEN DETERMINED BY THE NATIONAL INSTITUTE FOR CLINICAL EXCELLENCE (NICE), AND CAN BE FOUND AT HTTP://WWW.RCOG.ORG.UK/GUIDELINES/ (FIGURE 1).

BASELINE FHR

The baseline FHR is defined as the heart rate between contractions over at least 10 minutes. This reflects the balance of the autonomic nervous system. With fetal maturation, the parasympathetic system becomes predominant as blood pressure increases, and the heart rate slows. At term, the FHR is between 110-150 bpm.

VARIABILITY

Beat-to-beat variability indicates the ability of the central nervous system to control and adapt the cardiovascular system. Loss of beat-to-beat variability is the most specific pattern of asphyxia (negative predictive value approx 99%) (16). A premortem CTG is defined as a tracing with no beat-to-beat variability.

SINUSOIDAL PATTERN

With severe fetal anemia, due to immunization or hemorrhage, the CTG can have a sinusoidal pattern, which is defined as periodic variations of FHR without beat-to-beat variability and no accelerations. This pattern can indicate cerebral injury.

ACCELERATIONS

Accelerations are intermittent increases in FHR of more than 15bpm, lasting for more than 15 seconds. They reflect normal oxygenation. A normal CTG should show at least 2 accelerations over 20 minutes.
Decelerations are defined as intermittent decreases in FHR of more than 15 bpm, lasting for more than 15 seconds.

- **Early decelerations** mirror the contractions and are vagally mediated. They are usually due to head compression, and are not associated with hypoxia or acidosis.

- **Variable decelerations** are the most common type (80%), and can occur before, during, after or in the absence of contractions. They are often benign, but can also be severe. Duration is the most important aspect of variable deceleration.
  - Non-complicated variable decelerations are defined as < 60 bpm and lasting < 60 s. These are well tolerated by the fetus and reflect changes of blood volume (cord compression during contractions) that do not cause hypoxia.
  - Complicated variable decelerations are defined as a FHR decrease of > 60 bpm or a duration of > 60 s. They are usually due to cord compression or an acute decrease of umbilical flow for longer periods of time and may result in hypoxia, especially if the contractions are prolonged.

- **Late decelerations** usually start after the onset of a contraction, with a return to baseline after the end of the contraction; they have a regular form. Size and depth of deceleration are not related to clinical significance. They are usually associated with an increase in basal FHR. They are often observed with abnormal uterine tone and increased frequency of contractions. Repetitive decelerations are critical, and cause uteroplacental insufficiency.

**In summary**

1. Electronic fetal monitoring has a low specificity (40-60%) (1)
2. Loss of variability for 1 hour is the most specific predictive parameter (17,18), and can result from:
   - Prematurity (< 28-32 weeks gestation)
   - Maternal intake of magnesium sulfate (13)
   - Maternal intake of systemic opioids, or general anesthesia (12)
3. Fetal scalp sampling is very useful for assessing fetal acidosis (base deficit, lactate) (3)
4. Base deficit increases with a long 2nd stage (19) and the use of ephedrine (7).
5. A large difference between the acid-base status of arterial and venous umbilical cord blood (base deficit) indicates acute hypoxic event (conversely a small difference indicates long-lasting hypoxia).
6. Effective analgesia (epidurals/CSE) reduces maternal hyperventilation and fetal acidosis (14).
7. Intrathcal opioids may increase uterine tone, resulting in fetal bradycardia, but do not increase the CS rate for non-reassuring FHR (NRFHR) (20).
8. When performing CS for NRFHR, there is no evidence that spinal/epidural anaesthesia worsen fetal acidosis (21), but ephedrine does, via fetal β-adrenergic stimulation.

REFERENCES