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
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

<table>
<thead>
<tr>
<th>Category</th>
<th>Definition</th>
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<tbody>
<tr>
<td>Normal</td>
<td>An FHR trace in which all four features are classified as reassuring</td>
</tr>
<tr>
<td>Suspicious</td>
<td>An FHR trace with one feature classified as non-reassuring and the remaining features classified as reassuring</td>
</tr>
<tr>
<td>Pathological</td>
<td>An FHR trace with two or more features classified as non-reassuring or one or more classified as abnormal</td>
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Table 1
Long standing or chronic hypoxia — this happens due to reduction in placental blood flow over a long period of time and is associated with underlying conditions such as pre-eclampsia or fetal growth restriction. In the antenatal period the fetus will cope for a significant period of time by redistribution of the blood flow to vital organs, reduction in growth or activity and buffering against lactic acid. Antenatal surveillance with Doppler ultrasound can help predict when the fetal decompensation is likely so that delivery can be recommended. In labour, such hypoxia is likely to be associated with a CTG trace with no baseline variability and repetitive shallow late decelerations.

Gradually developing hypoxia — in gradually developing hypoxia, accelerations do not appear, the baseline rate increases and the variability reduces with progress of time. The decelerations get deeper and wider with increasing hypoxia. Attending staff need to consider the clinical picture of parity, cervical dilatation, rate of progress and high risk factors, and either perform fetal scalp blood sampling (FBS) or consider delivery. The natural response for the fetus with hypoxic stress that previously had a reactive CTG would be the appearance of decelerations (variable due to cord compression or late due to placental insufficiency), the disappearance of accelerations (fetal response to conserve energy), gradual rise in the baseline rate (due to hypoxia and catecholamine surge), deepening and widening of the decelerations (with increasing hypoxia to the myocardium) and finally progressive reduction of the baseline variability (after a maximum baseline rate has been achieved and with further lack of oxygen there is depression of the autonomic nervous system).

Acute hypoxia — is characterized by a sudden reduction in placental/cord blood flow and develops over minutes. Causes include acute accidents such as cord accident, abruption, hypertonic contractions or uterine dehiscence and CTG often shows prolonged deceleration or bradycardia. Management demands rapid delivery or treatment of hyperstimulation to prevent death or long term damage.

Subacute hypoxia — this may occur due to recurrent cord compression in labour. It may be particularly worsened in situations like oligohydramnios or prolonged pregnancies especially in the second stage of labour.

Hypoxic insults that are slow in onset and persistent over time allow the fetus to make homeostatic adaptations. These protective adaptations begin to fail with the development of acidemia and at pH less than 7.0 the entire fetal and cerebral oxygen consumption fall substantially. Acidemia leads to loss of vascular tone, cardiac cell injury, depressed myocardial function, and hypotension with resultant ischaemic brain injury.

Case 2

A primigravida at 37 weeks with a diamniotic dichorionic (DCDA) twin pregnancy, conceived through IVF, presented in labour at 23:00 hours. The pregnancy had been otherwise normal and history revealed spontaneous rupture of membranes with passage of clear liquor at 10:00 hours in the morning. General examination findings were unremarkable. Presentation of twin 1 was cephalic and twin 2 was breech. The cervix was 3 cm dilated and well effaced. Both twins had a normal CTG trace (Figure 4a, twin 1 — blue and twin 2 — pink). Two hours later, at 01:00 hours, the woman requested epidural analgesia when a vaginal
examination revealed the cervix to be fully dilated. The woman was transferred to theatre for epidural analgesia and delivery. An epidural was sited by 02:30 hours and oxytocin augmentation was commenced in view of ‘mild’ uterine contractions (Figure 4b). Since the delivery was not imminent, a plan was made to allow time for passive descent of vertex and the woman was transferred out of theatre back to the room (Figure 4c). The rate of oxytocin infusion was increased as per the protocol (Figure 4d). Active pushing was commenced at 04:00 hours (Figure 4e) and oxytocin infusion further increased at 04:20 hours (Figure 4f). At 04:45 hours decision was made for a trial of instrumental delivery in view of failure to progress. However, this was delayed due to another emergency on the labour ward at the same time (Figure 4g). At 05:00 hours the oxytocin infusion

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**Figure 3 (a–h):** Primigravida at 41 weeks in labour with history of SROM for 36 hours. By 23:20 hours, the CTG trace had become pathological (DR — epidural analgesia, augmentation with oxytocin, C — 4—5 in 10 minutes, BR — 160—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). Gradually developing hypoxia (possibly due to cord compression) with rising baseline rate, severe atypical variable decelerations and diminished variability resulted in hypoxic damage to the fetus.
was increased further. When the midwife-in-charge reviewed the
woman at 05:30 hours, the oxytocin infusion was discontinued
due to concerns with the CTG trace (Figure 4h). The woman was
transferred again to the theatre at 06:00 hours and oxytocin was
restarted at 06:15 hours (Figure 4i). Twin 1 was delivered by
ventouse at 06:17 hours. The birth weight was 2170 g and Apgar
scores were nine at 1 minutes and nine at 5 minutes (Figure 4j).
Cord blood gases were — arterial pH 7.25, BE — 4 and venous pH
7.29, BE — 3. After delivery of the twin 1, the plan was to
continue oxytocin to allow for descent of breech twin 2 while the

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**Figure 4 (a–l):** Primigravida with DCDA twin pregnancy at 37 weeks in labour. Twin 1 (blue) was cephalic and twin 2 (pink) breech presentation. By 04:45 hours, the CTG trace for twin 2 was pathological (DR — epidural analgesia, twins, augmentation with oxytocin, C — 3–4 in 10 minutes, BR — 160–180/min and difficult to identify, A — Nil, VA — <5 for >90 minutes, D — repeated atypical variable decelerations, O — pathological CTG). Delayed delivery of twin 2 despite pathological CTG lead to hypoxic damage and neonatal death.
registrar sutured a bleeding paraurethral tear. However, due to failure of descent of the breech by 06:42 hours, twin 2 was delivered by emergency caesarean section at 06:52 hours (Figure 4g). Twin 2 had Apgar scores of two at 1 minute and six at 5 minutes (Figure 4k and l). The cord arterial blood could not be obtained for pH and the venous pH was 6.63 and BE −27. Birth weight was 2510 g. The caesarean delivery was complicated by maternal bladder injury requiring surgical repair and suprapubic catheter following the operation. Twin 2 was diagnosed with HIE grade 3 followed by neonatal death on day 20.

There are a number of key learning points which arise out of this case scenario. The woman could have been offered the epidural analgesia in the room and transferred to theatre once the delivery was imminent. Generally for a primigravida with epidural analgesia, the duration of passive descent in second stage of labour is 2 hours. This is followed by active pushing for another hour. In this case, the obstetric review in second stage occurred 4 hours after full dilatation of the cervix and the delivery of twin 1 was almost five and half hours since onset of second stage of labour. If the delivery unit was busy, further help should have been considered. The registrar should have been able to call the consultant to help “as an extra pair of hands” for timely delivery in theatre. There was very poor documentation of CTG reviews in the notes and hourly systematic assessments as per NICE guidelines had not occurred. Despite noting abnormalities in the CTG – no systematic classification had been followed and no action plan was documented. Beginning from 04:00 hours, the quality of CTG recording for the second twin (pink) was poor. By 04:45 hours, the CTG trace for twin 2 was pathological (DR – epidural analgesia, twins, augmentation with oxytocin, C − 3–4 in 10 minutes, BR − 160–180/min and difficult to identify, A − nil, VA − <5 for >90 minutes, D – repeated atypical variable decelerations, O – pathological CTG). Despite the increasing baseline rate, reduced baseline variability and repeated atypical variable decelerations, the woman was allowed to continue in the second stage with oxytocin infusion until intervention occurred at 06:15 hours. Following delivery of twin 1, there was extremely poor quality recording of twin 2 FHR. Despite evidence of pathological CTG with absent variability and deep prolonged atypical variable decelerations, no recourse to early delivery of twin 2 by caesarean was considered. Appropriate CTG assessment at onset of repeated FHR decelerations (04:45 hours) and timely caesarean delivery would have resulted in a better outcome for twin 2 and the mother.

Case 3

A 34-year-old primigravida presented to the labour ward at 42 weeks in spontaneous labour. She had been low risk throughout pregnancy and had received a membrane sweep at 41 + 1 weeks gestation. Ultrasonography at 41 + 3 weeks had revealed a healthy fetus with normal growth and good liquor volume. At admission at 22:30 hours, her general examination findings were normal (afebrile, pulse − 80/minutes). The cervix was 4 cm dilated with intact membranes. The CTG was normal (Figure 5a). Epidural analgesia was sited at 00:15 hours. At the second vaginal examination at 03:15 hours, cervix was 8 cm dilated, no membranes were felt and the position of vertex was occipito-posterior at spines (Figure 5b). At this point the woman was asked about history of SROM. Although there was no clear
history of gush of fluid, she reported feeling leakage of fluid per vaginum for past 24 hours. In view of this history, intravenous benzyl penicillin was started. At 03:35 hours, the woman’s temperature was noted to be 37.6°C with a pulse rate of 86/

Figure 5 (a–h): 34-year-old primigravida at 42 weeks in labour with uncertain history of duration of SROM and pyrexia in labour. The trace shows progressive increase in baseline rate with diminished variability and atypical variable decelerations. The CTG trace was pathological since 03:30 hours and had repeated atypical decelerations by (DR — epidural analgesia, pyrexia, prolonged SROM, meconium, C — 3–4 in 10 minutes, BR — 180–200/minutes, A — Nil, VA — <5 for >90 minutes, D — repeated severe atypical variable decelerations, O — pathological CTG) by 06:00 hours. Delivery of a depressed fetus occurred by caesarean section following failed forceps and eventually resulted in neonatal death. There was evidence of chorioamnionitis due to Staphylococcus aureus at postmortem examination.

minutes. Oral paracetamol and cooling measures were given. She started feeling unwell at 04:15 hours with complaints of rigours and headache. The obstetric and anaesthetic teams reviewed her at 04:50 hours. The cervix was 9 cm dilated,
position of vertex was right occipito-anterior and station was +1. Maternal temperature was 36.7 degrees but the pulse rate was 110–130/minutes. The CTG showed a corresponding fetal tachycardia (Figure 5c). A plan was made to administer intravenous fluids, perform blood cultures and reassess in 1 hour. At 05:15 hours, maternal temperature was 39 degrees (Figure 5d) and intravenous paracetamol was administered. At 05:50 hours, cervix was fully dilated. There was evidence of grade 2 meconium and position of vertex was right occipito-transverse at spine (Figure 5e). The consultant reviewed the case at 06:05 hours and decided to transfer to theatre for delivery. The woman was in theatre with an epidural top-up by 07:00 hours. Vaginal examination findings were — cervix fully dilated, occipito-posterior position at spines with +1 caput. Forceps delivery was attempted. However, delivery could not be accomplished despite 3 pulls of forceps (Figure 5f and g). At 07:20 hours — decision was made for emergency caesarean section which was complicated by difficult delivery of the baby (delivered by breech with fractured humerus). Apgar scores at birth were 0 at 1 and 5 minutes. The cord blood gases were — arterial pH 6.87, BE — 22 and venous pH 7.3, BE — 8. Birth weight was 3435 g. Treatment was withdrawn at 10 hours of age followed by neonatal death. Postmortem findings revealed recent diffuse hypoxic ischaemic brain injury. There was evidence of acute chorioamnionitis with Staphylococcus aureus isolated from lungs and spleen. The placenta contained abnormal perivillous fibrin deposition.

Hypoxia may not be the only damaging factor in labour but acute fetal inflammatory stress response to infection and pyrexia can lead to adverse perinatal outcomes. The patterns of CTG associated with such insults may vary considerably and need to be carefully interpreted based on the overall clinical scenario. The presence of infection/pyrexia has been found to be an important finding in fetuses that are destined to develop cerebral palsy. The fetal inflammatory response associated with maternal fever during labour, chorioamnionitis and funisitis has been implicated as a cause of later cerebral palsy. It is believed that inflammatory cytokines can cause cerebral ischaemia resulting in damage to the fetal central nervous system. This information needs to be factored in while managing cases with pathological CTGs in relevant clinical scenarios. In the given case scenario — the CTG trace was pathological since 03:30 hours and had repeated atypical decelerations by (DR — epidural analgesia, pyrexia, prolonged SROM, meconium, C = 3–4 in 10 minutes, BR — 180–200/minutes, A — Nil, VA — <5 for >90 minutes, D — repeated severe atypical variable decelerations, O — pathological CTG) by 06:00 hours. Given the overall clinical picture, there was a need for immediate delivery to avoid adverse outcomes. The case also illustrates the need to act promptly once a decision for delivery has been made due to fetal concerns. Sometimes the time taken to shift the woman to theatre and get her ready for operative delivery can make a big difference to the ultimate clinical outcome.

CTG monitoring, medico-legal issues and best practice recommendations

In October 2012, the NHS Litigation Authority (NHSLA) in UK published a report — ‘Ten Years of Maternity Claims: An Analysis of NHS Litigation Authority Data’ which provided an analysis of the various clinical situations that have led to maternity claims. The project analysed ten years of maternity claims with an incident date between 1st April 2000 and 31st March 2010 (5087 claims) with a total value of £3.1 billion. While Obstetrics and Gynaecology claims totalled approximately half of the number of surgical claims during the same period, the total value was more than double the total value of surgical claims and in fact exceeded the joint total value of claims from the next three biggest specialities (Surgery, Medicine and Accident and Emergency). The three most frequent categories of claim were those relating to management of labour (14.05%), caesarean section (13.24%) and cerebral palsy (10.65%). Two of these categories, namely cerebral palsy and management of labour along with CTG interpretation, were also the most expensive and together accounted for 70% of the total value of all the maternity claims.

Best practice recommendations and risk management issues related to EFM

While it is easy to perform retrospective analysis of patient notes with CTG trace and criticize the clinical management of a given case — the situation faced by the clinical team on ground is often not straightforward and easy. When things go wrong, there are multiple difficulties and system failures which contribute towards adverse outcomes in patients. Bearing this in mind, clinical teams should set themselves with realistic goals, with the ultimate aim of improving care of women in labour especially in the context of intrapartum fetal monitoring. The best approach to minimize adverse outcomes is to ensure that every member of the team adheres to evidence based guidelines and continually updates his or her knowledge of CTG interpretation and documentation.

Important points to consider while recording or evaluating a CTG trace are —

Patient identity

The name of the woman, the date and time of commencement of recording should be entered on every trace. The clock on the machine must always be checked.

Maternal pulsations

FHR should be auscultated prior to application of the electronic probe to avoid picking up maternal pulsations. In addition, the maternal pulse should be identified and recorded separately. Sudden significant shift in the baseline or low baseline FHR suggest recording of the maternal pulse rather than fetal heart rate. If there is doubt, ultrasound should be used to locate the fetal heart and a fetal scalp electrode may be a better alternative in such a situation. Slippage of transducer from tracking the fetal heart to a maternal pulse should be identified and recorded separately. In addition, the clock on the machine must always be checked.

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Poor quality of the trace

The FHR tracing is difficult to interpret when there is persistent signal loss. The situation should be corrected by adjusting the transducer, or obtaining the signal via a scalp electrode, or changing the connections and/or machine. If these actions do not rectify the problem, intermittent auscultation should be performed and this should be documented in the medical records.

Misinterpretation of CTGs

Numerous studies have found poor agreement between individuals and even some inconsistency when the same individual was asked to repeat his or her readings of CTGs at another time. CTG interpretation is subject to variation outside the extremes of normal and grossly pathological traces. It is important that each individual obstetrician or midwife makes an effort to learn how to interpret CTG findings correctly and maintains these skills through continued medical education from time to time. In the UK, NICE has attempted to standardize the interpretation of CTGs while in the United States, the National Institute of Child Health and Human Development (NICHD) workshop has proposed the three tier system of interpretation of FHR patterns.

Emphasis should be paid to observe for reactivity (accelerations) and cycling (quiet and active sleep cycles) that indicates a non-hypoxic fetus with a normal behavioural pattern. Absence of cycling may be due to drugs, infection, cerebral haemorrhage, chromosomal or congenital malformation, previous brain damage. A non-reactive trace with baseline variability < 5 bpm and shallow decelerations (<15 beats) for >90 minutes suggests pre-existing hypoxia. In the presence of a clinical picture like post term, growth restriction, absent fetal movements, antepartum haemorrhage or infection such a trace should prompt earlier delivery. A previously brain damaged fetus may or may not show cycling but the cord pH at birth may be normal; such babies may not show evidence of HIE but may exhibit signs of neurological damage that manifests later.

Inappropriate action with suspicious or pathological CTG

Once a diagnosis of suspicious or pathological FHR trace is made — action must be taken depending on the severity of CTG abnormality. This may mean continued observation, change in maternal position, administration of tocolytic, hydration, omission of oxytocin infusion in cases with suspicious traces and in addition fetal blood sampling/immediate operative delivery in cases with pathological trace. Accurate documentation of the time of observation and any other actions taken is very important from a medico-legal view point. The importance of considering the clinical picture in planning management is essential. Earlier action is needed in the presence of fetal growth restriction, preterm, post term, intrauterine infection and thick meconium with scanty fluid. Injudicious use of oxytocin, epidural and difficult operative delivery can give rise to further compromise when the CTG is pathological.

In the presence of an abruption, cord prolapse or scar rupture intervention should take place when the diagnosis is made as they warrant immediate delivery (within 15–30 minutes). In these situations the CTG may suddenly present with prolonged deceleration. In cases of bradycardia <80 bpm the pH can decline by 0.01 every minute and with prolonged decelerations that have transient recovery to the baseline rate the pH can decline by 0.01 every 2–3 minutes. Fetal scalp blood sampling (FBS) is an inappropriate action in such situations and is likely to compromise the baby. Special arrangements should be in place in each unit to deliver these cases as category 1 caesarean section.

In situations of pathological CTG due to uterine hyperstimulation especially that due to prostaglandins or in cases with normal labour with pathological heart rate pattern where a theatre is not available, a bolus dose of a tocolytic drug like terbutaline may help to abolish the uterine contractions helping the baby to be born in better condition despite the delay.

Role of adjuvant tests

Fetal scalp blood sampling (FBS) — CTG is an imperfect tool and many of the indicators used to identify early hypoxia are non-specific. Thus, if CTG is used as the sole method of intrapartum fetal surveillance, unnecessary operative deliveries will be performed. Fetal scalp blood sampling for pH analysis and lactate measurement is used to assess fetuses with pathological FHR patterns. FBS is recommended with pathological CTGs to identify those fetuses that require delivery. Ideally all maternity units where CTG is employed should have ready access, 24 hours a day to an accurate blood gas analyser.

Fetal ECG waveform analysis — Increasing number of centres are taking up the use of fetal ECG in combination with continuous EFM as an additional test of fetal wellbeing. The fetal ECG is recorded continuously from a specialized scalp electrode and analysed by computer technology. Fetal hypoxia can act on the fetal myocardium and cause alterations to the ST segment of the fetal ECG. The drawback of the method is that the technology relies considerably on human interpretation of the CTG to indicate the appropriate action when ST segment changes occur.

Overall clinical picture and pattern evolution of CTG

Generally, during the intrapartum period a hypoxia-induced reduction in FHR variability develops gradually (commonly over approximately 60 minutes) and occurs in the context of recurrent late, variable, or prolonged decelerations. It is important therefore to be able to appreciate the CTG pattern evolution and recognize the gradual changes in FHR pattern tracing over time. Comparison of a trace at given point in time with another part of the trace some time back especially that during the first 20 minutes in the same fetus can give vital information, which can assist in decision making.

It must always be remembered that with any given CTG trace, the clinical actions and decisions will vary depending on the overall clinical picture. A pathological CTG showing fetal tachycardia with atypical variable or late decelerations and reduced variability at 3–4 cm cervical dilatation in a primigravida might warrant a caesarean section, whereas at 7–8 cm it might indicate the need for fetal scalp blood sampling and an instrumental delivery in the second stage, if this can be carried out safely. Other clinical risk factors, such as thick meconium with scanty fluid, intrauterine growth restriction or intrauterine infection, cause a rapid decline in pH with a pathological CTG.
and warrant early action. The clinical picture should always have a major influence on the action planned when faced with a pathological CTG.

**Diagnosis to decision to deliver interval**

This begins when FHR changes associated with presumed fetal acidaemia are first noticed on the CTG or auscultation and ends when a decision for some form of action is taken for delivery of the new born. This interval is often the subject of huge debate in court of law while incriminating liability in cases of intrapartum fetal asphyxia. It is therefore essential that the bedside provider must recognize the variant FHR patterns correctly and communicate his or her findings accurately to the decision making members of the team. Similarly the obstetrician attending the call to review a pathological CTG trace needs to act promptly based on the trace and the clinical situation and document his or her findings clearly with a plan of action to follow.

**Teamwork and communicating findings**

Effective intrapartum FHR monitoring requires good teamwork. All members of the maternity team (doctors, midwives, nurses) should be aware of how FHR traces are interpreted, which FHR patterns are associated with actual or impending fetal acidaemia and within what time frame the senior team member should be notified of pathological FHR pattern. Accurate, consistent and timely communication plays an important role in the successful implementation of hospital protocols. Communications involving descriptions of FHR tracings should include a systematic description of the trace in addition to the pattern of CTG evolution, maternal medications as well as overall clinical condition of the mother.

**Storage of CTG**

CTGs should be stored for at least 25 years and the hospital should make adequate provision for safe storage and easy retrieval. The CTGs are recorded on thermo sensitive paper and tends to fade in 3–4 years time. Where possible, traces should be stored electronically to facilitate storage of large amount of data while saving on time, space and paper.

**Training in CTG interpretation**

It is essential that all maternity units provide a regular and structured programme on interpretation of CTGs for all midwives and doctors working on the labour ward. The programme should include mandatory refresher course on 6 monthly or annual basis as well as induction training whenever an individual takes up a new post. Participation in weekly case review meetings and discussions on CTG traces is one of the best ways of reinforcing knowledge.

**Audit and risk management**

Incident reporting of adverse outcomes and audit of poor outcome (poor Apgar scores, cord arterial pH, need for assisted ventilation, admission for neonatal intensive care and HIE) are essential to find out whether there is a failure in education and training, induction of personal, supervision, inadequate staffing level or system failure. Lessons learned from analysis of adverse obstetric events should be disseminated to all staff working on the labour ward.

**Supervision and support** — ensuring that staff is aware of when supervision is required and how to access a more senior opinion are important. The availability of more experienced senior medical staff to assist in difficult decisions may help to develop a system whereby error on the part of less experienced staff dealing with difficult situations can be identified before any harm occurs to the patient. Junior doctors and midwives must be supported in the development of their decision making processes and be confident enough to ask for assistance where necessary.

**Conclusion**

The majority of CTG related medico-legal cases have similar problems which can be attributed to a few factors such as — a) inability to interpret FHR trace, b) inappropriate action, c) technical aspects and d) record keeping. Adverse maternal and perinatal outcomes and the litigations arising out of these, not only have long-term consequences for the working lives of midwives or obstetricians, but have also been influential in changing practice trends such as rising caesarean rates.

Unfortunately obstetric litigation with its huge costs is a growing problem and for the foreseeable future, the CTG is here to stay. Accurate identification of FHR patterns based on national guidelines, good communication and evidence based management are likely to be the key to successful clinical outcomes. Recommendations for improving safety in maternity care, such as compulsory attendance at regular training sessions for CTG interpretation, good communication between midwives, doctors and women, up-to-date guidelines and protocols for clinical care and strict compliance with the higher levels of the CNST maternity standards remain a priority.
Ten years of maternity claims. An analysis of NHS litigation Authority data. NHS Litigation Authority, October 2012.

Practice points
- Consider the overall clinical situation along with CTG findings before making management decisions in labour.
- Hypoxia may not be the only damaging factor in labour. Infection and possible trauma in second stage should also be considered.

- A systematic review of the CTG trace as per NICE guidelines should be undertaken every hour in labour.
- Once the plan is made to expedite delivery this should occur in a timely manner.
- If labour ward is busy, additional help should be readily sought.
- Regular mandatory CTG training is essential for all labour ward staff.
- There should be a mechanism for the rapid review of adverse obstetric events and dissemination of key learning points to all staff.
- Evidence based clinical practice (as per the national guidelines) and good team work are essential to avoid adverse maternal and perinatal outcomes.