Pathophysiological interpretation of fetal heart rate tracings in clinical practice

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The onset of regular, strong, and progressive uterine contractions may result in both mechanical (compression of the fetal head and/or umbilical cord) and hypoxic (repetitive and sustained compression of the umbilical cord or reduction in uteroplacental oxygenation) stresses to a human fetus. Most fetuses are able to mount effective compensatory responses to avoid hypoxic-ischemic encephalopathy and perinatal death secondary to the onset of anaerobic metabolism within the myocardium, culminating in myocardial lactic acidosis. In addition, the presence of fetal hemoglobin, which has a higher affinity for oxygen even at low partial pressures of oxygen than the adult hemoglobin, especially increased amounts of fetal hemoglobin (ie, 180—220 g/L in fetuses vs 110—140 g/L in adults), helps the fetus to withstand hypoxic stresses during labor.

Different national and international guidelines are currently being used for intrapartum fetal heart rate interpretation. These traditional classification systems for fetal heart rate interpretation during labor are based on grouping certain features of fetal heart rate (ie, baseline fetal heart rate, baseline variability, accelerations, and decelerations) into different categories (eg, category I, II, and III tracings, "normal, suspicious, and pathologic" or "normal, intermediary, and abnormal"). These guidelines differ from each other because of the features included within different categories and because of their arbitrary time limits stipulated for each feature to warrant an obstetrical intervention. This approach fails to individualize care because the "ranges of normality" for stipulated parameters apply to the population of human fetuses and not to the individual fetus in question. Moreover, different fetuses have different reserves and compensatory responses and different intrauterine environments (presence of meconium staining of amniotic fluid, intrauterine inflammation, and the nature of uterine activity).

Pathophysiological interpretation of fetal heart rate tracing is based on the application of the knowledge of fetal responses to intrapartum mechanical and/or hypoxic stress in clinical practice. Both experimental animal studies and observational human studies suggest that, just like adults undertaking a treadmill exercise, human fetuses show predictable compensatory responses to a progressively evolving intrapartum hypoxic stress. These responses include the onset of decelerations to reduce myocardial workload and preserve aerobic metabolism, loss of accelerations to abolish nonessential somatic body movements, and catecholamine-mediated increases in the baseline fetal heart rate and effective redistribution and centralization to protect the fetal central organs (ie, the heart, brain, and adrenal glands), which are essential for intrauterine survival. Moreover, it is essential to incorporate the clinical context (progress of labor, fetal size and reserves, presence of meconium staining of amniotic fluid and intrauterine inflammation, and fetal anemia) and understand the features suggestive of fetal compromise in nonhypoxic pathways (eg, chorioamnionitis and fetomaternal hemorrhage). It is important to appreciate that the timely recognition of the speed of onset of intrapartum hypoxia (ie, acute, subacute, and gradually evolving) and preexisting uteroplacental insufficiency (ie, chronic hypoxia) on fetal heart rate tracing is crucial to improve perinatal outcomes.

Key words: acute hypoxia, baseline fetal heart rate, cardiotocography, catecholamine response, chorioamnionitis, decelerations, fetal heart rate variability, fetal heart trace tracing, gradually evolving hypoxia, redistribution, subacute hypoxia

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All authors were part of the editorial board and international consensus panel, which produced the first International Consensus Guidelines on Physiological Interpretation of Cardiotocography (CTG). E.C. conducts several master classes on physiological interpretation of CTG and fetal electrocardiogram (ECG) in the United Kingdom, Europe, Asia, and Australia and has been the co-organizer of the intrapartum fetal surveillance course at the Royal College of Obstetricians and Gynaecologists, and he was a member of the editorial board for the National Health Service e-learning on CTG. He was the course lead for the Baby Lifeline CTG master classes. Several organizers and hospitals of some of these master classes have received sponsorships from Philips, Neoventa, Euroking, Huntleigh, K2, Cardiac Services, and other industries to support these master classes. However, E.C. does not have any financial or managerial interests in any pharmaceutical or medical device industry. Moreover, E.C. was 1 of 3 members of the guideline development group that revised the International Federation of Gynecology and Obstetrics guidelines on CTG in 2015.

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Introduction

The journey of human labor involves the progressive increase in the frequency, duration, and strength of uterine contractions, which may lead to repetitive compression of the umbilical cord, fetal head, and maternal spiral arteries with intermittent reduction of oxygenation within the intervillous spaces. Caldeyro-Barcia et al[1] recognized the importance of uterine contractions and oxytocin on fetal well-being even before the introduction of electronic fetal heart rate (FHR) monitoring into routine clinical practice.[1–3] The journey of human labor is similar to an adult running an extended marathon for approximately 8 to 10 hours, but without the luxury of being exposed to atmospheric oxygen. Unlike an adult undertaking an anaerobic exercise, the human fetus is unable to meet his or her oxygen requirements intake by increasing the rate or the depth of respiration. To cope with the increasing hypoxic and mechanical stresses as a result of progressively worsening uterine contractions without sustaining a hypoxic-ischemic injury, the human fetus has developed several compensatory mechanisms to adapt to such an intrauterine environment devoid of atmospheric oxygen (Table 1).

Most human fetuses are able to withstand progressive worsening intrapartum hypoxic stress by mounting effective compensatory responses, similar to tachycardia and tachypnea by adults during anaerobic activities, such as running or cycling. In addition, fetuses have a significant extra placental reserve of approximately 40% to 50% to enable them to withstand heightened hypoxic stress with the onset of active maternal pushing during the second stage of labor. Therefore, they avoid hypoxic-ischemic encephalopathy (HIE) or a perinatal death during labor. However, a small minority of human fetuses may present with preexisting (eg, a chronic uteroplacental insufficiency) or coexisting (eg, chorioamnionitis) compromise, and they may not be able to withstand additional hypoxic and mechanical stresses during labor for prolonged periods. Approximately 60 years ago, Edward Hon published his observations on the FHR patterns that preceded fetal death.[4,5] However, it is important to appreciate that some fetuses may have reduced uteroplacental reserve (eg, late-onset placental failure leading to a relative uteroplacental insufficiency), and others may experience an adverse intrauterine (eg, meconium staining of amniotic fluid or intrauterine infection) or maternal (eg, sepsis, diabetic ketoacidosis, or sickle cell anemia) environment. In these situations, the onset of fetal stress to injury (or death) interval may be much shorter. Rarely, an acute intrapartum accident (eg, placental abruption, umbilical cord prolapse, or uterine rupture) may result in an acute and profound hypoxic stress with limited time to mount an effective compensatory response resulting in an increased risk of HIE or perinatal death.

Several classification systems are attempting to classify cardiotocography (CTG) traces into "category I, category II, and category III" (United States),[6] "normal, suspicious, and pathological" (National Institute for Health and Care Excellence [NICE][7] and International Federation of Gynecology and Obstetrics [FIGO][8]), "normal, intermediary, and abnormal" (ST analysis [STAN]),[9] and a 5-tier classification system,[10] in addition to several other guidelines produced by national societies and academic bodies. The individual parameters included within each classification system into different categories and the duration at which these parameters become considerable, meriting an intervention, are different. In contrast, the adult electrocardiogram (ECG) interpretation does not have such variation in interpretation worldwide. This illustrates the ongoing confusion concerning CTG interpretation because of overreliance on the classification of decelerations into different categories based on their morphology to determine the severity of intrapartum hypoxic stress, without considering the features suggestive of central organ oxygenation on the CTG trace. Several authors have recently questioned the usefulness of continued use of electronic FHR monitoring in clinical practice because of the increasing intrapartum operative interventions without any demonstrable benefit of improving long-term perinatal outcomes.[11–14] Therefore, there is an urgent need to move away from "pattern recognition"-based CTG interpretation to fetal physiology-based interpretation. This allows to reduce inter- and intraobserver variability and improve perinatal outcomes while reducing unnecessary intrapartum operative interventions, which may cause harm to the mother.

Interobserver variability and evidence regarding the effectiveness of current cardiotocography guidelines

Intrapartum monitoring using CTG was introduced in the late 1960s to identify inadequate oxygenation of the fetus and prevent hypoxic damage occurring during labor and it has now become the mainstay for intrapartum monitoring of fetal well-being.

CTG interpretation has historically relied on the interpretation of the morphology of the FHR pattern and particularly on the assessment of the baseline, variability, and decelerations, that is, parameters that have been reported to be abnormal in the event of fetal asphyxia.[8,15,16] However, such an approach has led to the classification as abnormal (hence suggestive of fetal hypoxia) all CTG traces whose features are not consistent with those commonly seen in nonhypoxic fetuses, with no attempt to understand the pathophysiology underlying the different types of CTG patterns. This has led to a dramatic increase in the incidence of intrapartum obstetrical interventions and particularly of cesarean deliveries. Unfortunately, this increase in operative interventions has not been associated with a concomitant decreased occurrence of adverse perinatal outcomes, including cerebral palsy and death.[6,8] One of the earliest studies conducted on 279 high-risk pregnancies subjected to continuous CTG monitoring during the first stage of labor demonstrated a low incidence of

TABLE 1
Intrauterine adaptation and fetal compensatory mechanisms

Mechanisms
• Development of "large placental lakes" as a result of trophoblastic invasion of the tunica media of the spiral arterioles.
• "Increased hemoglobin concentration" (18.0−22.0 g/dL) to enable greater oxygen binding and transport.
• Replacement of the β-chains of the adult hemoglobin with γ-chains to form the "fetal hemoglobin," which has a great affinity for oxygen and, therefore, retains oxygen even at very low partial pressures of oxygen within the intrauterine environment.
• "Increasing baseline fetal heart rate" of 110−160 bpm to obtain adequate oxygen from the placenta to ensure optimum organ perfusion while immersed in a pool of amniotic fluid with limited oxygen exposure.
• A series of "cardioprotective reflexes" (decelerations) to withstand hypoxic and mechanical stresses during labor.
• More importantly, the human fetus is equipped with an efficient "catecholamine-mediated response" to increase the cardiac output and to ensure a rapid and effective redistribution of oxygenated blood from "nonessential" organs to essential central organs (ie, the brain, heart, and adrenal glands), thus avoiding hypoxic-ischemic injury to central organs.

Jia. Fetal heart rate tracing interpretation in clinical practice. Am J Obstet Gynecol 2023.

acidemia and a fetal pH of >7.25 in more than 60% of fetuses showing pathological CTG features, such as bradycardia and decelerations.[17] This demonstrates that many of the abnormalities of the CTG pattern, and the related obstetrical interventions performed because of suspected intrapartum distress, are not associated with fetal or neonatal acidemia. More recent data suggest that more than 95% of the CTG traces classified as pathological are not associated with cerebral palsy,[16,18] and the positive predictive value of a pathological CTG is lower than the estimated prevalence of cerebral palsy in the general pediatric population (1−2 per 1000 neonates).

Does the routine use of electronic fetal heart rate monitoring improve perinatal outcomes?

Although a meta-analysis by Vintzileos et al[19] demonstrated a reduction in intrapartum stillbirth with the use of electronic FHR monitoring in 1995, subsequent studies evaluating the role of intrapartum CTG in the prediction of fetal acidemia and prevention of the hypoxic damage of the fetus have consistently demonstrated a low accuracy of the test, regardless of the classification adopted for the interpretation of the FHR pattern. The available evidence, including data from a Cochrane review, supports the use of continuous CTG monitoring during labor only in pregnancies at high risk of intrapartum fetal hypoxia, but not in those identified as low risk.[8,20,21] More specifically, the use of continuous CTG in the general population has not been found to improve neonatal outcomes in terms of acidemia at birth, Apgar score of <4 at 5 minutes, neonatal intensive care unit admission, HIE, death, and cerebral palsy compared with the use of intermittent auscultation.[20] Here, a 50% reduction of neonatal seizures was reported; however, this was not associated with improved long-term neurologic outcomes. In contrast, continuous CTG monitoring was associated with 15% and 63% increases in the occurrence of instrumental and cesarean deliveries, respectively.[22]

The impact of interobserver and intraobserver variation in interpretation of fetal heart rate tracings

CTG misinterpretation has been advocated as the major determinant of the increased frequency of obstetrical intervention during labor. This can be accounted for by the fact that the interpretation of the CTG pattern is highly subjective, irrespective of the guideline adopted,[23−25] even though an improved agreement has been suggested with the use of the FIGO 2015 classification.[24] Several studies evaluating the intra- and interoperator reproducibility of the CTG analysis have demonstrated a high variability,[26−28] irrespective of the level of experience.[23]

A study involving 3 experienced obstetricians demonstrated a fair interobserver agreement (proportions of agreement [*pa*], 0.56−0.71) in the event of normal CTG features according to the FIGO classifications. However, the agreement in the event of abnormalities of the FHR pattern was shown to be exceedingly poor (*pa*, 0.14−0.45).[29] Concerning the variability in the interpretation of the FHR parameters, a study by Schiermeier et al[30] demonstrated that only the baseline FHR showed a fair agreement (*pa*, 0.49−1.01) among 43 different obstetricians. This has been corroborated by similar studies involving experts.[31]

Furthermore, the knowledge of the neonatal outcome has been demonstrated to impact the CTG interpretation.[32−35] Several authors demonstrated a poor intraobserver reproducibility of the FHR pattern evaluated before and after the ascertainment of the delivery outcome, with the only exception of the baseline FHR.[34] The 2009 guidelines on fetal intrapartum monitoring issued by the American College of Obstetricians and Gynecologists (ACOG) confirmed the high intra- and interoperator variability in the interpretation of the CTG pattern and concluded that "reinterpretation of the FHR tracing, especially if the neonatal outcome is known, may not be reliable" (level of evidence B).[6]

Does the use of computerized analysis of fetal heart rate tracings during labor improve perinatal outcomes?

The computerized analysis of the different components of the FHR has been proposed to reduce the variability in the interpretation of the CTG. However, available evidence has failed to demonstrate any difference in the identification of adverse perinatal and long-term outcomes vs conventional intrapartum CTG (FHR tracing) monitoring analysis.[36]

Lack of scientific evidence of benefit for continuous electronic fetal heart rate monitoring during labor: what is the problem?

Based on the current scientific evidence, the routine adoption of the CTG for the monitoring of fetal well-being during labor is debatable. Nonetheless, a critical appraisal of the published literature suggests that no trial or review is adequately powered to demonstrate the beneficial role of continuous CTG in the prevention of perinatal death. Looking at the most recent studies included in the systematic review published by the Cochrane Library,[22] increased sensitivity (97% vs 34%) and positive predictive value (37% vs 22%) for neonatal acidemia of continuous CTG compared with intermittent auscultation has been demonstrated.[37] In addition, continuous CTG may assist in identifying the timing of the hypoxic injury as a systematic review of the CTG traces of the neonates with confirmed brain damage showed that most fetuses demonstrated an abnormal CTG pattern before the onset of labor.[16–18,38,39] In such cases, the CTG cannot prevent the preexisting damage but can only prevent further hypoxic injury from occurring during labor. Moreover, a normal umbilical cord pH in the context of an abnormal CTG trace can be accounted as a false positive in a scientific study. However, it is unknown whether the obstetrical intervention had prevented the occurrence of adverse outcomes resulting in a normal umbilical cord pH.[37] Finally, it has to be acknowledged that, to date, no randomized trial has compared CTG with no monitoring concerning labor outcomes.

Although CTG is currently widely used for the intrapartum monitoring of fetal well-being, available evidence from randomized and clinical trials does not support its role in improving perinatal outcomes.[37] Therefore, the absence of evidence supporting the widespread use of the CTG is more likely to be dependent on studies that have failed to demonstrate its clinical benefits. Nonetheless, epidemiologic data have shown a decrease in the incidence of intrapartum deaths after the clinical implementation of the test.[37] Intra- and interobserver variability of the CTG interpretation are likely to be the main determinants of the poor clinical performance of the test in terms of increased incidence of obstetrical intervention, which is not concomitant to a reduction of the adverse perinatal outcomes. Overreliance on a morphologic classification of decelerations without considering fetal physiological responses to intrapartum hypoxic stress may compound existing inter- and intraobserver variability. Recently, Vintzileos and Smulian[40] have advocated the use of the evolution and duration of FHR patterns while interpreting FHR changes in clinical practice.

TABLE 2
The chronic hypoxia checklist: Is this fetus fit to undertake the journey of labor?

CTG feature	Yes	No
1 Is the baseline FHR "stable," and "appropriate" for the gestational age?		
2 Is there normal variability and cycling?		
3 Are there accelerations?		
4 Are shallow or late decelerations (ie, uteroplacental insufficiency) "absent"?		
5 No evidence of risk factors which may increase fetal compromise (meconium, maternal pyrexia, chorioamnionitis, vaginal bleeding, fetal growth restriction, etc.)		
If the answers to all 5 questions are "yes," then continue CTG monitoring. If not, immediately seek senior input.		

CTG, cardiotocography; *FHR*, fetal heart rate.

Jia. Fetal heart rate tracing interpretation in clinical practice. Am J Obstet Gynecol 2023.

What is physiological interpretation of cardiotocography?

Physiological interpretation of CTG was first developed at St George's University Hospital, London,[41] and then subsequently implemented in other maternity units in the United Kingdom with demonstrable improvements in perinatal outcomes.[42–44] Physiological interpretation of CTG is based on excluding features of chronic hypoxia and preexisting fetal compromise on the CTG trace at the beginning of recording by using a "checklist" (Table 2) and then determining the fetal compensatory responses to ongoing intrapartum hypoxic and mechanical stresses and the type of intrapartum hypoxia (Table 3). CTG trace is scrutinized for features suggestive of evidence of the onset of decompensation of the fetal central organs. The first International Consensus Guidelines on Physiological Interpretation of CTG was published by 34 CTG experts from 14 countries in 2018.[45] Currently, out of approximately 130 maternity units in the United Kingdom, approximately 20 have implemented the principles of physiological interpretation of CTG (https://www.google.com/maps/d/viewer?mid=1bNWUDAvaDE1gm72-IN9FSsvoKjYEeiun&ll=53.739161790863044%2C-3.105274058304048&z=6). In addition, physiological interpretation of CTG has been implemented in some hospitals in France, Denmark, Republic of Ireland, China, Australia, Oman, Italy, and Spain.

Principles of physiological interpretation of fetal heart rate tracings during labor

An adult would immediately increase the rate and depth of respiration to oxygenate the myocardium, whenever he or she is exposed to hypoxic stress. This immediate reflex response is aimed at maintaining an aerobic metabolism and avoiding the onset of anaerobic metabolism, which will lead to the production of lactate. The gradual buildup of lactate within myocardial fibers would result in an energy crisis (2 adenosine triphosphates [ATPs] vs 36 ATPs), culminating in myocardial failure and

TABLE 3
Types of intrapartum fetal hypoxia and their features

Type	Feature
Gradually evolving hypoxia	This gradually evolving hypoxic stress may arise from either repetitive compression of the umbilical cord or repetitive reduction in uteroplacental circulation by ongoing uterine contractions. As labor progresses, uterine contractions become progressively stronger, longer, and more frequent, especially if oxytocin is used to augment labor, increasing the risk of fetal decompensation, if corrective actions are not taken to modify the uterine contractions. In a "nonstimulated" labor, a gradually evolving hypoxia may occur in the presence of fetal growth restriction (reduced placental reserves with or without oligohydramnios leading to sustained compression of the umbilical cord) or if fetal metabolic demands are higher (eg, macrosomic fetus, terminal villus hypoplasia in gestational diabetes mellitus, poor placental function in postdate pregnancies, or chorioamnionitis with increased metabolic demand). A gradually evolving hypoxia is characterized by an immediate response to protect the myocardial workload (ie, onset of decelerations, which become progressively wider and deeper similar to the rate and depth of respiration of an adult subjected to progressive hypoxic exercise in the gym as hypoxic stress worsens), an attempt to conserve oxygen and glucose by restricting the somatic body movements (ie, loss of accelerations), an attempt to obtain more oxygen and glucose from the placenta and pumping these to the vital organs at a faster rate (ie, catecholamine-mediated increase in the baseline fetal heart rate), and redistribution of oxygenated blood to "essential" central organs (the heart, brain, and adrenal glands), by peripheral vasoconstriction and "centralization" of blood flow.
Subacute hypoxia (Figure 10)	Subacute hypoxia occurs when the intensity of the hypoxic stress is greater than a gradually evolving hypoxic stress during labor and less than an acute profound hypoxic stress, with the fetus progressively spending less time at his or her normal baseline than during the decelerations, typically $<$30 s at the baseline and $>$90 s during decelerations This reduces the time available for placental gas exchange and maintaining oxygenation to fetal central organs. It is frequently associated with increased variability (zigzag pattern).
Acute hypoxia (Figure 11)	Prolonged deceleration $>$3 mins. Acute hypoxia is caused by sudden cessation of oxygenation to the fetus and the attempt by the fetus to immediately drop the heart rate and to sustain it for more than 3 min to prevent myocardial hypoxia and acidosis This is because continued myocardial activity in the absence of oxygen would lead to the onset of anaerobic metabolism and the production of lactic acid within the myocardium resulting in a terminal myocardial failure.

In all types of intrapartum hypoxia secondary to excessive uterine activity, intrauterine resuscitation with tocolysis should be always considered to improve uteroplacental oxygenation (ie, intrauterine resuscitation).

Jia. Fetal heart rate tracing interpretation in clinical practice. Am J Obstet Gynecol 2023.

terminal bradycardia. The human fetus, similar to an adult, would show predictable compensatory responses to protect his or her central organs if he or she is exposed to any hypoxic stress during the intrauterine life. However, the intrauterine environment is devoid of any atmospheric oxygen. Therefore, unlike adults, the fetus will not have the option to increase the rate and depth of respiration to obtain more oxygen and maintain the aerobic balance within the myocardium. Alternatively, the fetus in the absence of oxygen will rapidly reduce the myocardial workload to avoid the onset of anaerobic metabolism. This is similar to an adult resting on a sofa at home and having no pain in his or her calf muscles as a result of lactic acidosis, compared with adults exercising in the gym and feeling pain in their calf muscles because of the production of lactic acid as a result of the anaerobic exercise. Lactate is produced only when the oxygen demand of a muscle outstrips its blood supply, leading to a shift from aerobic to anaerobic metabolism.

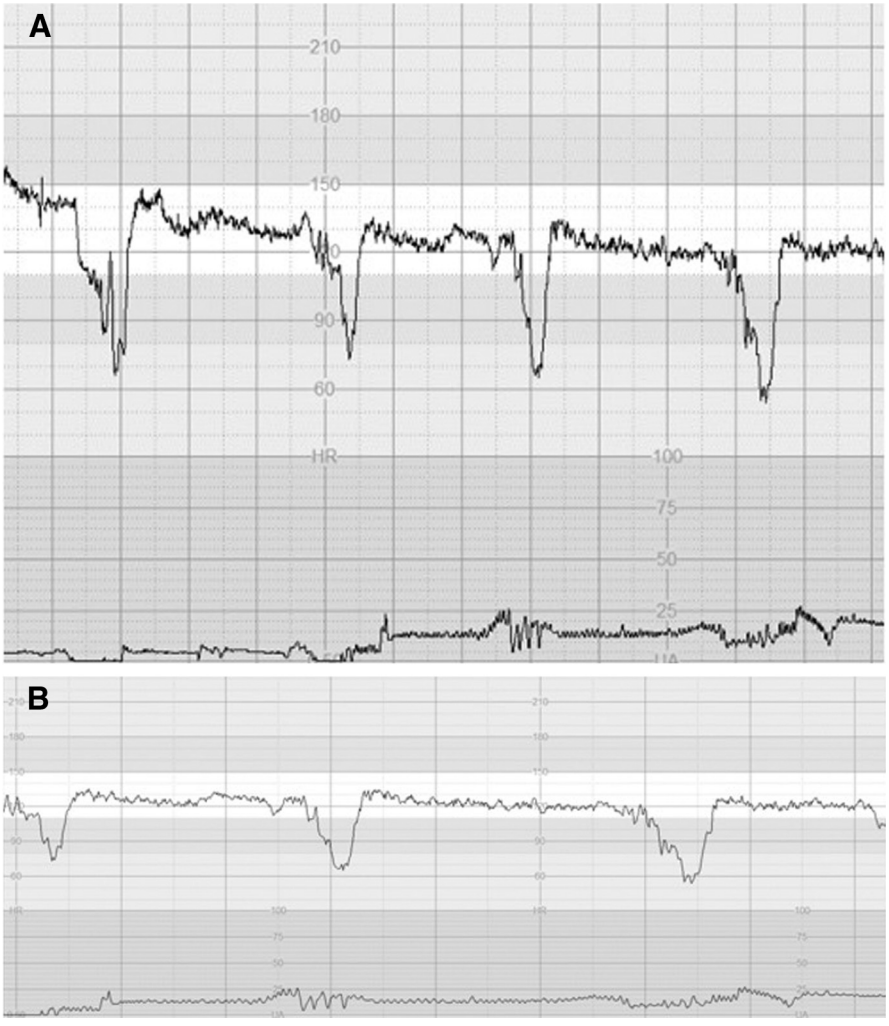
Application of fetal responses to hypoxic and mechanical stresses in clinical practice

The fetal reflex responses to protect the myocardial workload by lowering the fetal baseline heart rate, called "decelerations," should not be considered "pathological" purely based on their apparent morphology. Compression of the umbilical cord causes an acute and transient reduction in the oxygen concentration (ie, hypoxemia) and/or transient increase in the fetal blood pressure because of the intense compression of the umbilical arteries and resultant increase in systemic vascular resistance. These mechanisms stimulate peripheral chemoreceptors (ie, hypoxemia) and/or baroreceptors (ie, systemic blood pressure) resulting in a rapid drop in the FHR, followed by a quick recovery to the baseline. Depending on the intensity of umbilical compression, and the duration of compression, the depth and duration of such "quick" decelerations may vary. Moreover, if there are loops of the umbilical cord around the fetal neck, or oligohydramnios, sustained compression of the umbilical cord may give rise to "U, V, and W" patterns. Therefore, one should move away from the illogical practice of "naming and shaming" these decelerations as "typical, atypical, complicated, uncomplicated, reassuring, and nonreassuring" decelerations. Similarly, one should not create arbitrary time limits to classify CTG traces into "suspicious" or "pathological." These decelerations, which recover rapidly to the baseline FHR, irrespective of how "ugly" they seem, are not associated with fetal acidosis.[8,16,45] The fetal compensatory response to these transient, intermittent interruptions of oxygenation and/or rapid changes in fetal systemic blood pressure should be determined by scrutinizing the CTG trace between these

decelerations. Although earlier animal experimental studies have suggested that fetal carotid baroreceptors played a role in these "quick" decelerations,[46–48] recent animal studies have disputed the role of baroreceptors in the causation of these "quick" decelerations.[49] Some suggest that the predominant mechanism for such rapid and abrupt changes in the heart rate, followed by its rapid recovery is mediated through the peripheral chemoreflex and not the baroreflex.[46] Animal studies often fail to replicate the stress that a human fetus experiences during labor: when a uterus contracts, the umbilical cord gets compressed, and the uteroplacental oxygenation is reduced at the same time. Therefore, animal experimental models, which determine the effects of isolated, repetitive compression of the umbilical cord, often miss the real complexity of the labor ward that the clinicians experience in their day-to-day life. These "quick" decelerations may be entirely mediated by the peripheral chemoreflex as recently suggested by some.[37] Based on earlier animal experiments,[46–48] it is also possible that the baroreceptors do play a role, as accepted by international consensus panels for both FIGO and Physiological CTG Guidelines.[8,45] Therefore, for simplicity, the authors recommend that these "quick" reflex responses are termed as "baroreceptor decelerations" (Figure 1) to help midwives and obstetricians when they undertake FHR interpretation in their day-to-day clinical practice. If these decelerations are sustained, then this prolonged decrease in the FHR would lead to a reduction in cardiac output and, therefore, organ perfusion. If such prolonged and repetitive decelerations continue, the fetal compensatory responses may likely be overridden with time, predisposing them to hypoxic-ischemic injury. Many studies have attempted to correlate the "deceleration areas" with perinatal outcomes.[50–52]

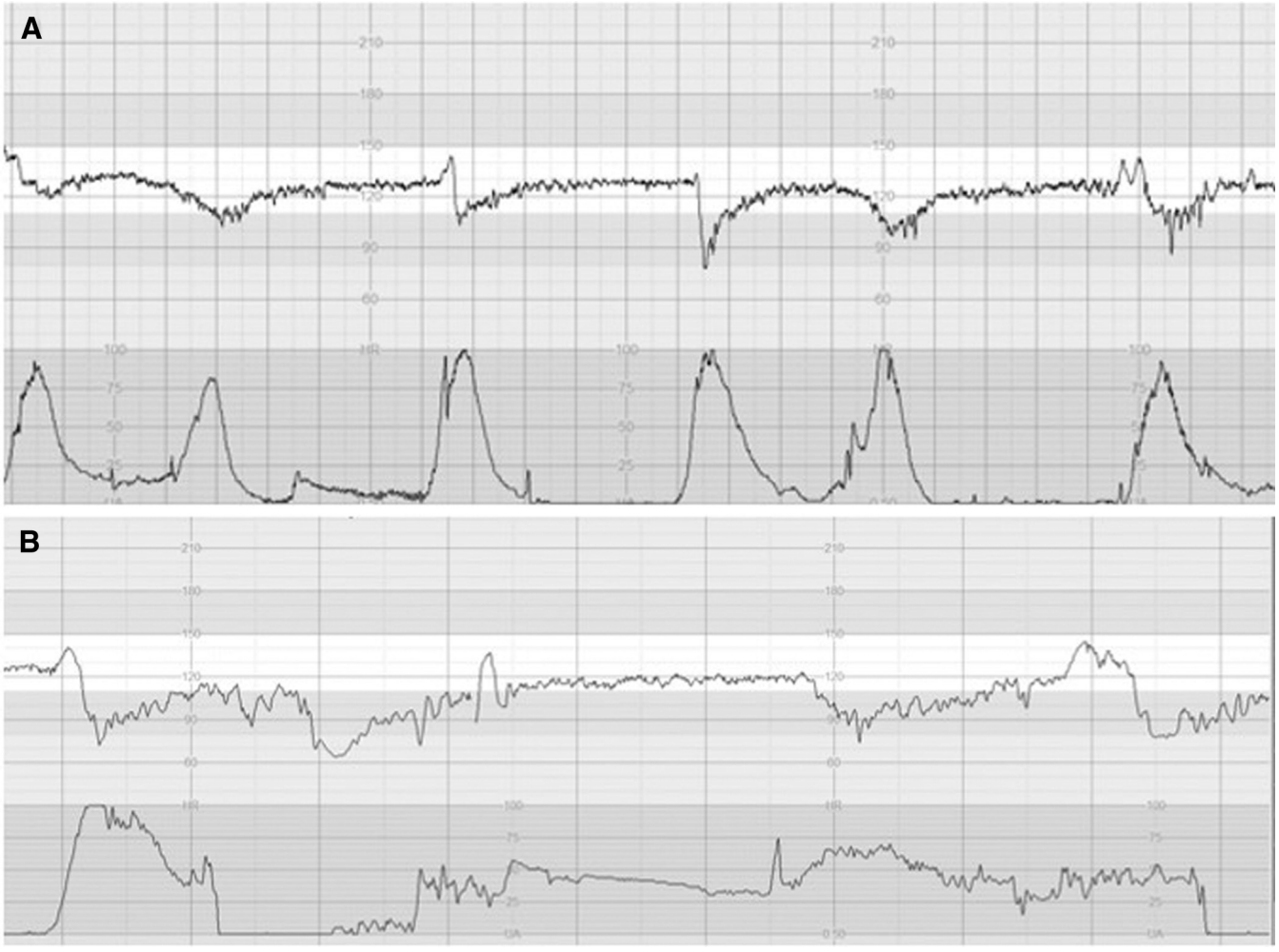
If there is uteroplacental insufficiency with reduced placental pools because of either a structural abnormality of the placenta (eg, preeclampsia, placental infarction, thrombosis, villous hypoplasia, or fibrin deposition) or a functional reduction in oxygenation of the placental pools (eg, injudicious use of oxytocin), then uterine contractions would result in a reduction in the oxygen concentration, accumulation of carbon dioxide, and transient shift to anaerobic metabolism resulting in transient acidosis. These chemicals (low oxygen tension, high carbon dioxide concentration, and high hydrogen ion concentration) would stimulate chemoreceptors. In uteroplacental insufficiency, unlike the compression of the umbilical cord, which happens quickly, it takes time for this altered chemical composition to bind to the receptors resulting in a gradual fall in the FHR. Similarly, when the myometrium relaxes, fresh oxygenated blood has to come into the placental venous sinuses from the maternal spiral arterioles to gradually "wash out" the accumulated chemicals from their respective chemoreceptors. Therefore, the recovery to the normal baseline FHR will not be quick in this case, but more gradual (Figure 2). In the past, these slowly recovering decelerations were termed "late decelerations."[53] It is important to appreciate that these "late" decelerations mediated through the

FIGURE 1
Quick decelerations



Note the "quick" decelerations that reach the nadir within 30 seconds and recover immediately to the baseline FHR without any delay because of umbilical cord compression. **A,** 1 cm/min. **B,** 3 cm/min.
FHR, fetal heart rate.
Jia. Fetal heart rate tracing interpretation in clinical practice. Am J Obstet Gynecol 2023.

FIGURE 2
Delay in recovery to the baseline FHR



Note the delay in recovery to the baseline FHR in cases of ongoing uteroplacental insufficiency. **A,** 1 cm/min. **B,** 3 cm/min.

FHR, fetal heart rate.

Jia. Fetal heart rate tracing interpretation in clinical practice. Am J Obstet Gynecol 2023.

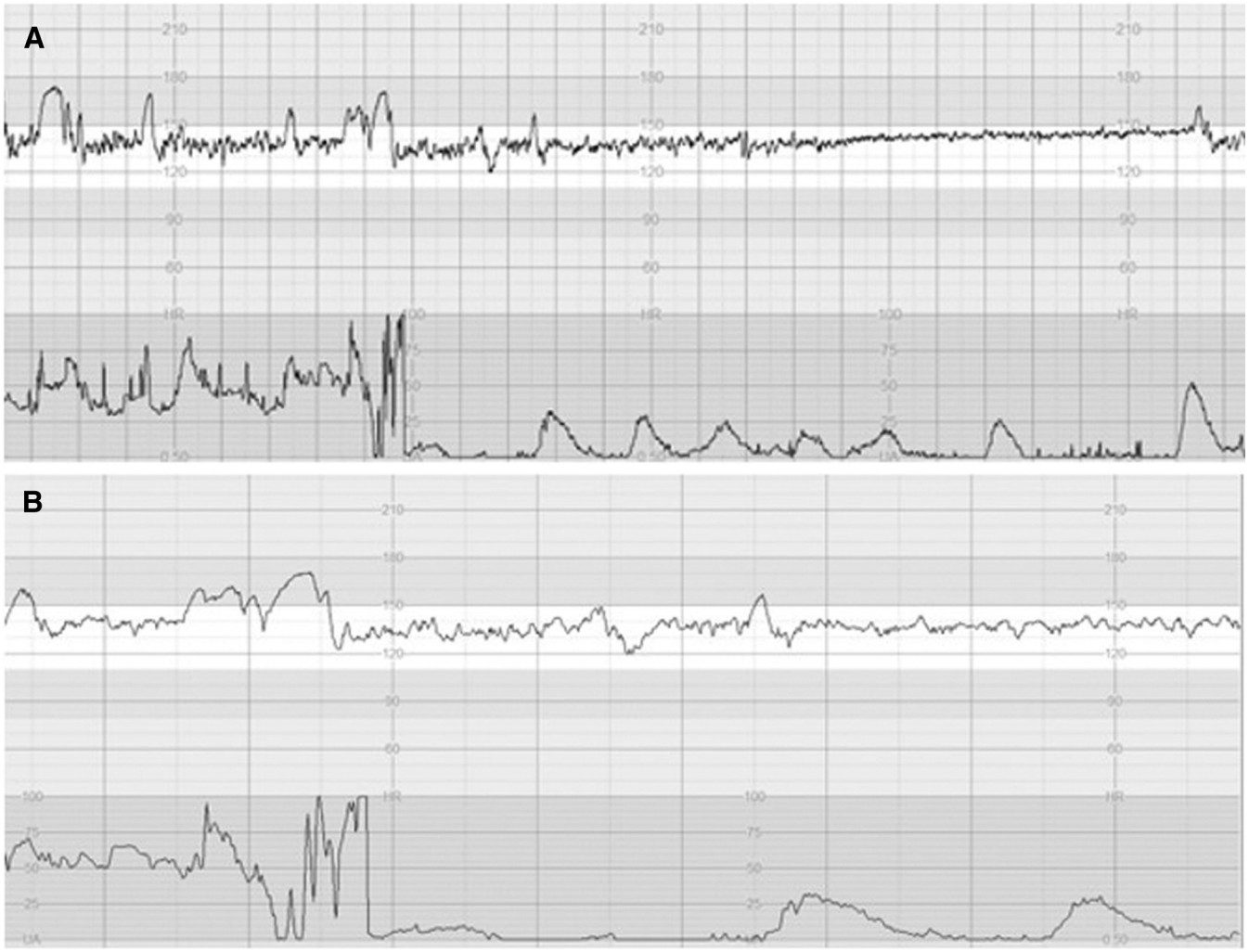
chemoreceptors indicate the underlying pathophysiology (ie, uteroplacental insufficiency).

Clinicians need to scrutinize the features on the CTG trace between the decelerations to determine the fetal compensatory response to ongoing stress. If reduced uteroplacental oxygenation was to be suspected, corrective actions should be taken (eg, reducing or stopping oxytocin to improve uteroplacental oxygenation). Moreover, the overall clinical picture should always be considered prior to making management decisions regarding the continuation of labor (ie, parity, rate of progress of labor, the reserve of the individual fetus to withstand further hypoxic stress, depending on the stage of labor). If corrective actions cannot be taken to reverse the situation, then, delivery should be expedited.

For simplicity, the authors recommend that these decelerations with "delayed recovery" (Figure 2) are termed "chemoreceptor decelerations" to help avoid confusion among midwives and obstetricians in their day-to-day clinical practice.

Therefore, in daily clinical practice, irrespective of the morphology of these decelerations, clinicians ought to appreciate that these are reflex responses to protect the myocardial workload and to maintain the heart in a positive aerobic balance whenever there is a transient reduction in oxygenation. The "quickly recovering baroreceptor-decelerations" (Figure 1) are not associated with acidosis, whereas the "slowly recovering chemoreceptor decelerations" (Figure 2) are associated with uteroplacental insufficiency and transient episodes of acidosis. However, in between these decelerations, if the fetus continues to show a stable baseline FHR, then the fetal myocardium is in a positive aerobic balance. Similarly, if the baseline FHR

FIGURE 3
Normal FHR trace with a stable baseline FHR, reassuring variability, presence of true accelerations, and absence of decelerations



The phenomenon of cycling (alternative periods of active and quiet fetal sleep) indicates a nondepressed fetal central nervous system. **A,** 1 cm/min. **B,** 3 cm/min.

FHR, fetal heart rate.

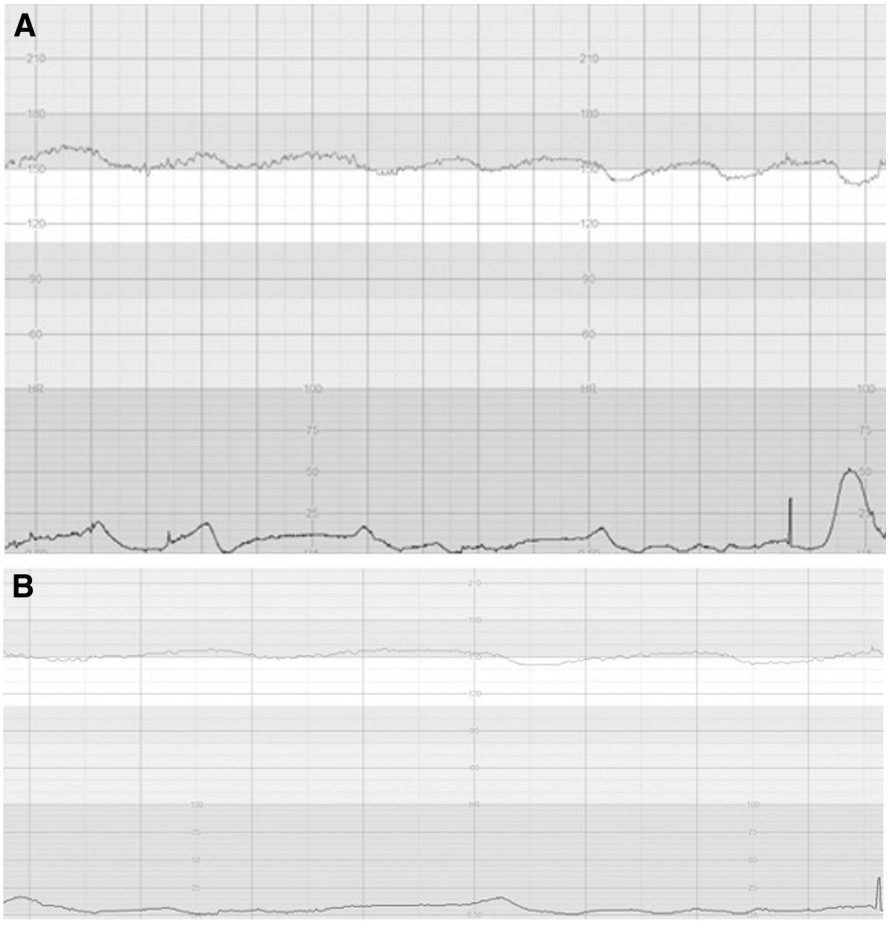
Jia. Fetal heart rate tracing interpretation in clinical practice. Am J Obstet Gynecol 2023.

variability is reassuring between the decelerations, then there is sufficient perfusion through the carotid arteries to the brain, suggestive of the absence of central nervous system (CNS) depression or acidosis.

Baseline fetal heart rate

CTG trace should be reviewed for 10 minutes, after excluding accelerations and decelerations to determine the average baseline FHR (Figure 3). Baseline FHR reflects the number of times the fetal heart chambers must beat to meet the ongoing metabolic demands of the fetus. When reviewing a CTG trace, clinicians should determine the stability of the baseline, the actual rate, and any changes in the baseline compared with the previous recording. If the myocardium has sufficient glycogen reserves and remains in an aerobic metabolism, the baseline heart rate would continue to be stable, irrespective of the rate (Figure 3). Therefore, the stability of the baseline is the most important feature of the CTG trace as it reflects the oxygenation of the most important central organ, the "pump." An unstable or "wavy" baseline indicates a negative myocardial energy balance and a failing myocardium (Figure 4). If observed, immediate actions should be taken to improve fetal oxygenation by reducing or stopping ongoing uterine activity. If this is not possible or appropriate (eg, clinical evidence of placental abruption), delivery should be expedited immediately. The normal range of FHR for term human

FIGURE 4
Myocardial negative energy balance



Note the unstable or "wavy" baseline indicative of myocardial negative energy balance. **A,** 1 cm/min. **B,** 3 cm/min.

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fetuses is considered to be between 110 and 160 bpm.[6–8,45] Nevertheless, it is important to appreciate that the FHR may change during labor when the metabolic demand of the individual fetus increases. Therefore, a ≥10% increase in the baseline FHR should be considered abnormal for the individual fetus in question,[33] and an evolving hypoxia with a catecholamine response[16,54] and/or chorioamnionitis[55,56] should be excluded. Romero et al[57] have shown that chorioamnionitis is a fetal disease with a fetal inflammatory response.[57–60] Therefore, >10% sustained increase in the baseline FHR without any preceding decelerations should be viewed with a high index of suspicion for ongoing fetal inflammatory response. Because of the progressive maturity of the parasympathetic nervous system as gestation advances, there is a gradual and progressive reduction in the baseline FHR with advancing gestational age.[61] Therefore, a fetus at term would be expected to have a baseline FHR toward the lower limit of the normal range (ie, 100–140 bpm).[61,62] Therefore, the question: "Is the baseline FHR appropriate for the gestational age of this fetus?" (Table 2) should always be asked while interpreting the CTG trace.[45,54] Higher than expected baseline FHR (>150 bpm) >40 weeks of gestation has been reported to be associated with poor perinatal outcomes.[63] One should not forget that fetal cardiac arrhythmias, such as supraventricular tachycardia (Figure 5) and congenital heart blocks, may also increase or decrease the baseline FHR.

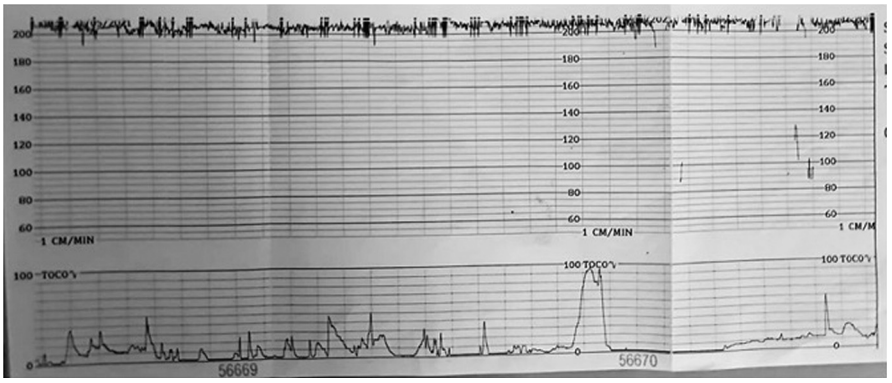
Baseline fetal heart rate variability

The bandwidth or the beat-to-beat fluctuation across the baseline is called the variability (Figure 3), and it reflects the integrity of the autonomic nervous system centers in the fetal brain (ie, the sympathetic center attempts to increase the heart rate, and the parasympathetic center attempts to reduce it). Therefore, a normal baseline FHR variability (FHRV) indicates a nondepressed fetal CNS, and conversely, a reduced baseline FHRV indicates an ongoing fetal CNS depression. The normal range for a population of human fetuses is considered to be between 5 and 25 bpm.[6,8,45] However, changes in the baseline FHRV for the individual fetus (eg, a reduction from 20 to 7 bpm) should be considered abnormal, even though it may still be within the "normal range" for the population of fetuses. Several nonhypoxic causes (eg, fetal deep sleep cycle; maternal hypoglycemia; medications, such as opiates and magnesium sulfate; and fetal neuroinflammation in chorioamnionitis) can depress the fetal CNS. However, one should always exclude metabolic acidosis suppressing the fetal autonomic centers in the brain leading to loss of baseline FHRV.[16,54,64] Rarely, there may be transplacental passage of acid from the maternal compartment (eg, diabetic ketoacidosis or severe maternal sepsis) leading to a reduction in variability. In a term fetus, if the reduced baseline FHRV is secondary to ongoing hypoxia, then the baseline FHR should be higher than expected for the gestational age because of the release of catecholamines to increase the cardiac output and redistribute the blood flow to the central organs before the onset of decompensation (loss of baseline FHRV).[16,45,54,64] Alternative phases of active and quiet sleep (REM and non-REM) give rise to FHR cycling. "Cycling" is one of the most important features of the CTG trace and is defined as the physiological alternating epochs of normal and reduced heart rate variability

(Figure 3). The presence of cycling on the CTG trace indicates fetal well-being during labor,[65] and its absence is associated with poor perinatal outcomes.[66,67] The absence of cycling has been reported in neuroinflammation secondary to chorioamnionitis,[56] and it may occur even before the onset of maternal fever.[67] Vintzileos et al[68] demonstrated the importance of FHR cycling in preterm gestation, and this phenomenon must be considered during labor in term fetuses to exclude depression of the fetal CNS.[65,69] In supraventricular tachycardia, the baseline FHRV may seem suppressed with the apparent loss of cycling (Figure 5).

In rapidly evolving hypoxia, the baseline FHRV increases secondary to an autonomic CNS instability leading to excessive variability of >25 bpm. It is termed "saltatory" pattern when it is uniformly increased and persists for >30 minutes.[8,45] A true saltatory pattern with >25 bpm lasting for >30 minutes is rare, and in many cases, the variability may be uniformly increased, but not >25 bpm. Such a uniform increase in the baseline variability usually occurs during the antenatal period when a fetus recovers from an acute insult,[70] and it is rare during labor.[71] This is because of ongoing contractions with interruptions of blood flow; a uniform increase lasting for >30 minutes is not possible. Alternatively, an erratic up and down fluctuation of >25 bpm, called the "zigzag" pattern (Figure 6), usually occurs during labor.[71] It has been shown that the presence of the zigzag pattern lasting for >2 minutes is associated with poor perinatal outcomes.[71,72] It has been recently reported that the zigzag pattern may also occur when there is autonomic instability secondary to fetal neuroinflammation in chorioamnionitis[55,56] and maternal COVID-19 likely because of the transplacental passage of the maternal cytokine storm.[73] In addition, increased variability has been shown to be associated with abnormal umbilical arterial pH.[74] It is important to appreciate that the baseline FHRV should not be considered in isolation but in conjunction with the changes in the baseline FHR.[15,16,54,75] Moreover, the absence of cycling and changes in the variability over time in the same fetus should be considered ominous. The reduction in baseline variability and the absence of cycling may also be seen in nonhypoxic causes of fetal compromise,

FIGURE 5
Cardiac arrhythmia (supaventricular tachycardia or SVT)



Note the absence of decelerations.

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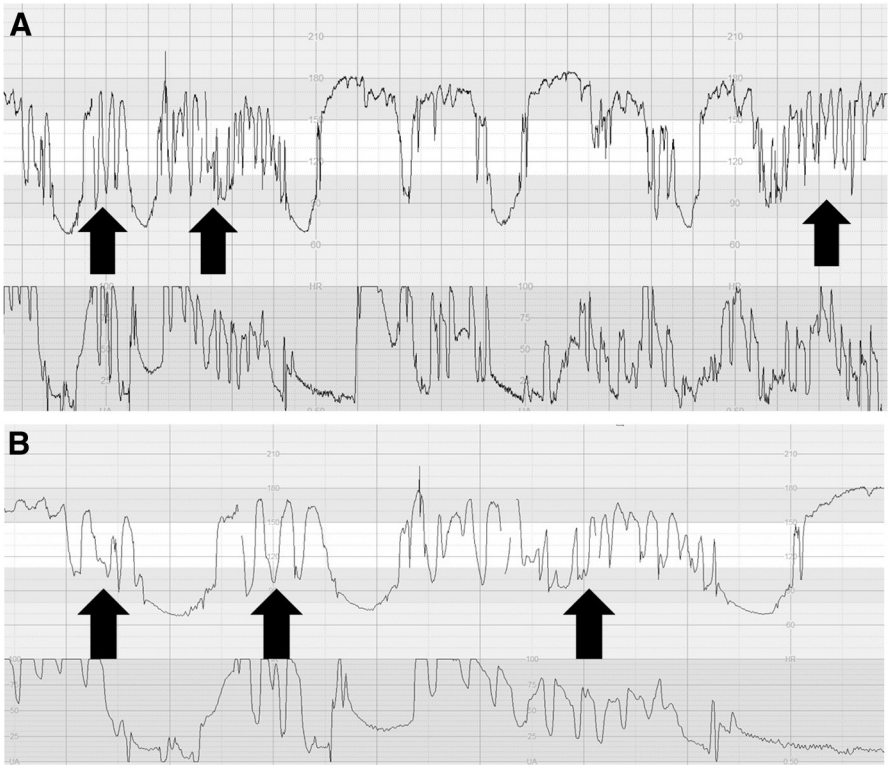
FIGURE 6
ZigZag Pattern



Note the transient, abrupt, up-down fluctuation across the baseline, called the "zigzag" pattern because of autonomic instability. A, 1 cm/min. B, 3 cm/min.

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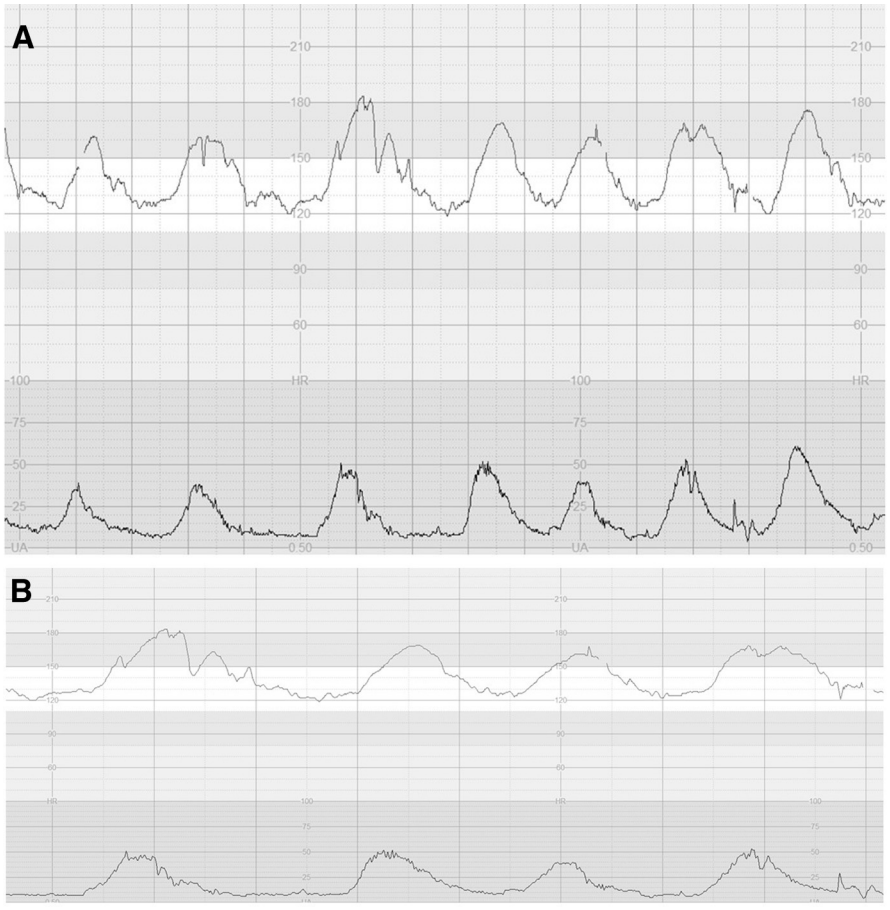
FIGURE 7
Double mountain peak sign



Note the large amplitude accelerations coinciding with ongoing uterine contractions and persisting throughout the contractions indicative of erroneous recording of the maternal heart rate as fetal heart rate.

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such as an intrauterine fetal stroke. In such cases, the baseline FHR would also be high secondary to increased fetal intracranial tension, similar to an adult with stroke.

Accelerations

An abrupt and transient increase in the baseline heart rate, approximately 15 bpm lasting for approximately 15 seconds in a term fetus is referred to as an acceleration.[6,8,45] They reflect the integrity of the fetal somatic nervous system and are usually associated with fetal gross body movements.[16,54,64] True accelerations arise from a stable baseline FHR with a normal variability and should return to the same stable baseline (Figure 3). They should not be part of any decelerations (ie, shoulders or "overshoots"). The presence of true, repetitive accelerations (>2 in 20 minutes) is indicative of the absence of hypoxia and acidosis because a fetus often restricts the somatic body movements to conserve oxygen and nutrients in the presence of hypoxic stress.[6,8,16,45,54,64] Several spurious or false transient increases in the baseline FHR may mimic true accelerations.[76] During the second stage of labor, one should be very cautious of the "double mountain peak" sign[76] when large amplitude accelerations coincide with ongoing uterine contractions (Figure 7) because of erroneous monitoring of the maternal heart rate as the FHR.[54,64,77–79] If such a "double mountain peak" sign is observed during the second stage of labor, immediate steps should be taken to confirm the FHR.

Decelerations

As described earlier, decelerations are reflex responses to protect the myocardial workload and the attempt by the fetus to continue to maintain an aerobic metabolism within the myocardium when there is hypoxic (uteroplacental insufficiency) or mechanical (umbilical cord compression) stress. Transient and repetitive reduction of oxygenation secondary to ongoing uterine contractions would result in intermittent decelerations. As these decelerations become wider or deeper, they are often termed "atypical" or "complicated" variable decelerations (Figure 8). If the intervening baseline FHR and variability remain normal, these "ugly" decelerations do not require any urgent intervention. "Chemoreceptor-mediated" late decelerations are associated with uteroplacental insufficiency (Figure 2) and, therefore, may lead to fetal acidosis over time if they are repetitive and prolonged. In contrast to intermittent decelerations, if there is an acute and profound reduction in fetal oxygenation, the fetus attempts to protect the myocardium from acidosis by abruptly lowering the heart rate and sustaining it for >3 minutes, resulting in a prolonged deceleration (Figure 9). The management of intermittent and prolonged decelerations is discussed under different types of hypoxia (Tables 2 and 3).

Fetal catecholamine response

Human fetuses have a remarkable ability to rapidly increase the heart rate by releasing adrenaline and noradrenaline (ie, catecholamines) to pump blood at a faster rate to the placenta and central organs to continue to maintain oxygenation. In addition, catecholamines help redistribute blood from peripheral, "nonessential" organs to essential central organs (ie, "centralization") to avoid anaerobic metabolism and production of lactic acidosis in the fetal central organs ("birth asphyxia"). It is important to appreciate that in a gradually evolving

hypoxia, the fetal catecholamine response should be preceded by repetitive decelerations as the fetus attempts to protect the myocardial workload first, if there is any hypoxic or mechanical stress.

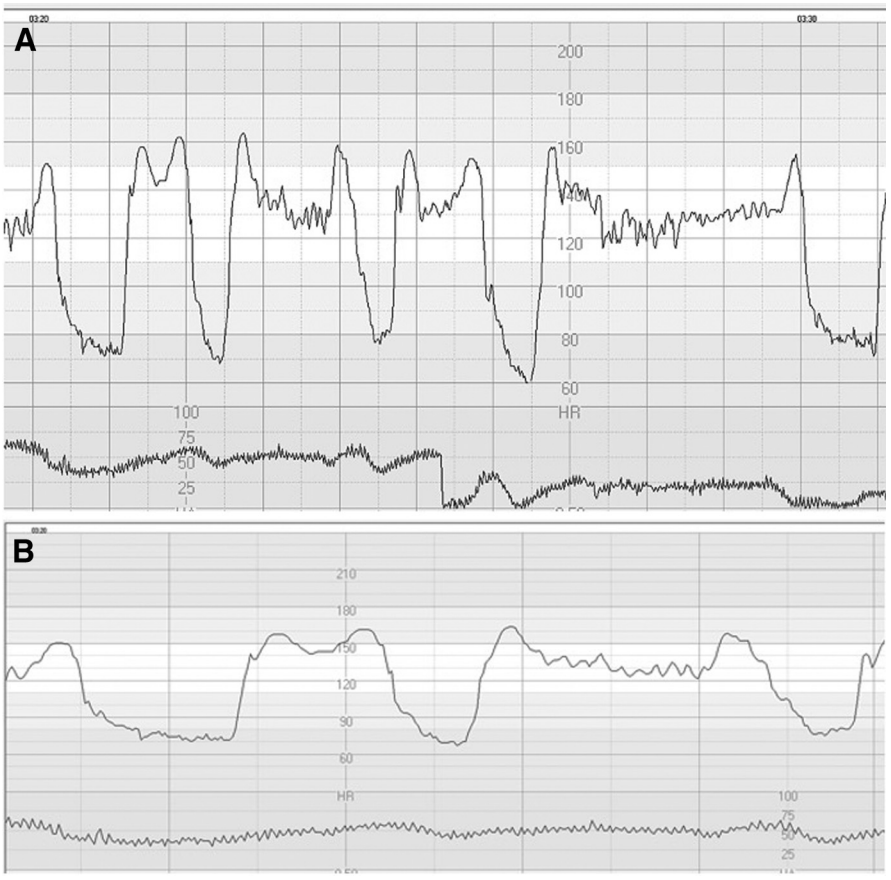
Chronic hypoxia and preexisting fetal compromise

The triad of catecholamine-mediated increase in the baseline FHR to increase placental oxygenation and increase central organ perfusion; repetitive shallow decelerations because of uteroplacental insufficiency, even in response to Braxton-Hicks contractions; and reduced baseline variability because of CNS depression and/or acidosis indicates chronic hypoxia (Figure 10).[16,54,64,80] Other useful pointers include the maternal history of reduced fetal movements and the presence of thick meconium (indicative of oligohydramnios secondary to a chronic uteroplacental insufficiency). If there is evidence of chronic hypoxia on the CTG trace, then the fetus will be unable to withstand any further hypoxic stress, and therefore, delivery should be expedited. A tocolytic should be used to abolish uterine contractions to improve placental perfusion before delivery. The "chronic hypoxia checklist"[80] (Table 2) recommended by the International Consensus Guidelines on Physiological Interpretation of CTG[45] aimed to identify fetuses who are not fit to undertake the journey of labor so that delivery could be expedited to avoid hypoxic-ischemic injury or perinatal death.

Unusual fetal heart rate patterns

It is important to appreciate that CTG was introduced to timely recognize the onset of fetal hypoxic stress; however, a human fetus is exposed to several nonhypoxic pathways of compromise and neurologic injury. In chronic fetal anemia and acidosis, a typical sinusoidal pattern may be seen.[80,81] However, in cases of an acute fetomaternal hemorrhage and resultant fetal hypotension, an atypical sinusoidal pattern or the "Poole shark teeth" pattern (Figure 11) may be seen because of autonomic instability.[16,54,81] The latter may also be seen in placental abruption and ruptured vasa previa and mandates an urgent delivery.[45] The presence of a sinusoidal pattern interspersed with epochs of normal variability and accelerations is termed "pseudosinusoidal" pattern and is often because of fetal thumb sucking or repetitive glottic movements.[45,72,81]

FIGURE 8
"Atypical" or "complicated" variable decelerations with quick recovery



A, 1 cm/min. B, 3 cm/min.

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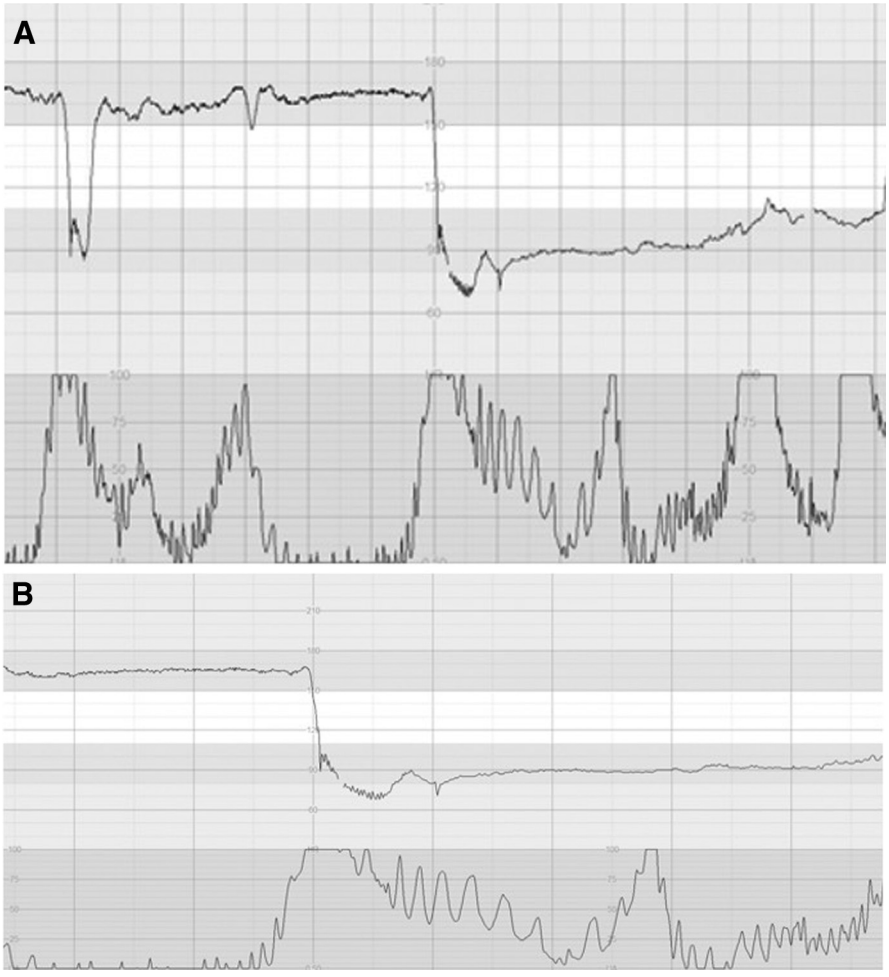
Recognition of different types of intrapartum fetal hypoxic stress

It is essential to apply the principles of fetal physiological response and recognize the types of intrapartum hypoxia while interpreting CTG traces[16,54,64,82] to overcome the limitations of current guidelines on CTG interpretation, which are based on "pattern recognition" to avoid adverse maternal and perinatal outcomes because of inter- and intraobserver variability.[59]

Of note, 3 types of intrapartum hypoxic stresses (acute, subacute, and gradually evolving) should be recognized on the CTG traces (Table 3), based on the different intensities, evolution, and duration of ongoing hypoxic stress.[16,45,54,64,83] The features of different types of intrapartum hypoxia (Table 3) are similar to adults undertaking a hypoxic exercise on a treadmill: one's ability to compensate effectively would depend on the speed of the treadmill (ie, intensity), duration of exercise, and the individual physiological reserve. The rate and depth of respiration and changes in the heart rate would reflect these variables. However, similar to an adult undertaking a treadmill exercise, a fetus may be exposed to a combination of hypoxic stresses, for example, a gradually evolving hypoxic

FIGURE 9
Onset of prolonged deceleration because of an acute hypoxic stress



Note the onset of prolonged deceleration because of an acute hypoxic stress. In umbilical cord prolapse, often repetitive, "quick" (cord compression) decelerations before the onset of terminal bradycardia. **A,** 1 cm/min. **B,** 3 cm/min.

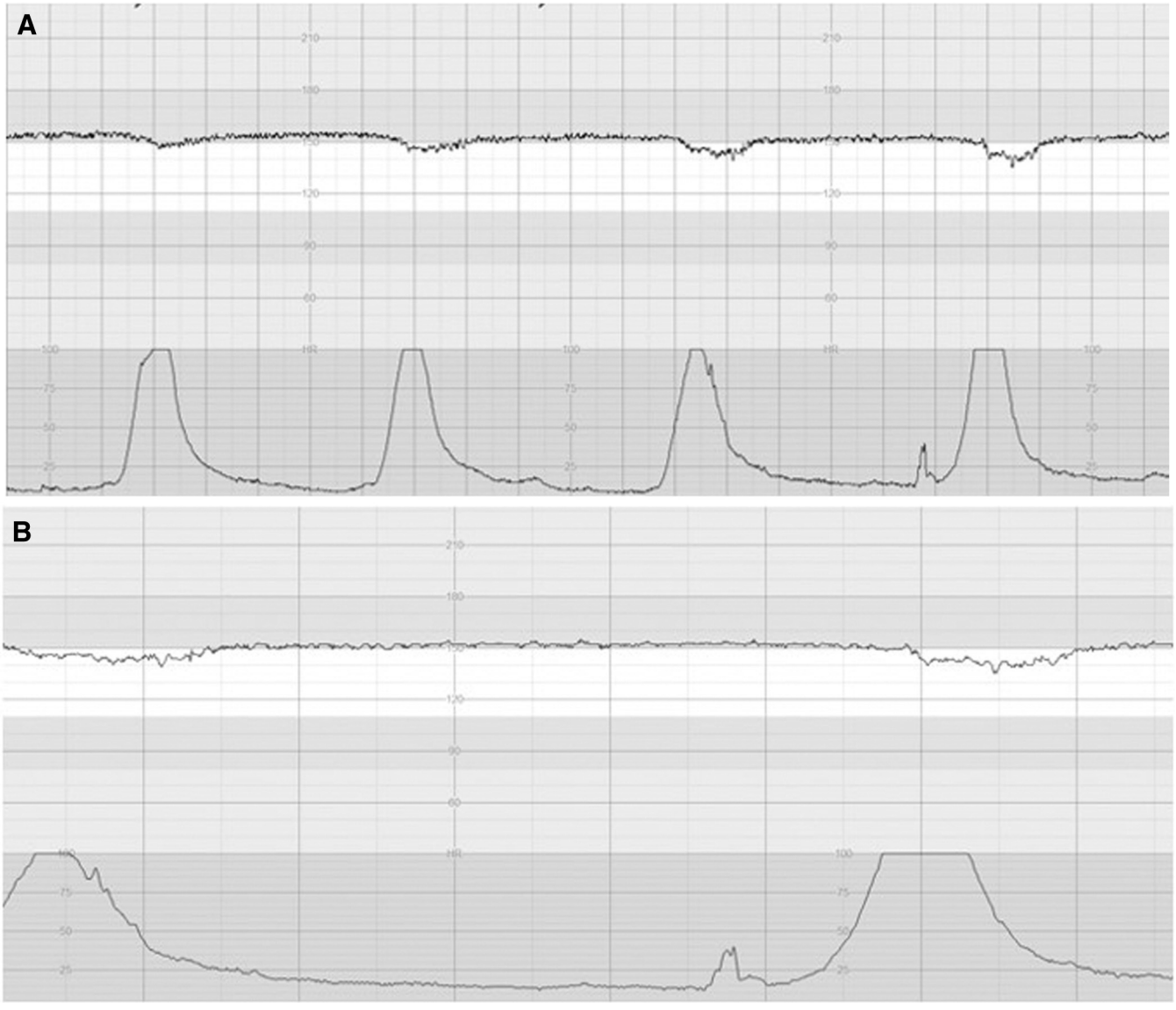
Jia. Fetal heart rate tracing interpretation in clinical practice. Am J Obstet Gynecol 2023.

stress may become subacute hypoxic stress (Figure 12) with the onset of active maternal pushing or commencement of oxytocin infusion, and this may culminate in acute hypoxia (Figure 9) with the onset of myocardial acidosis resulting in terminal bradycardia. It is important to have a systematic approach to the management of acute hypoxia (Table 4).

Recently, Jia et al[84] reported that it was possible to predict the next change in the CTG trace in fetuses being exposed to gradually evolving hypoxia by application of the knowledge of fetal physiology while interpreting CTG traces. Moreover, this study confirmed that even if there are repetitive variables or late decelerations, with or without catecholamine-induced increases in the baseline FHR, all fetuses were born without metabolic acidosis at birth. This study concluded that the onset of abnormal variability after repetitive variable or late decelerations and a catecholamine response were associated with neonatal acidosis in approximately 29% of cases.[84] These conclusions by Jia et al[84] were very similar to the study from Canada published in 2003, which concluded that if the baseline variability was normal, there was no significant risk of neonatal acidosis at birth despite ongoing repetitive variable or late decelerations, although if the reduced baseline variability persisted for more than 60 minutes, approximately 31% of cases had evidence of acidosis at birth.[85] Table 5 illustrates the "next" steps in a gradually evolving hypoxic stress during labor after a series of predictable changes "ABCDE."[16]

A gradually evolving hypoxia may become a "subacute hypoxia" (Table 3) with the onset of active maternal pushing or with the injudicious use of oxytocin infusion to augment labor. Albertson et al[86] analyzed 1800 CTG traces and reported that subacute hypoxia occurred in 10% of all cases within 30 minutes of active maternal pushing and 42% occurred in induced labor. Gracia-Perez-Bonfils et al[71] reported that because of the autonomic instability as a result of rapidly evolving "subacute hypoxia" during the second stage of labor, there is an erratic up and down fluctuation of the FHR across the baseline, increasing the baseline variability, called the "zigzag" pattern (Figure 7). They reported an 11-fold increase in the neonatal admission if the zigzag pattern persisted for more than 2 minutes, and immediate action was not taken to rapidly reverse the hypoxic stress.[72] Tarvonen et al[72] recently confirmed the same findings in a larger series of 5000 CTG traces, where they analyzed the perinatal outcomes in fetuses with the "zigzag" pattern. This erratic increase in the baseline FHRV (ie, the "zigzag pattern") should be differentiated from the uniform increase in the baseline FHRV of >25 bpm, which persists for more than 30 minutes, and it is called the "saltatory pattern." The latter is very rare during labor[54,71,72] as uterine contractions do not permit such a "uniform" increase in the baseline variability. The saltatory pattern has been described after an acute antenatal insult, and it is associated with brain damage in experimental animal studies.[37] Therefore, during active maternal pushing, clinicians should scan and scrutinize for the "zigzag" pattern (Figure 7) and not the saltatory pattern. Albertson et al[87] have reported that on the analysis of 1800 CTG traces, 40% of fetuses experiencing

FIGURE 10
Chronic hypoxia



Note the triad of higher than expected baseline FHR for the gestational age, reduced baseline FHR variability, and the presence of repetitive shallow decelerations. **A,** 1 cm/min. **B,** 3 cm/min.

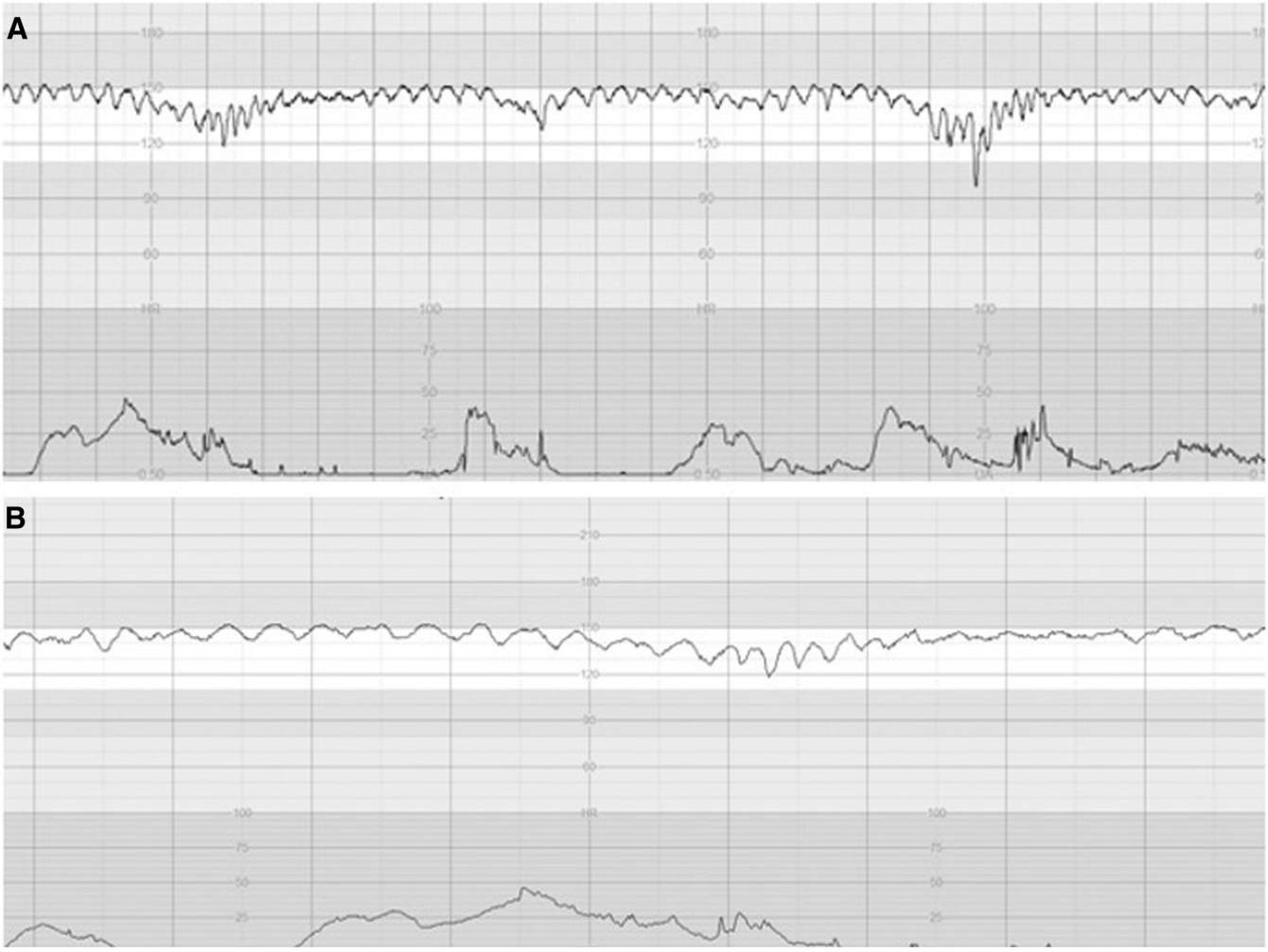
FHR, fetal heart rate.

Jia. Fetal heart rate tracing interpretation in clinical practice. Am J Obstet Gynecol 2023.

subacute hypoxic patterns demonstrated fetal ECG changes. These findings suggested that if subacute hypoxia is not rapidly reversed by stopping the ongoing stress by reducing myometrial contractions, myocardial decompensation may occur because of the negative myocardial energy balance (fetal ECG changes), leading to the production of lactic acid and terminal bradycardia. Therefore, if subacute hypoxia cannot be reversed with conservative measures, then delivery should be expedited to avoid poor perinatal outcomes.

Recently, an experimental animal study has suggested that deceleration area and deceleration capacity (an integrated measure of the magnitude and the frequency of FHR decelerations) were both associated with fetal hypotension.[88] Deceleration area was found to be an important predictor of neonatal acidemia in an observational study, which analyzed the nonreassuring FHR tracings during the second stage of labor.[89] These findings should not be of any surprise to those using physiological interpretation of CTG for the reason that if the deceleration area increases, then the fetal cardiac output reduces, resulting in reduced systemic blood pressure and organ perfusion.

FIGURE 11
Atypical sinusoidal pattern or the "Poole shark teeth" pattern (acute fetal hypovolemia)



A, 1 cm/min. **B,** 3 cm/min.

Jia. Fetal heart rate tracing interpretation in clinical practice. Am J Obstet Gynecol 2023.

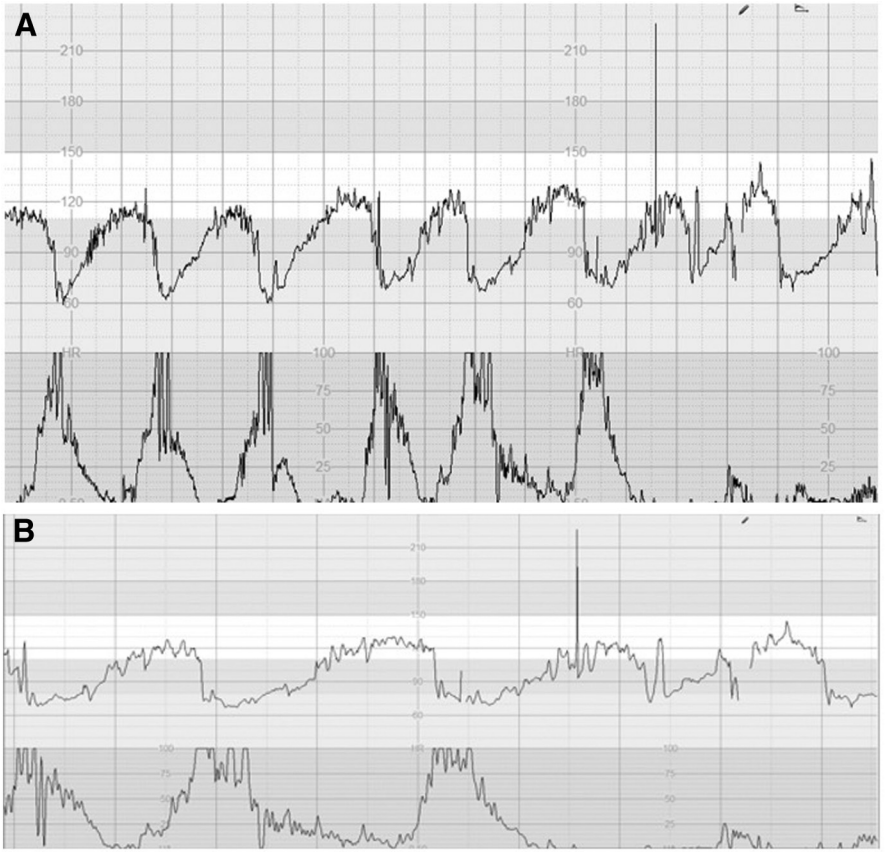
The knowledge of fetal pathophysiological responses to different degrees of hypoxic stress and recognizing the corresponding features on the CTG trace will assist clinicians to predict the "next" change on the CTG trace. As recommended by the International Consensus Guidelines on Physiological Interpretation of CTG traces,[45] CTG traces should be classified according to the type of ongoing fetal hypoxia. This approach will enable timely and appropriate actions to prevent hypoxic-ischemic injuries while avoiding unnecessary interventions because of the panic as a result of the classification of CTG traces into "pathological" or "category III," based on pattern recognition. Yatham et al[90] have reported that if the types of fetal hypoxia are used to interpret CTG traces compared with the traditional classification system using "normal, suspicious, and pathological," then the interobserver agreement can be increased from approximately <50% to 81%.

The importance of the clinical context when interpreting fetal heart rate tracings

Physiological interpretation of CTG is always carried in the context of the wider clinical context, because a growth-restricted fetus may not withstand the same intensity and duration of hypoxic stress compared with a term well-grown fetus. Therefore, the onset of stress to injury time will be much shorter. FHR tracing usually shows a baseline FHR higher than expected for the gestational age or an abrupt increase in the baseline of >10%, without repetitive preceding decelerations (Figure 13). Features of fetal neuroinflammation (absence of cycling and zigzag and sinusoidal patterns) (Figure 13) are commonly observed on the CTG trace.[55,56] Superimposition of hypoxic stress in a fetus with chorioamnionitis will potentiate

fetal neurologic injury[55,56,91,92] and may increase the likelihood of meconium aspiration syndrome (MAS).[93] Romero et al[94] reported that bacteria and increased interleukin 6 levels were found in a group with meconium-stained amniotic fluid compared with the control group. Moreover, a combination of intra-amniotic infection with fetal inflammatory response syndrome is an important antecedent of MAS.[95] Therefore, the presence of meconium should be viewed with a high index of suspicion for ongoing fetal inflammation while interpreting FHR tracings. Similarly, a difficult operative vaginal birth (vacuum or forceps), in a fetus experiencing subacute hypoxic stress may significantly potentiate the effect of birth trauma on ongoing hypoxic stress, if the subacute stress is not reversed. A detailed discussion of the wider clinical picture is beyond the scope of this review article. Recently, Oikonomou and Chandraharan[54] proposed the "intrapartum journey planner" with "6 P's" (Table 6) to help incorporate the clinical picture while interpreting the CTG traces.

FIGURE 12
Subacute hypoxic pattern



Note the subacute hypoxic pattern: the time spent at the baseline is less than the time spent during the decelerations. **A,** 1 cm/min. **B,** 3 cm/min.

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Adjunctive technologies to fetal heart rate tracings to reduce operative interventions

Several "adjunctive technologies" were attempted to reduce the false-positive rate of FHR tracings and intrapartum operative interventions. A deeper understanding of fetal physiological responses to hypoxic and mechanical stresses during labor will obviate the need for adjunctive technologies. This is because catecholamine-induced peripheral vasoconstriction will lead to anaerobic metabolism in peripheral tissues, as a fetal compensatory response. Therefore, assessing the oxygenation of peripheral tissues, such as the skin of the fetal scalp, will not produce reliable information regarding the oxygenation of fetal central organs. According to current scientific evidence, it seems that there is no role for peripheral tests of fetal well-being, such as fetal pulse oximetry[96,97] and fetal scalp blood sampling with pH or lactate estimation to reduce cesarean delivery in contemporary obstetrical practice.[21,98–101] First, it has been argued that fetal scalp blood sampling does not fulfill the "first do no harm" principle.[83,97,102,103] Recent scientific evidence suggests that performing repetitive fetal scalp lactate doubles the emergency cesarean delivery rate.[104] A multicenter, prospective study has concluded that performing fetal scalp pH increases the operative intervention rate by 60% without improving perinatal outcomes.[105] Therefore, these peripheral tests of fetal well-being should no longer be recommended in clinical practice. Fetal ECG (ST-analyzer or STAN) is used to determine the catecholamine-mediated glycogenolysis and the onset of negative cardiac energy balance.[9,106,107] The recent meta-analysis of 6 randomized controlled trials (>26,000 women) has concluded that there was a 34% statistically significant reduction in the rate of metabolic acidosis and an 8% reduction in operative vaginal births.[108,109] Therefore, fetal ECG remains the only additional test of fetal well-being with robust scientific evidence suggestive of benefit. However, it should not be used by clinicians without understanding fetal physiological responses to hypoxic stress. In the authors' experience with the use of fetal ECG spanning for approximately 20 years, if STAN is used with the knowledge of fetal physiology and understanding of the different types of fetal hypoxic stress, then intrapartum interventions can be significantly reduced.[41,110,111]

Does physiological interpretation of cardiotocography really improve outcomes in real life?

It is important to appreciate that classification of CTG traces into "categories I,

TABLE 4
Recommended management of prolonged decelerations: the acute hypoxia management algorithm

Time	CTG features	Recommended management
0	Sudden drop in the baseline FHR, which is sustained	Immediate examination to exclude irreversible causes (accomplish an urgent delivery if present) and to correct potentially reversible causes
3 min	Confirm the presence of normal baseline FHRV and cycling before the onset of the prolonged deceleration and the variability within the first 3 min of the deceleration	In the absence of acute intrapartum accidents, if these criteria are satisfied, 90% of these prolonged decelerations will recover back to the normal baseline in 6 min and 95% in 9 min. Continue conservative measures (eliminating the uterotonic agent from the maternal environment and considering the need for acute tocolysis, changes in the maternal position to relieve a sustained umbilical cord compression, and administration of fluids in cases of acute maternal hypotension).
6 min	Scrutinize the CTG for evidence of recovery = the "step-up pattern" to reach normal baseline	The interventions to improve uteroplacental perfusion is having the desired effect. Continue observation. If there is no attempt at recover, scrutinize the CTG trace for loss of variability during the deceleration, and if the variability becomes reduced, then consider a concealed placental abruption or a concealed uterine rupture.
9 min	The prolonged deceleration has not recovered to its baseline (with or without a compensatory fetal tachycardia) or onset of reduced or increased baseline FHRV within the deceleration	Immediate transfer to the operating theater as 95% of prolonged decelerations should have recovered. Reassess the overall clinical picture, including the features observed on the CTG and the rate of progress in operating theater, to determine whether an urgent birth by the safest and quickest mode is required.

CTG, cardiotocography; *FHR*, fetal heart rate; *FHRV*, fetal heart rate variability.

Jia. Fetal heart rate tracing interpretation in clinical practice. Am J Obstet Gynecol 2023.

II, and III" or "normal, suspicious, and pathological" has not worked. There has been an increase in the cesarean delivery rate and operative vaginal births without a concomitant reduction in the rate of cerebral palsy and perinatal deaths.[22] In addition, recent reports have suggested that up to two-thirds of cases of HIE and perinatal deaths are potentially avoidable, with CTG misinterpretation contributing to approximately 33% of cases.[112] Improvement in FHR interpretation has been reported following CTG master classes in physiological interpretation of CTG (Table 7).[113] Several maternity units have demonstrated >50% reduction in the HIE after training staff and then implementing the principles of physiological interpretation of CTG (Table 6). The reduction in the HIE rate was consistent across hospitals after the implementation of the International Consensus Guidelines on Physiological Interpretation of CTG (Figure 14). In addition to multidisciplinary training through physiological CTG master classes, local clinical

TABLE 5
Evolution of CTG changes in a gradually evolving hypoxic stress "ABCDE"[7]

CTG change	Mechanism
Onset of decelerations (and progressive widening and deepening of decelerations as hypoxic stress progressively worsens)	Cardioprotective reflex response to protect the myocardial workload to avoid anaerobic metabolism when oxygen supply is intermittently interrupted
A = loss of accelerations	Restriction of somatic body movements to conserve oxygen and nutrients to ensure continuous supply to "essential" central organs
B = baseline heart rate increases	Release of catecholamine to increase the heart rate to obtain more oxygen and nutrients from the placenta and to perfuse the central organs at a higher rate between the decelerations
C = compensated stress response	Increase in the tissue perfusion and effective redistribution through the catecholamine-mediated compensatory response is sufficient to maintain aerobic metabolism in central organs
D = decompensation	Unstable baseline FHR (myocardial anaerobic metabolism) and/or an abnormal variability (hypoxia to the autonomic nervous system centers in the brain stem)
E = end stage	Myocardial acidosis leading to the "step-ladder" pattern to death

CTG, cardiotocography; *FHR*, fetal heart rate.

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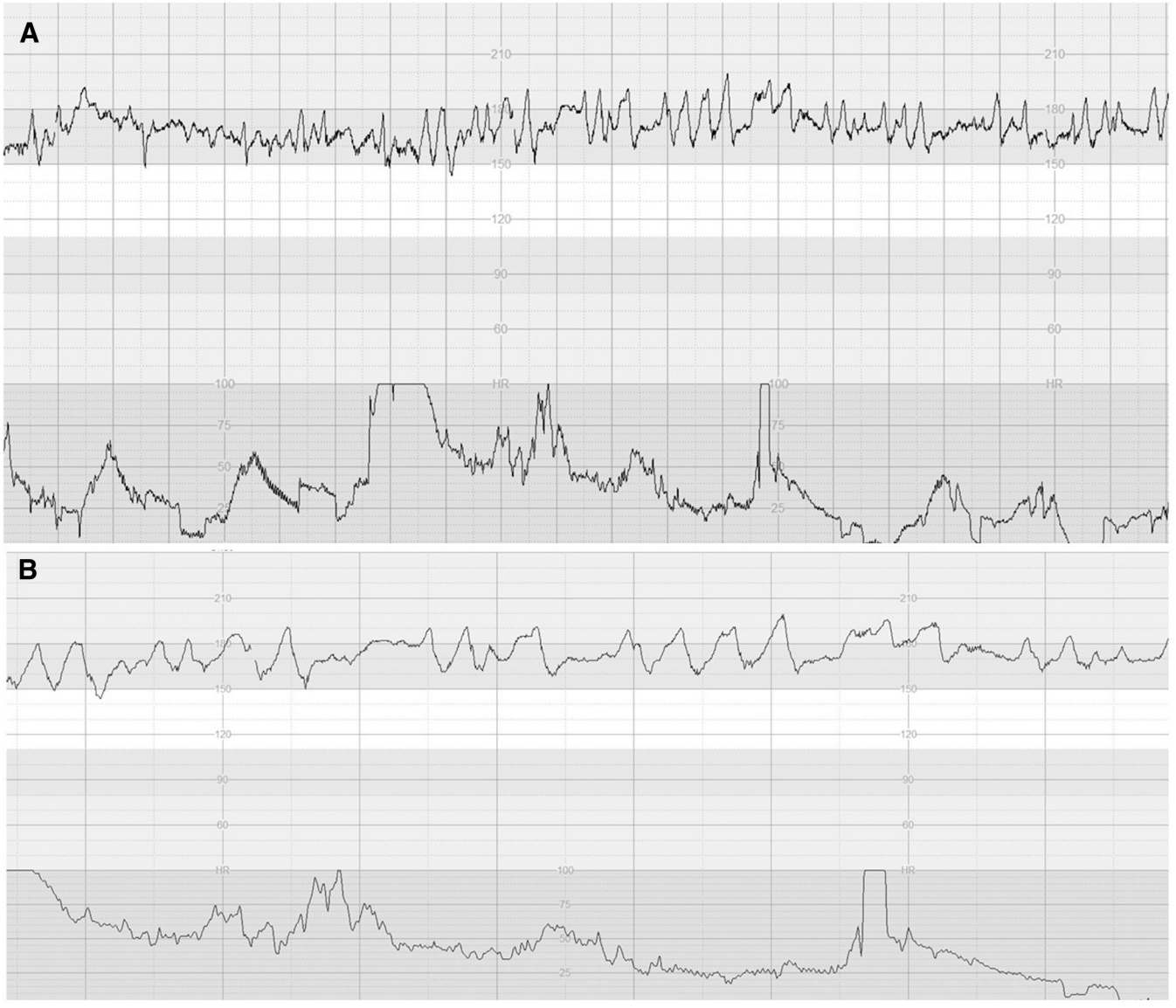
FIGURE 13
"ZigZag" pattern at a higher baseline in chorioamnionitis



Note the increased baseline for the given gestational age (41 4/7 weeks of gestation), apparent normal variability but with absence of repetitive decelerations and cycling, and transient "zigzag" pattern in chorioamnionitis. **A,** 1 cm/min. **B,** 3 cm/min.

Jia. Fetal heart rate tracing interpretation in clinical practice. Am J Obstet Gynecol 2023.

leadership and multidisciplinary team engagement and support of the senior management were responsible for reducing the HIE rate by >50% at these hospitals compared with the use of national guidelines based on "pattern recognition" (Figure 15). A recent study reported that physiological interpretation of CTG has a higher capacity to predict neonatal acidemia compared with the ACOG, NICE, and FIGO guidelines.[114] More recently, Pereira et al[115] reported that there was an association between the different types of intrapartum hypoxia and patterns of hypoxic injury on the magnetic resonance imaging scan when using the physiological classification of the CTG. In a large multicentric study on 436 neonates with acidemia, Di Pasquo et al[116] have shown that using the physiological classification of the CTG, the frequency of adverse outcomes seems related to the duration and the type of the hypoxic injury, being higher in fetuses showing features of antepartum chronic hypoxia.

Intrapartum fetal monitoring: back to the future and undoing the past

In the past, guidelines were developed concentrating on the morphology of decelerations as determinants of fetal

TABLE 6
Intrapartum journey planner: the "6 P's"

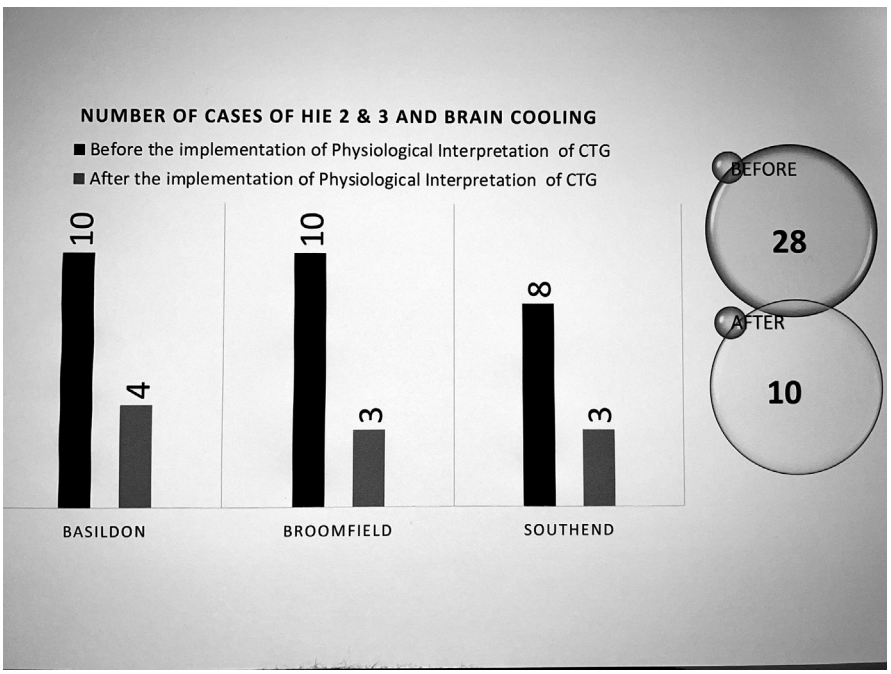
6 P's	Feature
Problems (meconium, oxytocin-induced hyperstimulation, infection, maternal pyrexia, relative uteroplacental insufficiency, and unfavorable maternal environment, such as diabetic ketoacidosis)	The cardiotocography trace forms an important part of this overall "intrapartum journey planner" to determine whether it is appropriate to expose the fetus to continuing uterine contractions and resultant intermittent reduction in fetal oxygenation.
Parity	
Power (spontaneous labor vs induced or augmented labor)	
Progress of labor during the last 4 h	
Passenger: too large, small, malposition, relative cephalon-pelvic disproportion with caput or molding)	
Predicted time of birth	

Jia. Fetal heart rate tracing interpretation in clinical practice. Am J Obstet Gynecol 2023.

well-being. In some countries, the paper speed was increased to 3 cm/min (Figure 4) as it was believed that this will help identify late decelerations. However, this approach of concentrating on the decelerations missed the recognition of fetal response to stress and the types of fetal hypoxia. If the same CTG trace (Figure 4) is run at 1 cm/min, it becomes easier to identify the baseline FHRV (Figure 6) and perceive a change when it gets progressively reduced. It has been reported that 31% of fetuses born with acidemia had a category I tracing 30 minutes before birth.[117] This illustrated the limitations of the current guidelines attempting to group certain features of the FHR tracings into different categories to prevent neonatal acidemia.

A journey of a human fetus, which takes approximately 8 to 10 hours should not be boxed into categories based on arbitrarily chosen parameters. CTG guidelines and classification systems should not be different in different countries and regions, as the physiological responses of a human fetus are the same irrespective of where the mother goes into labor. A deeper understanding of physiological responses to hypoxic and mechanical stresses and the type of hypoxia will help avoid unnecessary intrapartum interventions while reducing the rate of HIE and perinatal deaths. In all types of fetal hypoxia, except in cases of uterine rupture and placental abruption with maternal hypotension and tachycardia (ie, risk of cardiac arrhythmia) or maternal cardiac disease, intrauterine resuscitation with tocolysis should be always considered to improve uteroplacental oxygenation (ie, intrauterine resuscitation).[8,16,45,54,64,118,119] It is important to appreciate that FHR tachycardia may occur, which is usually more marked above 160 bpm, following a more recent insult of the CNS insult. However, the FHR may be normal (110−160 bpm) or with a marginal tachycardia when the injury to the CNS occurs several

FIGURE 14
Reduction n the rate of HIE (categories II and III) at Basildon, Broomfield, and Southend University Hospitals (approximately 12,500 births per year)



CTG, cardiotocography; *HIE*, hypoxic-ischemic encephalopathy.

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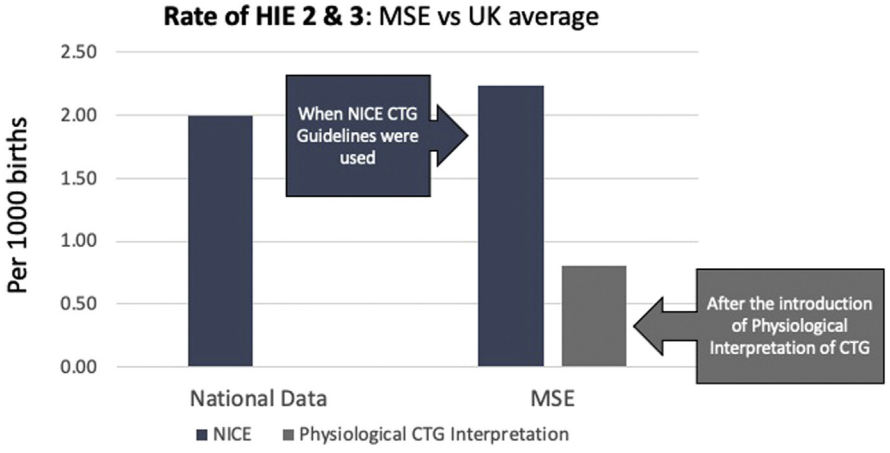
FIGURE 15
Rate of HIE at Mid and South Essex NHS Foundation Trust, United Kingdom, after the implementation of physiological interpretation of CTG



CTG, cardiotocography; *HIE*, hypoxic-ischemic encephalopathy.

Jia. Fetal heart rate tracing interpretation in clinical practice. Am J Obstet Gynecol 2023.

weeks earlier. The latter pattern represents a static encephalopathy, and there will not be even any shallow decelerations as the fetus has lost every capacity to respond, and this static encephalopathy cannot be modified significantly by aggressive peripartum operative interventions.[120] ■

TABLE 7
Maternity units demonstrating >50% reduction in the rate of intrapartum hypoxic-ischemic encephalopathy after the implementation of physiological interpretation of cardiotocography, which was first developed at St George's Hospital, London

Maternal units
Basildon University Hospital, Essex, United Kingdom
Broomfield Hospital, Essex, United Kingdom
Glangwili Hospital, Hywel Dda University Health Board, Wales, United Kingdom
Kingston Hospital, London, United Kingdom
Lister Hospital, Stevenage, United Kingdom
Newport Hospital, Wales, United Kingdom
Peterborough City Hospital, United Kingdom
South Warwickshire Hospital, United Kingdom
Southend University Hospital, Essex, United Kingdom
Sultan Qaboos Hospital, Salalah, Oman
University Hospital Limerick, Republic of Ireland

Jia. Fetal heart rate tracing interpretation in clinical practice. Am J Obstet Gynecol 2023.

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