Electronic fetal heart rate monitoring in current and future practice

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Introduction

Electronic fetal heart rate monitoring (EFM) is aimed at assessing fetal wellbeing during pregnancy and labor. Although, the vast majority of fetuses cope well during labor, the journey through the birth canal is stressful and fetuses mount a ‘stress response’ during labor. Fetuses with utero-placental insufficiency develop hypoxia in labor that may be acute (developing over minutes) or sub-acute (developing gradually). Some fetuses may be hypoxic prior to entering labor. Fetal monitoring during labor should identify the fetuses at risk of hypoxic damage, so that appropriate intervention could be instituted to optimise perinatal outcome. Such an approach is likely to prevent neurological injury, including cerebral palsy. Indiscriminate use of electronic fetal heart rate monitoring in low-risk labor is known to increase operative interventions without any beneficial perinatal outcome.

It is important to distinguish a fetus that exhibits a stress-response to labor, from a one that shows a ‘distress’ or hypoxic response. Failure to do so increases unnecessary intervention in the former, while increasing the morbidity and mortality in the latter. One of the drawbacks of electronic fetal monitoring is the high sensitivity and low specificity, leading to increased false positive rates. This would mean that even if the CTG looks abnormal, the fetus may not be hypoxic. Hence, relying entirely on the CTG as a mode of fetal monitoring and acting on an abnormal CTG is likely to increase the operative delivery rates for normoxic fetuses, without improving perinatal outcome. Consideration of the clinical situation when interpreting the CTG and timely, appropriate action based on the findings may help prevent birth asphyxia.

In current obstetric practice, additional tests of fetal well-being like fetal blood sampling (FBS), fetal ECG, fetal pulse oximetry and fetal scalp blood lactate levels are employed to reduce the false positive rate of the CTG. Such an approach is likely to increase our ability to identify hypoxic fetuses, that actually need an intervention and to avoid unnecessary intervention to those fetuses, which are not subjected to a hypoxic insult.

Future research and technological developments are likely to revolve around the understanding of the short term and long term variability of the fetal heart rate, which may enhance our ability to further reduce the false positive rates of the currently available methods of electronic fetal heart rate monitoring.

Who should have electronic fetal heart rate monitoring (EFM) in labor?
EFM should be offered to high-risk women in labor. Use of EFM in low-risk women is not recommended as it is associated with increased obstetric intervention without any improvement in the perinatal outcome.

Continuous EFM during labor is associated with a reduction in neonatal seizures, but no significant differences in cerebral palsy, infant mortality or other standard measures of neonatal well-being. However, continuous EFM was associated with an increase in cesarean sections and instrumental vaginal births. Table 1 shows common indications for electronic fetal monitoring in current obstetric practice. It is important to change to continuous EFM, if abnormal fetal heart rate (baseline below 110 or above 160 or the presence of any decelerations) is detected on intermittent auscultation.

Table 1. Indications for electronic fetal heart rate monitoring in labor

<table>
<thead>
<tr>
<th>Maternal problems</th>
<th>Fetal problems</th>
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<tbody>
<tr>
<td>Induced labor</td>
<td>Prematurity</td>
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<tr>
<td>Diabetes</td>
<td>Oligohydramnios (possible cord compression / utero-placental insufficiency)</td>
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<td>Prolonged rupture of membranes (&gt;24 hrs)</td>
<td>Fetal growth restriction</td>
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<tr>
<td>Antepartum hemorrhage (possible placental abruption)</td>
<td>Abnormal doppler artery velocimetry</td>
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<tr>
<td>Previous cesarean section (possible scar dehiscence)</td>
<td>Multiple pregnancy</td>
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<tr>
<td>Pre-eclampsia (placental insufficiency)</td>
<td>Meconium-stained liquor</td>
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<tr>
<td>Post-term pregnancy (&gt;42 weeks)</td>
<td>Intrauterine infection</td>
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<tr>
<td>Other maternal medical disease (systemic lupus erythematosis (SLE) ; renal disease)</td>
<td>Intrapartum risk factors</td>
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<tr>
<td></td>
<td>Vaginal bleeding in labor</td>
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<tr>
<td></td>
<td>Oxytocin augmentation</td>
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<td></td>
<td>Epidural analgesia</td>
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<tr>
<td></td>
<td>Maternal pyrexia</td>
</tr>
<tr>
<td></td>
<td>Fresh meconium-stained liquor</td>
</tr>
<tr>
<td></td>
<td>Any deceleration or abnormal baseline rate on intermittent auscultation</td>
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</tbody>
</table>

EFM : Technological aspects

The machine

It is very important to familiarise with the machine and to understand specifications and the functions, especially the buttons and keys. Right type of paper that is compatible with the machine should be loaded. The clock should be set correctly, especially to ensure the alterations of the clock at the end of spring and autumn in some countries. The transducers and the maternal pulse should be checked and the fetal heart auscultated, prior to the application of the ultrasound transducer. The ‘toco’ transducer should be placed on the abdomen on the prominent part of the uterus below the fundus to record uterine contractions. Facilities to record fetal movements, as perceived by the mother may be available and this should be connected to the right port. Recording fetal movements is useful in the antenatal period. Care should be taken to avoid damage to the leads and the wires of the CTG machine.

The trace

It is very important to set the paper speed at the correct rate. In most countries, including the U.K., it is set at 1 cm/minute, while in the U.S.A., it is set at 3 cm/minute. Confusion can be caused if paper speed is incorrectly set, as the appearance of the CTG may change. For example, increase the speed from 1 to 3 cm/min may create the impression that the variability has reduced. This may result in an incorrect classification and unnecessary intervention. The patient’s details and maternal pulse should also be recorded. It is also important to record vaginal examinations, applications of scalp electrode etc. so as to optimise interpretation.

Commencement of electronic fetal monitoring in labor

Intrapartum fetal monitoring should be discussed with the woman, who is considered to be ‘high-risk’ and a clear plan of management should be made. This should be recorded in the antenatal notes. The woman’s informed choice should be respected, after counselling. The exact time of commencement of CTG would depend on individual clinical circumstances and the perceived risk of hypoxic insult to the fetus during labor. For example, if intrauterine growth restriction (IUGR) has been diagnosed antenatally, monitoring may be commenced earlier in labor, as these fetuses are likely to have a reduced placental reserve. There may be a further reduction in placental perfusion during uterine contractions that may potentiate the pre-existing utero-placental insufficiency, thereby increasing the risk of hypoxia.
It is important to switch to electronic fetal heart rate monitoring, if the patient develops vaginal bleeding, pyrexia or fresh meconium-stained liquor during labor.

The use of oxytocin for augmentation of labor, epidural analgesia, any deceleration or an abnormal baseline rate on intermittent auscultation increase the risk of intrapartum hypoxia.

**Role of admission CTG (cardiotocograph)**

Use of electronic fetal heart rate monitoring at the time of admission in labor has been employed by some centers to identify fetuses that are at an increased risk of hypoxia. Although, this may provide a ‘snap-shot’ view of fetal well-being at the time of admission, it may not identify those fetuses, that would subsequently develop intrapartum hypoxia. Currently, there is no evidence to support the use of admission CTG. Meta-analyses of available trials\(^3\) suggests that women with admission CTG were more likely to have epidural analgesia (RR 1.2 [95% CI 1.1 to 1.4]), continuous EFM (RR 1.3 [95% CI 1.2 to 1.5]) and fetal blood sampling (RR 1.3 [95% CI 1.1 to 1.5]). There is evidence that women with continuous EFM were more likely to have an instrumental birth (RR 1.1 [95% CI 1.0 to 1.3]) and caesarean section (RR 1.2 [95% CI 1.0 to 1.4]), compared with the auscultation group, although there were no differences in augmentation rates (RR 1.1 [95% CI 0.9 to 1.2]), perinatal mortality (RR 1.1 [95% CI 0.2 to 7.1]) or other neonatal morbidities.

It may have a role in obstetric units with a heavy workload (>10,000 deliveries/year) with limited resources to help in ‘Triaging’ fetuses.

In developed countries with good antenatal care, such fetuses may have been picked up by serial ultrasound or doppler scans, alleviating the need for an admission CTG.

**Interpretation of CTG**

There are four features that should be noted while interpreting a CTG trace. These are baseline fetal heart rate (FHR), baseline variability and the presence of accelerations and decelerations. The definition and classification of these features by NICE\(^4\) are given in Table 2.

The individual features of the CTG mentioned above, should be categorized into ‘re-assuring’, ‘non-reassuring’ and ‘abnormal’. This should be followed by the classification of the CTG into ‘normal’, ‘suspicious’ or ‘pathological’ (Table 3). This classification may help clinicians to understand and communicate issues relating to fetal well-being in an objective manner. Terminologies like ‘bad CTG’, ‘fetal distress’, ‘reassuring or non-reassuring CTG’ should be avoided.

Both the ‘cardiac’ and ‘toco’ (uterine activity) aspects of the CTG should be assessed (Figure 1). Uterine hyperstimulation, tetanic contractions and sudden loss of uterine activity may provide a vital clue to the underlying pathology responsible for an abnormal CTG. When planning an intervention based on the changes on a CTG trace, consideration of the risk factors (Table 1) that may increase the chances of an intrapartum asphyxiial injury is essential.
Normal CTG

All the four parameters of the fetal heart trace should be within the ‘reassuring category’ for the CTG to be classified as ‘normal’. It is easy to recognize a normal CTG. It has been shown that the risk of fetal hypoxia and acidosis is extremely small when the CTG is normal. False negatives may sometimes occur (poor outcome despite of a normal CTG) and these may be due to factors other than hypoxia such as intrauterine infection, maternal pyrexia and fetal congenital or metabolic problems that may influence fetal outcome. In some of these cases the CTG could be abnormal and these insults can cause perinatal neurological injury independently or may aggravate the effects of intrapartum hypoxia. Changes of the baseline heart rate and variability over time may be easily missed, if only the segment in question is examined at a time. To avoid this pitfall, the entire CTG recording should be assessed during interpretation.

Figure 2 shows a normal CTG. Presence of accelerations (>2 episodes in 20 minutes, each rising at least 15 beats per minute above the baseline rate lasting for >15 seconds) usually associated with fetal movements, appears to suggest an intact somatic nervous system. Normal baseline variability indicates an intact autonomic nervous system.

Consideration of the clinical situation

The entire clinical picture should be considered, while reading and interpreting the CTG. These include the stage and progress of labor, ability of the fetus to withstand further hypoxia (i.e. physiological reserve of the fetus, thick scanty meconium, prematurity, antepartum or intrapartum bleeding, pyrexia, infection), uterine activity, oxytocin augmentation and feasibility of assisted vaginal delivery. If an abnormality is noted, intervention does not always warrant immediate delivery. Simple measures such as hydration, changing maternal position, reduction or temporarily stopping oxytocin infusion and rarely acute tocolysis may help to restore the CTG trace back to normal.

Changes in baseline fetal heart rate (FHR)

Baseline changes include baseline tachycardia (an increase in the baseline FHR above 160 beats / min) and baseline bradycardia (baseline FHR below 110 beats/min). The former may be physiological in a preterm fetus due to the immaturity of the parasympathetic system or secondary to maternal pyrexia, dehydration or rarely due to medications (e.g. beta mimetics). Epidural analgesia has also been found to be associated with intrapartum fever, leading to fetal tachycardia.

Maternal pyrexia contributing to fetal tachycardia may be due to an infective process. Fetal neurological injury may occur secondary to infection and infection per se may reduce the threshold for hypoxic brain damage. Various infective organisms can cause localized degeneration or damage to brain tissue (porencephaly), leading to neurological damage and cerebral palsy.

A rise in baseline fetal heart rate may also be an ominous sign of fetal hypoxia secondary to utero-placental insufficiency. In this situation, a fetus tries to increase the cardiac output mainly by increasing the heart rate to supply the vital organs with oxygen and nutrients. Association of baseline tachycardia with other abnormal features (reduced baseline variability or deceleration) is termed ‘complicated baseline tachycardia’.

A decrease in the fetal heart rate below 110 beats / minute is called baseline bradycardia, which may be a physiological finding in a post-term fetus and the fetus is unlikely to be hypoxic in this situation, provided there are accelerations and normal baseline variability. By definition, the low baseline should persist for more than
10 minutes, to be termed ‘baseline bradycardia’. This should be distinguished from transient decrease in the baseline below 80 beats/min, which is called ‘prolonged deceleration’. If it lasts for less than three minutes, it is considered ‘suspicious’ and if such a prolonged deceleration lasts for more than three minutes, it is considered to be ‘abnormal’. The latter is an indication to consider remedial actions or delivery.

Repeated ‘prolonged decelerations’, when the FHR is <80 beats/min for longer periods than at the baseline are likely to lead to rapid development of hypoxia. (sub-acute hypoxia) within 30-60 minutes.2

Baseline variability

The ‘band-width’ of variation of the heart rate above and below the baseline on the CTG trace is called baseline variability. This reflects the ‘integrity’ of the central nervous system centers that are responsible for the control of the fetal heart rate: the sympathetic and parasympathetic nervous systems. The normal baseline variability of 5-25 beats implies that cerebral hypoxia is unlikely. Reduced baseline variability of 0-5 beats may represent a quiet sleep phase or may indicate hypoxia to the central nervous system.

It may also be due to drugs (CNS depressants), infection or cerebral hemorrhage. An increase in the baseline variability (more than 25 beats/min) is termed ‘saltatory’ pattern and should be considered to be an abnormal feature, which needs closer observation, especially if it is associated with decelerations and may warrant further investigations or action.

Accelerations

A transient increase in the fetal heart rate > 15 beats from the baseline rate and lasting for > 15 seconds is termed ‘acceleration’. Accelerations “appear” to reflect the integrity of the somatic nervous system as they are almost always associated with fetal movements (FM) and are not present in the absence of fetal movements due to hypoxia, infection or cerebral hemorrhage. Accelerations are absent during “fetal sleep” and presence of accelerations rules out fetal acidosis.3 According to the NICE guidelines, absence of accelerations is of uncertain significance and one has to identify causes that may abolish accelerations such as hypoxia, infection, medication, brain hemorrhage etc. Figure 2 shows typical accelerations.

Decelerations: types and significance

A transient decrease of the fetal heart rate below the baseline heart rate (15 beats per min (bpm) and lasting for more than 15 seconds) is termed ‘deceleration’. The decelerations are classified as early, late and variable in relation to the uterine contractions. It is important to appreciate that labor is a complex process with several patho-physiological processes i.e. reduced placental reserve, head or cord compression can occur together at any given time. Hence, it is possible to have decelerations that have characteristics different from the three standard types described below.

Early decelerations (figure 3)

These are termed ‘mirror-image’ decelerations: they start with the onset of uterine contractions, reach the nadir with the peak of contractions and return to the baseline at the end of the contraction. They are due to head compression and the resultant stimulation of the parasympathetic nervous system and hence are observed during the late first stage or second stage of labor.

Late decelerations (figure 4)

Late decelerations are so termed because they are late: both the onset of deceleration as well as the subsequent recovery to the baseline rate. The nadir of these decelerations is seen after the peak of uterine contraction and they return to baseline at least 20 seconds after the contraction wanes off. Presence of late decelerations indicates utero-placental insufficiency, mediated through the fetal chemo-receptor mechanism secondary to fetal hypoxemia, hypercarbia and acidosis. Further tests of fetal wellbeing are indicated to exclude fetal hypoxia, if it is intended to continue with labor. Figure 4 shows late decelerations with loss of variability. The probability of fetal hypoxia is very high, with such a ‘pre-terminal trace’.
Variable decelerations

Variable decelerations are the commonest decelerations occurring during labor and they are so named because they vary in shape, form and timing in relation to the uterine contractions. These are due to cord compression.

A ‘typical’ variable deceleration consists of a slight rise in the fetal heart rate (called ‘shouldering’) both before and after the deceleration. Simple or uncomplicated variable deceleration lasts for less than 60 seconds and the drop from the baseline rate is < 60 beats, in the presence of normal baseline heart rate and variability (Figure 5).

Variable decelerations that do not conform to the typical features as described above are termed ‘atypical’. As shown in Figure 6, these may last for more than 60 seconds and may lose their shouldering, have a slow recovery, have an ‘overshoot’ or be combined with a late deceleration.

Oligohydramnios may cause variable decelerations and a recent Cochrane Review on amniofusion for potential or suspected umbilical cord compression during labor concluded that amniofusion is useful to reduce the variable decelerations and to reduce cesarean section mainly for fetal distress diagnosed by fetal heart rate monitoring alone.

Classification of the CTG using the recent NICE guidelines is given in Tables 2&3.

Electronic fetal monitoring: the shortfalls

CTG has very high sensitivity and is capable of excluding intrapartum hypoxia, if all the features are in the ‘re-assuring’ category. However, the ‘trade-off’ is that the CTG has a high false positive rate. Hence, it is a poor predictor of fetal hypoxia and metabolic acidosis. Beard et al reported that even with significant abnormalities in the CTG, the risk of fetal acidosis in fetal blood sampling was only 50%. An earlier prospective study of 6825 patients found the positive predictive value of an abnormal CTG trace for babies who needed intermittent positive pressure ventilation to be 8.7%. The same study reported a positive predictive value of an abnormal CTG trace to be 27.4% for all babies with an apgar score of less than 7 at five minutes. The false negatives may be due to adverse factors other than hypoxia. These include fetal infection, metabolic abnormalities, head compression and the effects of analgesia in labor.
Maternal heart rate trace

It is important to ensure that it is the fetal heart rate that is being recorded during labor. Failure to do so may result in poor perinatal outcome, due to a false sense of security. Maternal heart rate may be erroneously recorded, especially in late first stage or second stage of labor. This is because the transducer is often repositioned downwards, as the labor progresses and the fetal head descends into the pelvis, in order to ‘get a good signal’. At this stage, the transducer may pick up signals from the maternal iliac vessels resulting in the recording of the maternal heart rate.

As shown in Fig 7, the maternal heart rate trace has a greater variability and accelerations are characterised by increased amplitude and duration. The most important differentiating feature is that fetal heart rate often shows a deceleration during contraction, especially in advanced labor. In contrast, the maternal heart rate shows accelerations during contractions. This is similar to the heart rate response during physical exercise and reflects the ‘work’ that is performed by the mother during labor. Pain, maternal anxiety and stress during uterine contractions also contribute to the increase in the maternal heart rate.

![Figure 7. Maternal heart rate trace showing increased variability and accelerations with contractions.](image-url)
It is important to check the maternal pulse, if this is suspected to exclude such an erroneous recording. It is recommended that prior to instituting electronic fetal heart rate recording, fetal heart rate should be auscultated with a Pinard’s fetoscope to avoid recording the maternal heart trace at the outset.

Pathological CTG: need for ‘additional tests of fetal wellbeing’

In view of the problem of high ‘false positive’ results with the CTG, reduction in unnecessary intervention based on abnormalities in EFM during labor is essential. Several additional tests of fetal wellbeing have been described14. These include fetal blood sampling (FBS), fetal ECG (ST- Analyser), fetal scalp lactate and pulse oximetry.

Fetal scalp blood sampling (FBS)

Estimation of the pH on the sample of blood taken from the fetal scalp may help detect acidosis and hence reduce the false positive rate of the CTG. It is relatively a simple procedure but requires facilities for blood gas analysis. It is not recommended in fetuses with bleeding disorders or in cases where there is a risk of vertical transmission of infections such as in pregnancies complicated with HIV and hepatitis B. It is important to evaluate both the pH and the base excess (BE) as the base excess will provide an idea of the degree of depletion of the buffering system. This will in turn indicate the degree of metabolic acidosis.

**pH**

<table>
<thead>
<tr>
<th>Normal</th>
<th>&gt;7.25</th>
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<tbody>
<tr>
<td>Suspicious</td>
<td>7.20-7.24 (Repeat FBS in 30 – 60 min)</td>
</tr>
<tr>
<td>Abnormal</td>
<td>&lt;7.20</td>
</tr>
</tbody>
</table>

**Base excess**

<table>
<thead>
<tr>
<th>Normal</th>
<th>&lt; 8 mmol/l</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abnormal</td>
<td>&gt; 12 mmol/l</td>
</tr>
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Although, the use of fetal blood sampling may reduce operative delivery rates, there is no evidence to suggest that it confers any improvement to the overall neonatal outcome 3.

ECG waveform analysis (ST Analyser -STAN)

ST-Analyzer (or STAN) helps us to identify fetal hypoxia by detecting the changes in the ST segment of the fetal electrocardiograph (ECG), which reflects myocardial hypoxemia. In response to hypoxia, there is a catecholamine surge, which promotes glycogenolysis in the myocardium. The influx of glucose with the potassium into the cells causes changes in the T wave. This change is reflected by the increase in the T/QRS ratio. These are analyzed by the computer to give us an indication of myocardial oxygenation status. Used in conjunction with EFM, this technique may help us to detect fetuses that show a reaction to hypoxia. Unlike the scalp pH, STAN provides information about the oxygen status in a central organ (myocardium), which may also reflect cerebral oxygenation. Two randomized controlled trials comparing the use of STAN with the CTG versus CTG alone have shown a reduction in the operative delivery rates and metabolic acidosis with the use of STAN monitoring15. Good correlation has been shown between fetal scalp PH and the ST-changes (events) that are observed using STAN16.

Fetal pulse oximetry (FSpO2)

Fetal pulse oximetry is based on the principle of differential rates of absorption of infrared beam of light by oxygenated and de-oxygenated hemoglobin at two different wavelengths, during an arterial pulsation cycle.

It aims to measure the oxygen content of hemoglobin in tissues, which is a measure of the unabsorbed red and near infra-red light. A reading of SpO2 above 30 % is associated with good fetal outcome. FSpO2 correlates well with both the CTG and FBS and its predictive value is similar to that of FBS for an umbilical artery pH of <7.20. It improves the specificity of the CTG and enables a direct and continuous assessment of fetal oxygenation. Its drawbacks include technical difficulties in placing the device in the presence of a high presenting part and inability to get continuous reading due to contact sensor17. A recent Cochrane review has shown the safety of its use but has not shown a reduction in the overall caesarean section rate18.

Blood lactate

One of the drawbacks of fetal scalp pH estimation is its high failure rate because a larger sample of blood is needed for analysis. Recently, it has been shown that it takes about 18 minutes on average, to obtain a fetal scalp pH estimation19. In 9% of patients this procedure took over 30 minutes19. Unfortunately, this may result
in a further delay in delivery, when there is a suspicion of fetal compromise.

Fetal scalp blood lactate is aimed at overcoming these problems with FBS, as the amount of blood sample that is required is 5µL instead of 35-50µL needed for pH and blood gases20. It is aimed at directly measuring lactic acid, which is the end product of anaerobic metabolism21. The recent NICE Guidelines on intrapartum care has suggested that there is insufficient evidence to suggest any correlation between blood lactate levels and long term neonatal outcome4.

**EFM : the future**

Electronic fetal heart rate monitoring involves appropriate selection of patients, recognition of normal and abnormal CTG patterns, incorporation of clinical picture and the institution of timely and appropriate intervention. It is therefore open to human error, which accounts for the majority of cases of intrapartum obstetric litigation22. Attempts have been made recently to avoid relying on human factors for interpretation, by using computerized fetal heart rate parameters during labor. However, these did not correlate well with the degree of metabolic acidosis in the umbilical artery at birth23. More recently, there has been a renewed interest in further analysis of fetal heart rate variability during labor. A computerized analysis of fetal heart rate variability using the ‘matching pursuit’ (MP) technique has been tried, with some success24. This involves the use of an adaptive time-frequency model, called the ‘matching pursuit’ to evaluate the fluctuations in the frequency of the fetal heart rate. It has been shown that the ‘low-low frequency (LLF) parameter appeared to recognize the cases with lower pH with a sensitivity and specificity, of 78.5% and 52.3%, respectively24. Although, currently there is limited evidence to support the use of computerized electronic fetal heart rate monitoring during labor, future research is likely to help greater understanding of the short and long term variability of the fetal heart rate. These include ‘detrended analysis’ of fetal heart rate variability, which has shown some promise in fetus with intrauterine growth restriction 25.

**Conclusion**

Continuous electronic fetal heart rate monitoring during labor is associated with a reduction in neonatal seizures, but no significant differences in cerebral palsy, infant mortality or other standard measures of neonatal wellbeing4. However, continuous cardiotocography was associated with an increase in caesarean sections and instrumental vaginal births1. Hence, in current practice, electronic fetal heart rate monitoring (EFM) should be reserved for high risk pregnancies, with potential threat of intrapartum fetal hypoxia. Recognition of abnormalities of the fetal heart rate as well as instituting timely and appropriate action, are both essential to improve the perinatal outcome26. The role of education and training cannot be over-emphasized. Although, it is likely that there may be exciting developments with regard to the understanding and usage of short term variability of the fetal heart rate in clinical practice in the future, it is important to consider the basic pathophysiological processes, which may be contributing to fetal heart rate abnormalities as well as the entire clinical picture, while interpreting the trace.

Points to ponder while interpreting the CTG

- Accelerations and normal baseline variability are hallmarks of fetal health
- Periods of decreased variability may represent fetal sleep.
- Cycling refers to periods of alternating activity and quiescence (sleep) phase and reflects fetal wellbeing.
- Hypoxic fetuses may have a normal baseline FHR of 110-160 bpm with no accelerations and baseline variability of <5 bpm for >40 min
- In the presence of baseline variability <5 bpm even shallow decelerations <15 bpm are ominous in a non-reactive trace
- Hypoxia can be worsened by oxytocin, epidural analgesia and difficult operative deliveries
- During labor, if decelerations are absent, asphyxia is unlikely.
- Abnormal patterns may represent effects of drugs, fetal anomaly, infection not only hypoxia.
- It is important to monitor uterine activity during the use of oxytocin to avoid uterine hyperstimulation

**References**


