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Continuous cardiotocography during labour: Analysis, classification and management

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Fetal Physiological Score

The use of continuous intrapartum electronic fetal heart rate monitoring (EFM) using a cardiotocograph (CTG) was developed to enable obstetricians and midwives to analyse the changes of fetal heart rate during labour so as to institute timely intervention to avoid intrapartum hypoxic–ischaemic injury. Although CTG was initially developed as a screening tool to predict fetal hypoxia, its positive predictive value for intrapartum fetal hypoxia is approximately only 30%. Even though different international classifications have been developed with the aim of defining combinations of features that help predict intrapartum fetal hypoxia, the false-positive rate of the CTG is high (60%). Moreover, there has not been a demonstrable improvement in the rate of cerebral palsy or perinatal deaths since the introduction of CTG into clinical practice approximately 45 years ago. However, there has been a significant increase in intrapartum caesarean section and operative vaginal delivery rates. Unfortunately, existing guidelines employ the visual interpretation of CTG based on 'pattern recognition', which is fraught with inter- and intra-observer variability. Therefore, clinicians need to understand the physiology behind fetal heart rate changes and to respond to them accordingly, instead of purely relying on guidelines for management. It is very likely that such a 'physiology-based' approach would reduce unnecessary operative

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interventions and improve perinatal outcomes whilst reducing the need for 'additional tests' of fetal well-being.

Introduction

Electronic fetal heart rate monitoring (EFM) involves the use of a cardiotocograph (CTG) to record the fetal heart rate (FHR) so as to determine the fetal well-being in order to detect signs of intrapartum hypoxia.

EFM was introduced in the late 1960s, and the first equipment used phonocardiography to record the FHR, and this was later substituted by Doppler signals with significant improvement on the quality of the signals [1]. It was initially introduced to prevent brain injury secondary to intrapartum fetal hypoxia. Unfortunately, the rates of cerebral palsy have remained stable over the last 50 years. Moreover, the rates of caesarean section and instrumental deliveries have been continuously increasing over the last 40 years [2].

CTG not only has a 60% false-positive rate [3], but also has a high intra-observer variability [4]. The knowledge of neonatal outcomes also influences retrospective classification of the CTG traces [5]. Therefore, a good understanding of fetal physiology is essential to adequately interpret and manage the findings on the CTG, irrespective of the availability of other 'additional tests' (fetal scalp blood sampling or FBS, fetal scalp lactate, fetal pulse oximetry and analysis of the fetal electrocardiogram (ECG) using ST-Analyser or STAN). It is paramount to analyse the CTG trace in the context of the existing and evolving clinical picture during labour and not in isolation. Risk factors such as prolonged spontaneous rupture of membranes (SROMs), prematurity, intrauterine growth restriction (IUGR), infection or the presence of meconium-stained liquor, the use of oxytocin for failure to the progress of labour and the presence of a uterine scar need to be considered whilst interpreting the CTG trace. Therefore, the overall management needs to be modified in the presence of an 'a priori' reassuring CTG. In addition, a critical analysis of the CTG trace needs to be made to differentiate a fetus that is compensating well with the ongoing hypoxic and/or mechanical stress from a fetus that is unable to compensate or has begun the process of decompensation, based on the features observed on the CTG trace. Failure to understand the fetal physiology during labour and to correlate the CTG patterns with the clinical picture may result in an increase in unnecessary operative interventions and/or an increase in the risk of intrapartum hypoxic injury leading to hypoxic–ischaemic encephalopathy (HIE), severe metabolic acidosis culminating in long-term neurological sequelae (cerebral palsy) or perinatal death.

In modern obstetric practice, in view of the high false-positive rate of CTG, several additional tests of fetal well-being have been developed in an attempt to improve the detection rate of hypoxia. FBS, analysis of the fetal ECG using STAN, fetal pulse oximetry and fetal scalp blood lactate levels have been attempted with variable success rates.

Technical aspects

The FHR is recorded using a transducer placed on the maternal abdomen (external monitoring) or using an electrode placed on the fetal scalp (internal monitoring), and it is printed on a paper in a similar way to an ECG. This is the 'cardiac' part of the CTG. The external transducer is an ultrasound device that uses the Doppler principle. There is a second transducer, the 'toco' component, which is also placed on the maternal abdomen below the uterine fundus, and it records the contractions. It is important to be aware that this transducer gives us information about the frequency and duration of the uterine contractions, but not about their strength. The amplitude or the 'height' of the recording merely reflects a change in the tension of the anterior abdominal wall. Currently, there are intrauterine pressure catheters that can be placed inside the uterus once the membranes are ruptured, and they detect the strength of the contractions as well as the frequency and duration [6].

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Before starting CTG recording, it is mandatory to check the maternal pulse to avoid erroneous recording of maternal heart rate as fetal [7]. External FHR monitoring is less reliable than internal as it is more likely to have signal loss, record maternal heart rate or produce other signal artefacts, especially during the second stage of labour. If there is a suspicion that the maternal heart rate is being monitored at any point in labour, it should be checked immediately, and internal monitoring using a fetal scalp electrode (FSE) should be used, if appropriate. There are contraindications for internal monitoring in the presence of infections such as human immunodeficiency virus (HIV) and hepatitis B due to the risk of vertical transmission or in fetuses with suspected or confirmed bleeding disorders [6].

When starting the CTG monitoring, it is important to ensure that 'paper speed' is set correctly. In most countries, it is set at 1 cm/min; however, in the USA, it is set at 3 cm/min, and some European centres use 2 cm/min. Failure to set the speed correctly will result in errors on the correct interpretation of the FHR variability as well as in identifying the depth and duration of decelerations.

Analysis

There are four features that need to be analysed on a CTG: baseline rate, accelerations, variability and decelerations.

a) Baseline heart rate

It is defined as the mean FHR after excluding accelerations and decelerations. It is analysed over 5–10 min and expressed in beats per minute (bpm). The normal range is considered to be 110–160 bpm. It is regulated by the combined action of the sympathetic and parasympathetic nervous systems. Consequently, preterm fetuses with a less developed parasympathetic system will commonly show a higher baseline rate compared with term or post-term fetuses when the parasympathetic system is well developed, and they will usually have baseline rates between 110 and 130 bpm. It is not uncommon for a post-term fetus to have a baseline heart rate between 90 and 110 bpm as a consequence of maturity of the parasympathetic nervous system. This should be considered as normal if other parameters on the CTG trace are reassuring.

An increase in the baseline rate of >160 bpm persisting for >10 min is called baseline tachycardia. It can be a reflection of maternal tachycardia due to dehydration or maternal pyrexia (infection) or more rarely secondary to a fetal arrhythmia. It can also be related to chronic hypoxia when it occurs in combination with reduced baseline FHR variability and shallow decelerations.

A baseline FHR of <100 bpm, which persists for >10 min, is called baseline bradycardia, and in the presence of accelerations, good variability and absence of decelerations, it may reflect postmaturity. However, conduction defects of the heart (heart blocks), sympatholytic drugs and acute hypoxia to the myocardium may also present with a baseline bradycardia.

b) Accelerations

It is a transient increase in the baseline of >15 bpm, lasting for 15 s or more and returning to the normal baseline. The presence of two or more accelerations over a 20-min period is a reassuring feature suggestive of fetal well-being as they are caused by the somatic nervous system activity usually associated with fetal movements. They are absent in the presence of fetal sleep, chronic hypoxia, drugs (e.g., pethidine), infection and brain haemorrhage (intrauterine fetal stroke). The erroneous monitoring of maternal heart rate may result in accelerations of greater magnitude and/or duration coinciding with uterine contractions.

c) Variability

It is the bandwidth variation of the baseline, which is determined after excluding accelerations and decelerations. It is maintained by the interaction between the sympathetic and the parasympathetic

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systems. Hence, the presence of good baseline variability gives information about the integrity of the autonomic nervous system. It is classified as reduced (<5 bpm), normal (5–25 bpm) and saltatory (>25 bpm).

A normal variability is unlikely to be associated with cerebral hypoxia. During periods of fetal sleep, it can be reduced, but it will be associated with periods of normal variability. The combination of these two patterns is called 'cycling', and it is a reassuring feature of fetal well-being (Fig. 1). Periods of reduced variability can also be associated with drugs (central nervous system (CNS) depressants), or it may be a sign of hypoxia in the CNS. Correlation with the clinical picture is essential to perform a differential diagnosis.

The saltatory pattern (baseline variability of >25 bpm) may be associated with a rapidly evolving hypoxia usually with active maternal pushing or with the use of oxytocin infusion to augment labor [8]. Although the exact mechanism is still debated, it is proposed that it reflects attempts by the autonomic nervous system to maintain the stability of the baseline heart rate when there is a rapidly evolving hypoxic stress. It should be considered an abnormal feature in the presence of late or variable decelerations, especially with a 'subacute pattern' on the CTG trace (Fig. 2). In this case, immediate action should be taken to relieve hypoxic stress by recommending the cessation of active pushing or by stopping oxytocin as appropriate to improve fetal oxygenation.

d) **Decelerations**

They are defined as a transient decrease of the FHR of >15 bpm lasting for >15 s. Traditionally, they have been classified in relation to the uterine contractions as early, late and variable. However, clinicians need to understand that decelerations are a reflex response of a fetus to the ongoing hypoxic or mechanical stresses in labour to protect against fetal stroke (variable decelerations) or to hypoxic injury to fetal myocardium (late decelerations). This is because, unlike an adult, a fetus is not exposed to atmospheric oxygen, and, therefore, it is unable to increase the rate and depth of respiration to oxygenate the myocardium to maintain a positive energy balance when exposed to hypoxic stress. Therefore, the only way a fetus could immediately protect the energy balance within the myocardium is by rapidly slowing its own heart rate to reduce oxygen consumption and to improve its coronary blood flow during intrapartum hypoxic stress.

Clinicians also need to understand that, although guidelines classify decelerations as 'early', 'variable' and 'late', in reality, a combination of decelerations may occur as uterine contractions may compress the fetal head and the umbilical cord at the same time [9].

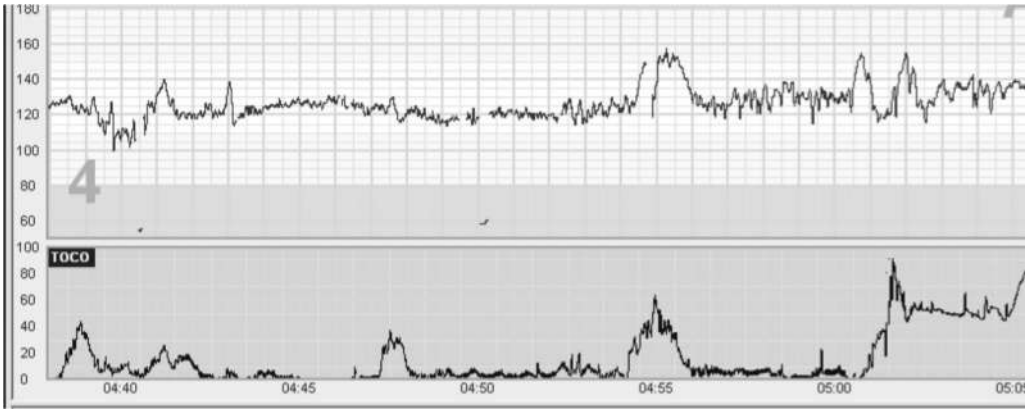

Fig. 1. CTG showing 'cycling': alternating periods of quiescence (reduced variability) and periods of activity (accelerations, variability of 5–25 bpm).

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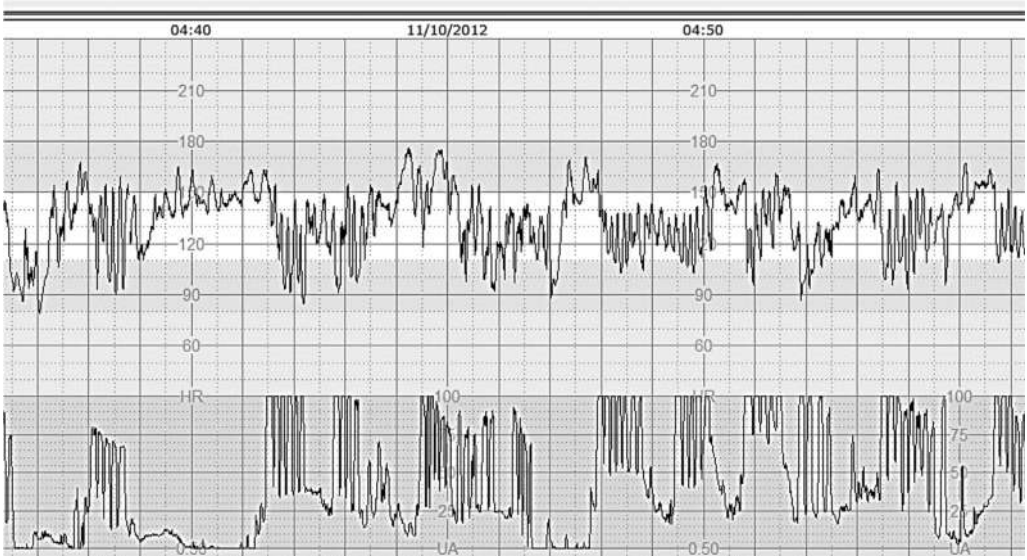

Fig. 2. Saltatory pattern suggestive of rapidly evolving hypoxia to the central nervous system, and this is usually seen with injudicious use of oxytocin or during active maternal pushing as was in this case.

1. *Early decelerations*

They start with the onset of uterine contraction, reach their nadir at the peak of the contraction and return to the normal baseline when the contraction ceases. They are associated with head compression, and they are generally benign. They are present on the late first stage or second stage of labour, but they only represent <2% of the decelerations.

2. *Late decelerations*

The deceleration occurs 'late' in relation to the contraction. The nadir of the deceleration occurs after the peak of the contraction, and the heart rate returns to the baseline after the contraction has finished. Usually, there is a 10–20-s 'lag time' for the deceleration to return to the normal baseline, and, therefore, they are termed 'late decelerations'. They are related to fetal hypoxemia, hypercarbia and acidosis, which stimulate the central and peripheral chemoreceptors. Fetal well-being should be assessed by determining the baseline FHR and variability, which denote the oxygenation of central organs (brain and the heart). Additional tests of fetal well-being may need to be considered if there is a progressive rise in the baseline FHR with reassuring variability, if a decision is made to continue labour. Any change in the baseline variability associated with preceding late decelerations and/or a rise in baseline FHR requires an immediate intervention such as intrauterine resuscitation if appropriate or immediate delivery.

3. *Variable decelerations*

They are the most common type of deceleration during labour. They vary in shape, length, size and timing in relation to the ongoing uterine contractions. They are related to umbilical cord compression, and they are mediated by a 'baroreceptor' mechanism. There are different nomenclatures depending on the classification used.

A 'typical' or an 'uncomplicated' variable deceleration has four components; initially, there is a slight rise in the FHR ('shouldering') followed by a sharp fall from the baseline (<60 bpm) with a quick

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rise that gives a second 'shouldering' and a final recovery to the baseline. They last for <60 s. The pathophysiology behind these decelerations is a compression of the umbilical cord in the absence of acidosis; the first shouldering is caused by the selective compression of the thin-walled umbilical vein (the fetus receives less blood from the placenta, whilst it continues to pump its blood through umbilical arteries and increases the heart rate to compensate for ongoing fetal hypovolaemia and hypotension); the sharp fall is secondary to the compression of the umbilical arteries (the fetus reduces the heart rate as a protective mechanism when systemic hypertension occurs to avoid stroke), and the recovery to the baseline occurs as soon as the compression ends. It is a baroreceptor-mediated response.

An 'atypical' variable deceleration does not have the features of the 'typical' deceleration described earlier. The initial and final shouldering disappear; drop in the FHR of >60 bpm may denote the complete occlusion of the umbilical cord, and the recovery to the original baseline may be delayed. They may have an 'overshoot', which may indicate ongoing fetal hypotension secondary to the intense and prolonged compression of the umbilical cord. The pathophysiology of these decelerations is a combination of baroreceptor- and chemoreceptor-mediated response. If these decelerations are recurrent, there may be a component of acidosis with time.

Classification

There are several classifications described by different societies, and these include the following: the FIGO (International Federation of Gynecology and Obstetrics) [10], NICE (National Institute of Health and Care Excellence) [11], from the UK, and ACOG (American Congress of Obstetricians and Gynecologist) [12] from the USA.

Although the features and terminologies used in these classification systems show a significant variation, there are some basic general principles on CTG interpretation. Regardless of the type of decelerations and their duration, in general, if the fetus shows stable baseline and reassuring variability in between the decelerations (i.e., fetal response to stress), then the risk of acidosis is low [9,13]. This is because the baseline FHR is maintained by the fetal myocardium, modified by the brain, and the baseline variability reflects the integrity of the autonomic nervous system (sympathetic and parasympathetic). Therefore, if the baseline FHR and variability are both reassuring, then, the central organs (the myocardium and the brain) are well oxygenated.

Decelerations lasting for >60 s and showing gradual recovery to baseline are more likely to be associated with fetal acidosis if they are persistent and recurrent, and, especially, if they are associated with a change in baseline FHR.

Clinicians should appreciate that intrapartum hypoxia is an evolving dynamic process. Therefore, whenever possible, intrauterine environment should be improved in the first instance irrespective of the classification of CTG trace. Understanding the types of intrapartum fetal hypoxia and fetal reserve is also vital to optimize outcomes.

We have used the FIGO classification as this was developed by representatives from ACOG, Royal College of Obstetricians and Gynaecologists (RCOG) and the FIGO with the input from over 40 national societies in obstetrics and gynaecology around the World (Table 1).

Management

Labour is a very stressful process for the fetus as uterine contractions compress the fetal head, different body parts and the umbilical cord, as well as it may cause a reduction in the utero-placental circulation. Therefore, a fetus will mount a stress response to compensate for the ongoing mechanical and hypoxic stresses.

Obstetricians and midwives have to understand the fetal response to stress based on the features observed on the CTG trace before instituting any intervention. The aim of management is to identify a fetus that is unable to maintain a successful compensatory response to the ongoing hypoxic stress, or that has exhausted all its resources.

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Table 1
FIGO CTG Classification 2015 [7] with Permission from Prof Diogo Ayres-de-Campos.

	Normal	Suspicious	Pathological
Baseline	110–160 bpm	Lacking at least one characteristic of normality, but with no pathological features	<100 bpm
Variability	5–25 bpm		Reduced variability for >50 min, increased variability for >30 min, or sinusoidal pattern for >30 min
Decelerations	No repetitive[a] decelerations		Repetitive late or prolonged decelerations during >30 min or 20 min if reduced variability, or one prolonged deceleration with >5 min
Interpretation	Fetus with no hypoxia/acidosis	Fetus with a low probability of having hypoxia/acidosis	Fetus with a high probability of having hypoxia/acidosis
Clinical Management	No intervention necessary to improve fetal oxygenation state	Action to correct reversible causes if identified, close monitoring or additional methods to evaluate fetal oxygenation (Chapter 4)	Immediate action to correct reversible causes, additional methods to evaluate fetal oxygenation (Chapter 4), or if this is not possible expedite delivery. In acute situations (cord prolapse, uterine rupture or placental abruption) immediate delivery should be accomplished.

[a] Repetitive decelerations refers to occurrence of decelerations for at least 50% of uterine contractions.

1. Understanding fetal physiopathology in labour

The process of labour generates changes in the blood supply to the fetus, and, therefore, the fetus reacts by activating its compensatory mechanisms. It is important to understand the normal physiology during labour to correctly interpret the features observed on the CTG trace. At the same time, it is essential to bear in mind that every fetus is different with an individual fetal reserve, and, therefore, it may react differently to the same hypoxic or mechanical stress. In addition, the rapidity at which hypoxia evolves also determines the ability of the fetus to mount a successful compensatory response to avoid hypoxic–ischaemic injury during labour. A well-grown, term fetus usually has good reserves, and its compensatory mechanisms are more effective than those of a growth-restricted or a preterm fetus [14]. In addition, other factors that are present during labour such as prolonged rupture of membranes with chorioamnionitis, anhydramnios, meconium-stained liquor, maternal infection or pyrexia and the speed of evolution of hypoxia are likely to modify the responses of the fetuses as well as to determine the perinatal outcome.

FHR is regulated by the somatic and the autonomic nervous systems. Voluntary fetal movements reflect the integrity of the somatic nervous system, and it is for this reason that the presence of accelerations is considered to reflect fetal well-being. This is because if a fetus is exposed to hypoxia, it would reduce non-essential movements of muscles to conserve energy leading to the disappearance of accelerations on the CTG trace.

The balance between the sympathetic and parasympathetic system maintains the variability of the FHR. The presence of good variability reflects the oxygenation and integrity of the fetal brain centres.

When head compression occurs during uterine contractions, the parasympathetic system activated as the dura mater, which is richly supplied by the vagus nerve, is stimulated. This leads to an immediate decrease in the heart rate that returns to the normal baseline when the head compression finishes. This results in early decelerations, and no component of hypoxia is present in this situation.

In the presence of utero-placental insufficiency, the fetus switches its metabolism from aerobic to anaerobic due to the lack of oxygen. This leads to metabolic acidosis with the accumulation of carbon dioxide and hydrogen ions (lactic acid). Under this scenario, the changes in the chemical composition of the blood (increased carbon dioxide and acid) activate the chemoreceptors present on the aortic and carotid bodies and in the brain. The FHR decreases as a compensatory mechanism, but the return of the well-oxygenated blood from the mother is necessary to 'wash out' all the accumulated acid and carbon dioxide, thereby gradually relieving the stimulus of the

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chemoreceptors. This results in a slow recovery of the FHR to its normal baseline that is characteristic of the late decelerations [9,13].

When the fetus is exposed to an evolving hypoxia, the next defense mechanism is to release catecholamines (adrenaline and noradrenaline) produced by the adrenal glands. This results in an increase in the baseline FHR.

Other factors that modify the FHR are maternal (pyrexia, hypovolaemia, dehydration and tachycardia), mechanical (vaginal examinations), medications (beta-sympathomimetics and opioids) and fetal (infection, fetal arrhythmias or cardiac malformations).

2. Understanding types of hypoxia

Based on the intensity and duration of hypoxic stress during labour, three types of intrapartum hypoxia had been described, and the management should be tailed according to the type of hypoxia to optimize fetal outcome (Fig. 3):

- *Gradually evolving hypoxia*

The hypoxic stress evolves over time (hours) giving the fetus the time to use its compensatory mechanisms effectively so as to avoid hypoxic damage. The first feature that appears on the CTG is the presence of decelerations. If the hypoxic stress continues (spontaneous contractions or oxytocin induced), the accelerations disappear in an attempt of the fetus to conserve its oxygen and energy substrates. The next compensatory mechanism is the release of catecholamines to increase the heart rate and its cardiac output to ensure good perfusion of the vital organs (heart, brain and adrenal glands). This results in an increase in the baseline heart rate on the CTG trace. If the hypoxic insult continues, depending on the fetal reserve and the intensity and duration of hypoxia, fetal decompensation may ensue. Lack of oxygenation via the carotid arteries would lead to hypoxia to the brain. As the perfusion to the brain is compromised, loss on the baseline variability would be observed on the CTG trace. Finally, lack of oxygenation of the coronary arteries that arise at the root of the ascending aorta would lead to myocardial hypoxia and acidosis leading to a 'step-ladder pattern to death'. After such repeated attempts to return to the baseline, a terminal bradycardia will ensue leading to fetal death (Fig. 4).

- *Acute hypoxia*

It is characterized by a sudden drop in the baseline heart rate, and it is called 'single prolonged deceleration'. If it lasts for <3 min and returns to the normal baseline with good variability, it is considered a 'suspicious' feature. If it lasts for >3 min, it is considered an abnormal feature and requires an urgent intervention. In the first instance, it is essential to exclude three major intrapartum accidents (placental abruption, umbilical cord prolapse and uterine rupture) and two iatrogenic causes (hyperstimulation due to oxytocin or prostaglandins and maternal hypotension usually secondary supine hypotension or epidural analgesia). If there is any evidence of one of the three major accidents, delivery should be expedited via the safest and quickest way to save the fetus. It is important to remember that fetal pH drops at the rate of 0.01/min in the presence of acute hypoxia. If the cause of the acute hypoxia is iatrogenic, oxytocin infusion needs to be stopped immediately and 'intrauterine resuscitation' commenced. This includes the administration of intravenous fluids, postural changes (turning a woman to a left or right lateral position) and considering tocolytics (terbutaline, 250 μg subcutaneously) [15].

In the event of an acute hypoxia, the '3-, 6-, 9-, 12- and 15-min' rule should be applied: it involves the appropriate institution of intrauterine resuscitation by 6 min, move the patient to a theatre by 9 min, and if the CTG shows no signs of recovery, commence delivery by 12 min with the aim of delivering the baby by 15 min. This rule can only be applied if the acute intrapartum accidents and iatrogenic causes have been excluded and normal baseline variability was noted before the onset of deceleration and within the first 3 min of the deceleration [9].

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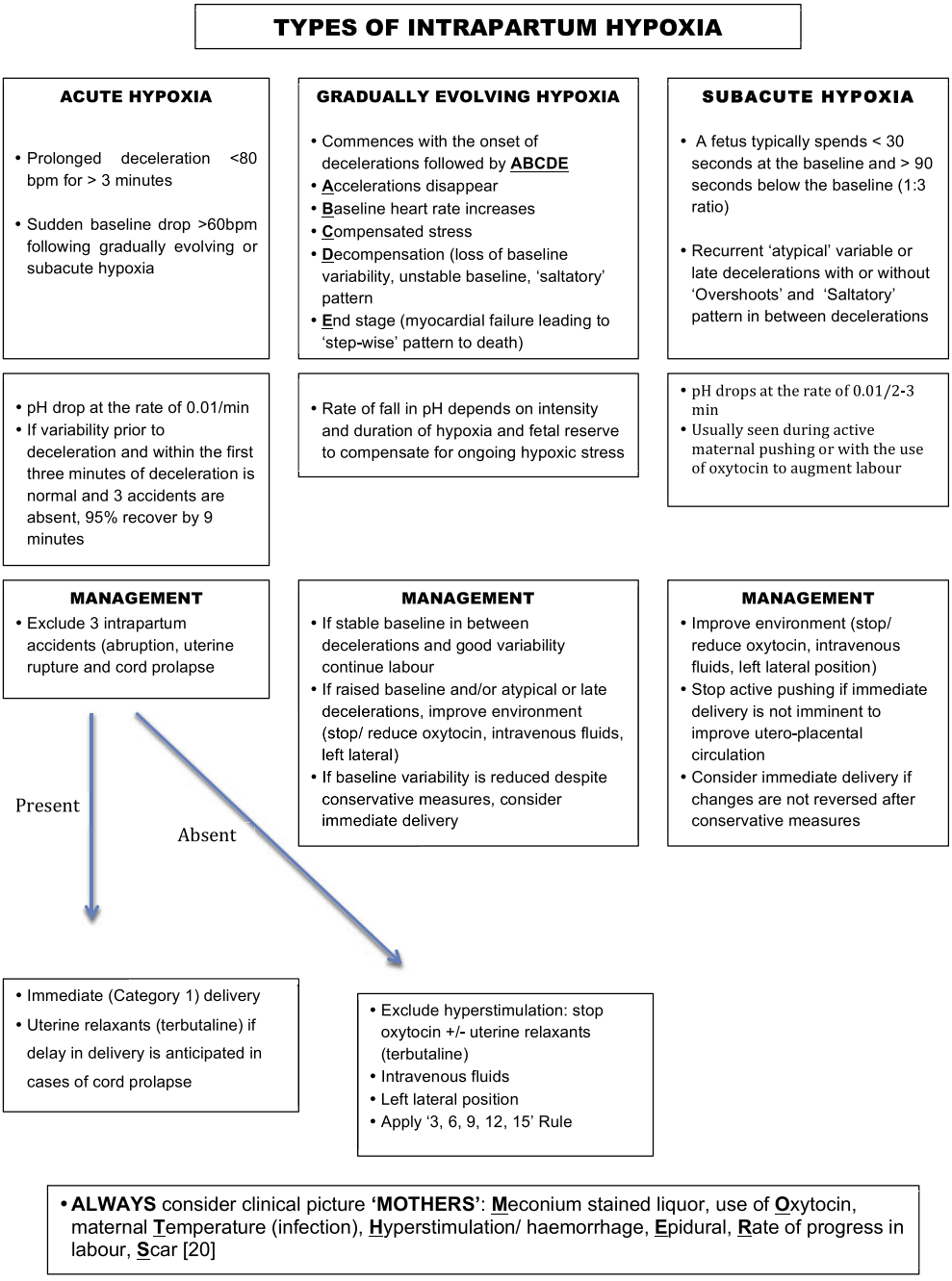

Fig. 3. Types of intrapartum hypoxia.

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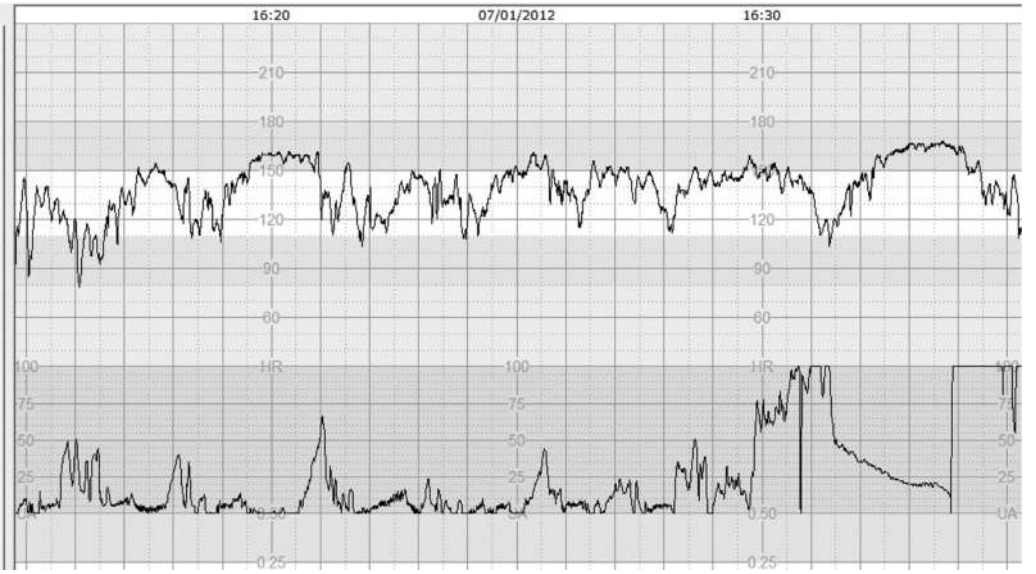

Fig. 4. Gradually evolving hypoxia. Note repetitive deceleration (hypoxic stress), absence of accelerations and a rise in the baseline fetal heart rate (catecholamine surge) to compensate for ongoing hypoxic stress. Note the presence of 'atypical' variable and late decelerations.

In the absence of the three major accidents, 90% of the prolonged decelerations will recover by 6 min and 95% by 9 min [16]. Features that indicate a high likelihood of recovery are the presence of good variability within the deceleration, a normal CTG before the deceleration and a heart rate of >60 bpm [17]. However, the presence of reduced variability before or within the first 3 min of the

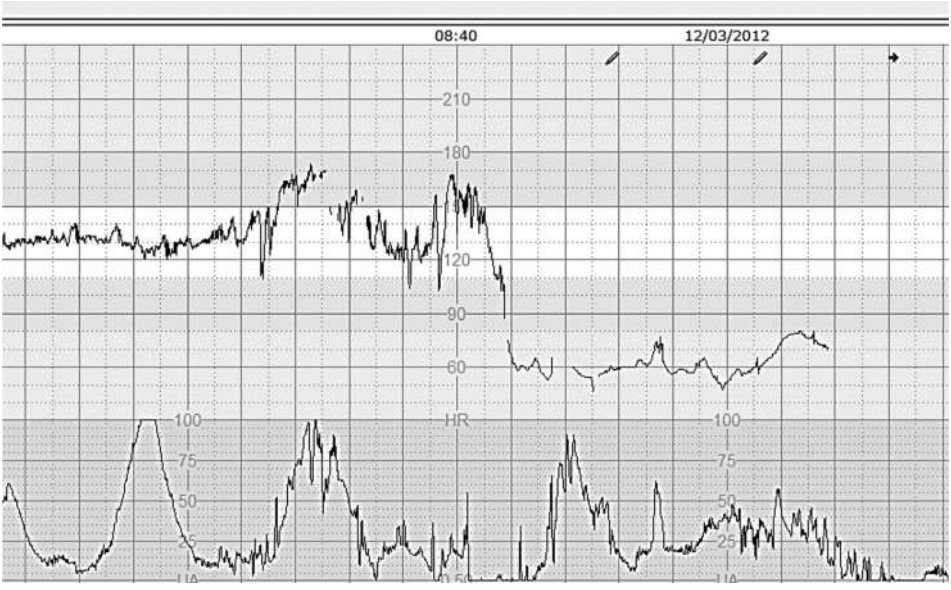

Fig. 5. Acute hypoxia due to uterine rupture. Note the sudden drop in the fetal heart rate below 80 bpm with total loss of baseline variability within 3 min of the deceleration.

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deceleration, repetitive late decelerations before the onset of the prolonged deceleration or a drop in the heart rate to >60 bpm are associated with a poor outcome [18], and the '3-, 6-, 9-, 12- and 15-minute' rule should not be applied. Measures should be undertaken to accomplish immediate delivery if this is the case (Fig. 5).

- *Subacute hypoxia*

It develops over 30–60 min, and it is characterized by the deepening and widening of ongoing decelerations, where the fetus spends more time within the deceleration (>90 s) than on the baseline (<30 s). Therefore, the time available on the baseline to wash off the acid and carbon dioxide and to obtain fresh oxygenated blood from the placenta becomes progressively shorter (Fig. 6). Fetal pH drops at the rate of 0.01/2–3 min. Management involves immediate intervention to improve the intrauterine environment (stopping or reducing oxytocin, and discouraging active maternal pushing for the next few contractions to ensure oxygenation of placental venous sinuses), because the failure to do so may result in an eventual acute hypoxia secondary to progressive myocardial hypoxia and acidosis.

- *Chronic hypoxia*

In this situation, a fetus has been exposed to a prolonged period of hypoxia during the antenatal period usually secondary to a chronic utero-placental insufficiency. Usual intrauterine adaptations include reduction in growth, movements and diversion of oxygenated blood and nutrients from non-vital organs to supply the vital organs. Failure of these compensatory

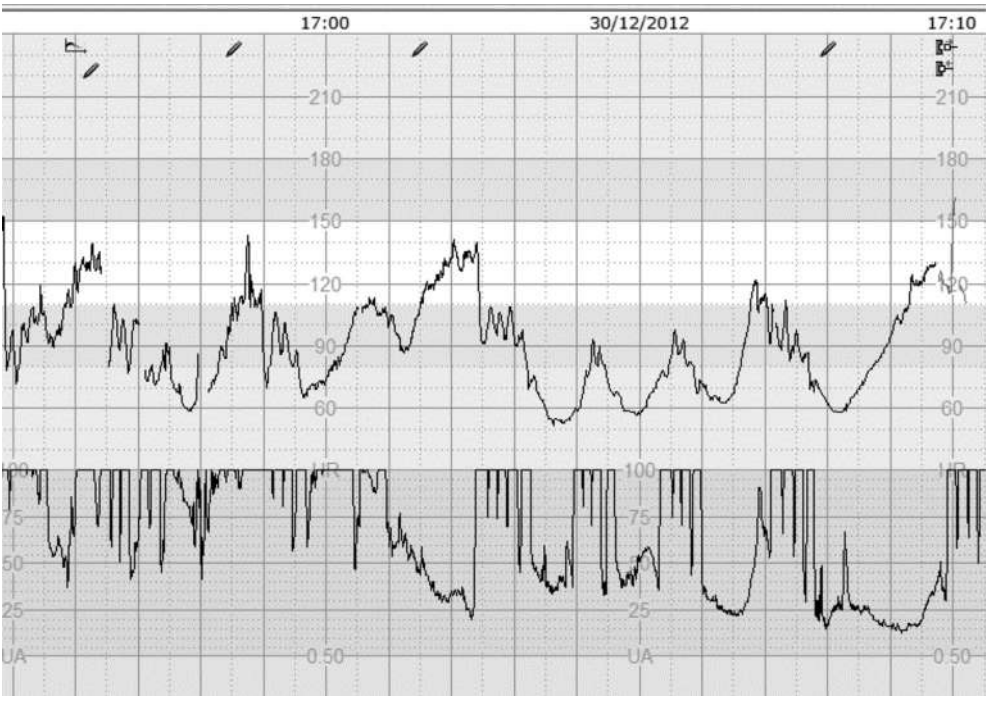

Fig. 6. Subacute hypoxia. The fetus spends progressively less time at the normal baseline (<30 s) as compared with the time spent during decelerations leading to a rapid fall in the pH. If this pattern continues, a terminal bradycardia may ensue.

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mechanisms will result in progressive hypoxia and acidosis of the fetal brain. Animal studies have shown that pre-existing chronic hypoxia adversely affects fetal compensatory response during labour [19]. The features observed on the CTG trace in chronic hypoxia include an increase in the baseline rate with reduced variability and the presence of shallow decelerations. Even though some brain damage may have already occurred, the presence of this CTG pattern requires immediate delivery. This is because with the onset of uterine contractions, and resultant intermittent umbilical cord compression and reduction in utero-placental circulation, there will be a further reduction in oxygenation leading to HIE as well as myocardial failure leading to a terminal bradycardia.

- *Other CTG patterns*

There are CTG patterns that correlate with specific clinical situations: sinusoidal typical and atypical, saltatory pattern and overshoots.

The sinusoidal pattern is defined by the NICE guidelines as a '*regular oscillation of the baseline long-term variability resembling a sine wave, lasting at least 10 minutes and has a relatively fixed period of 3-5 cycles per minute and an amplitude of 5-15 bpm above and below the baseline. Baseline variability is absent*' [11].

There are two different sinusoidal types:

- 'Typical' or smooth, the sine wave is rounded and symmetrical in shape. It is seen in fetal anaemia, and hence it can be present in the antenatal period. Depending on the gestational age and cause of the anaemia, intrauterine transfusion or delivery should be considered.
- 'Atypical', jagged, saw-tooth or the 'Poole Shark Teeth' Pattern. It is secondary to sudden feto-maternal haemorrhage, and, almost invariably, it is seen during labour. Immediate delivery is required, and in the majority of cases, the newborn will require a blood transfusion [8].

The saltatory pattern is defined as an FHR variability of >25 bpm lasting at least 1 min. It is a reflection of the 'fight' between the two components of the autonomic nervous system to maintain a stable baseline. It can be related to uterine hyperstimulation, cervical examination, ephedrine administration or repeated hypoxic insult especially during the active second stage of labour. If rapidly evolving hypoxia is the cause, then immediate action must be taken.

Lastly, an overshoot is described as an acceleration arising from a variable deceleration. They are caused by cord compression with adrenergic stimulation, but lack of vagal activation due to the ongoing fetal hypoxia and hypotension. They require action to improve the fetal environment [8].

3. Additional tests of fetal well-being

- *Fetal scalp blood sampling*

It involves taking a sample from the fetal scalp to determine if there is fetal acidosis by analysing the pH. It is a relatively simple test, but it requires a machine to determine the blood gases. A recent Cochrane review has concluded that FBS does not reduce the rate of caesarean section or instrumental delivery, and it does not influence the neonatal outcomes [20].

- *Fetal pulse oximetry*

The aim is to measure the content of oxygen in the haemoglobin in tissues. A value of >30% is related to a good fetal outcome. It improves the specificity of the CTG alone. However, it can be technically difficult to place the sensor and get a continuous reading due to loss of contact [21]. Recent Cochrane review has concluded that its use is safe, but it does not show a reduction in the overall caesarean section rate [22].

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- *Blood lactate*

It measures the lactic acid on an FBS. The advantage of this test compared with an FBS is that the amount of blood required is less (5 μL instead of 35–50 μL needed for pH) [24]. However, there is insufficient evidence of correlation between the lactate levels and the neonatal outcome, and hence the NICE guidelines do not recommend it in routine practice [11].

- *ST-Analyser*

It analyses changes in the fetal ECG complex secondary to hypoxia occurring during labour. It analyses the 'ST segment', 'T waves' and the T/QRS ratio with the aim of determining if there is myocardial hypoxia causing changes in these parameters. The physiology behind these changes is the glycogenolysis present in the myocardial cells in the presence of hypoxia. The influx of glucose and potassium into the cells causes changes on the T wave and therefore on the T/QRS ratio. The use of STAN needs to be in combination with the CTG. The advantage is that it provides information on the oxygenation of a central organ, the heart, rather than peripheral hypoxia as shown by the FBS. A recent meta-analysis has concluded that ST-Analyser reduces not only FBS rate but also the total operative delivery rate and neonatal metabolic acidosis rate [23].

- *Fetal Physiological Score (FPS)*

This scoring system is based on the fetal physiological response to hypoxic stress in the presence of 'pathological' (recurrent atypical variable or late) decelerations over a 30-min period [25]. It analyses the following features: the percentage increase of the baseline FHR (due to the release of adrenaline) compared with the original baseline, changes in variability (reduced variability or 'saltatory' pattern), inter-contraction interval (time available for the re-oxygenation of placental venous sinuses) and inter-deceleration interval (time available to perfuse vital organs as well as to obtain fresh oxygenated blood from the placenta). An FPS of >7 has been reported to be associated with normal APGAR scores and normal cord gases in 98% and 100% of the cases, respectively [26,27].

- *Role of computerized CTG*

The use of a computer to analyse CTG traces so as to alert clinicians has been shown to be beneficial in predicting the pH of umbilical artery at birth [28]. Currently, randomized controlled trials are underway to assess the usefulness of such computerized decision-support tools in improving perinatal outcomes. Recently, an algorithm has been developed for the computerized recognition of maternal heart rate so as to help avoid erroneous monitoring of maternal heart rate as fetal during labour [29].

Summary

Labour is a stressful process, and changes observed on the CTG trace may reflect fetal response to the ongoing hypoxic or mechanical stresses during labour such as compression of the umbilical cord or reduction in the placental blood flow. Continuous fetal monitoring is mandatory in any fetus considered to be at a 'high risk' of sustaining intrapartum hypoxic injury. It is essential to promptly diagnose 'accidents' related to labour (placental abruption, cord prolapse and uterine rupture) so as to institute timely and appropriate management to improve outcomes. Clinicians need to have appropriate knowledge of fetal physiology during labour to be able to correctly interpret CTG traces and to act on them. It is important to remember that, if appropriately interpreted, the CTG trace will provide information regarding the nature of the ongoing hypoxic and mechanical stress and fetal compensatory mechanisms. However, clinicians need to

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incorporate the wider clinical picture (meconium, intrapartum bleeding, maternal pyrexia or clinical chorioamnionitis), regardless of the classification of the CTG at a given time. Although clinical guidelines produced by various national and international bodies are useful in having a systematic approach to CTG interpretation and standardizing the terminology, understanding fetal physiology, incorporation of antepartum and intrapartum risk factors, use of additional tests of fetal well-being, if appropriate, are essential to improve perinatal outcomes and to reduce unnecessary operative interventions.

Practice points

- Intrapartum continuous fetal monitoring is mandatory in any labour considered as high risk. These include maternal problems (prolonged rupture of membranes, antepartum haemorrhage, previous caesarean section, any maternal disease that may adversely affect fetal oxygenation, post-term pregnancy, diabetes, obstetric cholestasis and pre-eclampsia), fetal problems (prematurity, oligohydramnios, IUGR/redistribution and multiple pregnancy) or intrapartum risk factors (meconium-stained liquor, significant vaginal bleeding, infection, use of oxytocin, epidural and any abnormal feature noted on intermittent auscultation).
- It is useful in clinical practice to have a systematic approach to analyse the CTG trace such as DR C BRAVADO', where DR = define risk (any risk factors present during labor for that particular patient), C = contractions (defines the number of contractions per 10 min), BR = baseline heart rate, A = accelerations, VA = variability, D = decelerations and O = overall (the final classification of the CTG trace according to the guidelines used).
- It is vital to consider four types of hypoxia whilst interpreting CTG traces: acute, subacute, gradually evolving and chronic hypoxia. Each of these has characteristic features, and it requires different management. It is important to understand that in the absence of intrapartum accidents (abruption, umbilical cord prolapse and uterine rupture), hypoxia during labour is an evolving process, and understanding the type of hypoxia will help in predicting the next feature that will appear on the CTG trace.
- When interpreting CTG traces, it is essential to individualize each fetus and to analyse the CTG patterns together with the clinical picture [30]. The presence of other factors such as prematurity, prolonged rupture of membranes, vaginal bleeding, maternal pyrexia and previous caesarean section amongst others needs specific management, and the isolated use of CTG classification can lead to fetal and maternal damage.

Research agenda

- Does computerized CTG improve decision-making in the labour ward?
- A randomized controlled trial to determine the effectiveness of physiology-based CTG interpretation versus the use of FBS and/or STAN in determining perinatal outcomes
- A prospective study to determine the usefulness and applicability of Fetal Physiological Score (FPS) in determining perinatal outcomes

Conflict of interest

None.

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