

Fetal Heart Rate Patterns: Monitoring, Interpretation, and Management

Intrapartum fetal heart rate (FHR) monitoring can help the physician identify and interpret changes in FHR patterns that may be associated with such fetal conditions as hypoxia, umbilical cord compression, tachycardia, and acidosis. The ability to interpret FHR patterns and understand their correlation with the fetus' condition allows the physician to institute management techniques, including maternal oxygenation, amnio-infusion, and tocolytic therapy. Current data indicate that FHR monitoring is equally effective whether done electronically or by auscultation.

Intrapartum fetal assessment by FHR monitoring is only one parameter of fetal well-being. It involves evaluation of the pattern as well as the rate, but it is not a substitute for informed clinical judgment.

Transient and repetitive episodes of hypoxemia and hypoxia, even at the level of the central nervous system (CNS), are extremely common during normal labor and are generally well tolerated by the fetus. Further, a progressive intrapartum decline in baseline fetal oxygenation and pH is virtually universal; levels of acidemia that would be ominous in an infant or adult are commonly seen in normal newborns. Only when hypoxia and resultant metabolic acidemia reach extreme levels is the fetus at risk for long-term neurologic impairment (1).

For the purposes of this bulletin, the following definitions will be used:

- Hypoxemia: Decreased oxygen content in blood
- Hypoxia: Decreased level of oxygen in tissue
- Acidemia: Increased concentration of hydrogen ions in the blood
- Acidosis: Increased concentration of hydrogen ions in tissue
- Asphyxia: Hypoxia with metabolic acidosis

Physiologic Basis

The fetus is well adapted to extracting oxygen from the maternal circulation even with the additional stress of normal labor and delivery. However, alterations in the fetoplacental unit resulting from labor or intrapartum complications may subject the fetus to decreased oxygenation, leading to potential damage to any susceptible organ systems or even fetal death.

Oxygen delivery is critically dependent on uterine blood flow. Uterine contractions decrease placental blood flow and result in intermittent episodes of decreased oxygen delivery. Normally, the fetus tolerates contractions without difficulty, but if the frequency, duration, or strength of contractions becomes excessive, fetal hypoxemia may result. Maternal position and the use of conduction anesthesia can also alter uterine blood flow and oxygen delivery during labor. Finally, labor may be complicated by conditions such as pre-eclampsia, abruptio placentae, chorioamnionitis, and other pathologic situations that can further alter blood flow and oxygen exchange within the placenta.

The umbilical cord is particularly vulnerable during labor because it can prolapse once membranes rupture or become compressed either through cord entanglement or secondary to oligohydramnios. While umbilical cord compression is common during labor, prolonged compression is infrequent but can seriously disrupt oxygen delivery and carbon dioxide removal and lead to acidosis or death.

Some fetuses are unusually susceptible to the effects of intrapartum hypoxemia, such as fetuses with growth retardation and those who are delivered prematurely. In these circumstances, hypoxia tends to progress more rapidly and is more likely to cause or aggravate metabolic acidemia which, in extreme cases, correlates

with poor long-term outcome. In severe cases, such hypoxia may lead to death (2, 3).

Experimentally induced hypoxia has been associated with consistent, predictable changes in the FHR (4). The fetal CNS is susceptible to hypoxia. Since the FHR and its alterations are most directly under CNS control through sympathetic and parasympathetic reflexes, alterations in the FHR can be sensitive indicators of fetal hypoxia (4, 5).

In some instances of decreased oxygenation, the pattern of the FHR change can identify the mechanism. Umbilical cord compression or, occasionally, head compression coincides with variable decelerations (6). These are defined as slowing of the FHR with abrupt onset and return and are frequently preceded and followed by small accelerations of the FHR. These decelerations vary in depth, duration, and shape on the tracing but generally coincide in timing and duration with the compression of the cord which, in turn, usually coincides with the timing of the uterine contractions.

Uterine contractions that result in decreases in fetal oxygenation exceeding that usually seen in labor may result in delayed or late decelerations. These are U-shaped decelerations of gradual onset and gradual return that are usually shallow (10–30 beats per minute [bpm]) and that reach their nadir after the peak of the contraction. In milder cases, they can be a reflex and the result of CNS hypoxia; in more severe cases, it has been postulated that they may be the result of direct myocardial depression.

Early decelerations are shallow and symmetrical with a pattern similar to that of late decelerations but reach their nadir at the same time as the peak of the contraction. They are seen in the active phase of labor, albeit infrequently. They are benign changes caused by fetal head compression.

Changes in the baseline FHR may also indicate the response of the fetus to an episode of hypoxia. Two specific parameters of the baseline FHR are important: rate and variability. The FHR at term usually ranges from 120–160 bpm. The initial response of the FHR to intermittent hypoxia is deceleration, but baseline tachycardia may develop if the hypoxia is prolonged and severe. Tachycardia also may be associated with conditions other than hypoxia (such as maternal fever, intraamniotic infection, and congenital heart disease). The presence of variability—or variation of successive beats in the FHR—is a useful indicator of fetal CNS integrity. In the absence of maternal sedation or extreme prematurity, decreasing variability—or flattening of the FHR baseline—may serve as a barometer of the fetal response to hypoxia. Because this is presumed to be a CNS response, in most situations, decelerations of the FHR will precede the loss of variability, indicating the

cause of neurologic depression. Many other factors, such as a fetal sleep cycle or medications, may decrease the activity of the CNS and the variability of the FHR. The development of decreased variability in the absence of decelerations is unlikely to be due to hypoxia (7).

Accelerations are common periodic changes in labor and are nearly always associated with fetal movement. These changes are virtually always reassuring and almost always confirm that the fetus is not acidotic at that time (8).

Guidelines for Performing Fetal Heart Rate Monitoring

The FHR may be evaluated by auscultation or by electronic monitoring. Auscultation is usually performed with a DeLee stethoscope or a Doppler ultrasound device.

Continuous FHR and contraction monitoring may be performed externally or internally. Most external monitors use a Doppler device with computerized logic to interpret and count the Doppler signals. Internal FHR monitoring is accomplished with a fetal electrode, which is a spiral wire placed directly on the fetal scalp or other presenting part. This method records the fetal electrocardiogram. In either case, the FHR is recorded continuously on the upper portion of a paper strip and every beat-to-beat interval is recorded as a rate. The lower portion of the strip records uterine contractions, which also may be monitored externally or internally. The most common paper speed in the United States is 3 cm/min.

Well-controlled studies have shown that intermittent auscultation of the FHR is equivalent to continuous electronic monitoring in assessing fetal condition when performed at specific intervals with a 1:1 nurse-to-patient ratio (9–14).

The intensity of FHR monitoring used during labor should be based on risk factors. When intensified monitoring is undertaken, such as when risk factors are present during labor, the fetal heart rate should be assessed according to the following guidelines:

- During the active phase of the first stage of labor: If auscultation is used the FHR should be evaluated and recorded at least every 15 minutes after a uterine contraction. If continuous electronic monitoring is used, the tracing should be evaluated at least every 15 minutes.
- During the second stage of labor: With auscultation, the FHR should be evaluated and recorded at least every 5 minutes. When electronic monitoring is used, FHR should also be evaluated at least every 5 minutes.

There are no comparative data indicating the optimal frequency at which intermittent auscultation should be performed in the absence of risk factors. One method is to evaluate and record the fetal heart rate at least every 30 minutes in the active phase of the first stage of labor and at least every 15 minutes in the second stage of labor.

With either auscultation or electronic monitoring, documentation of the evaluation is required. Auscultated FHR should be recorded in the chart after each observation. With electronic FHR monitoring, the monitor strip should be carefully labeled and the complete strip is usually retained, as with other medical records. Computer storage of fetal monitor records on devices such as laser discs which do not permit overwriting or revision appears to be a reasonable alternative, as do various methods of microfilm recording.

Documentation may consist of narrative notes or the use of comprehensive flow sheets detailing the periodic assessment. Specific responses to an abnormal FHR pattern such as further diagnostic procedures or therapeutic interventions also should be documented, as should the nature, date, and time of other pertinent events (eg, administration of medication or anesthesia).

Risks and Benefits

Currently, neither the most effective method of FHR monitoring nor the specific frequency or duration of monitoring to ensure optimal perinatal outcome has been identified by a significant body of scientific evidence. With the advent and liberal use of electronic FHR monitoring in the 1970s, there was great hope that intrapartum fetal death and morbidity associated with intrapartum asphyxia could be virtually eliminated. Retrospective studies of electronic FHR monitoring in both high- and low-risk populations were encouraging. A review of 11 studies including almost 40,000 electronically monitored patients and nearly 100,000 historical controls suggested a reduction in the intrapartum fetal death rate from 1.76/1,000 births in controls to 0.54/1,000 births in monitored patients (15). Similar reductions in neonatal death rates were also observed.

Subsequently, seven randomized, controlled trials have compared continuous electronic FHR monitoring with intermittent auscultation in both high- and low-risk patients; no differences in intrapartum fetal death rates were found (9–14, 16). It is significant that the intermittent auscultation groups in all but one of the seven studies had a dedicated 1:1 nurse-to-patient ratio. Nurses auscultated the FHR at least every 15 minutes in the first stage of labor and every 5 minutes in the second stage. If only the results of the studies with this intensity of

FHR auscultation are included, the intrapartum fetal death rate in auscultated women was only 0.5/1,000. This rate is nearly identical to those seen with electronic FHR monitoring in both prospective, randomized, controlled trials and retrospective, controlled studies (9–14).

In contrast, the most recently published randomized, controlled trial did show a significant reduction in perinatal deaths due to asphyxia in the electronically monitored group (17). It is not clear why this single study is so discordant with the others, but it does provide some promise that further studies may yet elucidate the real value of electronic FHR monitoring.

Likewise, a substantial body of evidence disproves the hypothesis that electronic fetal monitoring would reduce long-term neurologic impairment and cerebral palsy in newborns so monitored. Electronic FHR monitoring has been no more effective in reducing the rates of low Apgar scores at birth and long-term neurologic morbidity than has intensive intrapartum auscultative monitoring (as described here). One study did suggest that electronic FHR monitoring may decrease the rate of seizures in the newborn (14); however, this reduction did not persist into late childhood. On the other hand, another study showed a significant increase in cerebral palsy among premature infants monitored electronically during labor (18).

Certainly a correlation exists between abnormal FHR patterns and neurologic depression at birth; similarly, neonatal depression is correlated to some extent with adverse long-term neurologic outcome. It must be emphasized, however, that this correlation occurs only with prolonged and severe intrapartum fetal compromise. Indeed, the various methods of intrapartum fetal assessment currently used are not effective in predicting or preventing adverse long-term neurologic outcomes. Management of nonreassuring FHR patterns does not appear to affect the risk of subsequent cerebral palsy (19). This is due to the facts that neurologic abnormalities infrequently result from subtle events occurring during labor and delivery and, conversely, that most hypoxic and asphyxic episodes do not result in irreversible neurologic damage (19, 20).

The primary risk of electronic FHR monitoring is a potential increase in the cesarean delivery rate. This effect has been observed in both retrospective trials and the majority of prospective, randomized, controlled trials. More accurate interpretation of FHR monitoring, the use of fetal scalp blood pH monitoring, and possibly, the use of acoustic or scalp stimulation to elicit FHR accelerations can lead to more precise diagnosis of the condition of the fetus and, by inference, may lead to a decrease in the cesarean delivery rate. The use of amnioinfusion has also been shown in randomized,

controlled trials to lower the cesarean delivery rate for those patients with FHR patterns consistent with umbilical cord compression (21–23).

Interpretation and Management

A normal FHR pattern is reassuring and, when obtained by careful auscultation or electronic monitoring, is nearly always associated with a newborn who is vigorous at birth. Therefore, the terminology of *reassuring* implies that in the absence of patterns defined as nonreassuring, the fetus can be assumed—with a great deal of reliability—to have normal oxygen and acid–base status.

Conversely, nonreassuring patterns are quite nonspecific and cannot reliably predict whether a fetus will be well oxygenated, depressed, or acidotic. However, factors other than hypoxia may lead to a nonreassuring FHR. In addition, an abnormal FHR pattern associated with hypoxia may neither depict the severity of hypoxia nor predict how it will progress if labor is allowed to proceed.

The term *fetal distress*, while imprecise and inaccurate, has been applied so commonly to abnormal FHR patterns in labor that it is difficult to abandon. It is more helpful clinically to describe the fetal heart rate patterns in terms of type and severity, and to outline a management plan accordingly.

Interpretation of Fetal Heart Rate Patterns

The initial FHR pattern should be carefully evaluated for the presence or absence of accelerations, decelerations, and abnormalities of the baseline. In one study, the first 30 minutes of electronic FHR monitoring identified about 50% of all fetuses for whom cesarean delivery will be required for a nonreassuring FHR pattern or “fetal distress” (24). While the progression of decelerations will usually explain changes in the baseline later in labor, abnormalities of the baseline on admission, such as fetal tachycardia or loss of variability, are the most difficult to interpret, as data regarding previous changes are lacking.

Periodic changes in FHR are common in labor; they occur in response to contractions or fetal movement and include accelerations and decelerations. Variable decelerations are the most common decelerations seen in labor and indicate umbilical cord compression; they are generally associated with a favorable outcome (25). Only when they become persistent, progressively deeper, and longer lasting are they considered nonreassuring. Although progression is more important than absolute parameters, persisting variable decelerations to less than 70 bpm lasting greater than 60 seconds are generally concerning. In addition to prolonged and

deep variable decelerations, those with persistently slow return to baseline are also considered nonreassuring, as these reflect hypoxia persistent beyond the relaxation phase of the contraction (26). The response of the baseline FHR to the variable decelerations and the presence or absence of accelerations are important in formulating a management plan for the patient with significant variable decelerations. When nonreassuring variable decelerations are associated with the development of tachycardia and loss of variability, one begins to see substantial correlation with fetal acidosis.

Late decelerations may be secondary to transient fetal hypoxia in response to the decreased placental perfusion associated with uterine contractions. Occasional or intermittent late decelerations are not uncommon during labor. When late decelerations become persistent (ie, present with most contractions), they are considered nonreassuring, regardless of the depth of the deceleration. Late decelerations caused by reflex—those mediated by the CNS—generally become deeper as the degree of hypoxia becomes more severe. However, as metabolic acidosis develops from tissue hypoxia, late decelerations are believed to be the result of direct myocardial depression, and at this point, the depth of the late deceleration will not indicate the degree of hypoxia (27).

A prolonged deceleration, often incorrectly referred to as bradycardia, is an isolated, abrupt decrease in the FHR to levels below the baseline that lasts at least 60–90 seconds. These changes are always of concern and may be caused by virtually any mechanism that can lead to fetal hypoxia. The severity of the event causing the deceleration is usually reflected in the depth and duration of the deceleration, as well as the degree to which variability is lost during the deceleration. When such a deceleration returns to the baseline, especially with more profound episodes, a transient fetal tachycardia and loss of variability may occur while the fetus is recovering from hypoxia. The degree to which such decelerations are nonreassuring depends on their depth and duration, loss of variability, response of the fetus during the recovery period, and, most importantly, the frequency and progression of recurrence.

A sinusoidal heart rate pattern consists of a regular oscillation of the baseline long-term variability resembling a sine wave. This smooth, undulating pattern, lasting at least 10 minutes, has a relatively fixed period of three to five cycles per minute and an amplitude of 5–15 bpm above and below the baseline. Short-term variability is usually absent. This pattern may be associated with severe chronic, as opposed to acute, fetal anemia. It has also been described following the use of alphaprodine or other medications and, in such circumstances, may not represent fetal compromise. Additionally, severe hypoxia and acidosis occasionally manifest

as a sinusoidal FHR; the reason for this is as yet not understood. True sinusoidal patterns are quite rare. Unfortunately, small, frequent accelerations of low amplitude are easy to confuse with sinusoidal patterns. The former are benign and occur more frequently while the latter, if they meet the strict criteria of a sinusoidal FHR, are always nonreassuring (28).

Evaluation and Management of Nonreassuring Patterns

With a persistently nonreassuring FHR pattern in labor, the clinician should approach the evaluation and management in a four-step plan as follows:

1. When possible, determine the etiology of the pattern.
2. Attempt to correct the pattern by specifically correcting the primary problem or by instituting general measures aimed at improving fetal oxygenation and placental perfusion.
3. If attempts to correct the pattern are not successful, fetal scalp blood pH assessment may be considered.
4. Determine whether operative intervention is warranted and, if so, how urgently it is needed.

The search for the cause of the nonreassuring FHR pattern should be directed by the clinician's interpretation of the pattern. If there are late decelerations, then excessive uterine contractions, maternal hypotension, or maternal hypoxemia should be considered. For severe variable or prolonged decelerations, a pelvic examination should be performed immediately to rule out umbilical cord prolapse or rapid descent of the fetal head. If no causes of such decelerations are found, one can usually conclude that umbilical cord compression is responsible.

General measures that may improve fetal oxygenation and placental perfusion include administering maternal oxygen by a tight-fitting face mask, ensuring that the woman is in the lateral recumbent position, discontinuing oxytocin, and, if maternal intravascular volume status is in question, beginning intravenous hydration.

Oxygen Therapy

The arterial Po_2 in the fetus is normally about one fourth of the arterial Po_2 in the mother. Despite this low Po_2 , the fetal blood can carry a large amount of oxygen from the placenta because of the high concentration of fetal hemoglobin and its high affinity for oxygen. Oxygen is constantly and rapidly consumed and cannot be stored by the fetus. The fetus is thus dependent upon a constant supply of oxygen; a reduction in this supply cannot be tolerated for more than brief intervals.

When there is evidence of a nonreassuring pattern, administration of supplemental oxygen to the mother is

indicated. A significant increase in maternal oxygenation is accomplished with a tight-fitting face mask and an oxygen flow rate of 8–10 L/min. Although such administration results in only a small increase in fetal Po_2 , animal studies have suggested that a significant increase in fetal oxygen content may occur. Assuming both adequate placental exchange and delivery of oxygen through unobstructed umbilical cord circulation, the resultant increase in total fetal blood oxygen content is 30–40% or greater in animal studies, depending upon the degree to which the fetal Po_2 has fallen (29). When given for the usual duration of labor, maternal oxygen therapy has no known harmful effects on the fetus.

Maternal Position

Maternal position during labor can affect uterine blood flow and placental perfusion. In the supine position, there is an exaggeration of the lumbar lordotic curvature of the maternal spine facilitating compression of the vena cava and aortoiliac vessels by the gravid uterus. This results in decreased return of blood to the maternal heart leading directly to a fall in cardiac output, blood pressure, and uterine blood flow. In the supine position, aortic compression by the uterus may result in an increase in the incidence of late decelerations and a decrease in fetal scalp pH (30). The lateral recumbent position (either side) is best for maximizing cardiac output and uterine blood flow and is often associated with improvement in the FHR pattern (31). Other maternal positions may accomplish similar uterine displacement.

Epidural Block

Some degree of maternal hypotension is a relatively common complication of epidural block, occurring in 5–25% of procedures (32). Prophylactic intravascular volume expansion with 500–1,000 ml of lactated Ringer's injection is recommended prior to administration of an epidural anesthetic in order to diminish the likelihood of this complication. Treatment with an increase in intravenous fluids, left uterine displacement, or 2.5–10 mg of ephedrine intravenously or intramuscularly is recommended for hypotension occurring with administration of an epidural block. During the period of hypotension, uteroplacental perfusion may be compromised. This may be manifested by fetal tachycardia, prolonged decelerations, decreased beat-to-beat variability, late decelerations, or some combination of these.

The frequency of prolonged decelerations after administration of epidural analgesia has been reported to be 7.9–12.5% (33, 34). Uterine hypertonia with resultant prolonged decelerations has been observed in patients receiving epidural block during labor even in the absence of systemic hypotension (35). Management

of epidural-associated decelerations should focus on treatment of the specific cause—either the increased uterine tone or maternal hypotension.

Oxytocin

Careful use of oxytocin is necessary to minimize uterine hyperstimulation and potential maternal and fetal morbidity. If nonreassuring FHR changes occur in patients receiving oxytocin, the infusion should be decreased or discontinued. Restarting the infusion at a lower rate or increasing it in smaller increments may be better tolerated.

Amnioinfusion

Variable decelerations are frequently encountered in both the first and second stages of labor. Those occurring prior to fetal descent at 8–9 cm of dilatation are most frequently seen in patients with oligohydramnios.

In patients with decreased amniotic fluid volume in either preterm or term pregnancies, replacement of amniotic fluid with normal saline infused through a transcervical intrauterine pressure catheter has been reported to decrease both the frequency and severity of variable decelerations (22, 23, 36). Replacement of amniotic fluid may be elected therapeutically in patients with progressive variable decelerations. Although randomized, controlled trials are lacking, it is reasonable to replace amniotic fluid prophylactically at the onset of labor in patients with known oligohydramnios. Studies also have demonstrated that amnioinfusion results in reductions in rates of cesarean delivery for “fetal distress,” primarily due to variable decelerations, and fewer low Apgar scores at birth. Acute saline amnioinfusion has been reported to be an effective therapy that relieves most repetitive variable or prolonged intrapartum decelerations and is without apparent maternal or fetal risk (21). Investigators have also reported a decrease in newborn respiratory complications from meconium in patients who receive amnioinfusion. This results presumably from the dilutional effect of amnioinfusion and possibly from prevention of in utero fetal gasping that may occur during episodes of hypoxia caused by umbilical cord compression (37–39).

Generally, two techniques of amnioinfusion have been described: bolus infusion and continuous infusion. Originally described for patients requiring therapeutic amnioinfusion, bolus infusion of up to 800 ml can be administered at a rate of 10–15 ml/min until the decelerations abate; then, an extra 250 ml can be added (21). The infusion can be repeated when there is sudden or large fluid loss due to maternal position change or performance of the Valsalva maneuver or when an abnormal FHR tracing recurs. Ultrasound assessment

of amniotic fluid volume can also be used to determine the need for repeat infusion (39).

Alternatively, a continuous infusion may be performed. While this was originally described for prophylactic amnioinfusion (22), it may also be used therapeutically. Continuous amnioinfusion usually begins with a loading dose of 10 ml/min for 1 hour followed by a maintenance dose of 3 ml/min. Use of an infusion pump, although not essential, can more accurately control both the volume and rate of infusion.

Warmed saline has been used in prophylactic amnioinfusions of preterm patients, but warming of infusate has not been shown to be of any specific value in term or preterm patients. For the term patient, there do not appear to be any adverse effects on maternal or newborn temperature or electrolytes when room temperature saline amnioinfusion is administered (36).

Care should be taken to avoid overdistingending the uterine cavity. Increased basal uterine tone and sudden deterioration of FHR has been reported with infusion volumes of as little as 250 ml; abnormal FHR secondary to polyhydramnios has been reported following prolonged continuous amnioinfusion (40). With continuous amnioinfusion, intermittent discontinuation to assess basal uterine tone or the use of double-lumen uterine pressure catheters is recommended.

The onset of beneficial effects of amnioinfusion requires at least 20–30 minutes, so care should be taken when performing saline amnioinfusion to avoid delaying surgical intervention if there is no improvement in a significantly abnormal FHR. Preparations for expeditious delivery should be made simultaneously with saline amnioinfusion when worsening variable or prolonged decelerations occur.

Tocolytic Agents

Tocolytic agents are a potentially valuable tool in the management of certain intrapartum events. Changes in the FHR suggesting possible nonreassuring FHR patterns may accompany excessive uterine contractions. If a nonreassuring FHR pattern results from such excessive contractions, specific measures can be taken to decrease uterine activity. If oxytocin is being administered, the dose should be decreased or discontinued. In addition, a tocolytic agent sometimes may be injected. Terbutaline, 0.25 mg subcutaneously or 0.125–0.25 mg intravenously, has been used for this purpose. Both beta agonists and magnesium sulfate have been reported to be of value in rapidly improving fetal condition secondary to uterine relaxation during active labor (41, 42).

Even in the absence of uterine hypertonus, abnormal FHR patterns occurring in response to uterine contractions may be improved and newborn condition benefitted by the administration of tocolytic agents

(43). This is especially true when unavoidable delays in effecting operative delivery are encountered.

Certain potential maternal and fetal side effects need to be considered when tocolytic agents are administered for a nonreassuring FHR. Beta agonists elevate both serum glucose levels and maternal and fetal heart rate. However, the direct effect on FHR is minor, and any improvement in FHR from a nonreassuring pattern following acute beta agonist therapy is not due to a direct effect of therapy on the fetal heart, but rather the result of a decrease in the uterine activity. Maternal pulse pressure is widened, and peripheral vascular resistance decreases. Additionally, reinstating or augmenting uterine activity with oxytocin following acute administration of tocolytic agents may be necessary to reestablish a normal labor pattern. The administration of tocolytic therapy for nonreassuring FHR patterns should not delay necessary interventions.

Management of Persistent Nonreassuring Fetal Heart Rate Patterns

If the FHR pattern remains uncorrected, the decision to intervene depends on the clinician's assessment of the likelihood of severe hypoxia and the possibility of metabolic acidosis, as well as the estimated time to spontaneous delivery. For the fetus with persistent nonreassuring decelerations, normal FHR variability and the absence of tachycardia generally indicate the lack of acidosis. However, variability is difficult to quantify except in the extremes.

Persistent late decelerations or severe variable decelerations associated with the absence of variability are always nonreassuring and generally require prompt intervention unless they spontaneously resolve or can be corrected rapidly with immediate conservative measures (ie, oxygen, hydration, or maternal repositioning). The absence of variability or markedly decreased variability demonstrated on an external monitor is generally reliable. The presence of FHR variability is not confirmatory, however, and, in the presence of nonreassuring decelerations, a fetal electrode should be placed when possible.

The presence of spontaneous accelerations of greater than 15 bpm lasting at least 15 seconds virtually always ensures the absence of fetal acidosis. Fetal scalp stimulation or vibroacoustic stimulation can be used to induce accelerations; these also indicate the absence of acidosis (44, 45). Conversely, there is about a 50% chance of acidosis in the fetus who fails to respond to stimulation in the presence of an otherwise nonreassuring pattern (44, 45). In these fetuses, assessment of scalp blood pH, if available, may be used to clarify the acid-base status. This technique, while occasionally helpful, is used uncommonly in current obstetric practice (46).

If the FHR pattern remains worrisome, either induced accelerations or repeat assessment of scalp blood pH is required every 20–30 minutes for continued reassurance. In cases in which the FHR patterns are persistently nonreassuring and acidosis is present or cannot be ruled out, the fetus should be promptly delivered by the most expeditious route, whether abdominal or vaginal.

Summary

Because alterations in fetal oxygenation occur during labor and because many complications can occur during this critical period, some form of FHR evaluation should be provided for all patients. The choice of technique is based on various factors, including the resources available. Nonreassuring FHR patterns are common and quite nonspecific. By understanding the physiologic and pathophysiologic basis of FHR monitoring, as well as its capabilities and limitations, the clinician can reduce the need for interventions.

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This Technical Bulletin was developed under the direction of the Committee on Technical Bulletins of the American College of Obstetricians and Gynecologists as an educational aid to obstetricians and gynecologists. The committee wishes to thank Thomas J. Garite, MD, and Michael P. Nageotte, MD, for their assistance in the development of this bulletin. This Technical Bulletin does not define a standard of care, nor is it intended to dictate an exclusive course of management. It presents recognized methods and techniques of clinical practice for consideration by obstetrician–gynecologists for incorporation into their practices. Variations of practice taking into account the needs of the individual patient, resources, and limitations unique to the institution or type of practice may be appropriate. Requests for authorization to make photocopies should be directed to the Copyright Clearance Center, 222 Rosewood Drive, Danvers, MA 01923; telephone (508) 750-8400.

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ISSN 1074-8628

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