Pathophysiology of CTG

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Background

A fetus receives its blood supply from the placenta through the umbilical cord that contains two umbilical arteries and an umbilical vein. Blood supply to the placenta comes from uterine arteries of the mother which traverse the uterine muscle (myometrium) to supply the placental bed.

Labour threatens fetal oxygenation as each uterine contraction temporarily ‘squeezes’ all the blood vessels running within the myometrium (uterine muscle of the ‘wall’) leading to transient decrease in placental blood flow. In addition, stronger uterine contractions also may compress the umbilical cord leading to interruption of blood flow during contractions. A normal, well grown, healthy fetus is able to compensate for these hypoxic and mechanical stresses by decreasing the fetal heart rate (decelerations) to reduce energy demand of the heart muscle and by releasing adrenaline to increase the heart rate, reduce the blood supply to the peripheral tissue and increase the blood pressure to supply the central organs.

A Fetus receives oxygenated blood from the mother mainly during relaxation when uterine blood vessels are not ‘squeezed’ and the umbilical cord is not compressed. Therefore, any intervention that increases the uterine contraction and reduces the relaxation time (e.g. oxytocin or prostaglandins) endangers fetal oxygenation not only by increasing the risk of prolonged and repeated umbilical cord compression and by reducing blood supply to the placental bed, but also, by reducing the amount of time available for the fetus to replenish oxygen within the placental venous sinuses during a relaxation.
Types of Hypoxia during labour

During labour a fetus is exposed to three types of hypoxia: Acute, subacute or gradually evolving hypoxia based on the onset and progression of hypoxic stress.

Acute Hypoxia

Acute Hypoxia results in a sudden drop in baseline heart rate. It is termed ‘single prolonged deceleration’ if it lasts for less than 3 minutes and then recovers to normal baseline. If the deceleration lasts for more than 3 minutes, it is termed ‘prolonged decelerations lasting for more than 3 minutes’. If the heart rate remains below 80 beats/minute for over 10 minutes, then it is termed prolonged baseline bradycardia.

Figure below shows acute hypoxia

Clinicians should exclude three major ‘accidents’ during labour (abruption, cord prolapse and caesarean scar rupture) and two iatrogenic causes (uterine hyperstimulation due to oxytocin infusion and hypotension due to maternal hypotension). If there is any clinical evidence of these ‘accidents’, an immediate delivery should be undertaken to salvage the fetus.
This is because metabolic acidosis is likely to get worse with time due to continued reduction in the utero-placental circulation. In the presence of acute hypoxia, the fetal pH has been shown to drop at the rate of 0.01/minute. In cases of hyperstimulation, oxytocin infusion should be stopped immediately and tocolytics (terbutaline 250 mcg subcutaneously) administered, if required to abolish the uterine contraction.

If these three ‘accidents’ are excluded, it is reasonable to wait, if the variability of the heart rate during the episode of deceleration or bradycardia is normal and if the CTG prior to the deceleration was normal. Signs of recovery to the normal baseline (i.e. an ‘upward trend’ of the end of the deceleration, repeated attempts to reach the baseline) are also a positive feature. It is estimated that in the absence of the three ‘accidents’ of labour described above, over 90% of CTGs with prolonged bradycardia that are likely to recover to normal baseline in 6 minutes and up to 95% in 9 minutes.

The ‘3,6,9,12, & 15’ minute ‘guidance’ on the management of prolonged decelerations is based on this observation. This guidance involves instituting appropriate interventions (positioning, hydration, tocolysis, stopping syntocinon infusion) by 6 minutes and to move the patient to theatre by 9 minutes, if the CTG shows no recovery. It is recommended that attempts at delivery should commence by 12 minutes with the aim of delivering the fetus by 15 minutes.
Criteria to apply the 3,6,9,12,15 rule includes exclusion of intrapartum accidents, correction of iatrogenic causes and examining the CTG trace for a reduced variability prior to the onset of prolonged deceleration/ bradycardia and total loss of baseline fetal heart rate variability within 3 minutes of a prolonged deceleration. These features have been reported to be associated with a poor perinatal outcome. A recent study has confirmed that 98% of fetuses with terminal fetal heart rate decelerations are born with normal cord gases, if the intrapartum accidents are excluded and the above criteria were satisfied.

**Sub-acute Hypoxia**

In this situation, the fetus spends more time decelerating and progressively less time at the normal baseline fetal heart rate. Typically, the fetus spends less than 30 seconds at the baseline to ‘wash off’ carbon dioxide and acid and spends over 90 seconds building up carbon dioxide and acid. pH of the fetus has been shown to drop at the rate of 0.01 every 2-3 minutes.

Figure below shows Sub-acute Hypoxia- note the fetus spending less time on the baseline) and more time decelerating
**Gradually evolving hypoxia**

Hypoxic stress may develop over hours than minutes during labour and this may provide the fetus with the opportunity to utilize its compensatory mechanisms to avoid hypoxic injury. In this scenario, CTG would initially show decelerations followed by disappearance of accelerations as the fetus attempts to conserve energy by limiting muscle activity that may increase oxygen requirement. If the hypoxic insult continues, fetus then releases catecholamines to increase the heart rate and its cardiac output to supply vital organs.

Figure below shows sub-acute hypoxia: Note the decelerations (hypoxic stress), followed by a rise in baseline heart rate (due to release of adrenaline/noradrenaline from the adrenal glands).

Despite of fetal efforts at compensation, if the hypoxic insult persists, then decompensation ensues resulting in reduced perfusion of brain leading to loss of baseline variability. The CTG would now be termed 'pre-terminal' as the final event is reduction of myocardial oxygenation that results in gradual reduction of fetal heart rate.
This is often described as ‘step ladder pattern to death’ and signifies myocardial acidosis and failure of the autonomic centres of the brain to maintain a stable baseline heart rate.

**Long standing hypoxia and Pre-terminal traces**

In both these situation, the fetus has exhausted all its reserves or is unable to compensate (e.g. intra-uterine growth restriction). In the former, the hypoxic insult has occurred at some point during the antenatal period (i.e. prior to the onset of labour) and the CTG often shows a higher baseline with reduced variability and shallow decelerations with uterine contractions. Such uterine contractions during labour may cause further episodes of hypoxia and hence may worsen the existing cerebral damage. Prolonged bradycardia as well as total loss of variability (often with shallow decelerations) are often termed ‘pre-terminal CTG’ and these fetuses require immediate delivery.

Preterminal traces (below) have been reported to be associated with a perinatal mortality rate of up to 39%.
**Uterine Tachysystole and Uterine Hyperstimulation**

Uterine tachysystole refers to increase frequency of uterine contraction (> 6 in 10 minutes), whereas uterine hypertonus refers to increase tone or duration of uterine contractions.

Uterine hyperstimulation refers to any increase in uterine activity (frequency, strength and duration) that is associated with changes in the fetal heart rate on the CTG Trace. This is secondary to both repeated and prolonged umbilical cord compression as well as sustained reduction of oxygen supply to the placental venous sinuses secondary to increase in the duration of contraction and decreased relaxation time for replenishment of oxygen.

Figure below shows uterine hyperstimulation (although the number of uterine contraction was < 6 in 10 minutes, the contractions were lasting longer and stronger leading to changes on the CTG Trace)

Fetal decompensation can rapidly ensue during uterine hyperstimulation to rapid and progressive reduction in fetal oxygenation. Fetuses respond by reducing myocardial workload (decelerations) to conserve oxygen and
demonstrate variable decelerations (umbilical cord compression) and late
decelerations (lack of oxygen within the placental venous sinuses) and
attempt to increase the heart rate by releasing adrenaline in order to continue
perfusing the central organs (brain and the heart muscle). If these
compensatory mechanisms fail, a reduction of baseline variability (loss of
blood supply to brain centres) and a gradual and progressive reduction in
baseline fetal heart rate (lack of oxygen to the heart muscle which is unable to
keep pumping blood at the same rate) occurs. If timely and appropriate
interventions to reduce hypoxic stress and/or to expedite delivery are not
instituted, then myocardial failure occurs (terminal bradycardia).
References:


14. Chandraraharan E. Fetal scalp blood sampling during labour: is it a useful diagnostic test or a historical test that no longer has a place in modern clinical obstetrics? BJOG 2014; 121(9):1056-1062.


