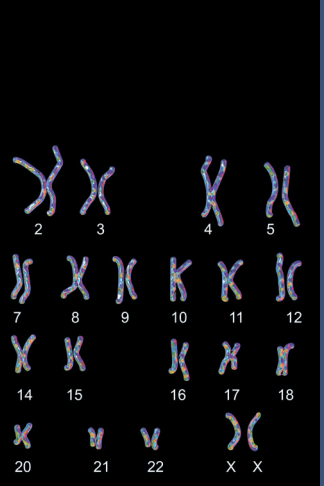

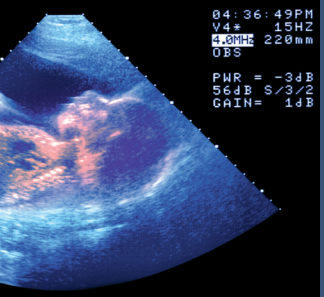

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Fetal Compromise in Labor

Mark I. Evans, Lawrence D. Devoe and Philip J. Steer

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FETAL COMPROMISE IN LABOR

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Abstract: Sixty years ago, the purpose of introducing electronic fetal heart rate monitoring (EFM) was to reduce the incidence of intrapartum stillbirth. However, by the early 1980s, with falling stillbirth rates, fetal blood sampling had been widely abandoned, as many considered that EFM was sufficient on its own. Unfortunately, while the sensitivity of EFM for the detection of potential fetal compromise is high, specificity is low, and there is a high false positive rate which has been associated with a rising cesarean section rate. The authors suggest that EFM is considered and analyzed as a classic screening test and not a diagnostic test. Furthermore, it requires contextualization with other risk factors to achieve improved performance. A new proposed metric, the Fetal Reserve Index, takes into account additional risk factors and has demonstrated significantly improved performance metrics. It is going through the phases of further development, evaluation, and wider clinical implementation.

Keywords: electronic fetal monitoring, Fetal Reserve Index, cerebral palsy, fetal acidosis, cesarean delivery rate

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Introduction

In previous editions of the book *High-Risk Pregnancy: Management Options*, this Element was published as a chapter titled 'Fetal Distress'. This is a term that is still commonly used, but it has always been difficult to define, leading to significant subjectivity in its use, and its retrospective attribution as a "diagnosis" when neonatal outcomes are suboptimal. For many years the term "fetal distress" has been taken to indicate the presence of hypoxia, leading to fetal acidosis. However, it has become clear that other clinical variables such as maternal/fetal temperature[1], chorioamnionitis[2], and passage of meconium into the amniotic fluid (which can lead to meconium aspiration syndrome) [3] can adversely affect the fetus during labor. External events can also contribute to fetal compromise, including trauma, cord prolapse, and head compression (which can occur from excessive molding even in spontaneous labor, but is more commonly associated with forceps and difficult cesarean deliveries)[4].

More recently, it has been demonstrated that formal addition of maternal, fetal, and obstetric risk factors, as well as the level of uterine contractility, can provide a contextualized evaluation of fetal heart rate (FHR) patterns and improve our ability to predict and possibly prevent poor perinatal outcomes. This approach requires a "paradigm shift," however, to conceptualize electronic fetal heart rate monitoring (EFM) or cardiotocography (CTG) as just one of the many screening tests commonly used in obstetrics. The concept of a "screening test" is widely appreciated in medicine and even in antenatal diagnosis, but CTG has yet to be properly appreciated as a screening test, rather than as a diagnostic test.

Simple, all-inclusive terms such as "fetal distress" should therefore be avoided. "Fetal distress" does not distinguish minor and inconsequential factors from catastrophic ones, or indicate the precise nature of the fetal compromise [5]. Such usage is similar to labeling everyone in an adult intensive care unit as being "ill" irrespective of whether they have cardiovascular, neurological, traumatic, or infectious problems. For this reason, this Element is retitled *Fetal Compromise*. It will address in turn the various factors which can lead to fetal compromise, both separately and in combination.

Monitoring and evaluating fetal well-being during labor are difficult, mostly because there is limited access to the baby. The easiest parameter to measure is the FHR. The first reported auscultation of the fetal heart sounds was by the French physician Marsac in the seventeenth century, then in 1818 by Francois Mayor, a Swiss surgeon, and in 1821 by a French nobleman, Jean-Alexandre Le Jumeau, Vicomte de Kergaradec. Each physician independently confirmed the audible beating of the fetal heart. An essay on "obstetric auscultation, or means

of detecting life or death of a fetus before birth" by Evory Kennedy of Dublin was published in 1834. By 1906, Cremer had described the detection of the fetal electrocardiogram (ECG), using electrodes placed on the mother's abdomen and in her vagina. However, this signal was weak and usually overwhelmed by electrical activity produced by the mother's rectus muscles. It was not until the 1960s that Edward Hon introduced a method using a fetal scalp electrode passed through the cervix which could produce a sufficiently large and clear signal for continuous intrapartum monitoring of the FHR. Using a less invasive approach, the first commercial "fetal monitor" (cardiotocograph [CTG]), designed by Konrad Hammacher in Germany and introduced commercially by Hewlett-Packard, initially used phonocardiography (picking up the fetal heart sounds with a microphone). Hon's pioneering work led to the option of monitoring the fetal heart using the ECG obtained via a fetal electrode. Doppler ultrasound to detect the fetal heart movement via the maternal abdomen was introduced in 1968 by a British company ("Sonicaid"). This approach became widely used in the 1970s.

Despite 50 years of increasingly sophisticated fetal heart signal processing, pulse rate alone cannot make a definitive diagnosis of fetal status. In an intensive neonatal care setting after birth, the medical staff monitor several physiological variables in addition to the heart rate, that include pulse oximetry, respiratory rate, and blood pressure. When pediatricians assess the initial condition of the neonate following delivery, they rely upon multiple measurements, including heart rate, respiratory effort, neurological performance (tone, reflex irritability), and peripheral circulatory function (color). Together, these measurements make up the Apgar score, which once was widely considered as the gold standard measure of "birth asphyxia"[6], and low scores were used to indicate hypoxia and acidosis[7]. However, as early as 1967, Beard and coworkers pointed out that the Apgar score "does not differentiate between asphyxial and non-asphyxial depression of the newborn"[8].

In 2005 the American College of Obstetricians and Gynecologists (ACOG), in a guideline decrying the inappropriate use of the terms "fetal distress" and "birth asphyxia," defined birth asphyxia as "intrapartum hypoxia sufficient to cause neurological damage"[9], which required all of the following four features to be present before such a diagnosis could reasonably be made:

- umbilical artery cord blood pH < 7.00
- 5-minute Apgar score ≤ 3
- moderate or severe neonatal encephalopathy
- multiorgan dysfunction (e.g. cardiovascular system [CVS], renal, pulmonary)

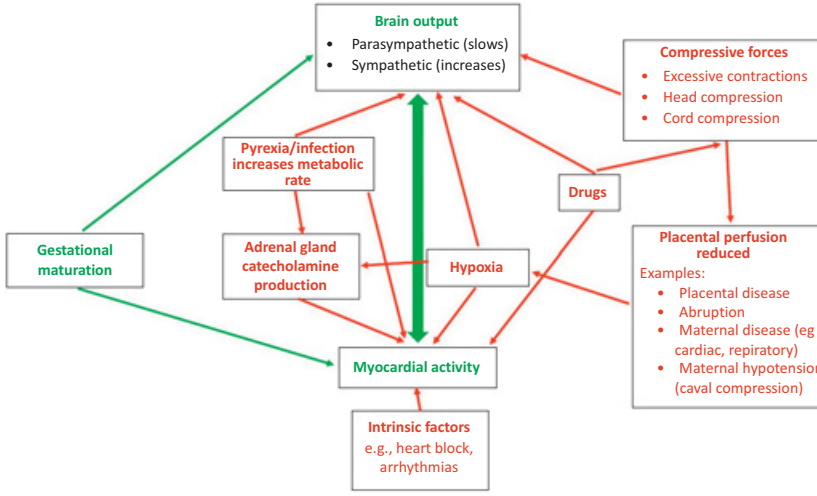

Figure 1 Factors influencing the fetal heart rate.

Since then, the complexity of defining "birth asphyxia" has become even more apparent, leading to a move to avoid the expression altogether (the 2005 guideline has since been withdrawn). For example, in 1982, Sykes and colleagues pointed out that there was a poor correlation between acidosis at birth (which they defined as an umbilical artery pH < 7.1 and base deficit >13 mmol/L) and a low Apgar score (only 27% of those babies with acidosis had a 1-minute Apgar score < 7, and only 21% of those with a 1-minute Apgar score < 7 were acidotic)[10]. It was subsequently reported that most babies who were depressed at birth and required resuscitation were, in fact, not acidotic, nor did they have an abnormal FHR pattern in labor[11]. Instead, their depression was often due to anesthetics given to the mother, trauma, meconium aspiration, and/or other stressors including maternal fever and/or chorioamnionitis.

Thus, FHR pattern analysis alone is not sufficient to evaluate intrapartum fetal condition but must be combined with other clinical features such as fetal growth restriction, length of labor, presence or absence of meconium in the amniotic fluid, and/or whether the mother is pyrexial (Figure 1).

The Physiology and Pathophysiology of Heart Rate Patterns

Fetal heart rate alterations are predominantly mediated by two mechanisms [12][13]:

- reflex slowing of the heart due to firing of the vagus nerve
- slowing of the heart by direct myocardial depression by the generation of lactate from anaerobic metabolism (due to inadequate oxygen supply)

Increases in the FHR can be caused by fetal release of catecholamines and stimulation of the sympathetic nervous system, by an increase in temperature (and therefore metabolic rate), or by various cytokines (as, for example, with infection). In addition, metabolic and endocrine factors and alterations in cerebral blood flow can indirectly influence the FHR pattern by affecting the cardiovascular control center in the brain (situated in the medulla). Unfortunately, it can be very difficult to identify the various influences leading to a pathological change in the FHR. Clinically, the most common changes seen are variable decelerations (secondary to head or umbilical cord compression that trigger FHR slowing by the vagus nerve. When umbilical cord compression occurs, cardiac output is reduced in order to prevent a potentially damaging rise in intracranial pressure due to a sudden increase in peripheral resistance as blood flow through the cord is cut off). Other less frequent alterations in the FHR pattern are late decelerations (secondary to hypoxia) and tachycardia (most commonly due to pyrexia, but sometimes due to catecholamine release). These alterations are described in more detail below.

The normal ranges quoted in this Element have been derived from a large body of observational data and interpreted by expert opinion. These data show that a normal FHR pattern has a good negative likelihood ratio, i.e. when it is normal there is a very low chance of hypoxia (and therefore of acidosis), i.e. a high negative predictive value. In contrast, when features of the FHR recording historically associated with adverse fetal or neonatal outcomes such as prolonged or severe bradycardia, prolonged decreased variability, and variable or late decelerations are seen, they are still commonly associated with babies born in good condition (a low positive predictive value). Thus, many FHR abnormalities are actually "false positives," a conclusion that can only be made reliably in retrospect.

It is therefore clear that the CTG (cardiotocography: continuous electronic assessment of the FHR and uterine contractions) should be regarded as a classic screening tool, and not a diagnostic test. Intrapartum FHR abnormalities are common, and trigger interventions in 10–20% of monitored labors. In contrast, severe perinatal asphyxia (causing death or severe neurological impairment) is very rare. When first introduced, CTG was designed to identify which patients should have a fetal blood sample (FBS) taken (usually from the scalp, occasionally from the buttock) to directly measure acid/base status. Despite, in retrospect (in our view) insufficient data, from the 1980s onward in most parts of the world, CTG interpretation alone was considered sufficient to predict acidosis, so FBS and pH estimation were widely abandoned. However, little attention was paid to the statistical performance metrics of CTG alone, i.e. how much using the CTG alone diminished the accuracy in predicting the neonatal condition.

There is no conclusive scientific evidence that the currently advocated normal ranges of multiple variables such as pH, Base Excess, PO_2, PCO_2, and others are the best ones on which to base clinical decision-making. The concept of "normal" is always liable to lead to an inappropriate metric when applied to physiological variables, because there is usually no sharp cutoff between "normal" and "abnormal." Instead, there is a Gaussian distribution around the mean value, such that the further a measurement is from the mean, the more likely it is to be associated with pathology. This is particularly true at the upper end of the range of FHR, where the likelihood of abnormality increases steadily within the range 150–180 beats per minutes (bpm). However, in this Element we have accepted the normal ranges recommended by the major clinical guidelines[14][15] as the "gold standard," although it could be argued that some of these ranges should be changed.

Baseline Fetal Heart Rate

The baseline fetal heart rate refers to the average recorded FHR after excluding accelerations and decelerations. It is calculated over a period of 5–10 minutes and is expressed in beats per minutes (bpm). Baseline FHR reflects the function of the fetal heart (myocardium) and the central nervous system centers (sympathetic and parasympathetic) and is modified by factors that act on the brain or the heart (e.g. β-sympathomimetic drugs). Therefore, a stable baseline FHR on a CTG trace (albeit with sufficient baseline variability, see below) generally reflects good oxygenation of the myocardium and the centers in the brain that control the heart rate. Although a wide range (110–160 bpm) is considered normal, baseline FHR varies from fetus to fetus, and therefore should be determined individually. Baseline FHR is higher in very early gestation and can be as high as 180 bpm at six weeks' gestation. The parasympathetic component of the central nervous system progressively matures with advancing gestation and decreases baseline FHR. Thus, a preterm fetus has a slightly higher average baseline FHR, due to unopposed activity of the sympathetic nervous system. However, most of this change has taken place by the beginning of the third trimester, and from 32 weeks onward there is no clinically significant change in the average or range of the baseline rate (Figure 2).

While some aspects of CTG should be interpreted in the moment, for example, a profound sustained bradycardia, most CTG assessments do not reflect "sentinel events" and require consideration of the trend of the baseline FHR over time. The fetal response to evolving intrapartum hypoxic stress involves a steady increase in catecholamine levels and therefore heart rate. For example, although a baseline FHR of 155 bpm is still within the "normal"

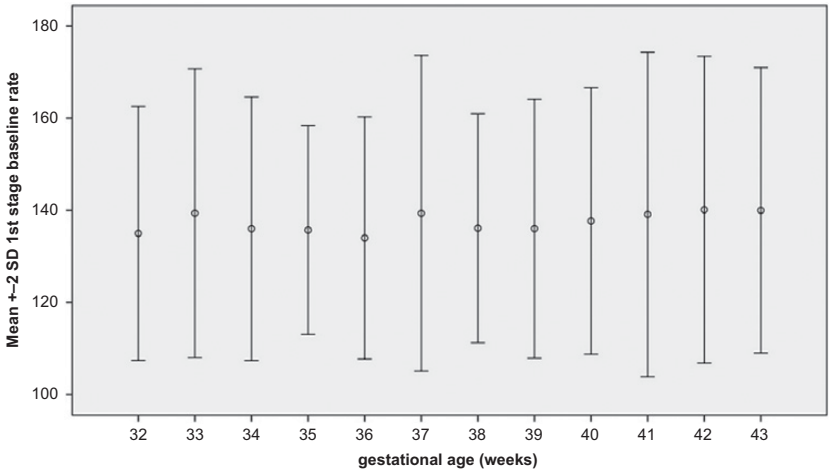

Figure 2 Mean FHR in the first stage of labor at gestational ages from 32 to 43 weeks. The range shown is ± two standard deviations (equivalent to the 2.3 and 97.7 percentiles). Data from the study by Steer PJ *et al*., *Obstet Gynecol* 1989; 74: 715–21[16].

range (100–160 bpm), an increase from a baseline rate of 110 bpm at the start of the CTG recording may reflect an ongoing stress response to hypoxia. A baseline tachycardia which is associated with preceding decelerations and/or a loss of baseline FHR variability should be appreciated as significant, and measures should be undertaken to improve fetal oxygenation whenever possible. Similar evolving patterns are seen with pH and Base Excess (BE) and will be addressed later.

Abnormalities of the electrical or conducting system of the heart may also lead to changes in baseline FHR (sinus tachycardia or atrioventricular heart blocks). A sudden and sustained fall in the baseline heart rate below 110 bpm is termed a prolonged deceleration. It may occur secondary to acute intrapartum accidents (placental abruption, umbilical cord prolapse, or uterine rupture) or due to correctable factors (maternal hypotension, umbilical cord compression, or uterine hyperstimulation). A prolonged deceleration persisting for more than 10 minutes is termed a baseline bradycardia.

A common error is to misinterpret a prolonged moderate bradycardia as a "wandering baseline." It should be remembered that fetuses (and adults) respond to stress with a tachycardia, and apparent falls in the "baseline rate" during labor should always be regarded with suspicion. The FHR at the end of a period of uterine relaxation (just before the next contraction) is often the best indicator of the true baseline.

Baseline Variability

Variation of the FHR above and below the baseline (often referred to as the "bandwidth") reflects the continuous interaction of the sympathetic and parasympathetic components of the central nervous system that regulate the FHR. Normal baseline variability of 5–25 bpm implies that these autonomic nervous system centers in the brain are not depressed and that fetal hypoxia is unlikely. However, this variability is not random, but has a specific undulating pattern in normal fetuses, with cycles every 15–20 seconds. When it becomes abnormally exaggerated, it can indicate hypoxemia[17] and predispose to the development of acidosis[18]. In some instances, this pattern can resemble a sinusoidal pattern, a so-called pseudo-sinusoidal pattern (Figures 3 and 4).

Loss of Variability

Moderate loss of variability is seen with acidosis secondary to hypoxia or metabolic conditions such as maternal ketoacidosis[19]. Complete loss of variability is associated with previous or ongoing brain damage[20], although it can occasionally result from other causes such as maternal exposure to depressant drugs (for example, magnesium sulfate[21], or occasionally opioids) (Figure 5).

Cycling

It is normal for a moderately reduced baseline FHR variability (i.e. < 5 bpm but > 2 bpm) to be seen for up to 40 minutes in the last trimester during quiet fetal

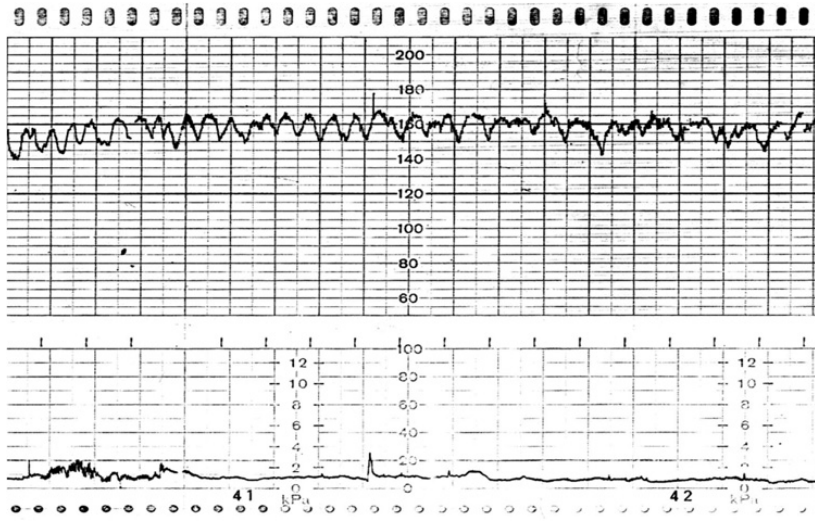

Figure 3 Pseudo-sinusoidal FHR pattern (tracing speed 1 cm/min).

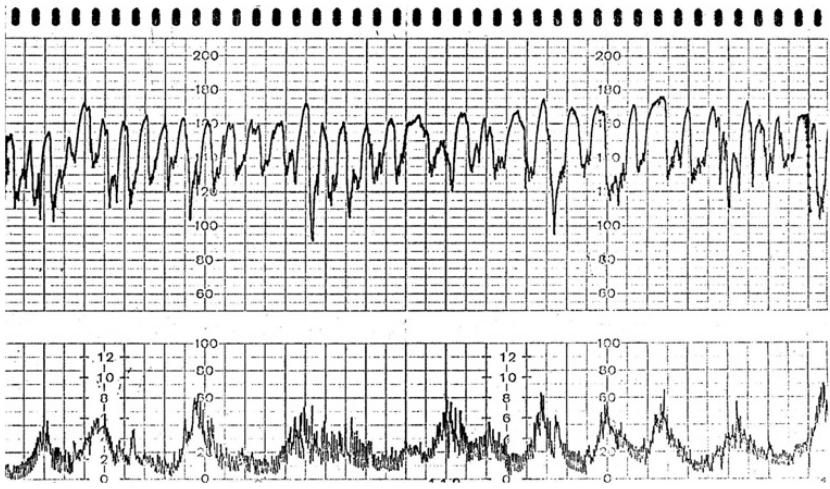

Figure 4 Exaggerated (abnormal) baseline variability (tracing speed 1 cm/min).

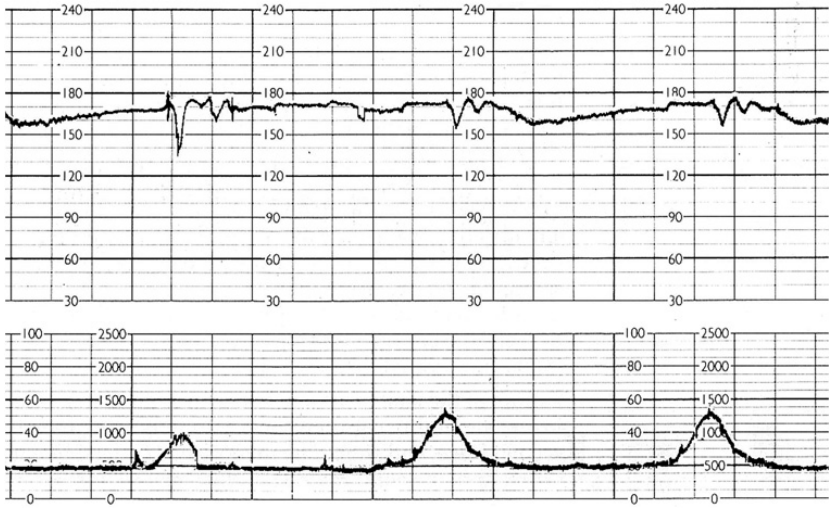

Figure 5 Severe loss of short-term baseline variability (tracing speed 1 cm/min).

sleep (quiescence or fetal behavioral state F1). In contrast, in active fetal sleep (fetal behavioral state F2) it is characterized by increased baseline variability in the presence of accelerations associated with fetal movement. The quiet/active fetal sleep states in mature fetuses alternate at intervals of 30–90 minutes. This alternating pattern is not usually seen before 28 weeks of gestation, and generally appears between 28 and 32 weeks[22][23] (Figure 6).

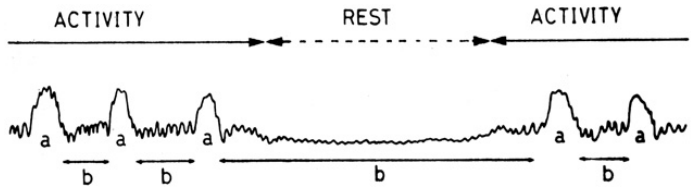

Figure 6 Activity rest cycles. From Wheeler T, Murrills A. Patterns of FHR during normal pregnancy. *Br J Obstet Gynaecol* 85:18–27, 1978[22].

Increased Variability

Increased baseline variability of > 25 bpm (the so-called "saltatory pattern") should be considered abnormal[17][18] (see Figure 4 above). This may be observed when intrapartum hypoxia evolves rapidly (e.g. secondary to oxytocin use or during active maternal pushing). Although its exact pathophysiology is uncertain, it has been suggested to be secondary to instability of the autonomic nervous system and represents an attempt by the fetus to maintain a stable baseline during a rapidly evolving hypoxic stress[24][25]. If a saltatory pattern is associated with atypical or late variable decelerations, immediate measures to improve uteroplacental oxygenation to the fetal brain (i.e. cessation of oxytocin infusion or maternal pushing) are recommended.

Accelerations

Accelerations are increases in the FHR of more than 15 bpm for more than 15 seconds (see Figure 6 above). Healthy preterm fetuses can show accelerations of lower amplitude and duration (10–15 bpm for 10 seconds). They are usually associated with fetal movements (but not directly due to movements as even paralyzing the fetus with a neuromuscular blocker does not prevent accelerations) and are therefore considered a reassuring sign. Absence of accelerations over a prolonged period is of uncertain significance when the rest of the FHR pattern is normal. However, in association with reduced baseline variability and decelerations, it is a marker for abnormal cerebral function. When the fetus becomes hypoxic, fetal body and breathing movements generally cease to conserve oxygen. Maternal "bearing down" (pushing efforts) in the second stage increases hypoxic stress; therefore, accelerations commonly disappear during the second stage of labor. A common error in the second stage is to interpret a rise in baseline rate between prolonged decelerations as accelerations.

As stated above, clear alternations between quiet and active fetal sleep patterns usually begin to appear between 28 and 32 weeks' gestation. Prior to

this gestational age range, the FHR pattern tends to exhibit somewhat reduced baseline variability when compared with the FHR patterns of more mature fetuses. Accelerations also tend to be less common. In contrast, it is not uncommon to see brief decelerations (lasting about 10 seconds or less) of relatively small amplitude (10–20 bpm) not associated with contractions. The etiology of these decelerations is not clearly understood. It has been suggested that they are due to traction on the umbilical cord because the fetus tends to be relatively more mobile earlier in gestation when the amniotic fluid volume is maximal compared with fetal volume. These decelerations are generally considered to be innocuous.

Decelerations

Slowing of the fetal heart below the baseline with a drop of at least 15 bpm and lasting at least 15 seconds is termed a deceleration. Decelerations are most commonly a fetal reflex response to compensate for a rise in intracranial pressure or blood pressure due to head compression or umbilical cord compression. Late decelerations generally indicate an hypoxic effect acting directly on the myocardium as well as a reflex change. Decelerations reflect the fetal compensatory response to maintain a positive energy balance of the myocardium during hypoxic stress, by reducing myocardial workload. Unlike a child or an adult who is exposed to external oxygen, a fetus is unable to increase oxygen supply to the myocardium by increasing the rate and depth of respiration.

Although different morphological types of decelerations have been described and have been used for classification of CTG traces, it is more important to understand the underlying pathophysiology of these decelerations and the fetal response to ongoing stress such as mechanical compression, uteroplacental insufficiency, or fetal hypertension, than to focus on precise classification.

Early Decelerations

True early decelerations are rare (0–2% of all decelerations) and are characterized by a uniform and repetitive slowing of the fetal heart, which begins at the start of the contraction, reaches its nadir at the peak of the contraction, and returns to its original baseline by the end of the contraction. They typically occur in the late first stage and second stage of labor in response to mechanical compression of the fetal head. As the dura mater is richly innervated by parasympathetic nerves, compression of the fetal head causes parasympathetic stimulation leading to slowing of the FHR and a reduction in fetal blood pressure, which serves to limit the rise in intracranial pressure. Direct stimulation of the cardiac inhibitory center in the brain may also occur during head

compression, and when the compression is relieved as the uterine contraction ceases, the heart rate returns to the baseline without any delay. Very small variable decelerations are impossible to distinguish from early decelerations.

Late Decelerations

These are characterized by a delayed recovery to the baseline heart rate after the cessation of uterine contractions, with the nadir at least 20 seconds after the peak of the contraction and a gradual return to baseline occurring after the end of the contraction (Figure 7).

Late decelerations are associated with uteroplacental insufficiency, leading to fetal hypoxemia, which stimulates the chemoreceptors (aortic and carotid bodies) situated in the arch of the aorta and the internal carotid artery. Stimulation of chemoreceptors by altered chemical composition of fetal blood (increased carbon dioxide, increased lactic acid, and/or low oxygen tension) triggers a vagal response leading to a drop in FHR. In addition, reduced perfusion of the myocardium eventually causes a switch from aerobic to anaerobic metabolism, resulting in a rise in lactic acid production. Just as is the case in skeletal muscle, a rise in lactate concentration inhibits myocardial contractility. As the uterine contraction ceases, freshly oxygenated blood from the placenta removes the ongoing stimulus to the chemoreceptors and improves myocardial oxygenation, and there is a gradual recovery of the FHR to the baseline.

Repetitive late decelerations are clinically ominous as they are usually associated with progressive fetal hypoxia which can lead to fetal metabolic acidosis. Interventions aimed at increasing the uteroplacental circulation (changing

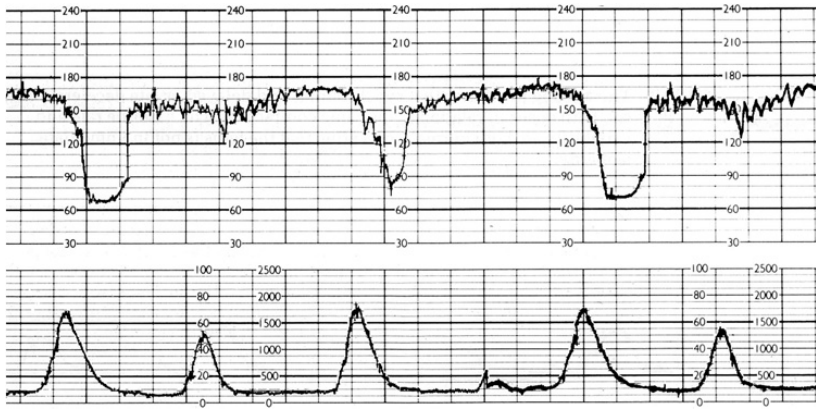

Figure 7 Late decelerations (tracing speed 1 cm/min). Note that the shorter contractions are associated with a temporary increase in heart rate variability, attributed to hypoxemia.

maternal position, administering intravenous fluids if there is any evidence of hypotension, stopping or reducing oxytocin infusions, and administering tocolytics if there is ongoing uterine hyperstimulation) may alleviate the problem and avoid an emergency delivery.

Variable Decelerations

Variable decelerations vary in shape, form, and timing in relation to the uterine contractions and primarily occur secondary to umbilical cord compression, although they are commonly synchronous with contractions. The umbilical cord is compressed to varying degrees and duration during each uterine contraction. At the onset of a uterine contraction, the umbilical vein is compressed before the umbilical artery, owing to its thinner wall and lower intraluminal pressure. This selective compression of the umbilical vein results in a reduced blood return to the fetus while blood is still being pumped to the placenta via the umbilical artery. Loss of fetal circulating blood volume and resultant hypotension lead to a transient increase in FHR (the initial "shoulder") as the fetus attempts to compensate for ongoing hypotension via activation of the sympathetic nervous system. As the uterine contraction gets stronger and reaches its peak, the umbilical arteries get compressed, leading to increased peripheral vascular resistance and fetal systemic hypertension. Stretching of the baroreceptors activates the parasympathetic center in the fetal brain which results in a swift drop in FHR, mediated via the vagus nerve. As the uterine contraction ceases, the umbilical arteries reopen, the systemic hypertension rapidly normalizes; the heart rate then swiftly returns to its original baseline. Continuing compression of the thin-walled umbilical vein after the umbilical arteries have opened up once more causes systemic hypotension secondary to fetal hypovolemia, which results in a second transient increase in FHR (the second "shoulder") as the fetus again attempts to compensate for ongoing hypotension via activation of the sympathetic nervous system.

Variable decelerations usually last for less than 60 seconds, and the amplitude of the drop is usually less than 60 bpm from the baseline. As they are due to umbilical cord compression rather than hypoxia, they are usually associated with a stable baseline within the normal range and good baseline variability.

However deep and prolonged, variable decelerations result in a reduction in overall cardiac output (as shown by measurements of "dip area"[26][27]), which can lead to secondary hypoxia and acidosis, a release of catecholamines, and a rise in the baseline rate (Figure 8).

Typical variable decelerations reflect a protective reflex response against fetal systemic hypertension secondary to acute umbilical cord compression, and they

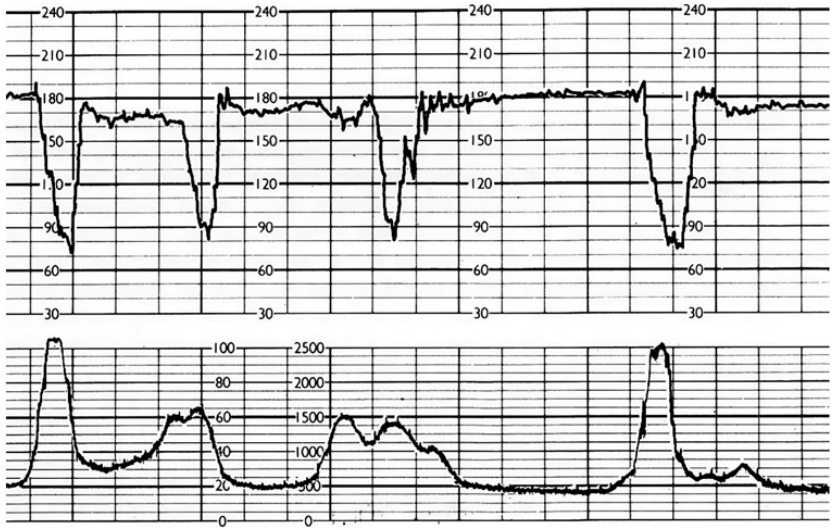

Figure 8 Large variable decelerations (tracing speed 1 cm/min).

can continue for a surprising length of time before fetal hypoxia develops. Although an arbitrary cutoff limit of 50% of contractions for 90 minutes has been suggested by some national guidelines, one needs to appreciate that if the intervening baseline FHR and variability are reassuring, fetal hypoxia is unlikely even if variable decelerations continue for longer than 90 minutes. Conversely, a growth-restricted fetus may show an increase in intervening baseline rate and/or a reduction in baseline FHR variability, necessitating an intervention to relieve umbilical cord compression before 90 minutes. As with the heart rate, taking into account the ongoing clinical context and specific risk factors provides a more accurate assessment of the overall clinical situation.

Complicated or atypical variable decelerations reflect an intense and prolonged umbilical cord compression. Increasing depth of variable decelerations (> 60 bpm) reflects the intensity of umbilical cord compression and resultant systemic hypertension, and increasing duration reflects the prolonged duration of umbilical cord compression or the presence of concurrent pathologic conditions, e.g. umbilical cord compression and a reduction in uteroplacental circulation occurring simultaneously in a growth-restricted fetus with oligohydramnios and uteroplacental insufficiency.

As the second stage of labor usually requires maternal expulsive effort, it is often associated with an abrupt increase of maternal heart rate during the contractions and active pushing. Therefore, "accelerations" seen on a CTG trace during the second stage of labor need to be viewed with caution, as it is very likely that the maternal heart rate (usually from maternal iliac vessels)

rather than FHR is being detected by the abdominal transducer (in fact, accelerations of the FHR are uncommon in the second stage). Features of a switch to maternal heart rate recording include a sudden drop in the baseline heart rate, an abrupt increase in baseline variability, and recurrent accelerations with larger amplitude (> 30 bpm) and duration (> 30 seconds) coinciding with uterine contractions. To reduce the likelihood of erroneous monitoring of maternal heart rate as if it were of fetal origin, the FHR transducer should be positioned in the midline rather than laterally during the second stage of labor. It is helpful to listen to the sound being generated by the Doppler signal from the ultrasound transducer. If the sound is simply "whoosh-whoosh" then this can be generated by any blood vessel, maternal or fetal. In this case, the transducer should be moved until the characteristic complex sound generated by the multiple structures in the fetal heart is heard (sometimes likened to the sound of a galloping horse). Autocorrelation techniques used in modern fetal monitors can decode this multiple signal into a single trigger for heart rate detection. As there are no other complex structures in the maternal abdomen capable of generating such a complex signal, its detection is a guarantee that the FHR is being recorded. Simultaneous recording of the maternal pulse rate with a pulse oximeter and the use of a fetal scalp electrode may also be useful, although if the fetus has a bradycardia and the mother has a tachycardia due to anxiety, the rates may be similar. If there is any doubt about the origin of the source being monitored, an ultrasound scan should be performed to confirm that the FHR signal is present.

Correlates of CTG Abnormalities

Gradually Evolving Hypoxia

Intrapartum hypoxic stress may evolve slowly over a number of hours, especially with the concomitant use of oxytocin to induce or augment labor. In such cases there may be sufficient time and adequate fetal physiological reserve for an effective compensatory response to the evolving hypoxic stress. The effectiveness of this compensatory response depends on the intensity and duration of the intrapartum hypoxia as well as the reserve of the individual fetus. The latter is modified by the maturity of the fetus, the presence of infection and/or meconium, whether growth restriction is present, and/or any preexisting prenatal insult. The rate of any deterioration will be accelerated if the mother is pyrexial. It should be remembered that the core temperature of the fetus is 0.8°C to 1°C higher than that of the mother and that the fetal metabolic rate and risk of metabolic acidosis is increased with maternal pyrexia[28][29].

The process of fetal deterioration is neither linear nor random. The first sign of any hypoxic or mechanical stress (such as head or cord compression) is a deceleration which occurs to reduce the myocardial workload. As hypoxia progresses, this compensatory process increases, with decelerations lasting long and becoming deeper. This delays the onset of anaerobic metabolism. The next feature is a loss of accelerations as the fetus reduces somatic nervous system activities such as movements, to conserve energy. If hypoxic stress continues, the fetus releases catecholamines from the adrenal glands to slowly increase the baseline heart rate in order to maintain oxygen supply to the vital organs (Figure 9). Catecholamines also cause intense peripheral vasoconstriction to divert oxygenated blood from nonessential organs (skin, scalp, and gut) and prioritize the supply to the heart and brain. Eventually, if hypoxia continues and if the compensatory mechanisms are unable to maintain a positive energy balance within the myocardium, decompensation will ensue with reduced perfusion of the brain characterized by the loss of baseline variability. Ultimately, myocardial decompensation will be manifested by a rapidly progressive reduction in the baseline FHR leading to a terminal bradycardia.

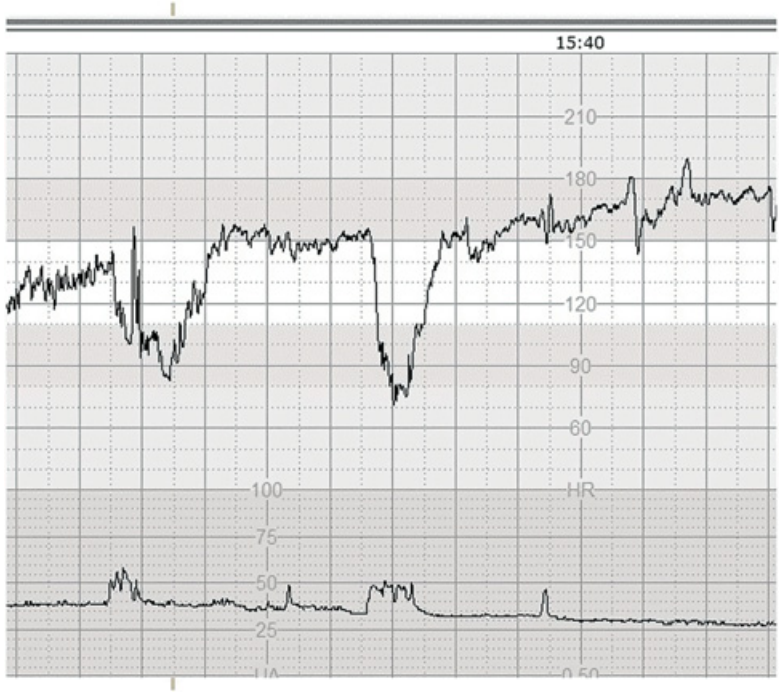

Figure 9 Evolving hypoxia. Note the progressively increasing baseline FHR secondary to the release of catecholamines.

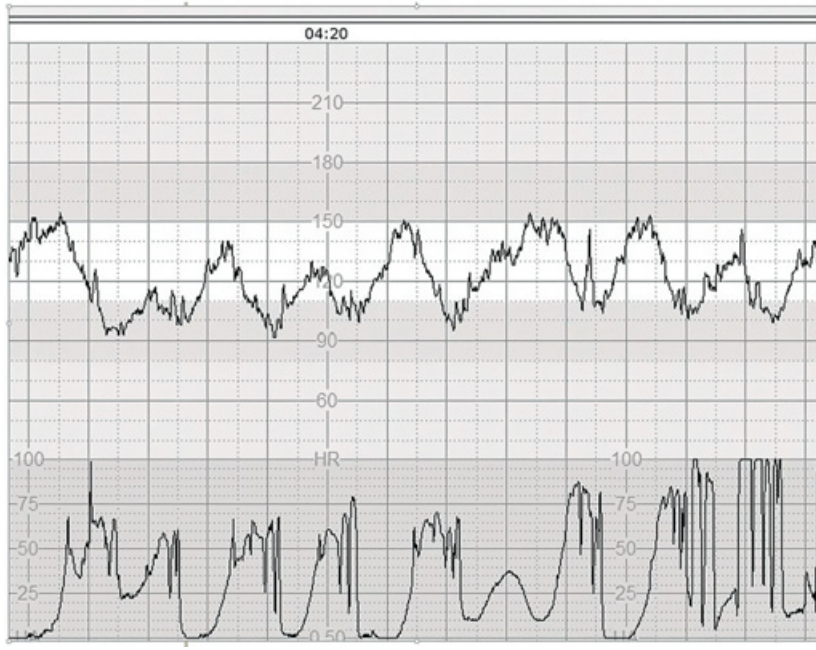

Figure 10 Subacute hypoxia. Note that the FHR spends less time at its normal baseline rate (150 bpm) than in decelerations, and the uterus is contracting for more than half the time (the inter-contraction interval is very short, thus reducing the time for oxygen transfer across the placenta). It would be quite easy to mistake the above tracing as having a baseline of 100–110 bpm with accelerations. The correct point at which to identify the baseline is just before a contraction, and in this case it is 150 bpm.

Subacute Hypoxia

This occurs when hypoxia evolves over 30–60 minutes and is most commonly caused by excessive use of oxytocin or active pushing during the second stage of labor. The FHR spends more time decelerating (> 90 seconds) and spends progressively less time at its normal baseline (< 30 seconds). Therefore, the time spent at the baseline, needed to eliminate metabolic waste products such as carbon dioxide and lactic acid and to enable effective reoxygenation, is progressively reduced. pH commonly will fall at an average rate of 0.01 every 2–3 minutes in the presence of a subacute hypoxic pattern on the CTG trace. Immediate actions should be taken to improve the uteroplacental circulation (Figure 10).

Traditional Diagnostics

Rational Application of Clinical Guidelines

A number of clinical guidelines have been produced by professional and national bodies worldwide (e.g. NICE[14], ACOG[15], and FIGO[30]) with different criteria and classifications. The lack of standardization exemplifies the ongoing controversy in relation to CTG interpretation based on "pattern recognition." While recognizing abnormal CTG patterns is necessary, it is not possible to make rational management decisions without considering pattern recognition within the context of the clinical situation and any possible underlying pathophysiology. For example, interpretation and management will differ substantially depending on whether the fetus is growth-restricted or normally grown, on the duration of active labor, and on the presence of complicating factors such as pyrexia, chorioamnionitis, or meconium staining of the amniotic fluid. This comprehensive approach is used in the Fetal Reserve Index and the fetal risk scores to be described later[31][32][33].

General Approach

If the CTG shows reassuring patterns in all four major domains (a reassuring baseline FHR and normal variability, presence of accelerations, and absence of any decelerations), fetal hypoxia is unlikely (less than 2%[34]) (Figure 11).

If there are decelerations, it is important to take steps to improve the intrauterine environment (turning the mother to the left lateral position, correcting any hypotension, stopping oxytocin infusion).

Assessing the fetal response to a hypoxic stress can be refined by examining the CTG trace between ongoing decelerations. If it shows a stable baseline FHR (between 110 bpm and 160 bpm) and reassuring variability (5–25 bpm), then the central organs (myocardium and brain) are probably still well oxygenated, and the chance of fetal acidosis is low.

If there is a rise in baseline FHR without any preceding decelerations, hypoxia is unlikely and other causes such as chorioamnionitis or maternal conditions (dehydration, pyrexia, infection, or even drugs such as pseudoephrine used as a maternal nasal decongestant) should be considered.

If there is an increase in baseline FHR with preceding decelerations and loss of accelerations, a gradually evolving hypoxia is more likely. Immediate measures to improve fetal oxygenation (reducing or stopping oxytocin, changing maternal position, or administering intravenous fluids) should be considered. If the interventions are effective, then the FHR should return to its original baseline.

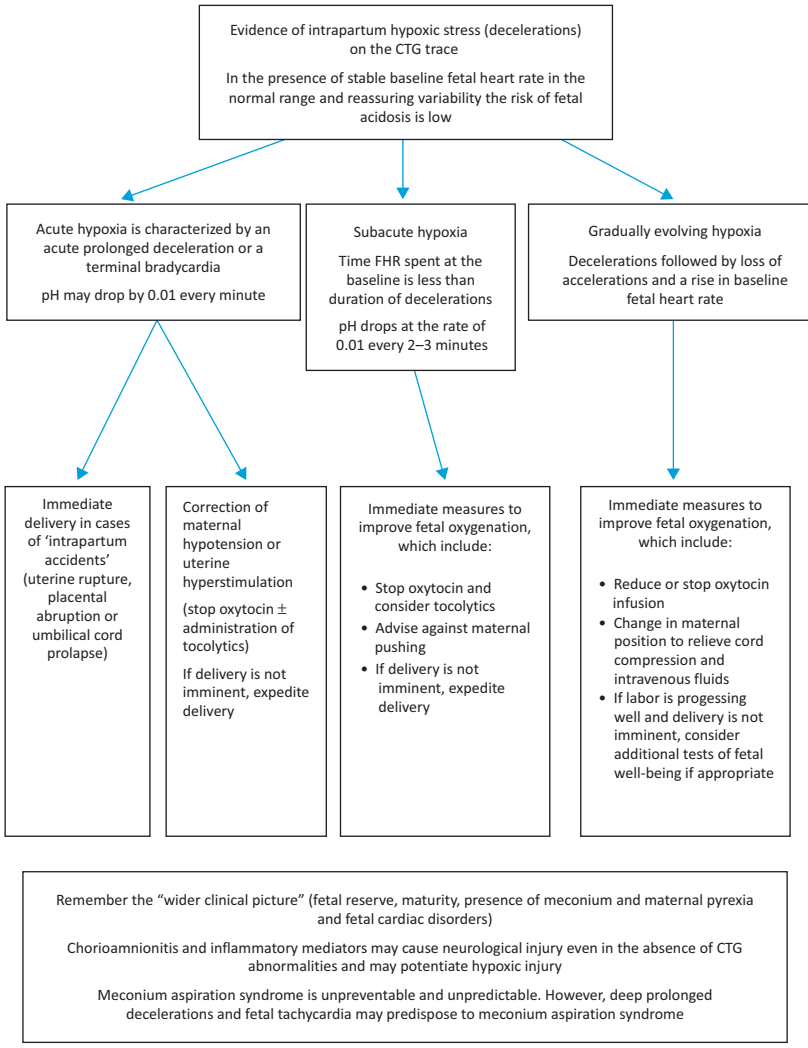

Figure 11 Algorithm for the management of fetal compromise in labor.

Diminished baseline variability, without any preceding decelerations or any increase in baseline heart rate, is likely to be due to a nonhypoxic cause such as quiet fetal sleep (i.e. F1), maternal drug administration (e.g. opioids), or, very rarely, fetal stroke due to intracranial hemorrhage or thrombosis. However, if such a decrease in baseline variability is preceded by decelerations and an increase in baseline FHR (due to catecholamine release), the onset of fetal decompensation following a gradually evolving hypoxia is likely. Immediate action to improve uteroplacental circulation should be undertaken, and if this is not possible, urgent delivery should be accomplished by the safest and the quickest route.

It is always important to interpret FHR patterns in their clinical context. For example, the growth-restricted fetus will have reduced reserves to survive hypoxic insults. The degree of reduced reserves generally depends on the degree of growth restriction. This means that the fetal buffering capacity and glycogen storage will restrict its ability to cope with ongoing hypoxia. Accordingly, the process of lactate generation and drop in pH in the presence of an abnormal FHR pattern will be more rapid than it would be in a well-grown fetus. There is no precise cutoff point of birthweight centile below which such problems occur, instead the rate of adverse outcome increases progressively with the reduction in fetal growth[35]. Moreover, a fall in fetal growth velocity toward term indicates increased risk even in babies above the tenth centile[36] [37]. Madden *et al*. in 2018 reported that babies at less than the fifth centile were almost twice as likely to be acidotic at birth, and almost four times as likely to die in the perinatal period than appropriately grown for gestional age (AGA) babies[38]. These findings question the validity of fixed criteria for the duration of an abnormal FHR pattern when assigning significance to them, because while growth-restricted babies develop acidosis rapidly when exposed to hypoxia, appropriately grown babies exposed to similar hypoxia can maintain their acid-base balance in a normal range for several hours[39]. It is essential that, at the onset of labor, a careful assessment of fetal growth/size (and by implication, metabolic reserve) is made, and the threshold for intervention adjusted accordingly.

In high income countries, maternal fever is most commonly due to the use of epidural anesthesia. However, it can also be due to chorioamnionitis, which should be suspected if there is at least one additional criterion (maternal leukocytosis, maternal and/or fetal tachycardia, uterine tenderness or foul-smelling discharge). The main FHR abnormality associated with fever is a tachycardia[40]. In one study of diagnosed chorioamnionitis, variable decelerations and loss of FHR cycling were noted in over 90% of cases, and 47% of cases showed what has been termed as pseudo-sinusoidal variability[40]. Chorioamnionitis is not, by itself, an indication for emergent delivery. The recommended management is the prompt administration to the mother of appropriate intravenous antibiotics such as amoxicillin and gentamicin[41]. These antibiotics are transferred across the placenta and also provide appropriate treatment for the fetus. Opinion number 712 of the ACOG committee (August 2017, reaffirmed 2022) suggests that "timely maternal management together with notification of the neonatal health care providers will facilitate appropriate evaluation and empiric antibiotic treatment when indicated. Intra-amniotic infection alone is rarely, if ever, an indication for cesarean delivery."[42]

If chorioamnionitis is suspected and the FHR pattern becomes abnormal (particularly if it involves a tachycardia), then the baby can become fatally compromised before there is any substantial drop in pH[43]. Interpretation of the FHR pattern also needs to consider the gestational age of the fetus.

The risk of a poor outcome in association with any given abnormal FHR pattern is substantially increased if there is also heavy meconium staining of the amniotic fluid. This is because perinatal hypoxia stimulates fetal gasping, which can lead to the meconium aspiration syndrome.

The Role of Uterine Contractions (the "Stress Factor")

Cardiotocograph interpretation should always include consideration of the frequency, strength, and duration of uterine contractions. Excessive uterine contractions may result in repeated and prolonged compression of the umbilical cord and/or the fetal head, as well as reducing the time available for reperfusion of the intervillous space with fresh oxygenated blood, leading to abnormalities in the FHR pattern. In spontaneous labor, fetal oxygen saturation is at its lowest 90 seconds after the peak of the contraction, and it takes approximately 90 seconds after a uterine contraction for it to return to its original level[44].

Uterine tachysystole refers to an excessive frequency of uterine contractions (now considered as > 4 in 10 minutes). Historically, the threshold for the diagnosis of excessive frequency was commonly > 5 in 10 minutes, which many consider to be too high[45][46], and although ACOG still considers up to 5 per 10 minutes averaged over a 30-minute period to be normal, the 2022 recommendations from NICE recommend that a safer upper limit is 4 in 10 minutes[14]. Persistent uterine tachysystole at a frequency of more than 4 contractions in 10 minutes causes a significant reduction in fetal oxygen saturation, especially if oxytocin is being used for induction or augmentation of labor [45]. Uterine hyperstimulation refers to any increase in the uterine activity (frequency, strength, or duration) that is associated with abnormalities in the FHR. Therefore, clinicians should be aware that even if the frequency of uterine contractions is < 5 in 10 minutes, if contractions last for more than 60 seconds or are unusually strong, there may be a reduction in fetal oxygenation leading to hypoxia and acidosis. It has been reported that when oxytocin is used to augment labor, if the interval between contractions is less than 2.3 minutes, there is a rapid reduction in oxygenated hemoglobin and a rapid rise in deoxygenated hemoglobin in the fetal brain[47]. Therefore, in the presence of uterine hyperstimulation, steps should be immediately taken to reduce or stop oxytocin infusion (or removal of a suppository containing prostaglandin used for labor induction if it is still in the vagina). If CTG changes persist despite these initial

interventions, the infusion of tocolytic agents (e.g. terbutaline 2.5 μg per minute for 20 minutes, increasing every 20 minutes in steps of 2.5 μg per minute until contractions have ceased, not exceeding 10 μg per minute) can be used to suppress uterine activity and improve uteroplacental circulation to avoid hypoxic-ischemic fetal brain injury.

Fetal Scalp Stimulation

Digital stimulation of the fetal scalp may elicit an acceleration during a vaginal examination or fetal scalp sampling for pH/lactate estimation, and this normally reflects good fetal oxygenation. A meta-analysis of studies of intrapartum fetal stimulation tests concluded that digital scalp stimulation was better at predicting fetal acidemia than vibroacoustic stimulation or fetal scalp puncture[48]. Absence of accelerations during digital scalp stimulation had approximately a 15% positive predictive value for fetal acidemia[48].

Therefore, in the presence of a pathological CTG trace, if an acceleration is noted during digital stimulation, then the likelihood of fetal acidosis is low. However, if there is no acceleration during digital stimulation, then the CTG trace should be carefully scrutinized to determine the stability of the baseline FHR and its variability. If appropriate, FBS and pH estimation can be considered. When there is loss of baseline variability with preceding decelerations and a rise in baseline FHR, delivery should be expedited.

Fetal Blood Sampling and pH/Lactate Estimation

When CTG was first introduced, it soon became apparent that many fetuses displayed intrapartum FHR patterns that could not be classified as reassuring, and yet upon delivery they showed no evidence of hypoxia or acidosis (Figure 12). Fetal blood sampling with pH estimation was introduced in the 1960s as a method of detecting fetal acidosis, prior to the use of CTG. It subsequently seemed logical that if the CTG pattern suggested fetal acidosis, then this could be checked by taking a FBS and measuring its pH. While a normal pH in a FBS largely excludes significant acidosis (high negative predictive value), a low pH still has a low positive predictive value as it can be due to compromise of the peripheral circulation in the absence of any central acidosis. For disorders with low incidence, even a screening test with good sensitivity will likely have a low positive predictive value. Moreover, measurement of fetal pH is often not relevant in many cases where the threat to the fetus is not primarily of hypoxia or acidosis. Examples include chorioamnionitis, a mechanical problem such as cephalopelvic disproportion, or meconium in the amniotic fluid.

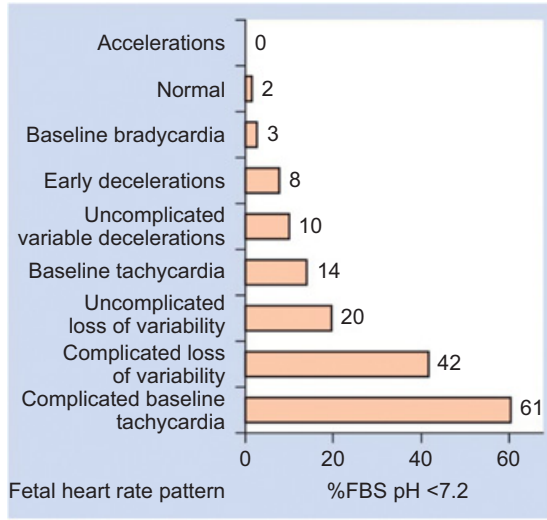

Figure 12 FHR pattern and the associated risk of acidosis. FBS, fetal blood samples. Data from Beard RW, Filshie GM, Knight CA, Roberts GM. The significance of the changes in the continuous fetal heart rate in the first stage of labor. *J Obstet Gynaecol Br Commonw* 1971; 78: 865–81.

Fetal Blood Sampling

Position

The lithotomy position should be avoided because of the risk of supine hypotension, which can result in iatrogenic hypoxia and acidosis in the fetus, leading to unnecessary operative delivery. The sampling is most comfortably performed with the woman in the left (or right) lateral position.

Procedure

Under aseptic conditions, an amnioscope is passed up the vagina to rest on the presenting part of the fetus. Sufficient pressure must be used to prevent sample contamination by excluding amniotic fluid. The fetal skin is then dried with a dental swab in a holder and sprayed with ethyl chloride. The evaporation of the ethyl chloride cools the skin, and as it warms up again a reactive hyperemia is produced, which promotes bleeding. The skin is smeared with a water-repellent gel (often silicone) so that when the skin is stabbed with a guarded 2 mm blade, a droplet of blood forms. This droplet is allowed to flow into a pre-heparinized thin glass tube by capillary action (it helps to tilt the tube slightly downward at the operator's end). Mouth-operated suction should not be used because of the risk of the operator ingesting potentially infected blood.

Analysis

A blood gas analyzer measures oxygen pressure (PO2), carbon dioxide pressure (PCO2), and pH, needed to calculate the base deficit. If the values are normal, but the FHR pattern remains abnormal, it will usually be necessary to repeat the sampling within 15–30 minutes. Alternatively, a lactate analyzer can be used to screen for metabolic acidosis[49]. It should be noted that unlike pH which is an absolute measurement, there are various techniques for measuring lactate and therefore normal ranges vary between different lactate analyzers, and this needs to be taken into account when interpreting the results[50].

If babies are born in poor condition and require resuscitation, it is important to establish whether this is being caused by metabolic acidosis. This diagnosis can be performed by taking blood from the umbilical cord promptly after birth. In some countries, it is recommended that such an assessment is made in all births, as a significant number of babies that are apparently normal at birth will be found to have a significant metabolic acidosis and this may require close monitoring of the neonate especially, for example, if the baby is growth-restricted.

Umbilical Cord Blood Gas Sampling

- A segment of cord is isolated between two sets of clamps after delivery.
- Changes in the pH, PCO_2, and PO_2 of cord blood occur slowly. Cord blood can be left at room temperature for up to one hour without significantly affecting the results and for several hours if left on ice.
- Commercially pre-heparinized blood gas syringes are used to take cord blood samples. Heparin is acidic, and if too much is used (> 10% of sample volume) it can cause significant errors.
- Blood should be taken from both artery and vein and the results checked to ensure both vessels have been sampled. If both results are very similar (≤ 0.02 pH units of one another), it is likely that the umbilical vein has been sampled twice (it is much easier to obtain a sample from the larger vein than the artery). If the venous pH is lower than the arterial pH, this is physiologically implausible, and it is likely that the samples have been inadvertently switched.
- Sample the cord vessels with the needle at an acute angle to the vessel, especially when sampling the umbilical artery. This makes it less likely that the needle has been inserted through the distal arterial wall into the vein.

Table 1 Normal values for umbilical cord blood sampling (mean, 5th, 95th centiles)

	Umbilical artery	Umbilical vein
pH	7.23 (7.1, 7.34)	7.32 (7.19, 7.43)
PO_2 (kPa)	2.9 (1.5, 4.9)	3.7 (2.1, 5.4)
PCO_2 (kPa)	6.1 (4.2, 8.4)	4.9 (3.5, 6.9)
BDecf (mmol/L)	−7.7 (−14.5, −1.8)	−6.4 (−11.7, −1.6)

Gestational length has no significant effect on cord blood gas values. BDecf, base deficit of extracellular fluid; PCO_2, carbon dioxide pressure; PO_2, oxygen pressure. From Eskes TK, Jongsma HW, Houx PC. Percentiles for gas values in human umbilical cord blood. *Eur J Obstet Gynecol Reprod Biol* 1983; 14: 341–6.

Analysis

- Normal values are given in Table 1.
- Table 2 illustrates the equivalent umbilical cord lactate level for a given pH value.
- Cord arterial values reflect fetal acid-base status, whereas those of the vein reflect maternal and placental status.
- The umbilical vein pH may be normal, but the arterial pH low, if there has been an acute interruption to umbilical blood flow (e.g. cord compression or if the fetal hypoxia has been of short duration).
- If both artery and vein have a low pH, the hypoxia is of longer duration and is usually due to metabolic acidemia.

Evidence of Benefit from CTG and FBS

Systematic reviews of CTG in labor have suggested that it reduces the incidence of intrapartum death due to hypoxia[51] and neonatal convulsions[52]. However, the numbers have been insufficient to address the question of whether the use of intrapartum CTG reduces the incidence of long-term damage such as cerebral palsy[53]. Moreover, FBS and pH estimation are cumbersome and time-consuming. Accuracy and convenience can be improved somewhat by measuring lactate rather than pH[54] because this requires much smaller samples (5 μL rather than 25 μL) and is not affected by air bubbles. However, the prognostic significance of lactate measurement is similar to that of pH. Because of the lack of robust scientific evidence of benefit, in the UK the 2022 (December 14) NICE guidelines no longer recommend the use of FBS and lactate/pH estimation to investigate cases of suspected fetal acidosis[14] as the evidence for its value is debatable. Although in some countries, particularly in Scandinavia, FBS is still

Table 2 Corresponding lactate values for umbilical cord pH (note that this is from a single study and the corresponding values for lactate can vary according to the machine used)

pH	Lactate
7.3	3.3
7.2	5.5
7.2	6.6
7.1	10
7.0	13.3

From Gjerris AC, Staer-Jensen J, Jørgensen JS, Bergholt T, Nickelsen C. Umbilical cord blood lactate: a valuable tool in the assessment of fetal metabolic acidosis. *Eur J Obstet Gynecol Reprod Biol* 2008; 139: 16–20.

used in about 5% of labors[55], it is considered reasonable and normal practice in many parts of the world (for example in the US) to abandon this procedure[15].

Intrauterine Resuscitation

If the FHR pattern suggests acute fetal hypoxia, it is important to try and correct this as rapidly as possible, while considering possible emergency delivery. The usual maneuvers comprise the following:

- Turning the mother to the left lateral position. This moves the uterus away from any possible compression of the inferior vena cava, thus avoiding supine hypotension, which can impair placental perfusion.
- Rapid infusion of 500 mL of crystalloid if there is any evidence of maternal hypotension. This is contraindicated in the presence of maternal cardiac disease.
- Reducing uterine hypercontractility by either stopping any infusion of oxytocin, and/or reducing the frequency and amplitude of contractions using a β-sympathomimetic such as terbutaline (subcutaneous dose of 0.25 mg, or intravenous infusion of 10–25 μg per minute). However, this drug is contraindicated in the presence of cardiac disease. While experience shows that this can sometimes produce a rapid improvement in the FHR pattern and may reduce the likelihood of emergency operative intervention, there is no evidence to support its use as an alternative to emergency delivery when this is indicated in cases of acute intrapartum accidents. However, a randomized controlled trial (RCT) concluded that tocolysis improved neonatal outcomes and reduced emergency interventions[56].

Indications for Continuous FHR Monitoring

This is a much-debated topic. It is controversial whether continuous electronic FHR monitoring should be used in all labors. In the absence of conclusive evidence from the previously performed RCTs, the UK guidelines support the use of intermittent auscultation with a Pinard stethoscope (or with a handheld battery-operated ultrasound Doppler device in situations such as maternal obesity, active birth, or birth in water pools), as long as there are no high-risk features in the pregnancy or labor[14]. There are many extensive lists of "high-risk features" that have been used to indicate the need for continuous intrapartum CTG, but none of them is comprehensive. It could be argued that the use of continuous CTG should remain a matter for clinical judgment.

In the event of a poor outcome, however, management is more difficult to defend without "hard evidence" from the CTG tracing of fetal well-being prior to delivery. In the United States, in part because of medicolegal concerns, continuous EFM is used in the vast majority of labors.

In the first stage of labor, the NICE guidelines recommend the birth attendant to "carry out intermittent auscultation immediately after a contraction for at least 1 minute, at least every 15 minutes, and record it as a single rate" (i.e. count the number of heart beats in 1 minute and record that number) [14]. Changing to continuous EFM is recommended if there is:

- suspected chorioamnionitis or sepsis, or a maternal temperature of ≥ 38 °C
- severe hypertension (≥ 160/110 mmHg)
- oxytocin use
- the presence of significant meconium
- fresh vaginal bleeding that develops during labor

In the second stage of labor, the NICE guidelines recommend that the birth attendant "perform intermittent auscultation of the FHR immediately after a contraction for at least 1 minute, at least every 5 minutes. Palpate the woman's pulse every 15 minutes to differentiate between the two heart rates"[14]. As explained previously, if the fetal and maternal heart rates are similar, this may not provide reliable differentiation. In the authors' experience, when using a handheld Doppler device to measure the FHR, it is important to ensure that the rate calculation is derived from the characteristic signals of the fetal heart (sharp and distinct, sometimes sounding like the hooves of a galloping horse), rather than to rely on the "whooshing" sound produced by reflections from blood vessels, which may be either fetal or maternal.

The Admission Cardiotocogram

Some practitioners have recommended performing a 30-minute CTG recording on admission of the parturient to the labor ward, and only converting to intermittent auscultation if this is normal. The efficacy of this approach has never been demonstrated by randomized trials of sufficient size, and therefore this remains a policy of unproven value.

Continuous CTG Monitoring Before 34 Weeks

The interpretation of the FHR pattern of the preterm fetus in labor is similar to that of its full-term counterpart. However, the FHR patterns present some subtle differences. Short-term baseline variability is often lower, and FHR accelerations are less frequent and smaller (the differentiation between quiet and active sleep patterns sometimes does not develop until 28–32 weeks' gestation). Small, brief (< 20 seconds) decelerations are often seen and are insignificant (cause unknown). Fetal blood sampling for pH estimation is contraindicated because of the increased risk of deep penetration of the scalp, resulting in excessive bleeding or even leakage of cerebrospinal fluid.

Limitations of CTG in Practice

Despite the worldwide use of CTG, the rate of neonatal neurologic impairment has not improved over the past 50 years despite a six-fold increase in the rate of cesarean deliveries. Even the most respected experts in the field have had to conclude that CTG as currently practiced has severe limitations. Steer and Lissauer reported in 1986 that 57% of babies requiring an expedited delivery had both a normal CTG and cord arterial blood pH[57]. There is only a weak association between umbilical cord gas values and neonatal outcomes as measured by the Apgar score[10][58]. Given that the commonly stated purpose of CTG is to predict hypoxia and acidosis, such conclusions are problematic. From the perspective of a screening test, the CTG has very poor performance metrics. Nevertheless, the current standard of care requirements will not abandon CTG even as practiced until there is something else that is clearly better.

Alternative Approaches

Computer Assessment of Fetal Heart Rate Patterns

For many years, it has been suggested that one of the problems with continuous CTG has been that some clinicians are either poorly trained in pattern recognition, or they have intrinsic difficulty with pattern recognition (an analogy is dyslexia and word recognition). For example, Ennis and Vincent in 1990 reported that in

14 of 64 cases of poor neonatal outcome reported to the UK Medical Protection Society, a CTG abnormality was not noticed, or ignored[59]. In 1991, Vincent *et al.* in a study of 41 cases of poor outcome notified to Action against Medical Accidents (UK) reported that "Inadequate fetal monitoring and insufficient supervision of junior doctors were implicated in a high proportion of accidents," and "some junior doctors and midwives cannot recognize abnormal CTG traces," and "most receive inadequate training in CTG monitoring"[60].

In 1998, the UK Confidential Enquiry into Stillbirth and Deaths in Infancy studied 567 cases of poor outcome and judged that there was substandard care in 72%, and that in 50% the poor outcome could possibly (28%) or probably (22%) have been prevented with better care[61]. They noted that "Fetal surveillance problems were the commonest cause [of problems in labor], with CTG interpretation … the most frequent criticism." A National Health Service Litigation Authority report in July 2009 said that misinterpretation of the CTG trace occurred in 34% of 100 stillbirth claims studied[62]. Thirty-three of the 39 clinicians involved were midwives or registrars (obstetricians in training) and "misinterpretation of a CTG trace was the most frequent example of negligence encountered in the study."

One potential solution to the failure to identify abnormal CTGs by junior clinicians is to use computer-assisted FHR pattern recognition. One of the first developments in this area was by Dawes and Redman who pioneered the development of a system (Oxford Sonicaid^{TM}) that was originally applied to antepartum FHR tracings to screen for fetal hypoxia and deterioration. It did not progress to use for intrapartum monitoring because it could not adequately differentiate normal from abnormal cases in that context[63]. Next, Devoe *et al*. developed a rule-based antepartum analytic system called NST-EXPERT which led to the development of a rule-based analytic system (TraceVue^{TM}) for FHR interpretation and alerting for FHR abnormalities that is still used worldwide, but studies showing that it led to improved perinatal outcomes are lacking[64].

Keith Greene and Robert Keith were early proponents of such an automated approach to FHR interpretation. They proposed an "expert system" to identify FHR pattern abnormalities[65]. A prospective randomized trial of their subsequent INFANT (INtelligent Fetal AssessmeNT) system commenced in January 2010, and finished in August 2013; the two-year follow-up was completed in 2015, and the results were published in *The Lancet*[66]. A total of 47,062 laboring women in whom continuous electronic FHR monitoring was indicated were randomized to decision (interpretation) support versus normal CTG monitoring. Both arms of the trial were run on the Guardian platform, which includes a central monitoring station for overview of the tracing by senior

staff. There was no evidence of a difference in the incidence of poor neonatal outcome between the groups: 0.7% (172) babies in the decision support group compared with 0.7% (171) babies in the no decision support group (adjusted RR 1.01, 95% CI 0.82–1.25). However, there were only three intrapartum stillbirths in the whole study (1/15,687 births). This compares with 1/2,200 births in the CTG arm of the 1985 Dublin trial[67] and 1/5,740 births in the UK in 2015[68]. The fact that the intrapartum stillbirth rate was seven times lower in the INFANT trial than in the Dublin trial suggests that over a quarter of a century care has dramatically improved such outcomes, although what has made the difference is not clear. The intrapartum stillbirth rate in the INFANT trial was also 2.7 times lower than in the UK as a whole, which may be attributable to a Hawthorne effect (in which individuals modify an aspect of their behavior in response to their awareness of being observed). Similarly, in the INFANT trial there were only 10 neonatal deaths (overall perinatal mortality 0.28/1,000 compared with 2.14/1,000 in the CTG arm of the 1985 Dublin trial and 0.39/1,000 in the UK in 2015). These data suggest that when well-trained clinicians assess the CTG frequently, fetal safety can be improved.

In the INFANT trial the standard of care was analyzed in 71 cases of stillbirth, neonatal death (28 days), or poor condition at birth plus metabolic acidosis (pH < 7.05 and base deficit ≥ 12 mmol/L). The overall incidence of grade 3 substandard care (better care likely to have avoided the poor outcome) was 38%. However, there was no difference between the decision support and control groups. The key failing was lack of attention to risk factors in addition to the abnormal CTG. For example, in the 71 cases, gestational age was > 41 weeks in 23%, low birth weight (< 2500 g) in 23%, maternal age > 35 years in 27%, BMI ≥ 30 in 32%. Fifty-four percent of labors were induced, and oxytocin was administered in 64%. Twenty-four percent of labors had meconium-stained amniotic fluid, and 11% of mothers were pyrexial. A key failing in many cases was failure to act promptly after a decision to deliver; in one case, the delay was more than 3.5 hours[69].

Another smaller trial of decision support also found that it did not improve perinatal outcomes[70]. The appropriate conclusion from these trials is that although it is clear that in a few (and often high-profile legal) cases there is a failure to recognize an FHR abnormality, if we are to reduce even further the commendably low current rate of poor perinatal outcome occurring during labor, we need to concentrate on improving recognition of the interaction of multiple risk factors such as fetal growth restriction, meconium passage, and mechanical problems in labor, and emphasize the importance of prompt response to them. This will require changes to the organization of care delivery to eliminate "system failures."

Hamilton and colleagues have developed a proprietary automated FHR analysis system (PeriCALM® Tracings™) sold in the United States. Its analytic software is based on ACOG Classification of FHR Categories and was visually validated by experts in FHR interpretation. It employs an artificial intelligence (AI) program to aid in the recognition of potentially pathologic EFM patterns and provides visual cues to alert caregivers to abnormal trends in FHR pattern and labor abnormalities. However, no large, randomized trials of this system have been conducted to date to investigate whether its use results in any improvement in neonatal outcomes[71].

A study of all the adverse outcomes (intrapartum stillbirth, early neonatal death, and severe brain injury, n = 1,136) in the whole of the United Kingdom throughout 2015 has been published by the RCOG – the "Each Baby Counts" study[68]. In the 727 cases with an adequate quality review, substandard care was judged to have occurred in 552 (76%). Fetal monitoring was a factor in 409 (74% of those with substandard care), including 115 cases (28%) involving intermittent auscultation. The review found that in addition to abnormal FHR patterns, there was an average of six additional high-risk features in each case, such as reduced fetal movements, fetal growth restriction, previous cesarean delivery, thick meconium staining of the amniotic fluid, suspected infection, and prolonged labor. The 2022 NICE intrapartum care guidelines emphasize that one should not make any decision about a woman's care in labor on the basis of cardiotocography alone, but also take into account maternal perceptions and observations, meconium or blood in the amniotic fluid, the frequency of contractions, the stage and progress of labor, the fetal response to digital scalp stimulation, and the results of FBS if undertaken (paraphrased for brevity)[14].

Role of the Fetal ECG (STAN)

The fetal ECG signal can be obtained from a fetal scalp electrode. Myocardial hypoxia causes changes in the ST segment and QRS complexes, and this test (fetal ECG or ST analysis) is aimed at measuring changes in response to intrapartum hypoxia in a central organ (fetal myocardium) rather than hypoxia in peripheral tissues[72]. The rationale is that the fetal myocardium is protected until the late stages of hypoxia by a fetal compensatory response through redistribution of available oxygen from peripheral organs to central organs.

Not only does ST analysis or STAN monitor the ST segment of a fetal ECG, it also computes the ratio of the height of the T wave to the height of the QRS complex (T/QRS ratio)[73]. This is because as the anaerobic metabolism sets in within the fetal myocardium, myocardial glycogen is broken down to

glucose to generate additional energy substrate to maintain a positive energy balance within the myocardium. During this process of glycogenolysis, the stored potassium within the glycogen is also released within the myocardial cells, leading to local hyperkalemia, which results in "tall" T waves and a rise in the T/QRS ratio, which, if significant, causes the computer to generate an "ST event" mark on the CTG[73] (Figure 13).

Once a decision has been made for continuous CTG, a fetal scalp electrode should be applied so that STAN monitoring can commence. The CTG trace prior to commencing STAN monitoring should have a stable baseline FHR and a reassuring baseline variability suggestive of good oxygenation of the central organs. This is because STAN technology works by determining the normal baseline ST segment and T/QRS ratio for the monitored fetus and then comparing subsequent ECG complexes with the calculated initial baseline measurements. Therefore, it is vital that the fetus retains its capacity to respond to hypoxia prior to beginning STAN monitoring. An unstable baseline FHR or reduced baseline variability indicates preexisting hypoxia of the central organs, while a preterminal trace already suggests a total loss of fetal compensation. Therefore, the fetus should be promptly delivered, and STAN monitoring is contraindicated in these clinical situations.

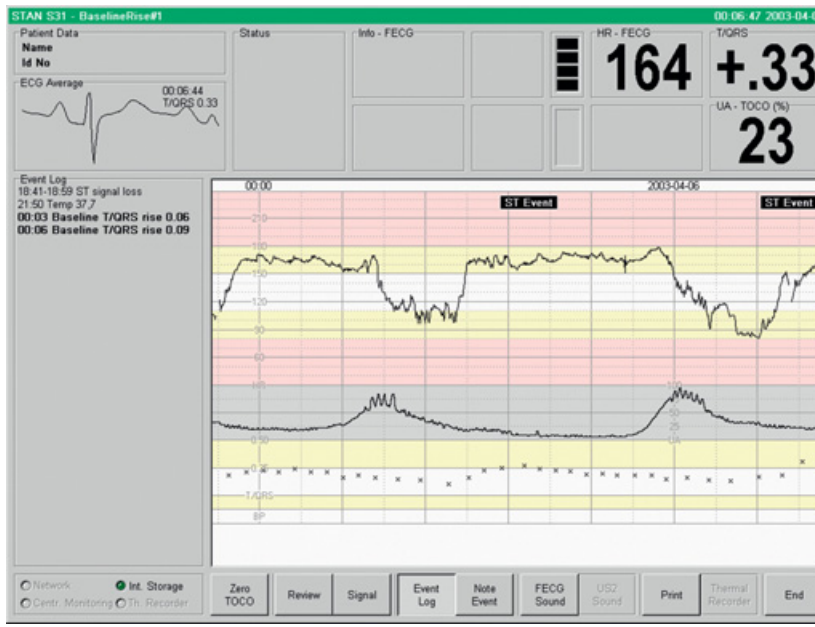

Figure 13 Display of STAN monitor screen showing FHR pattern and two ST events being triggered by the increase in baseline T/QRS ratios (courtesy of Neoventa Medical, AB).

Occurrence of an ST event during monitoring requires simultaneous appropriate classification (interpretation) of the CTG trace and consideration of the wider clinical picture. This remains the Achilles' heel of the STAN system because if clinicians are not trained in fetal physiology and CTG interpretation, an inappropriate action may be taken. In centers where STAN has significantly improved perinatal outcomes and has reduced intrapartum operative interventions, the primary focus has been on clinician training about fetal physiology and CTG interpretation.

To date there have been seven prospective RCTs using ST-segment analysis technology in five different countries; in addition, there have been a number of observational studies on STAN. Results have not been consistent across the RCTs with respect to rates of operative delivery and reduction in metabolic acidosis. A 2015 Cochrane Review considered six of these trials (16,295 women) on continuous fetal monitoring by CTG alone compared to the use of STAN[74]. It concluded that the use of STAN for intrapartum fetal monitoring resulted in fewer admissions to the special care baby unit, fewer fetal scalp samples during labor, and fewer operative vaginal deliveries. There was no significant reduction in neonatal metabolic acidosis or cesarean delivery rates (CDR). However, another meta-analysis that excluded a study that they considered had methodological flaws, and included another study excluded from the Cochrane Review, concluded that STAN reduced the incidence of metabolic acidosis by > 30%[75]. The disparate results mirror the wide variation seen in clinical practice and the criteria used for clinical trials as well as application of technology.

In 2015, a large American multicenter study including 11,108 patients reported that fetal ECG ST-segment analysis as an adjunct to conventional intrapartum EFM neither improved perinatal outcomes nor decreased operative delivery rates[76]. Several drawbacks of this largest RCT included the enrollment of a mostly low-risk population, low recruitment rates of under one patient a week in most of the participating centers (i.e. failure to gain sufficient experience with the technology), and the use of a different set of simplified guidelines for FHR interpretation[77][78]. A 2016 meta-analysis of six RCTs on fetal ECG (STAN) (26,446 women) included the large US trial. It concluded that, although there was no significant difference in operative vaginal delivery rates or CDR, there was a 36% reduction in the neonatal metabolic acidosis rate in babies monitored by fetal ECG[79]. The clinical significance of this reduced neonatal metabolic acidosis rate has also been highlighted[72], as has the role of intensive training on fetal physiology and mandatory competency testing in ensuring the effectiveness of this technology to reduce intrapartum operative interventions[80]. The take-home message is that all these approaches require

both the new technology and a higher level of clinician training than is usual. Thus, its public health effectiveness is limited. The most recent meta-analyis of STAN trials (including all of the above) fails to show an improvement in perinatal outcomes[81].

Additional Techniques of Monitoring Fetal Hypoxia and Acidosis

Attempts to use sophisticated technology to improve the assessment of fetal hypoxia have either proved too expensive and complex to be practical (e.g. near-infrared spectroscopy[82], transcutaneous PO^2 measurement[83]), or have been shown to be of no benefit when assessed in RCTs (e.g. pulse oximetry [84]). Attempts to measure fetal tissue pH continuously during labor were made in the 1970s and 1980s but proved to be impractical with the then current technology[85][86] and have not been pursued since as the value of measuring peripheral tissue pH in the fetus during labor remains uncertain.

Contextualization of CTG

CTG as a Screening Test

Developed in the late 1960 and early 1970s, CTG was introduced into clinical practice without any substantive published investigations comparing outcomes with and without the new technology. As previously discussed, if the original goal of CTG had stayed with the prevention of intrapartum stillbirth in high-risk pregnancies, it would have been a very successful innovation. However, as it was hoped to prevent neurologic handicap and cerebral palsy, this resulted in "mission creep" with the extension of CTG monitoring from high-risk pregnancies to all labors. The result was that the performance metrics dropped dramatically, and the incidence of cesarean deliveries mushroomed (a majority of which were, in retrospect, "false positive screens"). In fact, CTG is not a diagnostic tool but a classic screening test, i.e. it has false positives and false negatives. This misunderstanding of its appropriate use was not helped by the abandonment in the early 1980s of fetal scalp sampling (FBS) because the CTG was "just as good." The acceptance of the equivalence of CTG and FBS, from a public health perspective, has probably been deleterious to the care of women and babies. Furthermore, interpretation of the CTG is complex and nuanced, and the reality is that only a small percentage of providers can be considered experts. These considerations explain why alleged misinterpretation of CTG is the largest contributor to medical liability exposure and payouts in many parts of the world, including the United States, the UK, and South Africa.

How Much Cerebral Palsy Is of Genetic/Antenatal Origin?

One of the great debates driving much of the international malpractice crisis focuses on the proportion of cases with neonatal neurological compromise and cerebral palsy (CP) due to intrapartum management problems, antenatal events, and those that were predetermined by genetic causes. For decades, wildly divergent and unscientific "expert" testimony has been a prominent feature in courtrooms where medical liability cases were tried, which in numerous instances has led to enormous payouts to claimants. To clarify the issue, ACOG put together an expert committee in 2000 to look at the problem. One of us (MIE) was on the committee, which produced a monograph published in 2003[87]. This report concluded that the majority of CP – perhaps 90% – was genetic in origin and not related to intrapartum events. A second edition in 2014 added neuroimaging data, but did not fundamentally change the conclusions[88].

The extreme views that either all virtually cases of CP are genetic or that none are have fortunately become less frequently promoted, but they have not disappeared. Furthermore, the notion that most CP occurs before labor has begun must be further subdivided into genetic causes (usually undiagnosed before pregnancy and therefore not preventable) and other supposedly nonpreventable antenatal causes (such as maternal epilepsy) that might have been prevented with earlier recognition of risk [89]. The simplistic idea, partly promulgated by the ACOG monographs, was promoted by rigid criteria (e.g. a pH in arterial cord blood < 7.00) required to attribute CP to intrapartum events. Such a notion has been refuted by more recent data that found that most CP cases never exhibited Category III (the most severely abnormal) tracings which were thought to reflect severe acidosis[90].

A Japanese study of 1,069 babies with term CP concluded that only about 16% of them were preventable by earlier intervention, on the basis of having gone into labor with a normal FHR pattern but then developed the "Hon's pattern" of reassuring CTG at admission followed by decelerations, higher baseline, then decreased variability and low baseline (terminal bradycardia)[91]. They reported that about 26% of cases entered labor already having a bradycardia, or non-reassuring CTG without bradycardia, that remained abnormal throughout labor and delivery. Sixteen percent had an abrupt change of FHR in labor, often due to umbilical cord problems, but the authors concluded that these were not readily preventable. 19% of cases were considered unclassifiable and 18% of injuries were deemed to have begun after the delivery. The authors concluded that intrapartum hypoxic-ischemic events accounted for only about 30% of the severe CP cases, with only about half of these (Hon's pattern) potentially preventable. However, they did not consider genetics in their analysis, which renders their analysis problematic as a way of assessing the contribution of intrapartum events to adverse outcome.

Numerous genetic etiologies for CP were detailed even in the first edition of the ACOG monograph (mostly due to Mendelian and biochemical abnormalities) but these represented a small proportion of affected cases[87]. The rapid development of increasingly sophisticated laboratory techniques such as microarrays, whole exome sequencing (WES), and whole genome sequencing (WGS) has enabled more clarification of the molecular origins of hundreds more types of abnormality underlying the etiology of CP[92][93][94][95]. Recent papers using WES have suggested that approximately one third of CP cases have a molecular variant responsible for their phenotypic manifestations. Commentaries have suggested that all cases of CP should have WES as part of their workup[96][97][98][99]. This seems to us to be a reasonable or even mandatory conclusion[90][92][93][94][95][96][97][98]. The collective database of such cases will continue to rise over the next several years, and currently suggests that the proportion of children developing CP related to events in labor is about 35–38% (which will satisfy neither those who believe labor is essentially irrelevant, or claimants in lawsuits who believe almost all CP is related to labor events). About 35% will be shown to be genetic. Perhaps another 10% or so of cases will have a non-genetic antenatal origin, another 10% begin in the immediate postnatal period, and about 20% occur later from other causes such as infection[90][92][93][94][95][96][97][98] (Figure 14).

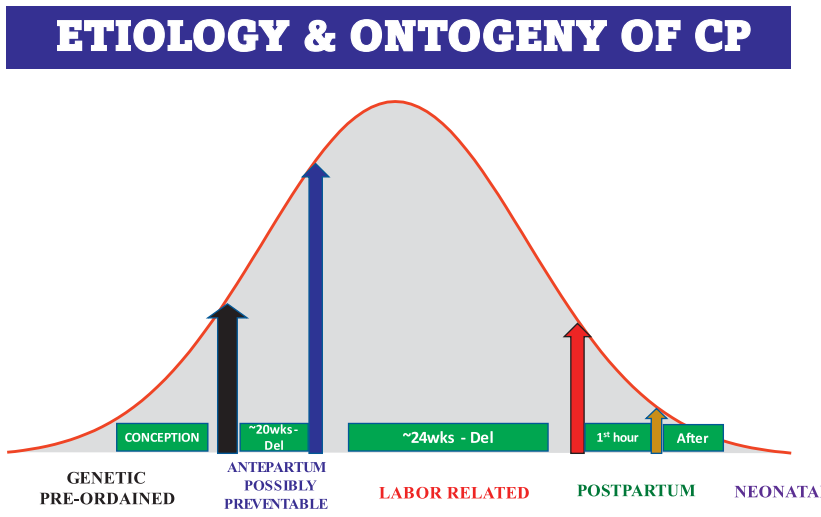

Figure 14 Distribution of CP cases. We categorize them into five groups (genetic, antenatal, labor related, postpartum, and neonatal). Exact percentages will evolve over time and will in part depend upon whether the definition includes the late postnatal cases or not [90][92][93][94][95][96][97][98].

Development of the Fetal Reserve Index

Limiting the evaluation of intrapartum fetal health by only using data from the CTG provides an incomplete picture, so it is not surprising that its statistical performance metrics are suboptimal. There are many clinical situations in which the interpretation of a lab test is contextualized by its environment. Combined screening for Down syndrome with NT screening, free β hCG, and PAPP-A produced a likelihood ratio that is then multiplied by the a priori maternal age risk. Thus, it took less deviation from normal to make a screen abnormal for a 33-year-old than for a 27-year-old. The principal goal for the Fetal Reserve Index (FRI) was to transform the subjective, nuanced, and often litigated interpretations of CTGs into a quantifiable, standardized, and objective metric that would have better quality control and, most importantly, have better statistical performance metrics for assessing risk of intrapartum fetal compromise and to do so earlier in in the process.

The FRI has three components:

1. **CTG (four parameters):** This uses the four individual components of the CTG that could be scored as a Yes/No or 0/1. Baseline heart rate, variability, accelerations, and decelerations are all evaluated according to current ACOG terminology.
2. **Uterine contraction frequency (one parameter):** The ACOG definition was not used for the number of maximum contractions. The FRI defines normal as ≤ 4 contractions per 10-minute period averaged over a half hour as compared to ACOG criteria which still allows ≤ 5. The NICE guidelines have also now (December 2022) adopted the ≤ 4 contractions cutoff[14].
3. **Risk Factors (three parameters):** The presence of maternal, fetal, or obstetrical risk factors are recorded (Table 3)[100][101].

Table 3 Fetal Reserve Index risk factors

Maternal risk factors	Fetal risk factors	Obstetrical risk
Decreased cardiac output/ vascular perfusion of the placenta a. Cardiac disease with risk of decreased cardiac output in pregnancy b. Hypertension (chronic and pregnancy induced) c. SLE	Abnormal Dopplers/ BPP	Fetal growth restriction/ macrosomia

Table 3 (cont.)

Maternal risk factors	Fetal risk factors	Obstetrical risk
Oxygen carrying capacity a. Pulmonary disorders (e.g asthma) b. Anemia and hemoglobinopathy	Genetic disorders	Oligohydramnios or polyhydramnios
Infection (chronic and acute)	Fetal arrhythmia	Bleeding and abruption
Chronic debilitating disease	Meconium passage	Previous cesarean delivery
Malabsorption/poor weight gain	Chorioamnionitis	Placental and umbilical cord anomalies
Endocrine – diabetes and thyroid disorders	Second stage of labor – active bearing down after full dilatation	Rupture of membranes (PPROM, SROM, AROM)
Advanced maternal age	Missing important data in labor (e.g lack of EFM in second stage)	Dystocia (slow (< 0.5 cm/hour) and arrested cervical dilatation)
Drug abuse, addiction, and smoking	Discontinuation of oxytocin infusion due to fetal intolerance	Malpresentation
Obesity – BMI > 35	"Conversion patterns" (acute prolonged tachycardia [> 170 bpm])	
Short stature (< 5'2")	Ominous overshoots Bradycardia (< 100 bpm)	

Abbreviations: EFM, electronic fetal monitoring; BMI, body mass index; SLE, systemic lupus erythematosus; PPROM, preterm premature rupture of membranes; SROM, spontaneous rupture of membranes; AROM, artificial rupture of membranes; BPP, biophysical profile.

In version 1.0 of the FRI, all eight parameters were weighted evenly such that when they are all normal – 8/8 – it produces a score of 100%. An abnormality of 2/8 parameters produces a score of 75%, and an abnormality

of 4/8 categories produces a score of 50%. For ease of interpretation, scores are then grouped: Green = 51–100%, Yellow = 26–50%, and Red ≤ 25% (Figure 15).

Red is considered a screen positive score. Reaching the Red zone is, however, not a cause for urgent intervention. Our data show that about 25% of all parturients reach an FRI score of 25% or less during labor. However, our data also show that for patients with good outcomes, the time in the Red zone is short – often in the second stage and averaging less than one hour. Reaching the Red zone is not a call for delivery but instead for expeditious evaluation by experienced personnel. A plan is made which usually consists of stopping oxytocin, administering oxygen, and repositioning the mother. Our Version 1.0 protocol allows 40 minutes to get out of the Red zone[100][101]. If not achieved, this triggers a 30 minute to delivery deadline. Version 2.0 is in preparation.

In our early studies, we evaluated 60 women who entered labor without evident risk factors (including Category I (normal) CTGs) but who went on to have a baby with CP with no apparent cause (pre or postnatally) for the CP except for intrapartum events. They all reached the Red zone early, and stayed there, on average, for five hours. None were in the Red zone for less than two hours, which we then used as the threshold for serious concern. We then compared these labors to 360 control labors with normal outcomes. Babies who developed CP had significantly lower cord blood pHs than did controls (7.03 vs. 7.21 for controls who reached the Red zone, and 7.24 for controls

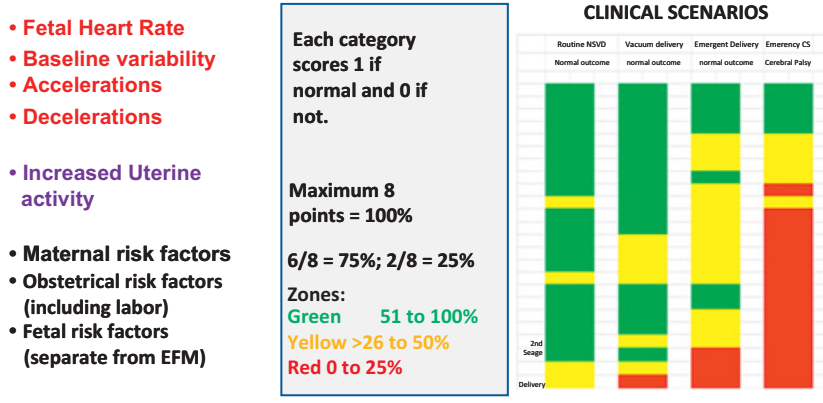

Figure 15 Summary of the Fetal Reserve Index concept. Left column: summary of risk factors. Centre column: scoring system. Right column: example time lines for FRI scores as labor progresses from early labor (top) to delivery (bottom).

who never did). Contrary to the ACOG monograph criteria for CP, most babies who developed CP did not have umbilical arterial cord blood pHs < 7.00[100]. Using the same labors, we evaluated the ability of three methods to predict babies who would develop CP. We used the 2003 ACOG monograph criteria, the CTG entry into Category III, and the FRI methodology. The monograph is actually a postnatal evaluation of related risk factors, but if we were to use them intrapartum, the sensitivity for predicting the development of CP would be 28%. Using Category III CTGs as the threshold for abnormality, the sensitivity was 45%, while the FRI detected all of these cases. Obviously, no test will ever be perfect, but clearly the FRI looked very promising, and merited further studies.

Contemporaneously, and independently, one of us (PJS) led a group which devised a similar score, calculated on an hourly basis, and incorporating the same features of the FHR plus tachysystole, with delay in progress, oxytocin augmentation, diagnosed or suspected fetal growth restriction or small for gestational age, new or increased meconium-stained liquor, and maternal pyrexia as additional risk factors to produce a combined score[32]. In 69 of the cases with an adverse outcome in the INFANT trial, 89% had at least four abnormal parameters more than one hour before birth, compared with only 26% in 198 consecutive labors with a normal outcome audited in four separate maternity units. Adding the hourly scores, 68% of labors with an abnormal neonatal outcome reached a score of more than 10, compared with only 17% in the normal outcome group. For the total score, the area under the receiver operator characteristic curve was 0.851 (any test with a value of greater than 0.7 is generally considered to be useful).

A retrospective study of 302,137 vaginal births at 37–42 weeks gestation, assessing by multivariable regression factors associated with a 5-minute Apgar score of < 7, has found significant odds ratios of abnormal CTG patterns (OR 2.40), meconium (OR 2.20), and pyrexia (OR 1.87)[33]. Importantly, when an abnormal CTG occurred in the presence of meconium, the odds ratio was significantly higher (OR 4.26) than for either CTG abnormality or meconium alone. Other risk factors for a low Apgar score included suspected fetal growth restriction (OR 1.34), induction of labor (OR 1.41), nulliparity (OR 1.48), maternal age < 25 years (OR 1.23), Black ethnicity (OR 1.21), and early or late post term compared with 39 and 40 weeks (OR 1.13 and OR 1.14). The authors highlighted the importance of considering abnormal CTG patterns in conjunction with these additional risk factors [33].

Continuing Studies and Broader Implications

In studies of the management of labor and delivery, "false positives" are those cases which have "fetal distress," require emergency delivery, may or may not show transient compromise, but ultimately have entirely normal outcomes. False positive predictions of CP produce unnecessary anxiety and stress for the mother, baby, entire family, and the medical staff. Our data suggest that normal (most) fetuses can tolerate some stress (here conceptualized as being in the Red zone) yet have enough "reserve" to withstand this stress without any long-term consequences. Further studies testing this hypothesis are needed. We found that the rate of emergency deliveries with ultimately normal outcomes increased markedly for fetuses in the Red zone for over one hour but less than two.

Physiologic Correlates to the FRI

In the 1970s and early 1980s, the role of CTG was in large part to determine which cases needed FBS for pH and BE. From the early 1980s and onward, FBS was largely abandoned as the prevailing opinion emerged that CTG was as accurate a predictor of neonatal condition as FBS pH and blood gas measurement, so the latter became unnecessary. The failure of CTG to reliably predict fetal status suggests otherwise, so direct comparisons are necessary. There is a dataset created under the direction of Edward Hon at the University of Southern California/LA County Hospital in the early 1970s. Several hundred patients were followed in the last one to two hours of labor with continuous monitoring, intrauterine pressure catheters, meticulous documentation of clinical events, and very liberal use of FBS any time anything of note occurred. Immediately after delivery, the babies were reconnected to a heart rate monitor, and umbilical artery catheterization was performed, allowing blood gas determinations at 0, 4, 8,16, 32, and 64 minutes. We have published several studies incorporating these data[31][100][101][102], which show that the fetus begins to demonstrate (subclinically at first) deterioration of acid/base homeostasis far earlier in the first stage of labor than is generally appreciated (Figure 16).

We took FBS BE values and categorized them by the cervical dilatation at the time the specimen was obtained. We then converted them into a multiples of the median (MoM) score for that dilatation. The BE fell as cervical dilation increased – i.e. further into the first stage of labor. For example, the median BE at 9 cm was -9 mMol/l, while the median BE value at 4 cm was -2.1 MoM [102]. Such a fetus would already be nearer to acidosis before going through the normal stresses of labor and therefore would be more likely to exhibit compromise during labor. We further showed that the FRI score closely follows the BE and is a more faithful surrogate for the BE than is the CTG category system.

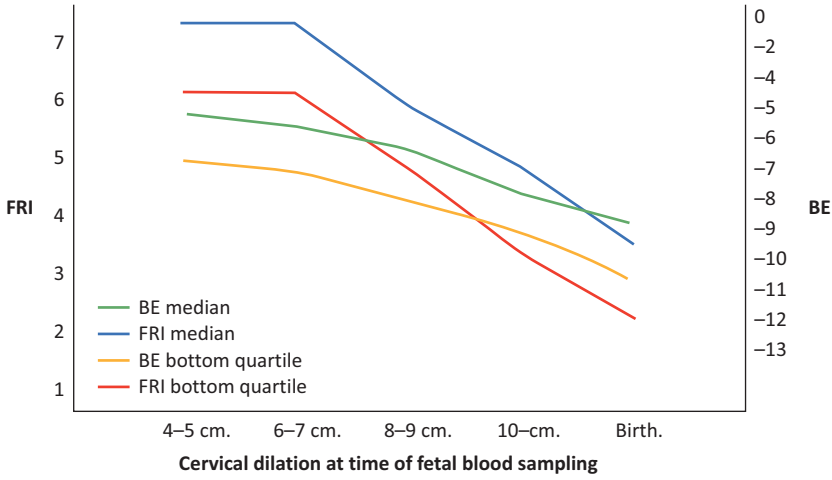

Figure 16 Base excess (mmol/l) measured from fetal blood samples through labor (data from original studies by Ed Hon and reanalyzed by Evans *et al*.[31] [91]), compared with median FRI values.

Postnatal Risks

The physiologic deterioration of fetal acid/base status progresses through the first stage, and continues to deteriorate in the second stage. We were surprised by the observation that fetal acid/basis status continued to deteriorate for several minutes after birth before beginning to return to normal[90] (see Figure 17).

As a first analysis, we divided patients by their last FRI score before delivery into three different groups, simplistically divided as reassuring (green), non-reassuring (yellow) and abnormal (red). The curves were parallel, but further out on the distribution such that for the abnormal group, the BE went below the −12 mMol/l threshold as a risk factor for neurologic compromise for an average of 20 minutes. Overall, one third of all patients had a BE of < −12 within the first 8 minutes.

In parallel, analysis of fetal and neonatal heart rate showed that following delivery, 85% of all neonates showed a tachycardia. Again, dividing the neonates by the last FRI score showed that for the abnormal group the tachycardia averaged 185 bpm, and their HR exceeded 160 bpm for 50 minutes for 50% of those patients (Figure 18).

In fact, if the first 10 minutes of the neonatal HR were the last 10 minutes of the fetal HR, 25% of cases would be considered as Category III (abnormal). These findings need further exploration, but they largely have not been appreciated because pediatricians worry more about bradycardia, while our data

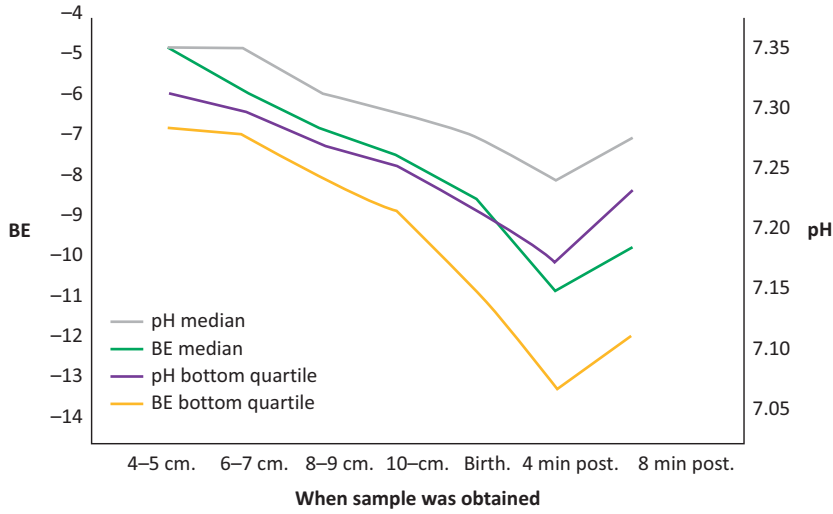

Figure 17 Base excess (mmol/l) measured from fetal blood samples through labor, at birth (umbilical cord artery blood), and in the immediate neonatal period (data from original studies by Ed Hon and reanalyzed by Evans *et al*.[31] [91]), compared with median FRI values.

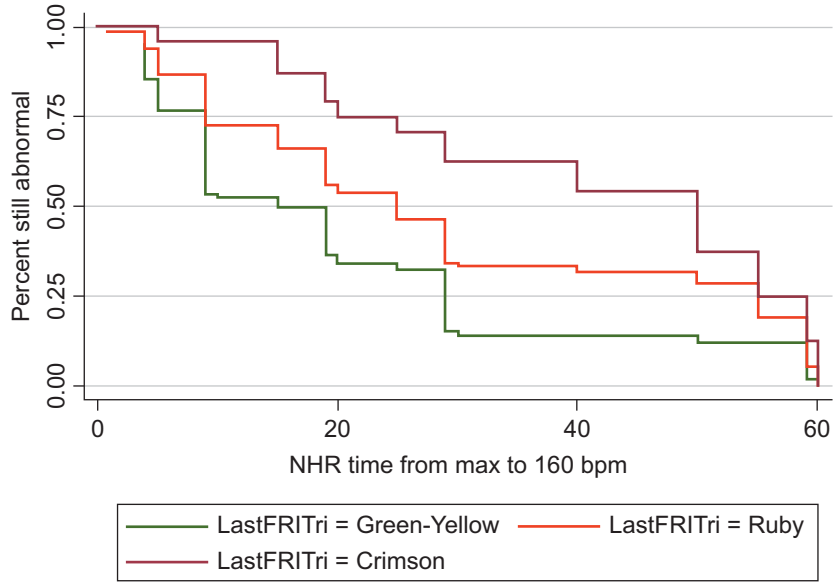

Figure 18 The time taken for a fetal tachycardia (>160 bpm) to fall to 160 bpm in the neonatal period, classified by the Fetal Reserve Index. Green = 51–100%, Ruby = > 12.5–25%, Crimson = 0–12.5%.

suggest that sustained (even moderate) tachycardia may be problematic. Our opinion is that monitoring of term neonates should be improved and continued for at least 30 minutes after delivery.

Reevaluating Some Old Conclusions

In the 1970s, the cesarean delivery rate was < 10%, and the forceps delivery rate approximated to 20%. Several publications, most notably those by Friedman's group, argued that midforceps were associated with a high level of risk of neurologic handicap and should be abandoned. Their sentinel study from the New England Collaborative Project showed that babies born by midforceps had lower IQ scores than those born by normal, spontaneous, vaginal deliveries. Forty years ago, a reanalysis by one of us (MIE) found several study design problems which, we argued, invalidated their findings[103]. In view of this history, we compared spontaneous versus midforceps deliveries in the Hon/USC dataset. The cord blood BE values of midforceps deliveries were, in fact, lower than the spontaneous deliveries. The initial interpretation of such would support Friedman. However, further analysis showed that the decrements in BE values were already there one hour *before* delivery suggesting a very different speculation: the forceps did not cause the compromise; the compromise led to the forceps (Figure 19).

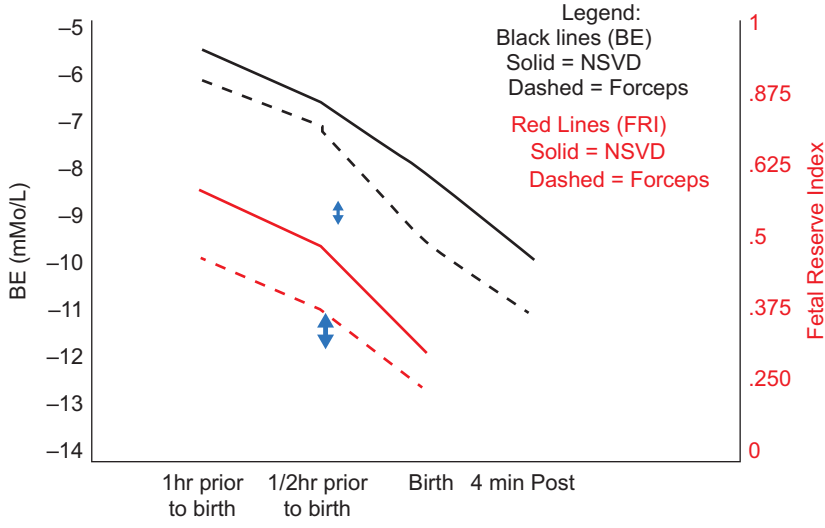

Figure 19 Fetal Reserve Index and BE values in relation to time before/after birth, classified by mode of delivery. NSVD = normal spontaneous vertex delivery.

Meconium Staining of the Amniotic Fluid

Meconium is found in the fetal gut from 10 weeks' gestation, but the passage of meconium is unusual (< 5%) before 34 weeks[3]. The presence of meconium in the amniotic fluid is strongly associated with fetal gastrointestinal maturity, and the incidence of meconium passage increases with gestational age, reaching approximately 30% at 40 weeks' gestation and 50% at 42 weeks [3]. The likelihood of meconium passage is increased in fetuses of Black African genetic geographical origin[3], which may be related to their accelerated maturity (average gestational length is about one week shorter than in white Europeans[104][105]. In the adult, involuntary defecation can occur in response to stress ("fight-or-flight" reaction). This is mediated centrally by the hypothalamus (and possibly by the amygdala), which in turn causes stimulation of the visceral parasympathetics. This mechanism may also occur in fetuses, as babies that exhibit stress, as characterized by abnormal FHR patterns, are about twice as likely to pass meconium during labor as those exhibiting normal FHR patterns, and the combination is associated with a two to threefold increase in adverse outcome[106]. In addition, there has increasingly been postulated a role for intrauterine inflammatory processes during labor, which not only predisposes to the passage of meconium, but increases its damaging effect upon the pulmonary endothelium[107]. However, in most cases where the baby passes meconium, the FHR pattern remains normal and the baby's condition at birth is not affected.

Risks

The risk associated with particulate ("thick") meconium is that if inhaled at birth, the bowel enzymes contained within the meconium "digest" the lungs, producing an inflammatory reaction[107]. The fetal airways are partially blocked by particulate matter, a combination that gives rise to meconium aspiration syndrome, which has a poor prognosis. This has been reported to occur to at least some degree in up to 30% of labors when there is significant meconium staining. This represents yet another reason to keep the fetus safe rather than having to rescue it when it is already in trouble.

In most cases, the presence of meconium indicates a mature fetus rather one who is "distressed." If the FHR pattern remains normal, the presence of meconium approximately doubles the incidence of low Apgar scores (which may in part be due to the airway being blocked by attempts to suction the pharynx, which in fact does not reduce the rate of meconium aspiration[108]) and has no effect on acid-base status (i.e. the babies are not more likely to be acidotic)[12]. Statistically, such a pattern suggests that meconium could be

useful as a part of a multicomponent algorithm (e.g. the FRI) but by itself supplies a limited proportion of the variances of abnormal outcome provided that the FHR pattern remains normal. However, if the FHR pattern becomes abnormal, the likelihood of both acidosis and neonatal morbidity (including neonatal death, neurologic morbidity, respiratory morbidity, hypotension that requires treatment, and sepsis) is increased approximately 2.5 times [109].

There is a reported association between the occurrence of intrahepaticcholestasis of pregnancy and meconium staining of the amniotic fluid[110], such that in affected cases meconium is passed into the amniotic fluid even in early gestations (20% before 35 weeks gestational age, compared with < 5% in normal pregnancies, peaking at 40% at 35 weeks, and then remaining at 15–20% until 40 weeks). Why this occurs, and whether it is related to the higher rate of stillbirth in pregnancies complicated by cholestasis, is unknown.

Management Options

When amniotic fluid is seen to drain, it should always be inspected carefully for the presence of meconium. If meconium is detected, continuous electronic FHR monitoring is recommended, if it is not already being employed. If the FHR pattern remains normal, expectant management can be appropriate. No specific action is necessary, except to avoid actions that might precipitate acute fetal hypoxia (supine hypotension, epidural hypotension, and uterine hyperstimulation with oxytocics). In particular, there is no indication for routine FBS and pH estimation as long as the FHR pattern is normal, because the likelihood of acidosis is not increased above what would be expected.

If the FHR pattern becomes abnormal, prompt delivery should be considered, because the risk of meconium aspiration is increased even if the pH is normal (secondary to intrauterine gasping induced by hypoxia that is not sufficiently prolonged to cause acidosis). Waiting until the fetus becomes acidotic probably increases the risk of meconium aspiration to an unacceptable level. An FBS should not be performed in this case, because the presence of bile acids in the meconium may result in erroneous results, and a normal result does not guarantee that acute problems will not precipitate meconium aspiration. At delivery, a pediatrician should be present if at all possible. If the baby is vigorous and cries promptly, there is no need for further action.

In the past, there was encouragement to perform amnioinfusion (flushing the amniotic cavity with warmed normal saline through a catheter passed through

the cervix). A 2014 meta-analysis of this approach found no evidence of benefit in settings where EFM was available[111], and therefore it is not currently recommended. However, a recent meta-analysis has challenged this recommendation and has suggested that prophylactic amnioinfusion could reduce meconium aspiration syndrome and the need for neonatal intensive care admission by as much as two thirds, and reduce the need for cesarean delivery by 40%[112].

Summary of Meconium Management Options

Meconium Staining of Amniotic Fluid

- *Background* [105][106]:
  - The main factor affecting the passage of meconium is gestational age
  - Hypoxia and acidosis increase the risk of meconium passage
  - The combination of severe hypoxia and aspiration of meconium causes lung damage
  - If the FHR pattern is normal, as gestation advances the presence of meconium is increasingly likely to be a normal finding
- The combination of meconium in the amniotic fluid and an abnormal FHR pattern approximately doubles the likelihood of a poor fetal outcome compared with the presence of an abnormal FHR pattern alone
- The combination can therefore be used as an indication for delivery without waiting for the pH to fall, as by the time this occurs the likelihood of a poor neonatal outcome will already have exceeded 50%
- A pediatrician should be present for delivery, but should not perform routine oropharyngeal suction in the absence of evidence of fetal hypoxia, because suction does not reduce the incidence of meconium aspiration syndrome
- Amnioinfusion with normal saline may not improve outcomes in settings where facilities for CTG monitoring are available (further evidence needed)

Pyrexia in Labor as a Risk Factor for Adverse Neonatal Outcome

Intrapartum maternal fever has long been known to be associated with a poor outcome. For example, Grether and Nelson reported in 1997 that maternal fever > 38°C in labor was associated with a ninefold increased risk of unexplained cerebral palsy[113]. Although they attributed this to "infection," the variable they analyzed was in fact maximum maternal temperature during labor. The following year, Fiona Stanley's group in Western Australia reported

an odds ratio of 3.82 for newborn encephalopathy when there was a maternal pyrexia[43]. In 2012, Greenwell *et al*.[1] reported a "dose response relationship" of poor outcome with maternal pyrexia in labor, with temperatures > 38.30°C giving an odds ratio of 4.8 for a 5 minute Apgar score less than 7 and an odds ratio of 6.5 for early onset neonatal seizures. Impey *et al.*[114] reported that the combination of maternal fever with acidosis on cord artery blood measurement resulted in an incidence of neonatal encephalopathy of 12.5% (one in eight). Although it is common to assume that a woman with intrapartum pyrexia has chorioamnionitis, studies show that only about 3.5% of pyrexias in labor are associated with evidence of neonatal sepsis[115]. Instead, most pyrexias in labor in modern practice are associated with the use of epidural anesthesia[116][117][118][119]. Nonetheless, there are plausible reasons for a raised maternal temperature being a risk factor even in the absence of infection, notably the increased metabolic rate which will increase the rate of metabolic acidosis generation in response to a hypoxic stress[120][121]. This hypothesis is supported by the observation that brain cooling helps to minimize the brain injury associated with encephalopathy[122][123][124][125]. It should also be remembered that the fetal core temperature is almost 1°C greater than that of the mother[28][29][126], so that if the mother is pyrexial, fetal temperature will often exceed 39°C[126]. In addition, pyrexia ≥ 37.5°C increases the incidence of an abnormal CTG from 19% to 50%, and the incidence of meconium staining from 16% to 30%. A combination of an abnormal CTG in labor, meconium staining of the amniotic fluid and a maternal pyrexia increases the likelihood of 5 minute Apgar scores ≤ 3 almost tenfold (from 0.12% to 1.15%), and intrapartum and neonatal death fourfold (1.3 per 1000 to 5.4 per 1000).

Although as explained above, most maternal pyrexias in current practice are secondary to the use of epidural anesthesia, there is currently no effective method of distinguishing such pyrexias from those due to chorioamnionitis. Therefore, safe practice requires that a significant pyrexia (≥ 37°C on two occasions one hour apart or a single observation of ≥ 38°C[127]) should be treated with administration of intravenous antibiotics to the mother[128]. Ampicillin and gentamicin are usually recommended as first-line treatment, although cephalosporins have also been used[128]. Provided that antibiotic treatment is being given, the duration of labor following the diagnosis of chorioamnionitis does not correlate with adverse outcomes following vaginal delivery[129] and clinical chorioamnionitis by itself is therefore not an indication for cesarean delivery[128][130].

Summary of Management Options

Pyrexia in Labor

- Isolated maternal temperature (e.g. regional analgesia for four or more hours, no evidence of chorioamnionitis (see below))
  - Ensure adequate hydration with oral or IV fluids
  - Cool using a fan or tepid sponge, reduce room temperature if possible
  - Investigations: full blood count, urea and electrolytes, C reactive protein, clotting, blood cultures, lactate and glucose in venous blood
  - COVID-19 test if WHO COVID-19 suspected case definition is met
  - Continuous FHR monitoring
- Suspected chorioamnionitis (FHR >= 160 bpm, maternal white cell count >= 15,000 per mm^3, offensive liquor, uterine tenderness)
  Management as above plus:
  - Maternal blood cultures
  - Intravenous infusion of amoxicillin and gentamycin (cephalosporin instead of amoxicillin if penicillin sensitivity suspected; vancomycin if penicillin allergy was anaphylactic)
  - Postdelivery placental surface/intramembrane swabs and fetal blood, ear and umbilical swabs, for culture
  - Ensure pediatrician informed and present for delivery

Prolapse of the Umbilical Cord

Prolapse of the umbilical cord is rare (around 2–3 per 1,000 births) and raises the risk of cord occlusion and acute fetal hypoxia. It is usually diagnosed at vaginal examination, although sometimes it is suspected because of the sudden appearance of deep variable decelerations of the FHR or sudden and profound fetal bradycardia. Cord prolapse is usually managed by prompt cesarean delivery, although if the patient is in the second stage of labor, the presenting part is below the level of the ischial spines, and easy, prompt vaginal delivery is anticipated, then a forceps delivery may be preferable. While the woman is being prepared for a cesarean delivery, it is usually recommended that she be placed in the knee–chest position facing downward or, alternatively (and often more practically), in steep Trendelenburg position. It may be necessary for a birth attendant to keep a gloved hand in the vagina to elevate the presenting part and relieve pressure on the cord. One technique first suggested in 1970 and since recommended by others is to fill the urinary bladder with 500–700 mL of saline, elevating the presenting part and relieving

pressure on the cord. The role of funic reduction (i.e. replacement of the umbilical cord) is uncertain[131], and handling of the umbilical cord may stimulate spasm of the umbilical vessels. This action may be harmful so this practice should be avoided. One study has suggested that it may have a beneficial role if umbilical cord prolapse occurs remote from delivery (e.g. in a home birth setting).

Summary of Management Options

Cord Prolapse

- Perform cesarean delivery while pressure on the cord is relieved by one of the following until delivery:
  - Manual elevation of the head away from the cord
  - Knee–chest position
  - Steep Trendelenburg position
  - Filling the bladder with up to 700 mL normal saline
- Instrumental delivery if the patient is in the second stage of labor, the presenting part is below the level of the ischial spines, and easy, prompt vaginal delivery is anticipated
- The value of funic reduction (manual replacement) is uncertain because few patients have been studied. It should be avoided, as it may stimulate spasm of umbilical cord vessels. However, it may be useful while transferring a woman from a home birth setting

Summary of Management Options

Screening for Fetal Compromise in Labor

- **Prevention of fetal hypoxia**
  - Avoid unnecessary induction of labor and excessive use of oxytocic agents
  - Preload with IV fluids in women who are having epidural analgesia in labor to reduce the risk of maternal hypotension
- **Pathophysiology (see Figure 1)**
  - Changes in the FHR are predominantly caused by two mechanisms:
    - Reflex slowing of the heart due to firing of the vagus nerve
    - Slowing of the heart due to direct myocardial depression by the generation of lactate from anaerobic metabolism (due to the absence of an adequate oxygen supply)

(cont.)

- Based on the rapidity of onset of hypoxia during labor, a fetus may be exposed to acute, subacute, or a gradually evolving hypoxia
- Remember that maternal drugs can influence FHR patterns

- **High-risk labors**
  - Indications for continuous FHR monitoring (see also Table 3):
    - Fetal problems
    - Maternal disease
    - Labor problems
    - Intrapartum fetal compromise

  Meta-analysis of RCTs of the use of continuous FHR monitoring in labor compared with intermittent auscultation shows significant reductions in the short-term neonatal morbidity rate but a significant increase in CDR associated with the use of CTG. However, studies are underpowered to show an effect on overall perinatal mortality or CP rates
- **Low-risk labors**
  - The advice from the American, Canadian, and NICE organizations is that intermittent auscultation can be used in such labors. However, there are no studies of sufficient size that have evaluated this approach
- **Admission FHR recording (admission test)**
  - Meta-analysis of systematic reviews have concluded that use of admission CTG does not improve perinatal outcomes but may increase operative interventions
- **Interpretation of FHR findings**
  - The four features to be noted/assessed are:
    - Baseline rate
    - Baseline variability
    - Presence/absence of accelerations
    - Presence/absence of variable or late decelerations
  - A normal FHR pattern is associated with a very low risk of hypoxia/acidosis
  - The FHR features that are associated with an adverse fetal or neonatal outcome are
    - prolonged or severe bradycardia
    - prolonged decreased variability, and
    - variable or late decelerations

(cont.)

- The CTG is a screening test. Most FHR abnormalities in labor have a low positive predictive value for fetal hypoxia/acidosis
- Baseline FHR should be individualized for each fetus while interpreting the CTG traces, and changes evolving over time need to be carefully scrutinized
- Any increase in uterine activity (frequency, strength, or duration of uterine contractions, rather than merely focusing on the number of contractions) associated with CTG changes should be considered as uterine hyperstimulation
- In each case consider:
  - The FHR characteristics
  - Uterine contraction frequency, strength, and duration
  - The clinical context, namely the presence of risk factors (see Table 3 and Figure 6)
- The maternal pulse may be mistakenly recorded as fetal. The maternal pulse should be regularly recorded by palpation to reduce this risk
- Additional tests of fetal well-being such as fetal scalp blood sampling for pH/lactate estimation should be used with caution as there is no robust scientific evidence to support their use
- Presence of an acceleration during digital stimulation of the fetal scalp may obviate the need for additional tests of fetal well-being

- **If FHR abnormality is detected**
  - Correct/avoid maternal vena caval compression
  - Give the mother oxygen by face mask
  - Correct hyperstimulation (stop oxytocics, use tocolytics)
  - Give IV fluids if the patient has epidural-induced hypotension
- **Human factors that adversely affect the outcome of EFM**
  - Delays in response times, and
  - Failure to interpret the findings accurately

  Education improves human responses, but this benefit may be lost over time if it is not refreshed on a regular basis.
- **Technical aspects**
  - EFM recordings should be retained
  - External cardiotocodynamometry can lead to difficulty in interpreting the timing of EFM abnormalities (e.g. due to movement, maternal obesity)

(cont.)

- **Medicolegal aspects**
  - The parturient must be fully informed of any increased risks (both common and rare but important), and the relevant management choices discussed. Whenever there are reasonable options, the parturient's choices must be respected

Medicolegal Aspects of Fetal Monitoring

Most mental handicap is not caused by intrapartum events. In 1985, the US National Institutes of Health reported that "the causes of severe mental retardation are primarily genetic, biochemical, viral, and developmental, and not related to birth events." While a commonly cited estimate is that 90% of such cases are unrelated to labor, we consider that the proportion with a genetic cause is closer to 35%[90][93][94][95][96][97][98][100][101][102]. Potentially preventable antenatal risk factors include maternal lifestyle, such as poor nutrition, cigarette smoking, alcohol, and drug abuse. After all these causes have been

Table 4 Criteria that should be fulfilled for long-term disability to be ascribed to intrapartum hypoxia

1. Was there evidence of severe, prolonged intrapartum dysfunction?
2. Is CP present?
3. Was the child severely ill as a newborn? Were there disturbances of feeding, tone, and consciousness, and evidence of involvement of other organ systems, of which renal involvement may be especially significant?
4. Have other potential explanations been excluded, such as:
   a. Congenital malformation
   b. Infection
   c. Metabolic abnormality
   d. Familial disease
   e. Microcephaly in the neonatal period
   f. Abnormal CT or MRI scan suggesting discrete lesions
   g. Maternal substance abuse (especially cocaine)
   h. Thyroid disease
   i. Genetic abnormalities such as abnormal microarray, WES, or Mendelian disorders

CT, computed tomography; MRI, magnetic resonance imaging.

From Nelson KB. Perspective on the role of perinatal asphyxia in neurologic outcome: its role in developmental deficits in children. *Can Med Assoc J* 1989;141(Suppl):3–10.

considered, there remains a strong association between pathological FHR traces in labor and long-term disability, in particular spastic quadriplegic CP. For example, in 1994 Gaffney *et al.* reported that of 339 cases of CP, 33 (10%) were associated with "failure to respond to signs of fetal distress"[132]. This figure rose to 50% when perinatal death occurred. Although 10% of normal controls had an FHR pattern classified at some time in the first stage of labor as "abnormal," this rose to 23% in cases of CP and 67% in cases of perinatal death.

In 1998, Karin Nelson defined the criteria to be fulfilled before long-term outcome can be linked with intrapartum events[133] (Table 4). If all these criteria are fulfilled, however, and there is evidence of failure to respond to an abnormal FHR pattern in labor, it is likely that courts in the developed world will find the care has been substandard and that damages will be awarded to the child and its family, to cover the cost of lifelong medical care. Such monetary awards often amount to many millions of pounds/dollars. In the annual report of NHS Resolution (formerly the National Health Service Litigation Authority) for 2021/22, 62% by value of all medicolegal claims received were related to maternity care, with a total value of £4.131 billion, or approximately £6,235 per birth[134]. Unpublished estimates (personal communications) in the United States suggest liability costs amount to about $40 billion per year. A 2023 British report suggested that the estimated total value of outstanding medicolegal claims is close to £40 billion ($50 billion) per year[135.]

A Swedish study found that the commonest finding of substandard practice was overlooking signs of asphyxia on the FHR tracing (71%), but also highlighted incautious use of oxytocin (71%) and a nonoptimal mode of delivery (52%)[136]. Studies have shown that even with training, 30–40% of multiple-choice questions about intrapartum care are answered incorrectly by midwives and about 15–20% by obstetricians[137]. Because of poor performance using current evaluation criteria, ACOG and other organizations have developed more structured testing of professionals before they are allowed to take responsibility for CTG interpretation. Part of the rationale for developing the FRI as a quantifiable, contextualized system was to overcome the currently widespread problem of inadequate CTG interpretation.

The previously referenced 2003 ACOG monograph stated that there were four "essential criteria" and five additional criteria before one could conclude that cerebral palsy/neonatal encephalopathy resulted from intrapartum brain injury[87]. Paradoxically, because such a high percentage of CP was felt not to be related to labor, as a result, many hospitals actually cut back on the number and expertise of providers supervising labor and delivery. The ACOG Category system was published in 2009 with Category I being very reassuring, Category III requiring immediate action such as delivery, and Category II suggesting close following but no clear management protocols[15]. It has very cumbersome

pathways, becomes difficult for most clinicians to follow, and as many as 80% of intrapartum CTGs will reach Category II, rendering it essentially useless as a screening test.

Communication with the Parturient and Her Family

Litigation is commonly driven by women and their families who believe they were not given enough information about risk factors and potential options regarding delivery. In 2015 a judgment in a landmark case heard at the Supreme Court in the UK emphasized the importance of giving women full information about the risks they face during labor, and their options[138]. Nadine Montgomery (a diabetic woman with short stature) was at term in her first pregnancy when her baby was predicted by ultrasound to weigh more than 4 kg. Guidelines[139] suggest that diabetic women with a diagnosis of fetal macrosomia might benefit from delivery by elective cesarean section, but this was not offered by the caregivers, nor was the possibility of shoulder dystocia mentioned. Shoulder dystocia did occur, the baby was born with severe asphyxia, and later developed CP. Two lower courts rejected a claim of negligence, on the basis that many obstetricians would have encouraged vaginal birth. However, the UK Supreme Court upheld an appeal in favor of the claimant, stating that pregnant people have a right to be told about any material risks before deciding how they wish to give birth. Importantly, they stated that the acceptable level of risk should not be judged on what the doctors think important, but instead on what the patient considers important. The "reasonable patient" standard of management has now been widely adopted in developed countries, including the USA[140]. Thus, caregivers who do not advise women and their families of all material risks (whether important because they are common, or because they are uncommon but particularly serious) are likely to be judged negligent in the event of a poor outcome. When there are options available, it must be left to the patient to decide which they prefer. The doctor must not decide for them, as this would be a breach of autonomy. If a cesarean section is a reasonable option, women are entitled to choose it .

Behavioral Aspects of Fetal Monitoring

The "Each Baby Counts" studies of adverse outcomes collated adverse outcomes throughout the United Kingdom in each of the four years from 2015 onwards[68]. The outcomes studied were intrapartum stillbirth (which in consecutive years were 126, 124, 130, and 121), early neonatal death (156, 145, 150, and 165), and severe brain injury (854, 854, 850, and 859). Clearly these figures hardly changed over time. In cases where there was an adequate quality

review (60–85%), the incidence of identifiable substandard care was persistently high: 76%, 71%, 72%, and 74%. In the 2015 study, while incorrect CTG interpretation was important and was considered to be a factor in 72% of such cases, auscultation alone (17%) and in conjunction with CTG monitoring (11%) were also often considered to be substandard. The 2015 report highlighted that not only did most cases with an adverse outcome have both antenatal and intrapartum complications, there was an average of six risk factors/complications per case rather than there being a single problem such as an abnormal CTG. The risk factors highlighted were reduced fetal movements, suspected or diagnosed fetal growth restriction, previous cesarean section, thick meconium staining of the amniotic fluid, suspected infection, vaginal bleeding, and prolonged labor. In the 2019 report, inadequate appreciation of these risk factors was the predominant failing in relation to substandard care, being present in 74% of cases with an adverse outcome. Errors of interpretation of the CTG were identified in 33%, but more important was failure to act upon a suspicious or pathological CTG (44%).

A key feature in the labor ward dynamic associated with an adverse outcome was loss of situational awareness, which involves being aware of what's going on around you so you can anticipate adverse events and take action to prevent them. The United States Coast Guard manual[141] lists loss of situational awareness as leading to confusion, failure to watch or look out for hazards (which in obstetrics includes errors such as failing to order blood prior to cesarean section for placenta previa), departure from regulations or guidelines (for example, failing to monitor the fetus when a regional block is started), use of improper procedures (for example, making too many attempts at instrumental vaginal delivery before resorting to cesarean section), unresolved discrepancies (for example, differing views about estimated fetal size), failing to meet planned targets (allowing labor to go on too long), ambiguity (not giving clear instructions about management plans), and fixation or preoccupation with a single variable (such as CTG pattern recognition), ignoring the presence of multiple additional risk factors.

Procedures to try and avoid loss of situational awareness include in the UK the concept of "buddying." This is a requirement to have an independent person evaluate progress in labor, including assessment of the CTG, on an hourly basis, which has now been instituted in most UK labor wards. Labor ward coordinators have been appointed, whose job is to take "a helicopter view" of events on the labor ward. An example is the objective of avoiding having three emergency cesarean sections materializing at the same time (e.g. by avoiding starting oxytocin or regional block or a nonurgent cesarean section when there are other labors experiencing problems).

The "Each Baby Counts" studies also emphasized the importance of good team communication. Deficiencies in team communication were identified in 49% of adverse outcomes. Useful insights here have been derived from "crew resource management", developed in the aviation industry. It is defined as the effective use of all available staff resources to ensure safe and efficient operation, reducing stress and thereby increasing efficiency. It emphasizes a "flat hierarchy" in which all those involved in labor, including the professionals, the parturient, and their family, are empowered to raise concerns and express their opinion about problems which arise. This helps to avoid the overlooking of developing risk factors until they present as a crisis. Should such concerns be raised, then a "huddle"[142] should be called. A huddle is a short, focused briefing which brings together representatives from across key staff groups to identify potential problems or safety issues, such as challenges to the safe flow of patients across a department or hospital. An approach which originally began in the aviation industry, huddles have been adopted as an effective way of working in the equally complex environment of healthcare. Huddles within a maternity department can enable the timely identification of high-risk deliveries and facilitate earlier discussions with receiving specialist units. Instead of multiple, repetitive, individual conversations, a single conversation at a huddle can precipitate earlier transfer of mothers to appropriate care.

Another important area to emphasize is the importance of professionals working together. The Morecambe Bay report[143] was instigated because of observed high rates of perinatal mortality. It was discovered that a group of midwives had developed a "silo" mentality in which their objective was "a natural birth" at almost any cost. The report highlighted the importance of good personnel management and ensuring effective teamwork on labor wards by educating staff about the need for empathy and consideration for one another as well as all the women in labor and their families. Good labor ward care should be seen as a collaborative exercise to which everyone contributes. The traditional model of a single professional caring for one woman at a time has advantages in terms of continuity but is vulnerable to staff burnout, excessive tiredness, and loss of situational awareness when labor has become protracted. In the same way that the aviation industry has become the safest way to travel through team working, our future labor wards will only be able to avoid most errors (and litigation) and push safety to even higher levels if we accept the principles of good management and teamwork.

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High-Risk Pregnancy: Management Options

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David James was Professor of Fetomaternal Medicine at the University of Nottingham from 1992–2009. The post involved clinical service, especially the management of high-risk pregnancies, guideline development, research and teaching and NHS management. From 2009–14 he was Clinical Director of Women's Health at the National Centre for Clinical Excellence for Women's and Children's Health. He was also Clinical Lead for the RCOG/RCM/eLfH eFM E-Learning Project. He is a recognised authority on the management of problem/complicated pregnancies with over 200 peer-reviewed publications. He has published 16 books, the best-known being *High-Risk Pregnancy: Management Options*.

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About the Series

Most pregnancies are uncomplicated. However, for some ('high-risk' pregnancies) an adverse outcome for the mother and/or the baby is more likely. Each Element in the series covers a specific high-risk problem/condition in pregnancy. The risks of the condition will be listed followed by an evidence-based review of the management options. Once the series is complete, the Elements will be collated and printed in a sixth edition of *High-Risk Pregnancy: Management Options*.

Cambridge Elements

High-Risk Pregnancy: Management Options

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