Intrapartum fetal surveillance

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Abstract

Electronic fetal monitoring (EFM) is the recommended method of intrapartum fetal surveillance for high risk pregnancies. Despite the questions about its efficacy and controversy regarding increased rates of operative delivery associated with its use, continuous cardiotocography (CTG) remains the predominant method of intrapartum fetal monitoring. The CTG trace also forms a central piece of documentary evidence in medico-legal cases related to intrapartum hypoxia and birth asphyxia.

Although CTG is sensitive in detecting abnormalities of fetal heart rate (FHR), its specificity for detection of fetal hypoxia remains low and therefore confirmatory tests such as fetal scalp blood sampling (FBS) or analysis of fetal electrocardiography (ECG) become necessary. Due to the rising costs of litigations related to birth asphyxia and increasing complexity of obstetric patient populations, it has become absolutely mandatory that all health professionals responsible for the care of women in labour are trained adequately in interpretation and documentation of CTG traces, as well as the guidelines for actions based on the assessment of the trace and overall clinical situation.

Confidential enquiries have always pointed to factors such as inability to interpret traces, failure to incorporate the clinical situation, delay in taking appropriate action and poor team working as contributors to adverse perinatal outcomes. In this article we discuss three case scenarios of adverse maternal and perinatal outcomes due to failure to adhere to basic principles of fetal monitoring and recommended actions as per the national guidelines. The key learning points and risk management issues are also discussed.

Keywords cardiotocography; CTG; EFM; fetal; hypoxia; intrapartum; monitoring; surveillance

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Introduction

Child birth could be a hazardous process and labour, which forms the most critical part of this journey, is a time when the fetus is subjected to maximum stress due to increasing frequency and duration of the uterine contractions. The principal aim of monitoring a fetus in labour is to detect changes in the fetal heart rate (FHR) that suggest a possibility of fetal hypoxia and metabolic acidosis so that timely action can be taken to prevent adverse outcomes. While intermittent auscultation continues to be the method of choice for intrapartum fetal monitoring in low risk pregnancies and in settings with limited resources, continuous EFM by CTG has formed the mainstay of fetal surveillance in high risk pregnancies in most of the developed world. However, intrapartum fetal surveillance with CTG has its own challenges. It is not uncommon in clinical practice to unexpectedly find normal size babies born asphyxiated while those delivered operatively for presumed 'fetal distress' may be born in good condition. There appears to be a consensus regarding the reassuring value of a normal reactive CTG pattern (Figure 1) with accelerations, normal baseline rate and normal baseline variability without any decelerations. On the other hand, patterns containing absent variability associated with persistent late decelerations, severe atypical variable decelerations or prolonged decelerations are considered ominous (Figure 2) and indicate the need for immediate delivery to avoid hypoxic damage. However, the clinician is often challenged with a CTG trace that falls between these two extremes, and needs to decide the further action depending on overall clinical assessment of the case.

Hypoxia due to repeated cord compression or decreased uteroplacental blood flow is not the only damaging factor during labour. Studies have suggested that fetal inflammatory response to infection and pyrexia can also cause central nervous system damage. The patterns of CTG associated with such insults may vary considerably and need to be carefully interpreted based on the overall clinical scenario.

In the United Kingdom, the National Institute for Health and Clinical Excellence (NICE) has published guidelines for standardized interpretation and classification of CTG traces and recommended actions based on the nature of the trace (Tables 1 and 2). In the examples that follow, we have applied a commonly followed systematic approach of interpreting a CTG trace based on characteristics such as DR – determine risk, C – contraction frequency, BR – baseline rate, A – accelerations, VA – variability, D – decelerations and O – overall impression.

Case 1

A primigravida at 41 weeks gestation was admitted to the delivery suite at 20:00 hours. The course of pregnancy had been uneventful and all antenatal investigations including ultrasonography were normal. She had spontaneous rupture of membranes (SROM) 36 hours earlier and had been contracting irregularly for12 hours. She was in established labour with 4–5 strong uterine contractions per 10 minutes and was requesting epidural analgesia. CTG was commenced at 20:07 hours and the trace was normal at this stage (Figure 3a), however, there was poor quality of recording between 20:24 and 20:48 hours when epidural analgesia was being sited (Figure 3b). A vaginal examination at 21:00 hours revealed a 2 cm dilated and fully effaced

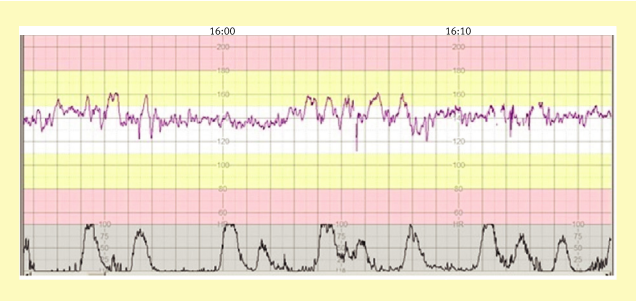

Figure 1 Normal reactive CTG with accelerations, normal baseline, good variability without any decelerations.

cervix (same finding as 12 hours before at first presentation to the triage). As there had been no change in cervical dilatation despite regular painful contractions, an oxytocin infusion was commenced at 21:20 hours at the rate of 1 mU/minutes (Figure 3c). At 22:50 hours, the rate of oxytocin infusion was doubled. Although a note was made of the decelerations on the CTG trace, the oxytocin infusion was further increased at 23:20 hours and again at 00:20 hours to 8 mU/minutes (Figure 3d and e). A repeat vaginal examination at 01:20 hours revealed the cervix to be fully dilated. At this point, the rate of oxytocin infusion was reduced to 4 mU/minutes because the contraction frequency exceeded 5 per 10 minutes (Figure 3f). In view of repeated decelerations, active pushing was commenced at 02:40 hours and there was spontaneous vertex delivery of a male baby at 03:12 hours with thick meconium stained liquor (Figure 3g and h). There was a loop of cord around the neck and Apgar scores were two at 1 minute and six at 5 minutes. The infant was resuscitated and transferred to the neonatal unit. The cord blood gases were – arterial pH 6.89, Base Excess (BE) – 24 and venous pH 7.06, BE – 17 and the birth weight was 3495 g. The baby was subsequently diagnosed with grade 2 hypoxic ischaemic encephalopathy (HIE).

This case is a typical example of gradually developing fetal hypoxia during labour. The initial part of CTG recording until 21:20 hours was normal (DR – epidural analgesia, augmentation with oxytocin, C – 4 in 10 minutes, BR – 150–155/minutes, A – Nil, VA – <5 for <40 minutes, D – nil, O – normal CTG). Since the woman was having four strong contractions in 10 minutes – oxytocin was not necessary and could have been withheld until the next vaginal examination to assess progress of cervical dilatation. By 23:20 hours, the CTG trace had become pathological (DR – epidural analgesia, augmentation with oxytocin, C – 4–5 in 10 minutes, BR – 160 to 165/minutes, A – Nil, VA – <5 for <40 minutes, D – typical variable decelerations with over 50% of contractions and few atypical variable decelerations occurring for over 90 minutes, O – pathological CTG). Despite the increasing baseline rate and repeated variable decelerations that were becoming deeper and wider, the oxytocin infusion was increased at 00:20 hours without any other test of fetal wellbeing such as FBS. At 01:20 hours, the CTG continued to be pathological with a base line rate of 160–170/minutes, normal baseline variability and atypical variable decelerations with overshoot. The contraction frequency was 6–7 per 10 minutes. By the time of commencement of active pushing at 02:40 hours, the trace was ominous and needed immediate delivery (DR – epidural analgesia, augmentation with oxytocin, C – 5–6 in 10 minutes, BR – 190–200/minutes, A – Nil, VA – <5 for >90 minutes, D – repeated atypical variable decelerations occurring for over 90 minutes, O – pathological CTG). This case illustrates the adverse perinatal outcome arising out of inability to interpret the CTG and not taking recommended actions based on the trace. A minimum of hourly formal systematic intrapartum CTG assessment has been recommended in every case by NICE guidelines. This was not followed in this case. NICE also recommends conservative measures such as change in position, stopping oxytocin infusion, tocolysis or hydration and reviewing the clinical situation, when the CTG trace becomes suspicious or pathological, and either FBS or timely delivery if the pathological trace does not respond to these measures. These guidelines were not followed in the above case leading to poor neonatal outcome.

Patterns of hypoxia resulting in birth injury

'Hypoxaemia' describes the condition where there is a reduction in the placental or cord blood flow causing a reduction in the level of oxygen in the peripheral arterial circulation of the fetus. This can happen in a normal labour with uterine contractions and majority of fetuses can cope well with such episodes for

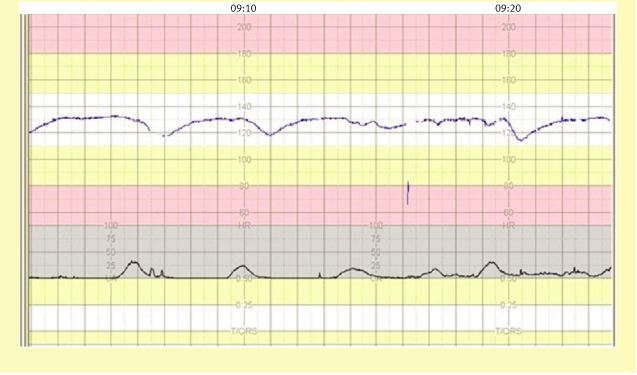

Figure 2 Pathological (pre-terminal) CTG with absent variability associated with shallow decelerations.

Definition of normal, suspicious and pathological FHR traces (NICE)

Category	Definition
Normal	An FHR trace in which all four features are classified as reassuring
Suspicious	An FHR trace with one feature classified as non-reassuring and the remaining features classified as reassuring
Pathological	An FHR trace with two or more features classified as non-reassuring or one or more classified as abnormal

Table 1