INTRODUCTION

Diogo Ayres-de-Campos, Sabaratnam Arulkumaran, for the FIGO intrapartum fetal monitoring expert consensus panel

Auscultation of the fetal heart rate (FHR) became part of routine intrapartum care in many countries during the 19th century, and remains an important form of fetal surveillance, particularly in low-risk pregnancies and in low-resource countries. Several technical breakthroughs that occurred in the 20th century led to the development of different forms of continuous electronic monitoring of the FHR and uterine contractions in the 1950s and early 1960s, and to the commercialisation of the technology known as cardiotocography (CTG) in the late 1960s. Cardiotocography (kardia=heart, tokos=labour, childbirth) is the term that best describes the continuous monitoring of the FHR and uterine contractions, but other designations such as electronic fetal monitoring are used in some countries. Fetal scalp blood sampling (FSBS) was introduced into clinical practice at around the same time as CTG, and other methods for intrapartum fetal surveillance were subsequently developed, including continuous fetal pH monitoring, fetal lactate measurement, fetal pulse oximetry, and ST waveform analysis, and some of these were successfully established. This guideline will focus on the clinical application of currently available methods for intrapartum fetal monitoring.

In 1985, the FIGO Subcommittee on Standards in Perinatal Medicine convened an expert consensus meeting in Switzerland to produce the “Guidelines for the use of Fetal Monitoring”, approved by FIGO’s Executive Board in 1986, and published in 1987. These
guidelines were an important landmark in the history of FHR monitoring, because they constituted the first wide-scale agreement on essential aspects of CTG monitoring, such as terminology, indications, acquisition techniques, and interpretation. Notwithstanding their decisive contribution to the field of fetal monitoring, with the passage of time some shortcomings have become evident, and the document has naturally become outdated.

The present guidelines were developed under FIGO’s Safe Motherhood and Newborn Health committee. In February 2013, all national member societies of FIGO were contacted by email and asked to appoint one subject matter expert with a wide knowledge of the fetal monitoring scientific literature, good written and spoken English, and available to provide written feedback by email in less than 15 days. By May 2013, 33 experts had been nominated by national scientific societies. A literature search was then conducted to identify a further list of experts who had published major clinical research in the field. Thirteen additional experts were invited according to this criterion. A geographical representation of the members of the consensus panel is presented in Figure 1.

**Figure 1.** Geographical representation of the members of the consensus panel.

The American College of Obstetricians and Gynecologists and the Royal College of Obstetricians and Gynaecologists were contacted in December 2012 for each to appoint one member of the writing committee for the “Cardiotocography” chapter, and the International Confederation of Midwives was contacted in July 2013 to nominate the authors of the “Intermittent auscultation” chapter.

The consensus process started in October 2013, and included three rounds for each chapter. Each round started with a draft version being sent by email to the panel members,
followed by written feedback from the panel within a time frame of three weeks. The received comments were considered by the authors and a revised manuscript was produced for the next round. After the three-round process was complete, the members of the panel were asked to read the final version and to give written consent for their name to be included in the panel list for that chapter. The consensus process for the four chapters was concluded in March 2015.

The purpose of these revised consensus guidelines is to update the existing ones, expanding their scope in order to include all currently available methods of intrapartum fetal monitoring, and using a language that is accessible to all healthcare professionals, independently of their previous expertise in the subject. The ultimate goal is to contribute to the improvement of intrapartum fetal monitoring throughout the world, thus reducing the burden of perinatal mortality and long-term sequelae, while at the same time avoiding unnecessary obstetrical intervention.

References

FIGO CONSENSUS GUIDELINES ON INTRAPARTUM FETAL MONITORING

Safe Motherhood and Newborn Health Committee
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PHYSIOLOGY OF FETAL OXYGENATION AND THE MAIN GOALS OF INTRAPARTUM FETAL MONITORING

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Introduction

This chapter focuses on the major aspects of the physiology of oxygen supply to the fetus and the main goals of intrapartum fetal monitoring: (1) timely identification of fetuses that are being inadequately oxygenated, to enable appropriate action before the occurrence of injury; (2) reassurance on adequate fetal oxygenation to avoid unnecessary obstetric interventions. It should be emphasized that in order to avoid adverse outcome, fetal surveillance requires a timely clinical response, and the ready availability of both adequate equipment and trained staff in intrapartum care.

The importance of oxygen supply to the fetus

All human cells require oxygen and glucose to maintain aerobic metabolism, their main source of energy production. Glucose can usually be stored and mobilised when needed, but total lack of oxygen supply for just a few minutes is enough to place the cells at risk. During fetal life, oxygen supply is entirely dependent on maternal respiration and circulation, placental perfusion, gas exchange across the placenta, umbilical and fetal circulations. Complications occurring at any of these levels may result in decreased oxygen concentration in
fetal arterial blood (hypoxemia) and ultimately in the tissues (hypoxia). Some degree of hypoxemia occurs in almost all fetuses during labour, but it is the intensity, duration and repetitive nature of the event, together with the individual variation in the capacity of each fetus to cope with the situation, that will determine the severity of the resulting hypoxia.

Difficulties in carbon dioxide (CO₂) elimination across the placenta will result in elevated CO₂ concentrations, and this gas will combine with water to increase carbonic acid (H₂CO₃) concentration, a phenomenon called respiratory acidemia. The process is quickly reversible with re-establishment of placental gas exchange, as CO₂ diffuses rapidly across the placenta. There is no evidence of injury from isolated respiratory acidemia.

When hypoxia occurs, cellular energy production can still be maintained for a limited time by anaerobic metabolism, but this process produces 19 times less energy and results in the accumulation of lactic acid inside the cell, and its dispersion to the extracellular fluid and fetal circulation. The increased concentration of hydrogen ions of intracellular origin in the fetal circulation is called metabolic acidemia, but it closely parallels hydrogen ion concentration in the tissues, so the term metabolic acidosis is frequently used as a synonym. The hydrogen ions of lactic acid are transferred very slowly across the placenta, but they are buffered by circulating bases, comprised mainly of bicarbonate, haemoglobin and plasma proteins. The depletion of these buffering agents (increasing base deficit, or base excess in negative numbers) indicates the growing inability to neutralise hydrogen ions, and their continued production will ultimately lead to the disruption of cellular enzyme systems and to tissue injury.

**Documentation of fetal hypoxia**

As oxygen concentration in the tissues cannot in practice be quantified, the occurrence of fetal hypoxia can only be assessed by the documentation of metabolic acidosis. Metabolic acidosis can be evaluated by sampling arterial and venous blood from the umbilical cord immediately after birth (see Annex 1 for a detailed description of the method), measuring pH and partial pressure of carbon dioxide (pCO₂), and the derived bicarbonate (HCO₃⁻) and base deficit (BD) values. Base deficit in the extracellular fluid (BD_{ecf}), as calculated from umbilical cord blood parameters using the Siggaard-Andersen formula, is believed by some experts to be the best representative of hydrogen ion concentration of metabolic origin in the different fetal compartments, but the slightly higher BD_{blood}, as calculated by blood gas analysers can also be used. It should however be noted that different blood gas analysers may use different algorithms to calculate BD_{blood}. Metabolic acidosis is defined as the measurement in umbilical artery blood of a pH value below 7.00 and a BD in excess of 12 mmol/l. However, there is already an association with adverse short-term newborn outcome when pH values are below 7.05 and BD_{ecf} values above 10 mmol/l. Alternatively, umbilical artery blood lactate concentration may be used to quantify metabolic acidosis, and values exceeding 10 mmol/l have been strongly associated with adverse short-term newborn outcome. However, analysing
devices are often calibrated differently or measure lactate concentrations in different blood compartments, so reference values may vary according to the device.  

Blood gas and lactate analysis in the umbilical cord or in the newborn circulation during the first minutes of life is currently the only way of quantifying objectively the occurrence of hypoxia/acidosis just prior to birth. Umbilical blood sampling is innocuous to the newborn and relatively inexpensive. The resulting information provides useful and immediate feedback to the labour ward staff and can enhance the team’s experience with intrapartum monitoring. Umbilical cord blood analysis is also frequently considered important evidence in medico-legal claims. Local guidelines should determine the clinical situations in which umbilical blood analysis should be performed, but if the technology and resources are available, it is recommended in all cases of suspected fetal hypoxia/acidosis and/or low Apgar scores. It should be noted that the presence of metabolic acidosis does not exclude other contributory factors in the causation of neonatal depression and/or subsequent handicap (e.g. prematurity, birth trauma, infection, meconium aspiration, certain congenital anomalies, pre-existing lesions, neonatal hypoxia). Similarly, the absence of metabolic acidosis at birth does not exclude the occurrence of hypoxia/acidosis during pregnancy or earlier in labour.

The Apgar score reflects the pulmonary, cardiovascular and neurological functions of the newborn, and is depressed when hypoxia is sufficiently intense and prolonged to affect these systems. The 1-minute Apgar score is a crucial parameter to decide the start of newborn resuscitation, but has a relatively low association with intrapartum hypoxia/acidemia. Low Apgar scores at both 1 and 5 minutes are expected when severe intrapartum hypoxia/acidemia occurs, but the 5-minute Apgar has a stronger association with short- and long-term neurological outcome and neonatal death. However, it is important to remember that Apgar scores are not affected by minor degrees of fetal hypoxia, score assignment is subject to some inter-observer disagreement, and values can be low due to non-hypoxic causes, such as prematurity, birth trauma, infection, meconium aspiration, certain congenital anomalies, pre-existing lesions, medication administered to the mother, and early neonatal interventions such as vigorous endotracheal aspiration.

What are we trying to avoid with intrapartum fetal monitoring?

Low intracellular pH and inadequate energy production caused by hypoxia/acidosis have the potential to compromise cell function and to cause cell death. However, the vast majority of fetuses born with metabolic acidosis, with or without decreased Apgar scores, recover quickly and will not incur any short- or long-term complications. In only a few cases will fetal hypoxia/acidosis be of sufficient intensity and duration to cause malfunction of important organs and systems, and thereby put the newborn at risk of death or long-term morbidity.

Short-term neurological dysfunction caused by intrapartum hypoxia/acidosis is called hypoxic-ischemic encephalopathy (HIE), and this diagnosis requires the confirmation of metabolic acidosis, low Apgar scores, early imaging evidence of cerebral edema, and the
appearance of changes in muscular tone, sucking movements, seizures or coma in the first 48 hours of life. In a simplified way, it can be divided into three grades (Sarnat & Sarnat classification): Grade 1: no seizures present; the vast majority of newborns do not develop major long-term neurological sequelae; Grade 2: seizures; associated with a 20-30% risk of death or major neurological sequelae; Grade 3: coma; the majority of newborns die or develop long-term neurological sequelae. Importantly, there are other non-hypoxic causes for neonatal encephalopathy, and the hypoxic-ischemic nature of this entity needs to be confirmed by the documentation of metabolic acidosis in the umbilical artery or in the newborn circulation during the first minutes of life. HIE may also be accompanied by dysfunction of the cardiovascular, gastrointestinal, haematological, pulmonary or renal systems.

Cerebral palsy of the spastic quadriplegic or dyskinetic type is the long-term neurological complication that is more commonly associated with intrapartum hypoxia/acidosis at term, but in developed countries only 10-20% of cerebral palsy cases are caused by birth asphyxia. Infection, congenital diseases, metabolic diseases, coagulation disorders, antepartum and post-natal hypoxia, and the complications associated with birth trauma and prematurity constitute the majority of causal situations. It may also be linked to a combination of antepartum and intrapartum events. To implicate intrapartum hypoxia/acidosis as the cause of cerebral palsy in term infants there is a need to document the joint occurrence of metabolic acidosis, low 1 and 5-minute Apgar scores, early onset grade 2 or 3 hypoxic-ischemic encephalopathy, early imaging studies showing evidence of an acute and non-focal cerebral anomaly, the development of spastic quadriplegic or dyskinetic types of cerebral palsy, and to exclude other identifiable etiologies (birth trauma, coagulation disorders, infection and genetic disorders).

While avoiding adverse fetal outcome related to hypoxia/acidemia is the main objective of intrapartum fetal monitoring, it is equally important that it does not result in unnecessary obstetrical intervention, as some of these procedures, such as instrumental vaginal delivery and caesarean section, are associated with increased maternal and fetal risks.

**Intrapartum events leading to fetal hypoxia**

Contractions compress the maternal blood vessels running inside the myometrium, decreasing placental perfusion, and this can result in a temporary reduction of maternal-fetal gas exchange. If during contractions the umbilical cord is compressed between fetal parts, or between fetal parts and the uterine wall, this will result in interference with blood circulation. The frequency, duration and intensity of uterine contractions are key determinants of the magnitude and effects of these disturbances. The interval between contractions is of particular importance for re-establishment of fetal oxygenation. There are data to suggest that in spontaneous labour it takes up to 90 seconds after a contraction for fetal oxygenation to be restored, while in oxytocin-augmented labours this recovery period averages 138 seconds. Excessive uterine activity (please see Chapter 3 for a definition) is often responsible for decreased fetal oxygenation, and where possible, should be avoided irrespective of FHR.
changes. Whether spontaneous or iatrogenic in nature, excessive uterine activity can usually be reversed by reducing or stopping oxytocin infusion and/or starting acute tocolysis with beta-adrenergic agonists (salbutamol, terbutaline, ritodrine) or atosiban, or nitroglycerine.

Other less frequent intrapartum complications can also affect fetal oxygenation. Some of these are of maternal origin, such as the occurrence of acute respiratory complications, a cardio-respiratory arrest following amniotic fluid embolism or pulmonary thromboembolism, or sudden maternal hypotension that may occur after epidural or spinal analgesia. Major placental abruption and uterine rupture will also severely impact fetal oxygenation, the latter due to acute maternal blood loss and/or to the disruption of placental blood supply. Several mechanical complications of delivery may cause compression of the umbilical cord and/or parts of the fetal circulation, such as umbilical cord prolapse, shoulder dystocia and retention of the after coming head in a breech delivery. It is also important to note that maternal supine position can lead to aorto-caval compression by the pregnant uterus, resulting in reduced placental gas exchange and temporary hypoxemia. Finally the rare occurrence of fetal hemorrhage, associated with ruptured vasa previa or fetal-maternal hemorrhage, will reduce the oxygen carrying capacity of the fetal circulation.

All of these complications require specific interventions for their resolution, to tackle the underlying cause and to determine the timing of delivery, with the objective of avoiding prolonged fetal hypoxia/acidosis, as well as unnecessary obstetric intervention. While the specific management of each of these situations is beyond the scope of this document, the general principles involved in the clinical reaction to the FHR patterns associated with these events are included in the following chapters.

Annex 1 – Umbilical blood sampling technique, interpretation, and pitfalls

Sampling of umbilical arterial and venous blood shortly after delivery is needed to document objectively the occurrence of fetal hypoxia/acidosis. Clamping of the cord is not necessary before vessels are sampled, but umbilical blood gas concentrations change quickly after birth, so this needs to be performed as soon as possible. Even if the cord is doubly clamped, sampling of vessels should be performed as soon as possible and preferably within 15 minutes, as blood gas and lactate values change significantly over time. Blood should be drawn, introducing as little air as possible, into two different 1 or 2 ml pre-heparinised syringes (if pre-heparinised syringes are not available, a small quantity of heparin can be drawn into normal syringes, and the excess heparin expelled before blood sampling). After blood is drawn, existing air bubbles should be removed from the syringes, these should be capped, rolled between the fingers to mix blood with heparin, and blood gas analysis should be performed in a calibrated apparatus within the next 30 minutes.

Umbilical arterial blood reflects the fetal acid-base status better than venous blood. However, it is important to obtain blood from both artery and vein in order to assure that a valid arterial sample is present. Sampling of the wrong vessel is not uncommon, particularly
when the needle crosses the artery to pierce the vein, and this can also result in mixed sampling. Arterial pH is lower than that of the vein, and when the difference in pH between the two blood samples is less than 0.02 and the difference in pCO₂ is less than 5 mm Hg or 0.7 kPa (kilopascal), then the samples are most likely mixed or were obtained from the same vessel. In addition, a pCO₂ < 22 mm Hg or 2.9 kPa is almost impossible to achieve in the umbilical artery, so such a value indicates likely contamination from the umbilical vein or from air.

Median umbilical artery pH in deliveries after 36 weeks of gestation is 7.25 (5th percentile 7.06; 95th percentile 7.37), median arterial BD 2.8 mmol/l (5th percentile -1.8; 95th percentile 11.48 mmol) When placental gas exchange is preserved, there is slow transfer of hydrogen ions in both directions, so maternal hyperventilation may result in an increase in fetal pH and likewise maternal acidemia will slowly result in fetal acidemia.

When gas exchange across the placenta is compromised or when there is significant umbilical cord occlusion, both increased CO₂ and decreased O₂ concentrations may occur in the fetus, and thus an acidemia of mixed respiratory and metabolic origin is documented. However, the metabolic component, reflected in the BD is the one with the greatest potential for harm, as it indicates decreased cellular oxygen concentration and reduced energy production.

References

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1. INTRODUCTION

The purpose of this chapter is to assist in the use and interpretation of intrapartum cardiotocography (CTG), as well as in the clinical management of specific CTG patterns. In the preparation of these guidelines, it has been assumed that all necessary resources, both human and material, required for intrapartum monitoring and clinical management are readily available. Unexpected complications may occur during labour, even in patients without prior evidence of risk, so maternity hospitals need to ensure the presence of trained staff, as well as appropriate facilities and equipment for an expedite delivery (in particular emergency cesarean section). CTG monitoring should never be regarded as a substitute for good clinical observation and judgement, or as an excuse for leaving the mother unattended during labour.

2. INDICATIONS

The evidence for the benefits of continuous CTG monitoring, as compared to intermittent auscultation, in both low and high-risk labours is scientifically inconclusive 1-2. When compared to intermittent auscultation, continuous CTG has been shown to decrease the occurrence of neonatal seizures, but no effect has been demonstrated on the incidence of overall perinatal mortality or cerebral palsy. However, these studies were carried out in the 1970s, 1980s, and early 1990s where equipment, clinical experience and interpretation criteria were very different from current practice, and they were clearly underpowered to evaluate differences in major outcomes 3. These issues are discussed in more detail below (see section 8 of this chapter). In spite of these limitations, most experts believe that continuous CTG monitoring should be considered in all situations where there is a high risk of fetal hypoxia/acidosis, whether due to maternal health conditions (such as vaginal haemorrhage and maternal pyrexia), abnormal fetal growth during pregnancy, epidural analgesia, meconium stained liquor, or the possibility of excessive uterine activity, as occurs with induced or augmented labour. Continuous CTG is also recommended when abnormalities are detected during intermittent fetal auscultation. The use of continuous intrapartum CTG in low-risk women is more controversial, although it has become standard of care in many countries. An alternative approach is to provide intermittent CTG monitoring alternating with fetal heart rate (FHR) auscultation. There
is some evidence to support that this is associated with similar neonatal outcomes in low-risk pregnancies. Intermittent monitoring should be carried out long enough to allow adequate evaluation of the basic CTG features (see below). The routine use of admission CTG for low-risk women on entrance to the labour ward has been associated with an increase in caesarean section rates and no improvement in perinatal outcomes, but studies were also underpowered to show such differences. In spite of the lack of evidence regarding benefit, this procedure has also become standard of care in many countries.

3. TRACING ACQUISITION

**Maternal position for CTG acquisition**

Maternal supine recumbent position can result in aorto-caval compression by the pregnant uterus, affecting placental perfusion and fetal oxygenation. Prolonged monitoring in this position should therefore be avoided. The lateral recumbent, half-sitting, and upright positions are preferable alternatives.

CTG acquisition can be performed by portable sensors that transmit signals wirelessly to a remote fetal monitor (telemetry). This solution has the advantage of allowing the mother to move freely during signal acquisition, rather than be restrained to bed or a sofa, and should therefore be the preferred option when available. Telemetry systems differ in the maximum distance allowed between patient and monitor for adequate signal transmission.

**Paper scales for CTG registration and viewing**

The horizontal scale for CTG registration and viewing is commonly called “paper speed” and available options are usually 1, 2 or 3 cm/min. In many countries throughout the world, 1 cm/min is selected, while in the Netherlands it is usually 2 cm/min, and in North America and Japan it is almost exclusively 3 cm/min. Some experts feel that 1 cm/min provides records of sufficient detail for clinical analysis, and this has the advantage of reducing tracing length. Other experts feel that the small details of CTG tracings are better evaluated using higher paper speeds. The vertical scale used for registration and viewing may also be different, and available alternatives are 20 or 30 bpm/cm.

The paper scales used in each centre should be the one with which healthcare professionals are most familiar, because tracing interpretation depends on pattern recognition and these patterns may appear very different. Inadvertent use of paper scales to which the staff is unaccustomed may lead to erroneous interpretations of CTG features. For example, at 3 cm/min variability appears reduced to a clinician familiar with the 1 cm/min scale, while it may appear exaggerated in the opposite situation (see examples below).

**External versus internal FHR monitoring**

External FHR monitoring uses a Doppler ultrasound transducer to detect the movement of cardiac structures. The resulting signal requires signal modulation and autocorrelation to provide adequate quality recordings. This process results in an approximation of the true heart rate intervals, but this is considered to be sufficiently accurate for analysis. External FHR monitoring is more prone to signal loss, to inadvertent monitoring of the maternal heart rate (Fig. 1), and to signal artefacts such as double-counting (Fig. 2) and half-counting, particularly during the second stage of labour. It may also not record fetal cardiac arrhythmias accurately.
Fig 1. Maternal heart rate monitoring in the last 9 min of the tracing. External FHR monitoring at 1 cm/min (top graph), 2 cm/min (middle graph) and 3 cm/min (bottom graph).

Fig 2. Double-counting of the FHR during decelerations (arrows). External FHR monitoring at 1 cm/min (top graph), 2 cm/min (middle graph) and 3 cm/min (bottom graph).

Internal FHR monitoring using a fetal electrode (usually known as scalp electrode, but it can also be applied to the breech) evaluates the time intervals between successive heart beats by identifying R waves on the fetal electrocardiogram QRS complex, and therefore measures ventricular depolarisation cycles. This method provides a more accurate evaluation of intervals between cardiac cycles, but it is more expensive because it requires a disposable electrode. It is very important that the fetal electrode is only applied after a clear identification of the presenting part and that delicate fetal structures such as the sutures and fontanels are avoided. Internal FHR monitoring requires ruptured membranes and has established contra-indications, mainly related to the increased risk of vertical transmission of infections. It should not be used in patients with active genital herpes infection, those who are seropositive to hepatitis B, C, D, E, or to human immunodeficiency virus, in suspected fetal blood disorders, when there is uncertainty about the presenting part, or when artificial rupture of membranes is inappropriate (i.e. an unengaged presentation). Fetal electrode placement should also preferably be avoided in very preterm fetuses (under 32 weeks gestation).

External FHR monitoring is the recommended initial method for routine intrapartum monitoring, provided that a recording of acceptable quality is obtained, i.e. that the basic CTG features can be identified. Minimum requirements for using this method are that careful repositioning of the probe is carried out during the second stage of labour, that in all atypical FHR
External versus internal monitoring of uterine contractions

External monitoring of uterine contractions using a tocodynamometer (toco) evaluates increased myometrial tension measured through the abdominal wall. Incorrect placement, reduced tension applied to the supporting elastic band, or abdominal adiposity may result in failed or inadequate registration of contractions. In addition, this technology only provides accurate information on the frequency of contractions. It is not possible to extract reliable information regarding the intensity and duration of contractions, nor on basal uterine tone.

Internal monitoring of uterine contractions using an intrauterine catheter provides quantitative information on the intensity and duration of contractions, as well as on basal uterine tone, but it is more expensive as the catheter is disposable, and requires ruptured membranes. Contra-indications include uterine haemorrhage of unknown cause and placenta praevia. It may also be associated with a small risk of fetal injury, placental haemorrhage, uterine perforation, and infection. The routine use of intrauterine pressure catheters has not been shown to be associated with improved outcomes in induced and augmented labour, and so it is not recommended for routine clinical use.

Simultaneous monitoring of the maternal heart rate

Simultaneous monitoring of the maternal heart rate (MHR) can be useful in specific maternal health conditions and in cases where it is difficult to distinguish between maternal and fetal heart rates (for example complete fetal heart block). Some CTG monitors provide the possibility of continuous MHR monitoring, either by electrocardiography or pulse oximetry. In some recent models, the latter technology has been incorporated in the tocodynamometer, allowing continuous MHR monitoring without the use of additional equipment. Providing that the technology is available and does not cause discomfort to the mother, simultaneous MHR monitoring should be considered when performing continuous CTG, especially during the second stage of labour, when tracings show accelerations coinciding with contractions and expulsive efforts, or when the MHR is elevated.

Monitoring of twins

Continuous external FHR monitoring of twin gestations during labour should preferably be performed with dual channel monitors that allow simultaneous monitoring of both FHRs, as duplicate monitoring of the same twin may occur and this can be picked up by observing almost identical tracings. Some monitors have embedded algorithms to alarm when this situation is suspected. During the second stage of labour, external FHR monitoring of twins is particularly affected by signal loss, and for this reason some experts believe that the presenting twin should preferably be monitored internally for better signal quality, if no contraindications to fetal electrode placement are present. Other experts believe that external monitoring of both twins is acceptable, provided that distinct and good quality FHR signals can be obtained.

Storage of tracings

All CTG tracings need to be identified with the patient name, place of recording, “paper speed”, date and time when acquisition started and ended. In hospitals where paper CTG recordings are used, the latter should be considered as part of the patient record and preserved as such. In hospitals using digital CTG archives, a secure file backup system needs to be in place, and all tracings should be readily available for review by the clinical staff.

4. ANALYSIS OF TRACINGS

CTG analysis starts with the evaluation of basic CTG features (baseline, variability, accelerations, decelerations and contractions) followed by overall CTG classification.
**BASELINE** – this is the mean level of the most horizontal and less oscillatory FHR segments. It is estimated in time periods of 10 minutes and expressed in beats per minute (bpm). The baseline value may vary between subsequent 10-minute sections.

*In tracings with unstable FHR signals, review of previous segments and/or evaluation of longer time periods may be necessary to estimate the baseline*, in particular during the 2nd stage of labour and to identify the fetal behavioural state of active wakefulness (see below – Fig. 3) that can lead to an erroneously high baseline estimation.

![Fetal behavioural state of active wakefulness](image)

**Fig 3.** Fetal behavioural state of active wakefulness. This pattern may lead to an erroneously high baseline estimation if it is identified at the top of accelerations. External FHR monitoring at 1 cm/min (top graph), 2 cm/min (middle graph) and 3 cm/min (bottom graph).

**Normal baseline** – a value between 110 and 160 bpm. *Preterm fetuses tend to have values towards the upper end of this range and post term fetuses towards the lower end. Some experts consider the normal baseline values at term to be between 110-150 bpm.*

**Tachycardia** – a baseline value above 160 bpm lasting more than 10 minutes. *Maternal pyrexia is the most frequent cause of fetal tachycardia, and it may be of extra-uterine origin or associated with intrauterine infection. Epidural analgesia may also cause a rise in maternal temperature resulting in fetal tachycardia*. In the initial stages of a non-acute fetal hypoxemia, catecholamine secretion may also result in tachycardia. Other less frequent causes are the administration of beta-agonist drugs (salbutamol, terbutaline, ritodrine, fenoterol), parasympathetic blockers (atropine, escopolamine), and fetal arrhythmias such as supraventricular tachycardia and atrial flutter.

**Bradycardia** – a baseline value below 110 bpm lasting more than 10 minutes. *Values between 100 and 110 bpm may occur in normal fetuses, especially in postdate pregnancies. Maternal hypothermia, administration of beta-blockers, and fetal arrhythmias such as atrial-ventricular block are other possible causes.*

**VARIABILITY** – refers to the oscillations in the FHR signal, evaluated as the average bandwidth amplitude of the signal in one-minute segments.

**Normal variability** – a bandwidth amplitude of 5-25 bpm.
**Reduced variability** – a bandwidth amplitude below 5 bpm for more than 50 minutes in baseline segments 21 (Figs. 4-5), or for more than 3 minutes during decelerations 22 (Figs. 8-9).

Reduced variability can occur due to central nervous system hypoxia/acidosis and resulting decreased sympathetic and parasympathetic activity, but it can also be due to previous cerebral injury 23, infection, administration of central nervous system depressants or parasympathetic blockers. During deep sleep, variability is usually in the lower range of normality, but the bandwidth amplitude is seldom under 5 bpm. There is a high degree of subjectivity in the visual evaluation of this parameter, and therefore careful re-evaluation is recommended in borderline situations. Following an initially normal CTG, reduced variability due to hypoxia is very unlikely to occur during labour without preceding or concomitant decelerations and a rise in the baseline.

*Fig 4.* Reduced variability. External FHR monitoring at 1 cm/min (top graph), 2 cm/min (middle graph) and 3 cm/min (bottom graph).
Reduced variability – the baseline is affected by contractions causing decreases in FHR that are close to fulfilling the criteria for decelerations, but the bandwidth remains reduced. Internal FHR monitoring at 1 cm/min (top graph), 2 cm/min (middle graph) and 3 cm/min (bottom graph).

**Increased variability (saltatory pattern)** – a bandwidth value exceeding 25 bpm lasting more than 30 minutes (Fig. 6).

The pathophysiology of this pattern is incompletely understood, but it may be seen linked with recurrent decelerations, when hypoxia/acidosis evolves very rapidly. It is presumed to be caused by fetal autonomic instability/hyperactive autonomic system 24.

**ACCELERATIONS** – abrupt (onset to peak in less than 30 seconds) increases in FHR above the baseline, of more than 15 bpm in amplitude, and lasting more than 15 seconds but less than 10 minutes.

Most accelerations coincide with fetal movements and are a sign of a neurologically responsive fetus that does not have hypoxia/acidosis. Before 32 weeks’ gestation, their amplitude and frequency may be lower (10 seconds and 10 bpm of amplitude). After 32-34 weeks, with the establishment of fetal behavioural states, accelerations rarely occur during periods of deep sleep, which can last up to 50 minutes 21. The absence of accelerations in an otherwise normal intrapartum CTG is of uncertain significance, but it is unlikely to indicate hypoxia/acidosis. Accelerations coinciding with uterine contractions, especially in the second stage of labour, suggest possible erroneous recording of the maternal heart rate, since the FHR more frequently decelerates with a contraction, while the maternal heart rate typically increases 9.
DECELERATIONS – decreases in the FHR below the baseline, of more than 15 bpm in amplitude, and lasting more than 15 seconds.

**Early decelerations** – decelerations that are shallow, short-lasting, with normal variability within the deceleration and are coincident with contractions. They are believed to be caused by fetal head compression and do not indicate fetal hypoxia/acidosis.

**Variable decelerations** (V-shaped) – decelerations that exhibit a rapid drop (onset to nadir in less than 30 seconds), good variability within the deceleration, rapid recovery to the baseline, varying size, shape and relationship to uterine contractions (Fig. 7). Variable decelerations constitute the majority of decelerations during labour, and they translate a baroreceptor-mediated response to increased arterial pressure, as occurs with umbilical cord compression. They are seldom associated with an important degree of fetal hypoxia/acidosis, unless they evolve to exhibit a U-shaped component, reduced variability within the deceleration (see late decelerations below), and/or their individual duration exceeds 3 minutes (see prolonged decelerations below).

![Image of variable decelerations](image)

**Fig 7.** Variable decelerations. Internal FHR monitoring at 1 cm/min (top graph), 2 cm/min (middle graph) and 3 cm/min (bottom graph).

**Late decelerations** (U-shaped and/or with reduced variability) – decelerations with a gradual onset and/or a gradual return to the baseline and/or reduced variability within the deceleration (Fig. 8). Gradual onset and return occurs when more than 30 seconds elapses between the beginning/end of a deceleration and its nadir. When contractions are adequately monitored, late decelerations start more than 20 seconds after the onset of a contraction, a nadir after the acme, and a return to the baseline after the end of the contraction.

These decelerations are indicative of a chemoreceptor-mediated response to fetal hypoxemia. In the presence of a tracing with no accelerations and reduced variability, the definition of late decelerations also includes those with an amplitude of 10-15 bpm.
**Fig 8.** Late decelerations in the second half of the tracing. External FHR monitoring at 1 cm/min (top graph), 2 cm/min (middle graph) and 3 cm/min (bottom graph).

**Prolonged decelerations** – lasting more than 3 minutes. These are likely to include a chemoreceptor-mediated component and thus to indicate hypoxemia. Decelerations exceeding 5 minutes, with FHR maintained <80 bpm and reduced variability within the deceleration (Fig. 9), are frequently associated with acute fetal hypoxia/acidosis and require emergent intervention.
**Fig 9.** Prolonged deceleration. External FHR monitoring at 1 cm/min (top graph), 2 cm/min (middle graph) and 3 cm/min (bottom graph).

**SINUSOIDAL PATTERN** – a regular, smooth, undulating signal, resembling a sine wave, with an amplitude of 5-15 bpm, and a frequency of 3-5 cycles per minute. This pattern lasts more than 30 minutes, and coincides with absent accelerations (Fig. 10).

The pathophysiological basis of the sinusoidal pattern is incompletely understood, but it occurs in association with severe fetal anemia, as is found in anti-D allo-immunisation, fetal-maternal hemorrhage, twin-to-twin transfusion syndrome and ruptured vasa praevia. It has also been described in cases of acute fetal hypoxia, infection, cardiac malformations, hydrocephalus and gastroschisis 31.

**Fig 10.** Sinusoidal pattern. External FHR monitoring at 1 cm/min (top graph), 2 cm/min (middle graph) and 3 cm/min (bottom graph).

**PSEUDO-SINUSOIDAL PATTERN** – a pattern resembling the sinusoidal pattern, but with a more jagged “saw-tooth” appearance, rather than the smooth sine-wave form (Fig. 11). Its duration seldom exceeds 30 minutes and it is characterised by normal patterns before and after.

This pattern has been described after analgesic administration to the mother, and during periods of fetal sucking and other mouth movements 32. It is sometimes difficult to distinguish the pseudo-sinusoidal pattern from the true sinusoidal pattern, leaving the short duration of the former as the most important variable to discriminate between the two.
**FETAL BEHAVIOURAL STATES** – refers to periods of fetal quiescence reflecting deep sleep (no eye movements), alternating with periods of active sleep (rapid eye movements) and wakefulness. The occurrence of different behavioural states is a hallmark of fetal neurological responsiveness and absence of hypoxia/acidosis. Deep sleep can last up to 50 minutes and is associated with a stable baseline, very rare accelerations, and borderline variability. Active sleep is the most frequent behavioural state, and is represented by a moderate number of accelerations and normal variability. Active wakefulness is rarer and represented by a large number of accelerations and normal variability (Fig. 1). In the latter pattern, accelerations may be so frequent as to cause difficulties in baseline estimation (see Fig 1 above). Transitions between the different patterns become clearer after 32-34 weeks of gestation, consequent to fetal nervous system maturation.

**CONTRACTIONS** – these are bell-shaped gradual increases in the uterine activity signal followed by roughly symmetric decreases, with 45-120 seconds in total duration. Contractions are essential for the progression of labour, but they compress the vessels running inside the myometrium and may transiently decrease placental perfusion and/or cause umbilical cord compression (see Chapter 1). With the tocodynamometer, only the frequency of contractions can be reliably evaluated, but increased intensity and duration can also contribute to FHR changes. **Tachysystole** – represents an excessive frequency of contractions and is defined as the occurrence of more than 5 contractions in 10 minutes, in two successive 10-minute periods, or averaged over a 30-minute period.

5. **TRACING CLASSIFICATION**

Tracing classification requires a previous evaluation of basic CTG features (see above). Tracings should be classified into one of three classes: normal, suspicious or pathological, according to the criteria presented in Table 1. Other classification systems including a larger number of tiers are recommended by some experts. Due to the changing nature of CTG signals during labour, re-evaluation of the tracing should be carried out at least every 30 minutes.
Table 1. CTG classification criteria, interpretation and recommended management. The presence of accelerations denotes a fetus that does not have hypoxia/acidosis, but their absence during labour is of uncertain significance. *Decelerations are repetitive in nature when they are associated with more than 50% of uterine contractions 29.

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Suspicious</th>
<th>Pathological</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Baseline</strong></td>
<td>110-160 bpm</td>
<td>Lacking at least one characteristic of normality, but with no pathological features</td>
<td>Reduced variability for &gt; 50 min, increased variability for &gt;30 min, or sinusoidal pattern for &gt; 30 min</td>
</tr>
<tr>
<td><strong>Variability</strong></td>
<td>5-25 bpm</td>
<td></td>
<td>Repetitive* late or prolonged decelerations during &gt; 30 min or 20 min if reduced variability, or one prolonged deceleration with &gt; 5 min</td>
</tr>
<tr>
<td><strong>Decelerations</strong></td>
<td>No repetitive* decelerations</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Interpretation</strong></td>
<td>Fetus with no hypoxia/acidosis</td>
<td>Fetus with a low probability of having hypoxia/acidosis</td>
<td>Fetus with a high probability of having hypoxia/acidosis</td>
</tr>
<tr>
<td><strong>Clinical Management</strong></td>
<td>No intervention necessary to improve fetal oxygenation state</td>
<td>Action to correct reversible causes if identified, close monitoring or additional methods to evaluate fetal oxygenation (chapter 4).</td>
<td>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.</td>
</tr>
</tbody>
</table>

6. CLINICAL DECISION

Several factors, including gestational age and medication administered to the mother, can affect FHR features (see above), so CTG analysis needs to be integrated with other clinical information for a comprehensive interpretation and adequate management. As a general rule, if the fetus continues to maintain a stable baseline and a reassuring variability, the risk of hypoxia to the central organs is very unlikely. However, the general principles that should guide clinical management are outlined in Table 1.

7. ACTION IN SITUATIONS OF SUSPECTED FETAL HYPOXIA/ACIDOSIS

When fetal hypoxia/acidosis is anticipated or suspected (suspicious and pathological tracings), and action is required to avoid adverse neonatal outcome, this does not necessarily mean an immediate cesarean section or instrumental vaginal delivery. The underlying cause for the appearance of the pattern can frequently be identified and the situation reversed, with subsequent recovery of adequate fetal oxygenation and the return to a normal tracing.

Excessive uterine activity is the most frequent cause of fetal hypoxia/acidosis (see Chapter 1) and it can be detected by documenting tachysystole in the CTG tracing and/or palpating the uterine fundus. It can usually be reversed by reducing or stopping oxytocin infusion, removing administered prostaglandins if possible, and/or starting acute tocolysis with beta-adrenergic agonists (salbutamol, terbutaline, ritodrine) 37-39, atosiban 40, or nitroglycerine 41. During the second stage of labour, maternal pushing efforts can also contribute to fetal hypoxia/acidosis and the mother can be asked to stop pushing until the situation is reversed.

Aorto-caval compression can occur in the supine position and lead to reduced placental perfusion. Excessive uterine activity may also be associated with the supine position 42,43, possibly due to the stimulation of the sacral plexus by the uterine weight. In these cases, turning the mother to her side is frequently followed by normalization of the CTG pattern. Transient cord compression is another common cause of CTG changes (variable decelerations), and these can sometimes be reverted by changing the maternal position or by performing amniinfusion 44.

Sudden maternal hypotension can also occur during labour, usually after epidural or spinal analgesia 45, and it is usually reversible by rapid fluid administration and/or an intravenous
ephedrine bolus. Other less frequent complications affecting the maternal respiration, maternal circulation, placenta, umbilical cord or the fetal circulation can also result in fetal hypoxia/acidosis (see Chapter 1), and their management is beyond the scope of this document.

Oxygen administration to the mother is widely used with the objective of improving fetal oxygenation and consequently normalising CTG patterns, but there is no evidence from randomised clinical trials that this intervention, when performed in isolation, is effective when maternal oxygenation is adequate. Intravenous fluids are also commonly used for the purpose of improving CTG patterns, but again there is no evidence from randomised clinical trials to suggest that this intervention is effective in normotensive women.

Good clinical judgement is required to diagnose the underlying cause for a suspicious or pathological CTG, to judge the reversibility of the conditions with which it is associated, and to determine the timing of delivery, with the objective of avoiding prolonged fetal hypoxia/acidosis, as well as unnecessary obstetric intervention. Additional methods may be used to evaluate fetal oxygenation, and these are considered in detail in chapter 4. When a suspicious or worsening CTG pattern is identified, the underlying cause should be addressed before a pathological tracing develops. If the situation does not revert and the pattern continues to deteriorate, consideration needs to be given for further evaluation or rapid delivery if a pathological pattern ensues.

During the second stage of labour, due to the additional effect of maternal pushing, hypoxia/acidosis may develop more rapidly. Therefore, urgent action should be undertaken to relieve the situation, including discontinuation of maternal pushing, and if there is no improvement, delivery should be expedited.

8. LIMITATIONS OF CARDIOTOCOGRAPHY

Cardiotocography has well documented limitations, and it is necessary to be aware of these for a safe use of the technology.

It has been well demonstrated that CTG analysis is subject to considerable intra- and inter-observer disagreement, even when experienced clinicians use widely accepted guidelines. The main aspects that are prone to observer disagreement are the identification and classification of decelerations, the evaluation of variability, and the classification of tracings as suspicious and pathological. The subjectivity of observer analysis has also been demonstrated in retrospective audit of tracings, where CTG features are frequently assessed to be more abnormal in cases with known adverse neonatal outcome.

Many studies have evaluated the ability of suspicious and pathological CTGs to predict the occurrence of hypoxia/acidosis. Different CTG interpretation criteria, different intervals between tracing abnormality and birth, and different criteria to define adverse outcome have been used, resulting in mixed findings. However, it is recognised that hypoxia/acidosis has not been documented shortly after a normal CTG tracing. On the other hand, suspicious and pathological tracings have a limited capacity to predict metabolic acidosis and low Apgar scores, i.e. a large percentage of cases with suspicious and pathological tracings do not have these outcomes. While there is a strong association between certain FHR patterns and hypoxia/acidosis, their capacity to discriminate between newborns with or without metabolic acidosis is limited. Thus, they are sensitive indicators, but have a low specificity and low positive predictive value. However, it should not be forgotten that the aim of intrapartum fetal monitoring is to identify situations that precede hypoxia/acidemia so as to avoid fetal injury. The subjectivity of CTG interpretation, and the fact that hypoxia is a continuum that may not reach the threshold of metabolic acidosis or injury are probably important contributing factors to these limitations.

A large number of randomised controlled trials have been conducted comparing continuous CTG monitoring with intermittent auscultation as screening methods for fetal hypoxia/acidosis during labour, in both low- and high-risk women. However, these trials took place in the 1970s, 1980s, and early 1990s, and used different CTG interpretation criteria, so it is difficult to establish how their results relate to current clinical practice. With these limitations in mind, they indicate a limited benefit of continuous CTG for fetal monitoring in all women during labour, as the only significant improvement was a 50% reduction in neonatal seizures (hypoxic-ischemic encephalopathy was not evaluated in most trials), and no differences were found in the incidences of overall perinatal mortality and cerebral palsy. However, it is widely recognised that the trials were underpowered to detect differences in these outcomes. Only a small proportion of perinatal deaths and cerebral palsies are caused by intrapartum hypoxia/acidosis, so a large number of cases is needed to show any benefit. On the other hand, continuous CTG was associated with a 63% increase in cesarean delivery and a 15% increase in instrumental vaginal deliveries.
Unnecessary obstetric intervention confers additional risks for the mother and newborn and the former may result from poor CTG interpretation, limited knowledge of the pathophysiology of fetal oxygenation, and inadequate clinical management. It is recognised that, for consistent implementation, clinical guidelines need to be as simple and objective as possible, to allow rapid decision-making even in complex and stressful situations. In addition, regular and structured training of the labour ward staff is essential to ensure proper use of this technology.

References

INTRODUCTION

Intermittent auscultation (IA) is defined as the technique of listening to the fetal heart rate (FHR) for short periods of time without a display of the resulting pattern. Whether it be used for intrapartum fetal monitoring in low-risk women or for all cases in settings where there are no available alternatives, all healthcare professionals attending labor and delivery need to be skilled at performing IA, interpreting its findings, and taking appropriate action. The main aim of this chapter is to describe the tools and techniques for IA in labor.

HISTORICAL BACKGROUND

Hippocrates is said to have described the technique of listening to the internal activity of the body by placing the ear on the skin proximal to the organ under examination. However, the perception of fetal heart sounds using this method was not reported until the 1600’s. Little notice appears to have been taken of fetal heart auscultation until 1818, when it was discussed by both Mayor and de Kergaradec, to determine whether the fetus was alive or dead. Interest then accelerated, and in 1833 Kennedy published a book on the subject of obstetric auscultation.

The first recorded use of an amplification device for auscultation of the adult heart rate is attributed to Laennec in 1816, who overcame the embarrassment of placing the ear on a young woman’s chest to hear her heart beat, by rolling sheets of paper into a tube and listening through this device. This tool was soon replicated in wood, and gained wide usage for fetal heart auscultation. The most common instrument currently used for this purpose is the Pinard stethoscope (Figs. 1 and 2), but in some countries, notably the
US, the DeLee stethoscope is used as an alternative (Fig. 3). In both cases, the technology has not changed much from the original design, in which a belled tube creates an amplification chamber for sound waves that are transmitted from the fetal heart to the examiner’s ear.

More recently, handheld electronic devices that rely on the Doppler effect have been used for IA (Fig. 4), a technology similar to the external FHR monitoring of cardiotocography (CTG). However, as described in Chapter 3, these devices do not transmit the actual sound produced by the fetal heart, but rather a representation of this, based on ultrasound-detected movements of fetal cardiac structures, that are then subject to signal modification and autocorrelation.

<table>
<thead>
<tr>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pinard stethoscope</td>
<td>Inexpensive</td>
</tr>
<tr>
<td></td>
<td>Readily available in most countries</td>
</tr>
<tr>
<td></td>
<td>No consumables needed</td>
</tr>
<tr>
<td>DeLee stethoscope</td>
<td>Inexpensive</td>
</tr>
<tr>
<td></td>
<td>Readily available in some countries</td>
</tr>
<tr>
<td></td>
<td>No consumables needed</td>
</tr>
<tr>
<td>Handheld Doppler</td>
<td>More comfortable for the woman</td>
</tr>
<tr>
<td></td>
<td>FHR audible to all present in the room</td>
</tr>
<tr>
<td></td>
<td>Can be used in various maternal positions and locations (e.g. in water)</td>
</tr>
<tr>
<td></td>
<td>May calculate and display FHR values</td>
</tr>
<tr>
<td></td>
<td>May be difficult to use in certain maternal positions</td>
</tr>
<tr>
<td></td>
<td>More costly to purchase and maintain (requires batteries)</td>
</tr>
<tr>
<td></td>
<td>Probe is very sensitive to mechanical damage</td>
</tr>
<tr>
<td></td>
<td>May display maternal heart rate</td>
</tr>
</tbody>
</table>

Table 1. Advantages and disadvantages of the instruments used for IA

**OBJECTIVES AND INDICATIONS**

As for other approaches to fetal monitoring, the main aim of IA is the timely identification of fetuses with hypoxia/acidosis to enable appropriate action before the occurrence of injury. It also allows the confirmation of normal FHR characteristics, so that unnecessary intervention will be avoided. Systematic reviews of randomised controlled trials carried out in the 1970s, 1980s and early 1990s, comparing IA with continuous CTG for intrapartum monitoring in both low- and high-risk women, have shown that CTG is associated with a lower risk of neonatal seizures, but with higher cesarean section and instrumental vaginal delivery rates. The limitations of this evidence are analysed in Chapter 3. There is currently no conclusive evidence for the benefits of continuous CTG versus IA monitoring in labour. There are also no trials comparing IA with no FHR auscultation during labor.

Based on expert opinion, IA should be recommended in all labours in settings where there is no access to CTG monitors or to the resources necessary for using them. When the resources for CTG monitoring are available, IA may be used for routine intrapartum monitoring in low-risk cases (Table 2). However, approximately half of the panel members believe that continuous CTG should be the option during the second stage of labour, although there is no direct scientific evidence to support this.

<table>
<thead>
<tr>
<th>Antepartum factors</th>
<th>Intrapartum factors</th>
</tr>
</thead>
</table>
No serious previous maternal health conditions
No maternal diabetes or pre-eclampsia
No antenatal vaginal hemorrhage
Normal fetal growth, amniotic fluid and Doppler
Normal antenatal CTGs
No previous uterine scar
Normal fetal movements
No rupture of membranes lasting > 24 hours
Singleton, term, cephalic presentation

| No serious previous maternal health conditions | Normal frequency of contractions |
| No maternal diabetes or pre-eclampsia | No labor induction or augmentation |
| No antenatal vaginal hemorrhage | No epidural analgesia |
| Normal fetal growth, amniotic fluid and Doppler | No abnormal vaginal hemorrhage |
| Normal antenatal CTGs | No fresh or thick meconium |
| No previous uterine scar | No maternal temperature > 38ºC |
| Normal fetal movements | Active first stage lasting < 12 hours |
| No rupture of membranes lasting > 24 hours | Second stage lasting < 1 hour |
| Singleton, term, cephalic presentation | Clearly audible FHR sounds in normal range |

Table 2. Conditions required for considering and maintaining IA in settings where CTG is available.

ADVANTAGES OF IA

Performing regular IA ensures frequent contact between healthcare professionals and the laboring woman, offering the opportunity for social and clinical support. It facilitates the assessment of other physical parameters such as maternal skin tone, temperature, breathing patterns, direct palpation of fetal movements and maternal contractions.

IA permits the fetal heart to be monitored in various positions and locations and favors the mobility of laboring women, which has been shown to benefit the progress of labor. Another benefit of IA is the easier availability and sustainability of the technology, which allows it to be undertaken in even the lowest resource settings.

DISADVANTAGES OF IA

It takes time to develop clinical expertise with IA when performed with a fetal stethoscope. Initially it may not be easy to recognize the fetal heart sounds, and later there is a slow learning curve for the identification of accelerations and decelerations. Even for the most experienced healthcare professionals, it is impossible to recognize subtle features of the FHR, such as variability. Using fetal stethoscopes, awkward positions sometimes need to be adopted for effective auscultation and therefore healthcare professionals should ensure good ergonomic position for themselves and the laboring woman when using IA. Also with these instruments, there is no independent record of the FHR and usually no confirmation of the findings by other healthcare professionals, or by those in the room. This may cause uncertainty in case reviews and medical-legal cases.

Many of these disadvantages are overcome by the use of a handheld Doppler. When the latter includes a display showing the FHR, even low variability may be suspected. On the other hand, as occurs with external FHR monitoring in CTG, the device can inadvertently pick up the maternal heart rate.

Whichever method of IA is used, it may be difficult to guarantee the continued availability of appropriately trained staff to attend laboring women in busy labor units.

TECHNIQUE FOR PERFORMING IA

Before IA is initiated, a clear explanation of the technique and its purpose should be provided to the laboring woman, and her consent obtained. This is followed by an assessment of the fetal position on abdominal palpation, and placement of the stethoscope or handheld Doppler over the fetal back, as this is where the heart rate will usually be heard most clearly. Searching for sounds produced by the fetal heart (usually compared to a “galloping horse”) rather than those created by fetal vessels (“whoosh” sounds) allows for a clearer distinction from maternal heart rate. Simultaneous evaluation of the maternal pulse provides additional reassurance that the FHR is being monitored. Just before and during IA, a hand is placed on the uterine fundus to determine the timing of uterine contractions and to detect fetal movements. If the fetal heart cannot be
identified unambiguously, ultrasound should be used when available to determine the FHR and to establish the optimal location for IA.

There are no studies comparing the benefit of different auscultation intervals. In large randomised trials comparing CTG with IA, the latter was usually performed every 15 minutes in the first stage and every 5 minutes or after every other contraction in the second stage. While it is recognized that recommendations for the scheduling of IA are based only on expert opinion, standardisation of procedures is important for planning of healthcare and for medical-legal purposes. The recommendations for performing IA are considered in Table 3.

<table>
<thead>
<tr>
<th>Features to evaluate</th>
<th>What to register</th>
</tr>
</thead>
<tbody>
<tr>
<td>FHR</td>
<td>Duration: for at least 60 seconds; for 3 contractions if the FHR is not always in the normal range (110-160 bpm). Timing: during and at least 30 seconds after a contraction. Interval: Every 15 minutes in the active phase of the 1st stage of labor. Every 5 minutes in the 2nd stage of labor. Baseline (as a single counted number in bpm), presence or absence of accelerations and decelerations.</td>
</tr>
<tr>
<td>Uterine contractions</td>
<td>Before and during FHR auscultation, in order to detect at least two contractions. Frequency in 10 minutes</td>
</tr>
<tr>
<td>Fetal movements</td>
<td>At the same time as evaluation of uterine contractions. Presence or absence</td>
</tr>
<tr>
<td>Maternal heart rate</td>
<td>At the time of FHR auscultation. Single counted number in bpm</td>
</tr>
</tbody>
</table>

**Table 3.** Practice recommendations for IA, uterine contraction and maternal heart rate monitoring during labor.

All features listed in Table 3 should be recorded in dedicated labor charts, to provide an ongoing account of their evolution, and to share information between caregivers who are or may become involved in the process.

**ABNORMAL FINDINGS AND THEIR MANAGEMENT**

**In settings where continuous CTG is available**

Abnormal findings on IA are listed in Table 4. If there is doubt as to the characterization of FHR findings, auscultation should be prolonged in order to cover at least 3 contractions.

<table>
<thead>
<tr>
<th>Baseline</th>
<th>Below 110 bpm or above 160 bpm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decelerations</td>
<td>Presence of repetitive or prolonged (&gt;3 minutes) decelerations</td>
</tr>
<tr>
<td>Contractions</td>
<td>More than 5 contractions in a 10 minute period</td>
</tr>
</tbody>
</table>

**Table 4.** Abnormal findings on IA.

A FHR value under 110 bpm lasting more than 3 minutes, when the rate has previously been normal, is very suggestive of a prolonged deceleration or of fetal bradycardia, and constitutes an indication for immediate continuous CTG. A FHR value exceeding 160 bpm during three contractions is very suggestive of fetal tachycardia, and constitutes an indication for continuous CTG.

Sometimes, decelerations occur due to the maternal supine position and resulting aorto-caval compression. Changing the maternal position may quickly revert the
situation. However, if a rapid normalization does not ensue, or if repetitive or prolonged decelerations are detected, continuous CTG should be started.

Most accelerations coincide with fetal movements detected by the mother and/or the healthcare professional, and are a sign of fetal wellbeing. However, accelerations occurring just after a contraction do not usually translate fetal movements and should motivate auscultation over at least 3 contractions in order to rule out the occurrence of decelerations.

An interval between two contractions of less than 2 minutes, should lead to evaluation of uterine contractions over 10 minutes. More than 5 contractions detected during this period is considered tachysystole (see Chapter 3). This constitutes an indication for continuous CTG, at least until the situation is reversed.

If assessment of the parameters described in Table 3 and the general behavior of the mother indicate the continuous wellbeing of both mother and baby, IA may continue to be the technique of choice for labor.

**In settings where continuous CTG is not available**

If a FHR value under 110 bpm lasting more than 5 minutes is detected, in the absence of maternal hypothermia, known fetal heart block, or beta-blocker therapy, consideration should be given to immediate delivery by cesarean section or instrumental vaginal delivery, according to obstetric conditions and local resources.

A FHR value exceeding 160 bpm during at least 3 contractions is suggestive of fetal tachycardia, and should motivate an evaluation of maternal temperature and signs of intrauterine infection. Beta-agonists drugs (salbutamol, terbutaline, ritodrine, fenoterol) and parasympathetic blockers (atropine, escopolamine) are other possible causes. With isolated fetal tachycardia, increased frequency of IA and treatment of pyrexia and/or infection need to be considered.

Repetitive decelerations are frequent during the second stage of labor and may occur as a result of aorto-caval, umbilical cord or fetal head compression. Changing the maternal position may revert the first two causes. However, if decelerations start more than 20 seconds after the onset of a contraction and take more than 30 seconds to recover to baseline values (late decelerations), or when decelerations last more than 3 minutes (prolonged decelerations), this is very suggestive of fetal hypoxia/acidosis. If an accompanying tachysystole is detected, consideration should be given to acute tocolysis with beta-adrenergic agonists (salbutamol, terbutaline, ritodrine), atosiban, or nitroglycerine (see Chapter 1), followed by continued auscultation to document the normalization of the pattern. Sudden maternal hypotension rarely happens during labour in the absence of conduction analgesia, but should it occur in association with a fetal deceleration, increased intravenous fluid administration turning the mother to her side and administering intravenous ephedrine will usually revert the situation. When late and/or prolonged decelerations are documented during the second stage of labour the mother should be asked to stop pushing until this pattern disappears. If there is no rapid reversal of late and/or prolonged decelerations, consideration should be given to immediate delivery, by cesarean section or instrumental vaginal delivery, according to obstetric conditions and local resources.

**References**


Figure 1

Figure 2

Figure 3
Figure 4
FIGO CONSENSUS GUIDELINES ON INTRAPARTUM FETAL MONITORING

Safe Motherhood and Newborn Health Committee
Co-ordinator: Diogo Ayres-de-Campos

ADJUNCTIVE TECHNOLOGIES

Gerard H.A. Visser, Diogo Ayres-de-Campos for the FIGO intrapartum fetal monitoring consensus panel.

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* nominated by FIGO associated national society; ** invited by FIGO based on literature search

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INTRODUCTION

As referred to in the previous chapter, cardiotocography (CTG) has a high sensitivity but only a limited specificity in predicting fetal hypoxia-acidosis. In other words, a normal CTG is reassuring regarding the state of fetal oxygenation, as hypoxia-acidosis is generally restricted to cases with suspicious or pathological patterns (see definitions in previous chapter), however, a large number of fetuses with the latter patterns will not have clinically important hypoxia-acidosis. To reduce such false-positive cases and unnecessary medical interventions, adjunctive technologies have been proposed to further assess fetal oxygenation. These technologies should indicate intervention at an early stage of evolving fetal hypoxia-acidosis in order to prevent rather than to predict poor newborn outcome. Several adjunctive technologies have been developed over the last decades, including fetal blood sampling (FBS), continuous pH and lactate monitoring, fetal stimulation (FS), pulse oximetry, and ST waveform analysis, and some of these have been successfully established.

Continuous fetal pH monitoring was developed in the 1970’s, but several technical difficulties arose, particularly because glass electrodes could break in the fetal scalp, and the technique was
subsequently abandoned. Fetal pulse oximetry was developed in the 1990’s, but the commercialisation of electrodes has subsequently been discontinued. A systematic review of four trials comparing CTG + fetal pulse oximetry with isolated CTG showed no difference in overall caesarean section rate (RR 0.99, 95% confidence intervals (CI) 0.86 to 1.13), while adverse fetal outcomes were rare in both groups ³. This chapter will focus on currently available adjunctive technologies for intrapartum fetal monitoring.

**FETAL BLOOD SAMPLING (FBS) FOR PH AND LACTATE MEASUREMENTS**

Fetal blood sampling (FBS) during labour was first introduced in 1962 and is currently used for assessment of fetal blood gases and/or lactate. Studies in fetal monkeys showed a good correlation of acid-base parameters between scalp and carotid blood ⁵, and human data have shown similar correlations between pH and lactate values obtained in scalp blood and those recorded shortly after birth in the umbilical artery and vein ⁶-¹⁰. However, correlation of these values with newborn outcome depends on the time interval between scalp sampling and birth ¹¹. It has been argued that fetal capillary blood is likely to be affected by the redistribution of circulation occurring during fetal hypoxemia, and it therefore may not adequately represent the central circulation ¹². There is however the opposite argument that this aspect favours FBS, because intrapartum fetal monitoring aims to identify fetuses in the early rather than in the late process of hypoxia.

**Indications**

FBS may be used in cases of suspicious or pathological CTG tracings (see Chapter 3). When pathological CTGs indicate a severe and acute event (see Chapter 3), immediate action should be taken, and FBS is not advised, as it would cause further delay.

**Technique**

To perform FBS a disposable or re-usable FBS set can be used. It is necessary for the membranes to be ruptured and cervical dilation should be at least 3 cm. A vaginal examination needs to be performed prior to the procedure, to assess the nature and position of the presenting part. The technique has similar contra-indications to those of the fetal electrode: active genital herpes infection, women seropositive to hepatitis B, C, D, E, or to human immunodeficiency virus, suspected fetal blood disorders, uncertainty about the presenting part, or when artificial rupture of membranes is inappropriate. An amnioscope (the diameter of which can vary according to cervical dilation) is inserted in the vagina, and the lighting equipment attached. With the amnioscope held tightly in place, the presenting part is dried using small swabs, and a thin layer of paraffin is applied to the presenting part, in order for blood to form a large drop and to prevent it from spreading over the skin, thus causing loss of CO₂ by diffusion. The incision on the fetal skin should not exceed 2 mm and after a blood drop is formed, it is collected in a heparin-coated capillary. When this is concluded, the incision
site is inspected for persistent bleeding, which can usually be resolved with continuous pressure. In about 10% of attempts no pH information is obtained, because of blood clotting within the capillary, insufficient blood obtained, air bubbles inside the capillary, or a blood gas measurer that is calibrating at the time the sample needs to be analysed. The failure rate when lactate analysis is performed is lower, at about 1.5% \(^{13,14}\). This is due to the need of approximately 5 microlitres for the latter, instead of the 50 microlitres required for blood gas assessment \(^{14-16}\).

**Interpretation of results**

In three studies conducted in the 1960s, scalp pH values were evaluated in a total of 180 women with normal CTG tracings \(^{17-19}\). During the first stage of labour the lowest reported values were between 7.18 and 7.21. Based on these data, fetal acidosis during the first stage of labour was defined as a pH<7.20. This was later confirmed in a larger study including 306 fetuses \(^{20}\).

In a large randomized controlled trial (RCT) comparing scalp pH and lactate measurements, the rate of operative deliveries was identical when cut-off values for intervention were set at pH<7.21 and lactate>4.8 mmol/l, and the latter value is commonly used to define the need for intervention \(^{16}\). However, cut-off values for lactate need to consider the apparatus used for measurement, and this value was the only one to have been evaluated in this manner, being established with the Lactate Pro™ meter (Arkray, Kyoto, Japan). Further studies should also consider sub-group analysis to establish cut-off values by gestational age and stage of labour \(^{13}\). The interpretation of pH and lactate values is shown in Table 1.

<table>
<thead>
<tr>
<th>pH</th>
<th>Lactate (mmol/l)</th>
<th>Interpretation</th>
</tr>
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<tbody>
<tr>
<td>&gt; 7.25</td>
<td>&lt; 4.2</td>
<td>Normal</td>
</tr>
<tr>
<td>7.20–7.25</td>
<td>4.2-4.8</td>
<td>Intermediate</td>
</tr>
<tr>
<td>&lt; 7.20</td>
<td>&gt; 4.8</td>
<td>Abnormal</td>
</tr>
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</table>

**Table 1.** Interpretation of FBS results regarding pH and lactate values (adapted from \(^{21}\))

Intervention is indicated in cases of pH<7.20 or lactate>4.8 mmol/l, and this should result in actions towards normalization of the CTG pattern or rapid delivery (see Chapter 3). When the pH is between 7.20 and 7.25, or lactate between 4.2 and 4.8 mmol/l \(^{22}\), measures should be taken to improve fetal oxygenation, and if the CTG abnormality persists or the pattern worsens, FBS should be repeated within 20-30 minutes. With a normal pH or lactate value no further action is usually required, but if the CTG remains grossly abnormal, FBS should be repeated within the next 60
minutes. A normal lactate measurement is strongly predictive of absent hypoxia/acidosis, when performed in the last hour of labour. With a continuously abnormal CTG pattern, even after three or more normal FBSs have been obtained, the fetus can still be safely delivered vaginally in about 60% of cases. When three adequate FBS results have been obtained, consideration of further testing is rarely needed.

**Does FBS improve fetal outcome?**

There is uncertainty on whether the use of FBS as an adjunct to CTG, measuring either pH or lactate, improves neonatal outcome and reduces intervention rates. The first meta-analysis of RCTs comparing continuous CTG with intermittent auscultation for intrapartum fetal monitoring, when analysing the three trials in which FBS was not used as an adjunctive technology, found an almost threfold increase in cesarean section rates in the CTG arm. In the six trials in which FBS was used as an adjunct to CTG (CTG+FBS) the cesarean section rate was only 30% higher than in the intermittent auscultation arm, while neonatal seizures were reduced by 50%. In the only trial in which CTG with and without FBS were directly compared, cesarean section rates were 11 and 18%, respectively, but this difference was not statistically significant. A recent Cochrane review based on seven trials with FBS as an adjunctive technology and five with CTG only, found a RR of 1.34 for cesarean section in the former and of 1.63 in the latter as compared to intermittent auscultation. Vaginal instrumental deliveries were somewhat higher in the CTG+FBS trials and acidosis in cord blood somewhat lower. A systematic review of the studies directly evaluating this technique concluded that, based on heterogeneous data of modest quality with somewhat inconsistent results, CTG+FBS “can provide additional information on fetal wellbeing” and “can reduce the risk of operative delivery.” The National Institute of Clinical Excellence guidelines of 2014 consider that use of FBS “may help to reduce the need for further, more serious interventions.” The guidelines of the Society of Obstetricians and Gynaecologists of Canada recommend FBS in association with CTG for uninterpretable or non-reassuring tracings, but consider the level of evidence to be moderate. Altogether these data suggest that CTG+FBS results in a reduction in cesarean sections when compared to CTG alone. However, more than 50 years after its introduction, a high quality RCT is still needed to evaluate the effect of CTG, with or without FBS on perinatal outcomes and intervention rates.

**Limitations and risks**

FBS use is mainly limited to Central and Northern Europe. The reason for the low global uptake of FBS may include the fact that it is not very patient- or user-friendly. Moreover, it is time-consuming with a median interval of 18 minutes between the decision to perform and the result. This interval is significantly shorter when using point-of-care devices, with a median sampling interval of two minutes for lactate analysis using micro-volume meters. A recent survey from Sweden
concluded that FBS was well tolerated by laboring women, and clinicians did not consider it difficult to perform \(^{31}\). Given the dynamic nature of fetal hypoxia/acidosis during labour, the information provided by FBS quickly becomes outdated, requiring repetitions of the method. It is also difficult to perform in early labour and carries a small risk of infection and bleeding. Moreover it requires laboratory support to evaluate blood gases and lactate, although bedside techniques have largely overcome this \(^{32}\). In the USA, FBS has virtually been abandoned following a paper suggesting that CTG, when properly interpreted, may be equal or superior in the prediction of both normal and adverse outcomes \(^{33}\).

**FETAL SCALP STIMULATION (FSS)**

This technique involves the stimulation of the fetal scalp, by rubbing it with the examiner’s fingers or using a forceps to clasp the fetal skin, or alternatively vibro-acoustic stimulation applied to the maternal abdomen. Digital scalp stimulation is the most widely used, as it is the easiest to perform, less invasive, and appears to have a similar predictive value for fetal hypoxia/acidosis to the other alternatives \(^{34}\). The main purpose of FSS is to evaluate fetuses showing reduced variability on the CTG, in order to distinguish between deep sleep and hypoxia/acidosis. It is of questionable value in other patterns. Observational studies have shown that when FSS leads to the appearance of an acceleration and subsequent normalisation of the fetal heart pattern, this should be regarded as a reassuring feature, with a negative predictive value that is similar to pH > 7.25 on FBS \(^{5,21}\). When FSS does not elicit the appearance of accelerations, or when accelerations occur but continued reduced variability ensues \(^{34}\), the positive predictive value for fetal hypoxia/acidosis is limited. In these situations continued monitoring and additional tests are necessary. It has been reported that, in settings where FBS is used, FSS may reduce its need by about 50% \(^{35}\).

**COMBINED CARDIOTOCOGRAPHIC-ELECTROCARDIOGRAPHIC ST (CTG+ST) MONITORING**

CTG+ST monitoring was commercialized in 2000, and combines continuous internal CTG monitoring with continuous analysis of the fetal electrocardiogram ST segment morphology. The monitor evaluates 30 heart cycles to construct an average electrocardiographic signal that is then used for morphologic analysis of the ST segment (STAN®, Neoventa, Gothemburg, Sweden). Information is obtained on the amplitude of the T-wave in relation to the QRS complex (T/QRS ratio) and on the shape of ST segments, which when showing an important part below the baseline, are named grade 2 and 3 biphasic STs. Extensive animal experiments performed in the 1970s showed that during hypoxia, ST segment changes precede the signs of failing cardiovascular function \(^{36,37}\). The monitor provides automatic warnings called “ST events”, when relevant changes are detected in ST segment
analysis. The theoretical advantages of CTG+ST monitoring over FBS are its less invasive nature, an easier applicability during early labour, and the display of continuous information.

**Indications**

CTG+ST monitoring may be used to provide additional information about cardiac oxygenation in cases of suspicious or pathological CTG tracings (see Chapter 3). When reduced variability and absent accelerations are already present on the CTG, ST information cannot be reliably used to indicate fetal hypoxia/acidosis (see below). With pathological CTGs indicating a severe and acute event (see Chapter 3), immediate action should be undertaken with or without the occurrence of ST events.

**Technique**

A fetal electrode is necessary to acquire continuous CTG+ST signals. Therefore the technique has similar contra-indication to internal CTG monitoring and to FBS (see Chapter 3 or section above on the contra-indications to FBS). The ST technology has not been extensively evaluated in gestational ages below 36 weeks.

**Interpretation of results**

Tracing interpretation needs to take into account the CTG pattern and the degree of ST changes. Specific guidelines were developed for CTG interpretation, inspired by the original FIGO guidelines of 1987, together with specific CTG+ST criteria for taking clinical action 38. The system’s automatic warnings of ‘ST events’ only occur when it detects changes in ECG morphology when compared to a previously existing state, and these changes may not be detectable if ECG morphology is already abnormal at the start of recording. Therefore, a “reactive CTG” (i.e. one showing normal variability and accelerations), or a normal FBS need to be documented at the start of monitoring, for a safe use of ST information. If FBS is not available, conservative measures to improve the CTG pattern can be considered (turning the laboring woman on her side, stopping oxytocin, acute tocolysis, reverting maternal hypotension if this was documented) before starting CTG+ST monitoring.

When the CTG is normal, “ST events” should be ignored, as in this setting they do not indicate fetal hypoxia/acidosis. A few cases have been described in which CTG tracings have gradually changed from normal to pathological, without the appearance of “ST events” 39. For this reason, any abnormal CTG lasting more than 60 minutes, or less if the CTG pattern deteriorates rapidly, requires assessment by a senior obstetrician, whether or not “ST events” occur. With a CTG showing persistently reduced variability or a pattern indicating a severe and acute hypoxic event, intervention is always required irrespective of ST data 38.

**Does CTG+ST monitoring improve fetal outcome?**
Six RCTs were published comparing CTG+ST monitoring with isolated CTG, for a total of more than 26,000 enrolled women. The first trial used an earlier version of the technology, the first five trials were conducted in Europe using FBS as an adjunctive technique, and the most recent trial was performed in the United States, where a simplified 3-tier CTG classification was used and FBS was not available. Several meta-analyses of the first five RCTs have been performed, but doubts remain as to whether the first trial should be included because of the different version of the technology, and whether a more recent study should be included because its entry criteria contradict the established CTG+ST guidelines.

All five European RCTs point to a reduction of FBS use in the CTG+ST arm of about 40%. Newborn metabolic acidosis was significantly lower in the CTG+ST arm in one of the larger trials, a similar trend was observed in two other large studies, and an opposite trend was seen in the two smaller trials. Operative deliveries (instrumental vaginal deliveries + cesarean sections) were significantly lower in the CTG+ST arm in one large study, showed a similar trend in another large study, and showed no difference in the remaining three studies. The 26-center USA trial enrolling 11,108 participants showed no differences in operative delivery or adverse neonatal outcome between the two arms.

A few centers have published data on neonatal outcome in the years following the introduction of the CTG+ST technology together with structured CTG training, reporting progressive declines in the incidence of metabolic acidosis, with stable or decreasing intervention rates. A causal relationship with the CTG+ST technology or with structured CTG training has not been established, but these unique outcomes deserve close attention. The importance of training and of prioritizing of the labour ward may have been underestimated. The ST technique is still relatively new and its guidelines were developed empirically. Further research is needed to evaluate whether changing management guidelines will improve the performance of the technique. Recently it has been suggested that biphasic STs do not add to the diagnostic value of the technique.

**Limitations and risks**

Clinical use of CTG+ST requires a relatively complex educational process. A CTG with normal variability and accelerations or a normal FBS is required at the start of monitoring for a confident evaluation of ST data, but even then hypoxia/acidosis can rarely develop during labour without the occurrence of ST events. Finally, ST events have been reported in about 50% of normally oxygenated fetuses, but only in 16% they were associated with abnormal CTG patterns warranting intervention according to the STAN guidelines.

**COMPUTER ANALYSIS OF FETAL MONITORING SIGNALS**
Computer analysis of CTGs was developed to overcome the poor interobserver agreement on tracing interpretation and to provide an objective evaluation of some CTG features that are difficult to assess visually, such as variability (Chapter 3). Over the last two decades, a small number of systems have been commercialised for computer analysis of intrapartum fetal monitoring signals, all in association with fetal central monitoring stations 58: IntelliSpace Perinatal®, incorporating the former OB TraceVue® (Philips Healthcare®, Eindhoven, Netherlands), Omnvieiw-SisPorto® 59 (Speculum, Lisbon, Portugal), PeriCALMTM 60 (LMS Medical systems, Montreal, Canada and PeriGen, Princeton, USA), INFANT® 61 (K2 Medical SystemsTM, Plymouth, United Kingdom), and Trium CTG Online® (GE Healthcare®, Little Chalfont, United Kingdom and Trium Analysis Online GmbH, Munich, Germany).

These systems incorporate real-time visual and sound alerts for healthcare professionals, based on the results of computer analysis of CTG or combined CTG+ST signals 59. These alerts are aimed at raising attention to specific findings and prompting tracing re-evaluation, with subsequent action if considered necessary. All systems use relatively similar colour-coding of alerts, and they refrain from providing clinical management recommendations. However, different mathematical algorithms are used, and computer analysis is based on different interpretation guidelines.

Published research evaluating these systems is still relatively scarce. Computer analysis has been compared with that of experts, generally yielding satisfactory results 62-66. Comparisons between the systems are difficult, as different numbers of observers and different observer experiences were selected. A small number of studies have evaluated the capacity of computer alerts to predict adverse neonatal outcomes 67-69. The results suggest that it is possible to achieve a good prediction of newborn acidemia with computer analysis of CTG tracings acquired shortly before birth. Again, comparisons between studies are hampered by different case selection criteria, and different choices of adverse neonatal outcome. Studies with larger sample sizes and direct comparisons of the different systems are lacking. Two of these systems have recently completed multicentre RCTs comparing them with standard CTG analysis 70,71, and their results are expected soon.

Computer analysis of intrapartum fetal monitoring signals is therefore a relatively new but promising technology, as optimization of the analysis algorithms will most likely continue. Currently, this technology should be used with caution, since further research is necessary to evaluate its capacity to detect fetal hypoxia/acidosis, and to prevent adverse outcomes.

**Conclusions**

There is still a lot of uncertainty regarding the use of the different adjunctive technologies in intrapartum fetal monitoring. FSS is easy to perform and can be useful when reduced variability is the main CTG feature, as the appearance of accelerations and a change to a normal pattern is very predictive of absent hypoxia/acidosis. However, the benefits of this technique have not been evaluated in randomised trials, so little is known about how it affects neonatal outcome or intervention rates. FBS may reduce the incidence of operative deliveries, although the level of evidence for this is
moderate, and there is no evidence that fetal outcomes are improved. CTG+ST monitoring results in a lower need for FBS and perhaps in a modest reduction in operative deliveries. There is conflicting evidence as to whether it improves perinatal outcome. Computer analysis provides a reproducible and quantifiable approach to CTG and CTG+ST interpretation. It is a promising method to evaluate how different features/patterns relate with fetal outcome and perhaps to prompt healthcare professionals to act upon certain findings. Further studies are needed to compare the different computer systems and to evaluate how this technology affects intervention and adverse outcome rates.

Some experts consider that a better understanding of the pathophysiology of the fetal response to reduced oxygenation during labour is the main requisite for intrapartum fetal monitoring, and when repetitive decelerations are present, the presence of a stable baseline and normal variability obviates the need for adjunctive technologies and reduces the false positive rate of CTGs. However, adjunctive technologies will still need to be considered in the remaining cases.

Further research and development is needed in this field, to remove the uncertainty that surrounds many of these adjunctive technologies and to provide more robust evidence on how they affect intervention and adverse outcome rates.

Conflicts of interest
Diogo Ayres-de-Campos and João Bernardes are co-developers of the Omniview-SisPorto system. They do not receive funding from commercialisation of the program, but the University of Porto receives royalties which are totally re-invested in research. Lawrence Devoe is a consultant for Neoventa Medical (Molndal, Sweden). Joscha Reinhard has received funding from Monica Healthcare Ltd (Nottingham, UK) for conduction of research on non-invasive electrocardiographic monitoring. Austin Ugwumadu has received honorarium from Neoventa for delivering lectures on fetal monitoring.

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