i An update to this article is included at the end

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FIGO GUIDELINES

FIGO consensus guidelines on intrapartum fetal monitoring: Cardiotocography☆,★

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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 labor, 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 delivery). 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 labor.

2. Indications

The evidence for the benefits of continuous CTG monitoring, as compared with intermittent auscultation, in both low- and high-risk labors is scientifically inconclusive [1,2]. When compared with 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 in 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 hemorrhage 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 labor. 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 [4]. 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 labor ward has been associated with an increase in cesarean delivery rates and no improvement in perinatal outcomes [5], 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

3.1. Maternal position for CTG acquisition

Maternal supine recumbent position can result in aortocaval 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 [6].

CTG acquisition can be performed by portable sensors that transmit signals wirelessly to a remote fetal monitor (telemetry). This solution

☆ Developed by the FIGO Safe Motherhood and Newborn Health Committee; coordinated by Diogo Ayres-de-Campos.

★ The views expressed in this document reflect the opinion of the individuals and not necessarily those of the institutions that they represent.

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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 [7].

3.2. 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 papers 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 center should be the ones 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).

3.3. 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 [8]. 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) [9], and to signal artefacts such as double-counting (Fig. 2) and half-counting [8], particularly during the second stage of labor. It may also not record fetal cardiac arrhythmias accurately.

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 depolarization 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 contraindications, 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 HIV [10,11], 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 of 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 labor, that in all atypical FHR tracings maternal heart rate monitoring is ruled out (see below), and if any doubt remains, fetal auscultation,

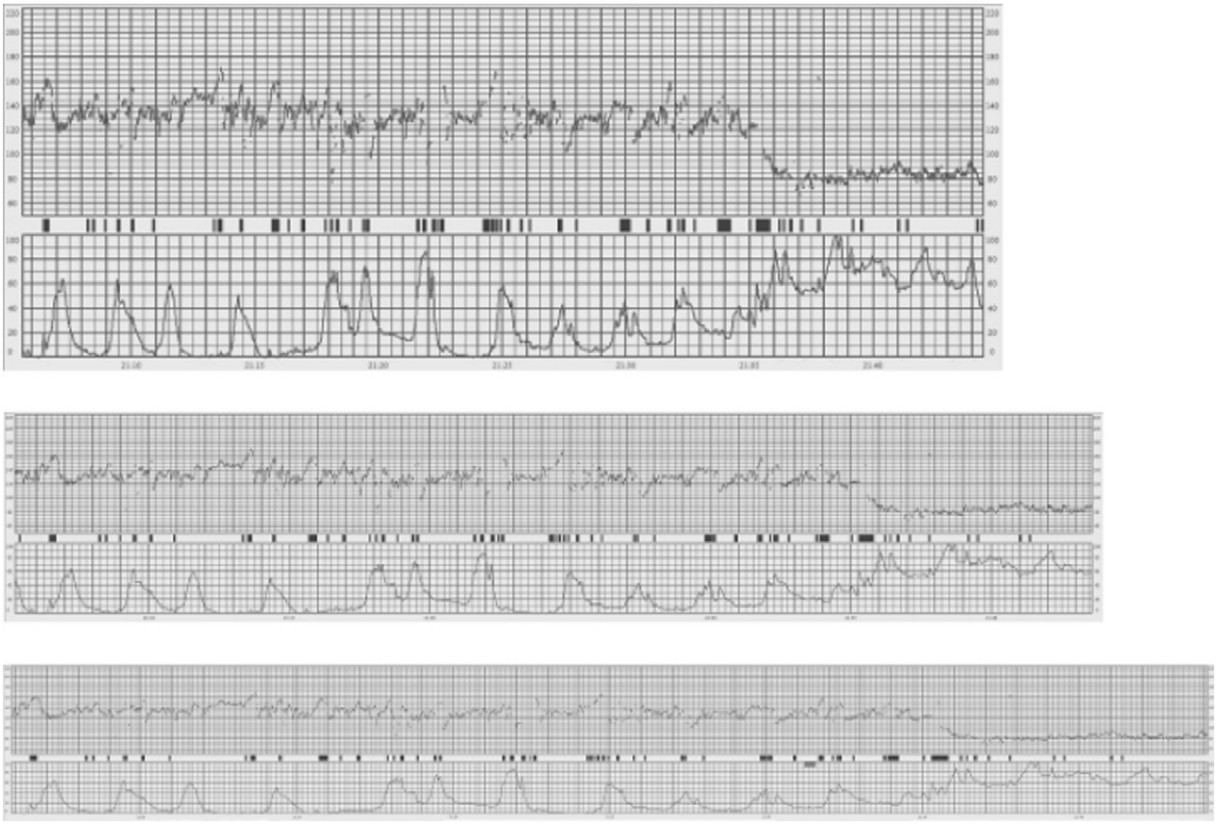


Fig 1. Maternal heart rate monitoring in the last 9 minutes of the tracing. External fetal heart rate monitoring at 1 cm/min (top graph), 2 cm/min (middle graph), and 3 cm/min (bottom graph).

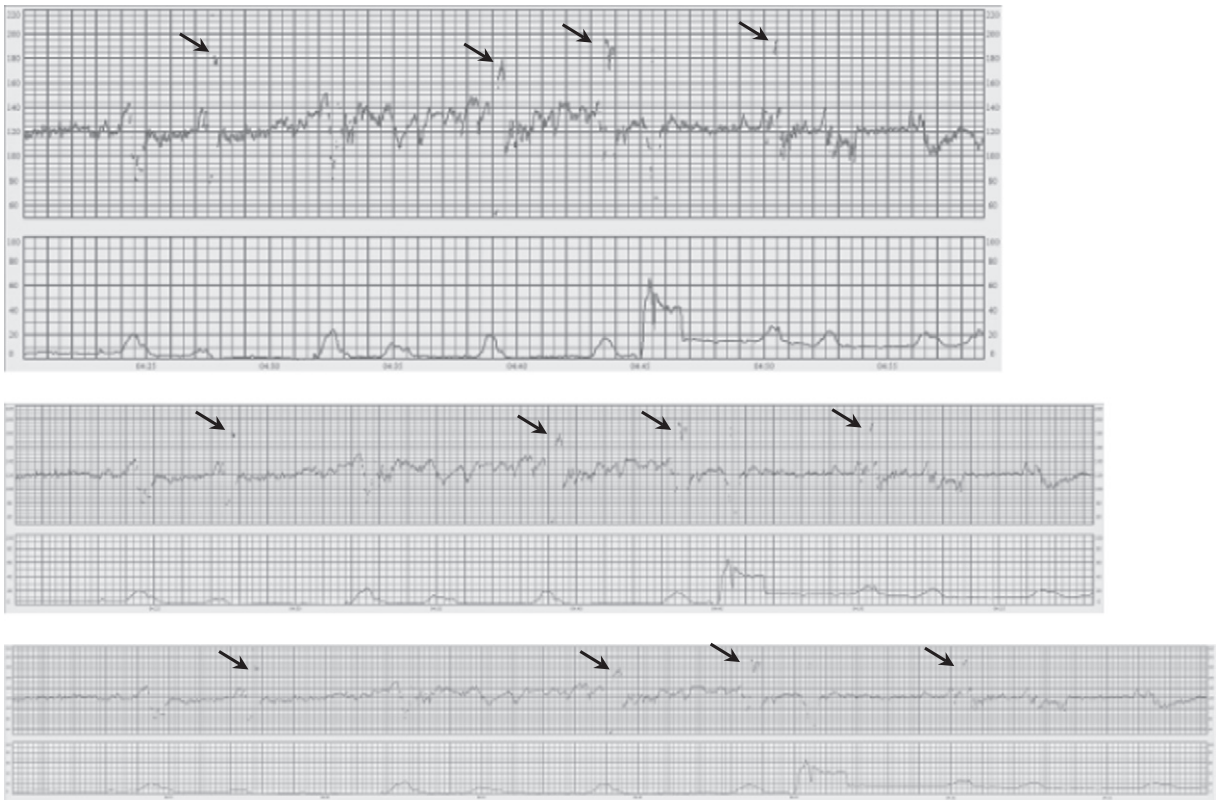


Fig 2. Double-counting of the fetal heart rate during decelerations (arrows). External fetal heart rate monitoring at 1 cm/min (top graph), 2 cm/min (middle graph), and 3 cm/min (bottom graph).

ultrasound evaluation, or internal FHR monitoring are performed. If an acceptable record cannot be obtained with external monitoring or if a cardiac arrhythmia is suspected, then internal monitoring should be used, in the absence of the previously mentioned contraindications.

3.4. 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. Contraindications include uterine hemorrhage of unknown cause and placenta previa. It may also be associated with a small risk of fetal injury, placental hemorrhage, uterine perforation, and infection [12]. The routine use of intrauterine pressure catheters has not been shown to be associated with improved outcomes in induced and augmented labor [13], and so it is not recommended for routine clinical use.

3.5. 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) [9]. 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 labor, when tracings show accelerations coinciding with contractions and expulsive efforts [9], or when the MHR is elevated.

3.6. Monitoring of twins

Continuous external FHR monitoring of twin gestations during labor 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 labor, 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 [14], 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.

3.7. Storage of tracings

All CTG tracings need to be identified with the patient name, place of recording, “paper speed,” and 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 [15], 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.

4.1. Evaluation of basic CTG features

4.1.1. 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 [16], in particular during the second stage of labor and to identify the fetal behavioral state of active wakefulness (Fig. 3) that can lead to erroneously high baseline estimation.

Normal baseline: a value between 110 and 160 bpm.
Preterm fetuses tend to have values toward 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 extrauterine origin or associated with intrauterine infection. Epidural analgesia may also cause a rise in maternal temperature resulting in fetal tachycardia [17]. 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 [18] (salbutamol, terbutaline, ritodrine, fenoterol), parasympathetic blockers (atropine, scopolamine), 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 [19], administration of beta-blockers [20], and fetal arrhythmias such as atrioventricular block are other possible causes.

4.1.2. Variability

This refers to the oscillations in the FHR signal, evaluated as the average bandwidth amplitude of the signal in 1-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 and 5), or for more than 3 minutes during decelerations [22] (see Figs. 8 and 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 labor without preceding or concomitant decelerations and a rise in the baseline.



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

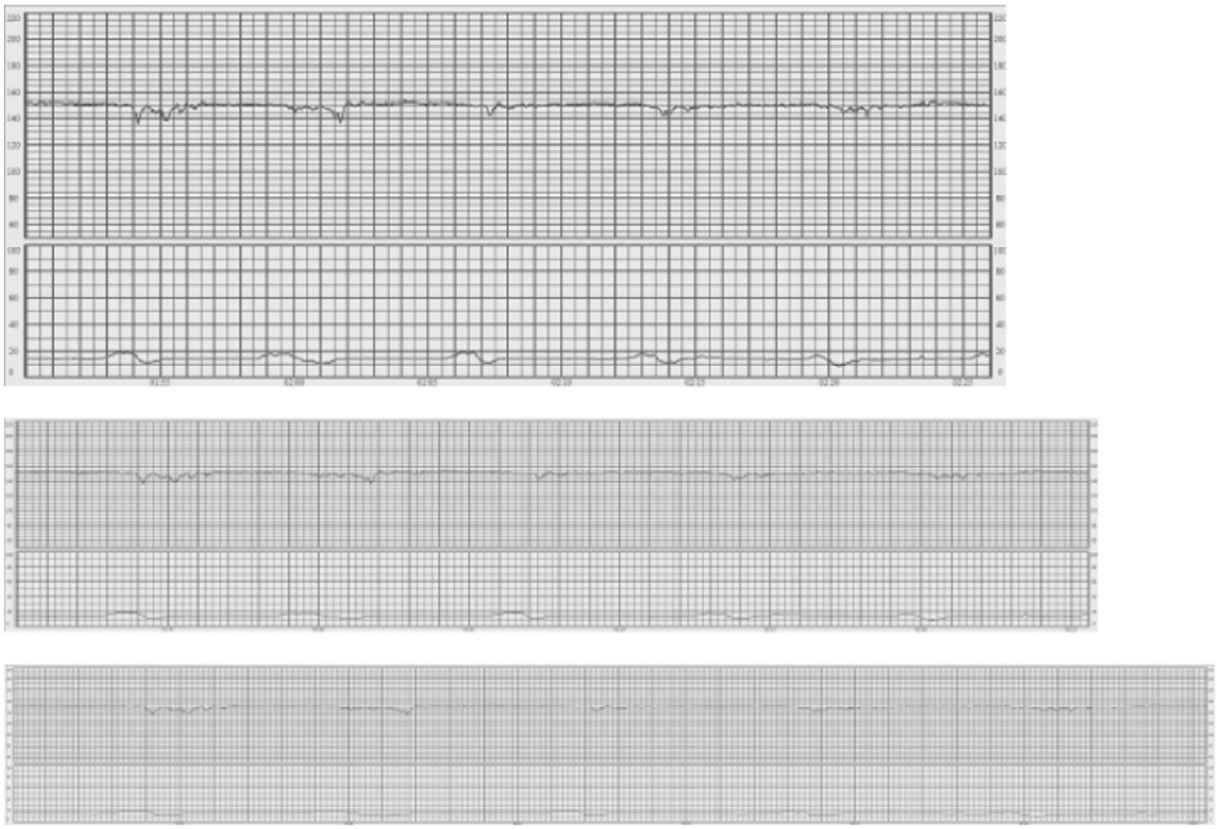


Fig 4. Reduced variability. External fetal heart rate monitoring at 1 cm/min (top graph), 2 cm/min (middle graph), and 3 cm/min (bottom graph).



Fig 5. Reduced variability. The baseline is affected by contractions causing decreases in fetal heart rate that are close to fulfilling the criteria for decelerations, but the bandwidth remains reduced. Internal fetal heart rate 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].

4.1.3. 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 of gestation, their amplitude and frequency may be lower (10 seconds and 10 bpm of amplitude). After 32—34 weeks, with the establishment of fetal behavioral 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 labor, 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].

4.1.4. 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 [25] 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 labor, and they translate a baroreceptor-mediated response to increased arterial pressure, as occurs with umbilical cord compression [26]. 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 [22,27] (see prolonged decelerations below).

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, have 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 [25,27]. 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.

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 at less than 80 bpm and reduced variability within the deceleration (Fig. 9), are frequently associated with acute fetal hypoxia/acidosis [22,28–30] and require emergent intervention.

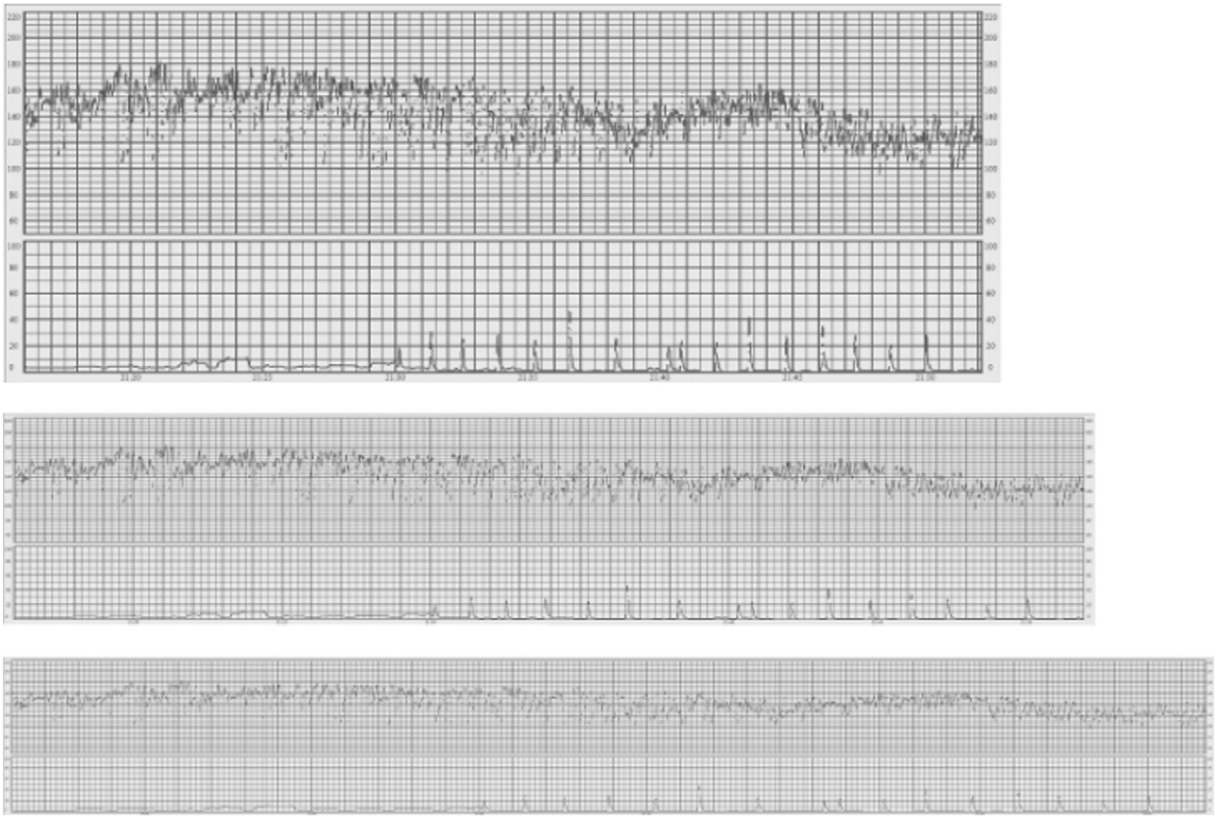


Fig 6. Increased variability: saltatory pattern. Internal fetal heart rate monitoring at 1 cm/min (top graph), 2 cm/min (middle graph), and 3 cm/min (bottom graph).



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

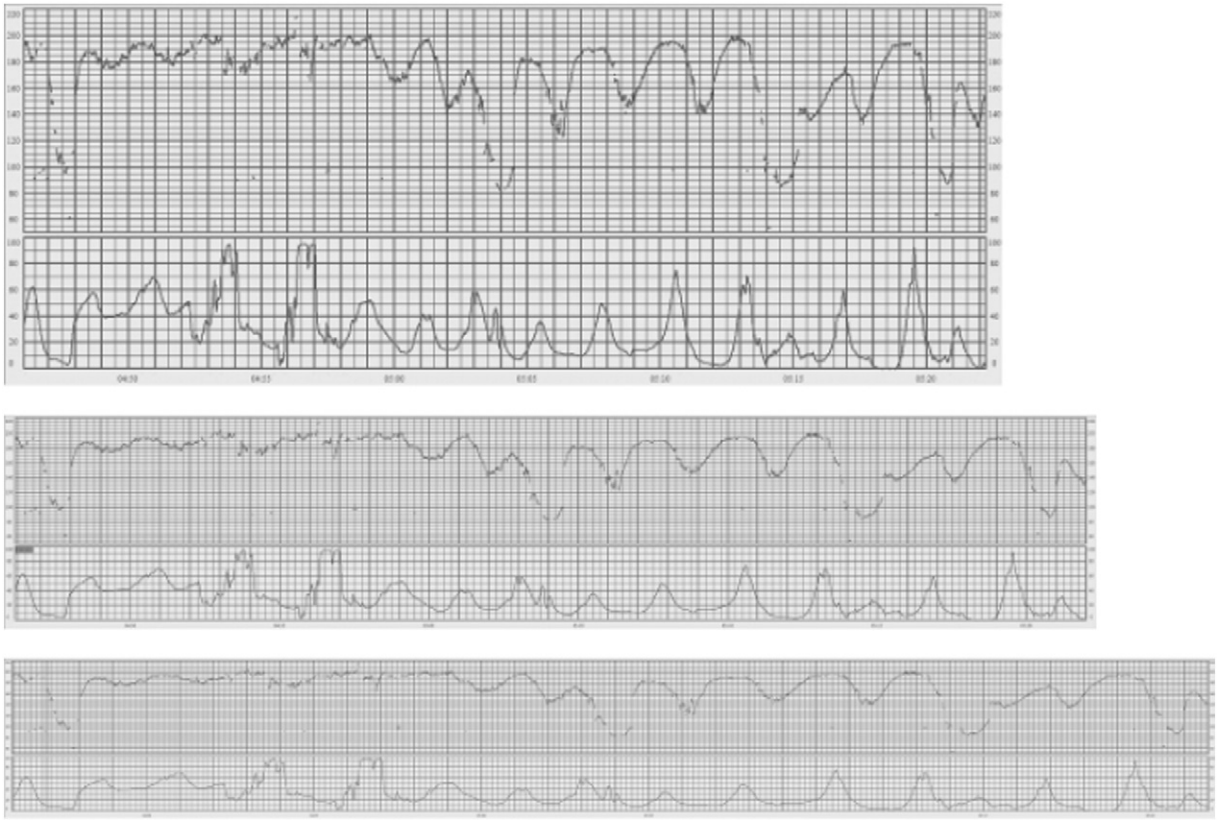


Fig 8. Late decelerations in the second half of the tracing. External fetal heart rate monitoring at 1 cm/min (top graph), 2 cm/min (middle graph), and 3 cm/min (bottom graph).

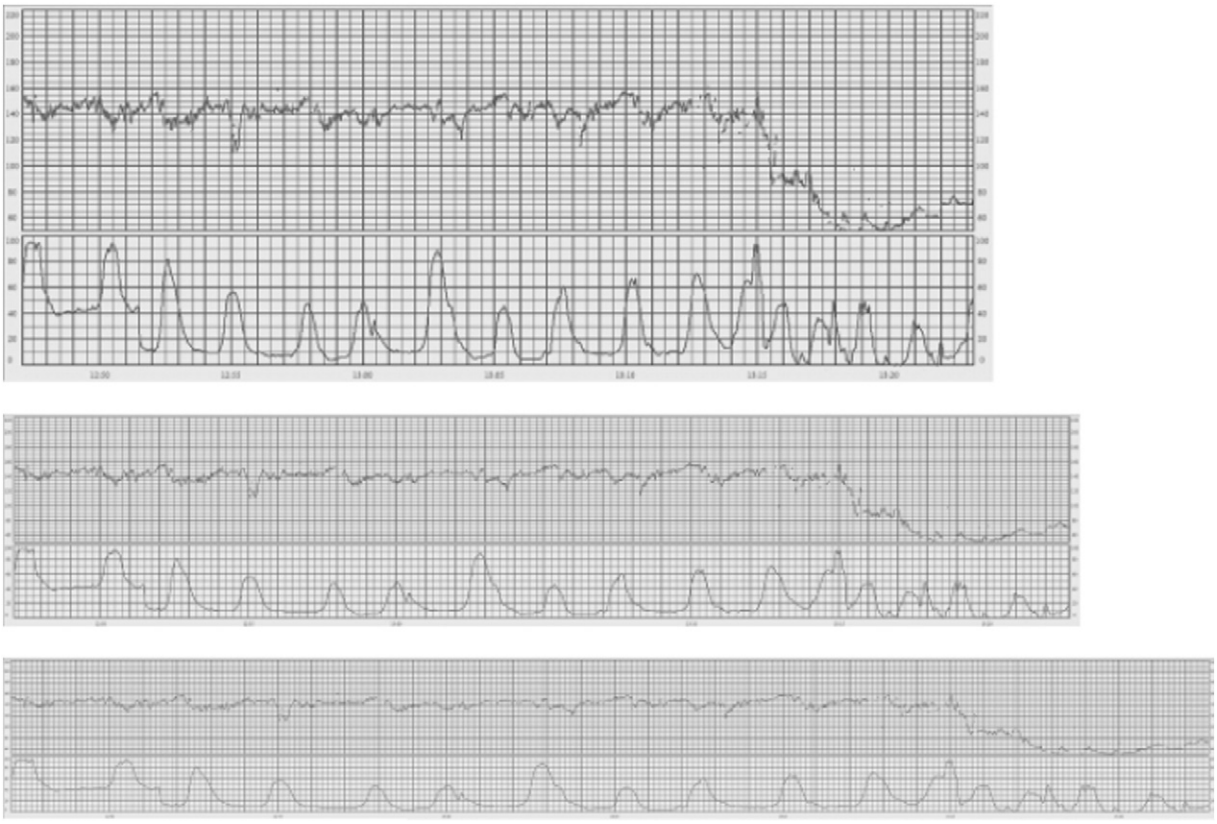


Fig 9. Prolonged deceleration. External fetal heart rate monitoring at 1 cm/min (top graph), 2 cm/min (middle graph), and 3 cm/min (bottom graph).

4.1.5. 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 alloimmunization, fetal-maternal hemorrhage, twin-to-twin transfusion syndrome, and ruptured vasa previa. It has also been described in cases of acute fetal hypoxia, infection, cardiac malformations, hydrocephalus, and gastroschisis [31].

4.1.6. Pseudosinusoidal 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 characterized 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 pseudosinusoidal pattern from the true sinusoidal pattern, leaving the short duration of the former as the most important variable to discriminate between the two.

4.1.7. Fetal behavioral states

This refers to periods of fetal quiescence reflecting deep sleep (no eye movements), alternating with periods of active sleep (rapid eye movements) and wakefulness [33,34]. The occurrence of different behavioral states is a hallmark of fetal neurological responsiveness and absence of hypoxia/acidosis. Deep sleep can last up to 50 minutes [21] and is associated with a stable baseline, very rare accelerations, and borderline variability. Active sleep is the most frequent behavioral 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). Transitions between the different patterns become clearer after 32–34 weeks of gestation, consequent to fetal nervous system maturation.

4.1.8. 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 labor, but they compress the vessels running inside the myometrium and may transiently decrease placental perfusion and/or cause umbilical cord compression [35]. With the tocodynamometer, only the frequency of contractions can be reliably evaluated, but increased intensity and duration can also contribute to FHR changes.

Tachysystole: This represents an excessive frequency of contractions and is defined as the occurrence of more than five 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 [36,37]. Due to the changing nature of CTG signals during labor, re-evaluation of the tracing should be carried out at least every 30 minutes.



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



Fig 11. Pseudosinusoidal pattern. External fetal heart rate monitoring at 1 cm/min (top graph), 2 cm/min (middle graph), and 3 cm/min (bottom graph).

Table 1
Cardiotocography classification criteria, interpretation, and recommended management.[a]

	Normal	Suspicious	Pathological
Baseline	110–160 bpm	Lacking at least one characteristic of normality, but with no pathological features	<100 bpm
Variability	5–25 bpm	Lacking at least one characteristic of normality, but with no pathological features	Reduced variability, increased variability, or sinusoidal pattern
Decelerations	No repetitive[b] decelerations	Lacking at least one characteristic of normality, but with no pathological features	Repetitive[b] late or prolonged decelerations during >30 min or 20 min if reduced variability, or one prolonged deceleration with >5 min
Interpretation	Fetus with no hypoxia/acidosis	Fetus with a low probability of having hypoxia/acidosis	Fetus with a high probability of having hypoxia/acidosis
Clinical management	No intervention necessary to improve fetal oxygenation state	Action to correct reversible causes if identified, close monitoring or additional methods to evaluate fetal oxygenation [49]	Immediate action to correct reversible causes, additional methods to evaluate fetal oxygenation [49], or if this is not possible expedite delivery. In acute situations (cord prolapse, uterine rupture, or placental abruption) immediate delivery should be accomplished.

[a] The presence of accelerations denotes a fetus that does not have hypoxia/acidosis, but their absence during labor is of uncertain significance.
[b] Decelerations are repetitive in nature when they are associated with more than 50% of uterine contractions [29].

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 delivery 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 [35] 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) [38–40], atosiban [41], or nitroglycerine [42]. During the second stage of labor, maternal pushing efforts can also contribute to fetal hypoxia/acidosis and the mother can be asked to stop pushing until the situation is reversed.

Aortocaval compression can occur in the supine position and lead to reduced placental perfusion. Excessive uterine activity may also be associated with the supine position [43,44], 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 amnioinfusion [45].

Sudden maternal hypotension can also occur during labor, usually after epidural or spinal analgesia [46], 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 [35], 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 normalizing CTG patterns, but there is no evidence from randomized clinical trials that this intervention, when performed in isolation, is effective when maternal oxygenation is adequate [47]. Intravenous fluids are also commonly used for the purpose of improving CTG patterns, but again there is no evidence from randomized clinical trials to suggest that this intervention is effective in normotensive women [48].

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 [49]. 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 labor, 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 safe use of the technology.

It has been well demonstrated that CTG analysis is subject to considerable intra- and interobserver disagreement, even when experienced clinicians use widely accepted guidelines [50–52]. The main aspects that are prone to observer disagreement are the identification and classification of decelerations, the evaluation of variability [51], and the classification of tracings as suspicious and pathological [51,52]. 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 [53].

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 [54]. However, it is recognized 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 [54]. 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 randomized controlled trials have been conducted comparing continuous CTG monitoring with intermittent auscultation as screening methods for fetal hypoxia/acidosis during labor, in both low- and high-risk women [1,2]. 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 labor, 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 recognized that the trials were underpowered to detect differences in these outcomes [3]. Only a small proportion of perinatal deaths and cerebral palsies are caused by intrapartum hypoxia/acidosis, so a large number of cases are 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 [1].

Unnecessary obstetric intervention confers additional risks for the mother and newborn [55,56], and the former may result from poor CTG interpretation, limited knowledge of the pathophysiology of fetal oxygenation, and inadequate clinical management. It is recognized 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 labor ward staff is essential to ensure proper use of this technology.

Conflict of interest

The authors have no conflicts of interest to declare.

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The authors regret that the name of the contributor T. Alhaidari previously appeared incorrectly. Additionally, the middle initial of the contributor F.G. Mirza was missing. The correct listing is as above.

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