INTRODUCTION

Normal human labor is characterized by regular uterine contractions and repeated episodes of transient interruption of fetal oxygenation. Most fetuses tolerate this process well, but some do not. The fetal heart rate (FHR) pattern helps to distinguish the former from the latter as it is an indirect marker of fetal cardiac and central nervous system responses to changes in blood pressure, blood gases, and acid-base status. (See "Nonstress test and contraction stress test", section on 'Physiologic basis of fetal heart rate changes'.)

The rationale for intrapartum FHR monitoring is that identification of FHR changes potentially associated with inadequate fetal oxygenation may enable timely intervention to reduce the likelihood of hypoxic injury or death. Although virtually all obstetric societies advise monitoring the FHR during labor, the benefit of this intervention has not been clearly demonstrated and this position is largely based upon expert opinion and medicolegal precedent.

The procedure for intrapartum FHR monitoring, physiology behind FHR accelerations and decelerations, classification of FHR tracings, and use of ancillary tests to evaluate the fetus will be discussed here. Intrapartum management of category I, II, and III FHR tracing is reviewed separately (see "Management of intrapartum category I, II, and III fetal heart rate tracings"). Antepartum FHR monitoring (nonstress test, contraction stress test) is also reviewed separately. (See "Nonstress test and contraction stress test").

DOES INTRAPARTUM FHR MONITORING IMPROVE OUTCOME?

Although some evidence suggests that intrapartum fetal monitoring is associated with a reduction in intrapartum death [1], a reduction in long-term neurologic impairment has not been proven. All available data are derived from trials comparing continuous electronic monitoring with intermittent auscultation. There are no randomized trials of intrapartum fetal monitoring versus no intrapartum fetal monitoring.

For both low- and high-risk pregnancies, continuous electronic FHR monitoring is not clearly superior to intermittent auscultation with respect to preventing death or poor long-term neurologic outcome, and has a high false-positive rate [2-4]. A 2017 systematic review that compared continuous electronic FHR monitoring with intermittent auscultation (13 randomized trials, >37,000 low- and high-risk pregnancies) reported the following major findings [4]:

- No significant differences between techniques were noted for the following newborn/childhood outcomes:
  - Acidemia (measured in cord blood) (relative risk [RR] 0.92, 95% CI 0.27-3.11)
  - Apgar score <4 at five minutes (RR 1.80, 95% CI 0.71-4.59)
  - Neonatal intensive care unit admission (RR 1.01, 95% CI 0.86-1.18)
  - Hypoxic ischemic encephalopathy (RR 0.46, 95% CI 0.04-5.03)
  - Perinatal mortality (RR 0.86, 95% CI 0.59-1.24)
  - Neurodevelopmental impairment at ≥12 months of age (RR 3.88, 95% CI 0.83-18.2)
  - Cerebral palsy (RR 1.75, 95% CI 0.84-3.63)

- Although use of continuous electronic FHR monitoring resulted in fewer neonatal seizures (RR 0.50, 95% CI 0.31-0.80), there were no differences in long-term neurologic outcomes [4, 5].

- Continuous electronic FHR monitoring resulted in more operative vaginal deliveries for abnormal FHR patterns or acidosis (RR 2.54, 95% CI 1.95-3.31), fewer spontaneous vaginal births (RR 0.91, 95% CI 0.86-0.96), and more cesarean deliveries for abnormal FHR patterns or acidosis (RR 2.38, 95% CI 1.89-3.01). Overall risks of instrumental vaginal and cesarean delivery were also statistically increased (RR 1.15 and 1.63, respectively). Data for low-risk and high-risk subgroups, preterm pregnancies, and high-quality trials were consistent with these overall results.

Literature review current through: Feb 2019. | This topic last updated: Nov 05, 2018.
One important limitation of these findings is that none of the more contemporary trials (which included a substantially larger number of patients) demonstrated a significant difference in the rate of cesarean delivery between patients monitored continuously and those monitored by intermittent auscultation, in contrast to all four of the randomized trials published before 1980, which demonstrated significantly higher rates of cesarean delivery in continuously monitored patients. Another important limitation is that a statistical reduction in the perinatal death rate is difficult to demonstrate because death is a rare outcome. In the 2017 meta-analysis, even the pooled results from multiple randomized trials lacked sufficient power to permit a definitive conclusion [4]. However, a previous meta-analysis that specifically evaluated perinatal deaths attributed to fetal hypoxia in low- and high-risk pregnancies concluded that this outcome was significantly less common in continuously monitored patients than those monitored with intermittent auscultation (7/9398 versus 17/9163; 0.7 versus 1.8 per 1000; odds ratio 0.41, 95% CI 0.17-0.98; nine trials) [1].

A statistical reduction in the incidence of cerebral palsy with continuous intrapartum FHR monitoring is also difficult to prove for several reasons. Many cases of cerebral palsy are due to ante partum, rather than intrapartum, events. In such cases, intrapartum interventions are unlikely to change the course of the disease [2]. Most FHR abnormalities are not associated with fetal acidemia or hypoxemia, and most fetal acidemia and hypoxemia does not result in neurologic disability. In fact, one study calculated that 99.8 percent of "abnormal" FHR tracings are not associated with the later development of cerebral palsy, yielding a positive predictive value that is equal to, or lower than, the prevalence of cerebral palsy in the general population [2]. Another potentially confounding factor is that a preexisting fetal neurologic disorder may be the cause, rather than the result, of an intrapartum FHR abnormality [6]. In many cases, the interventions that are undertaken in the setting of abnormal FHR tracings may alter the natural history of fetal oxygen deprivation, potentially decoupling FHR abnormalities from hypoxic neurologic sequelae such as cerebral palsy. Lastly, the degree of fetal oxygen deprivation that leads to long-term neurologic injury is close to that causing fetal death; thus, many severely depressed term fetuses may either survive intact or die, rather than survive with disability [7].

CANDIDATES FOR INTRAPARTUM FETAL MONITORING

Any pregnancy in which detection of a FHR abnormality would prompt intervention, such as conservative maneuvers to improve fetal oxygenation and/or cesarean delivery, is a candidate for intrapartum fetal monitoring.

Some degree of FHR monitoring during labor has become routine in the United States, even though the clinical benefit has not been established conclusively (see ‘Does intrapartum FHR monitoring improve outcome?’ above). It is unlikely that this practice will be abandoned in the near future because most patients and clinicians are reassured by normal FHR monitoring results, and most believe that detection of a FHR abnormality and prompt intervention is beneficial.

PROCEDURE

Continuous monitoring versus intermittent auscultation: Recommendations of selected national organizations — Although major national organizations endorse monitoring low-risk pregnancies with intermittent auscultation using a fetal stethoscope (Pinard, Laennec, fetoscope, or Doppler device), it is rarely performed in the United States because it provides limited information about FHR variability, accelerations, or decelerations and requires one-to-one nursing care, which is costly and impractical for most maternity units. The possible increase in operative delivery associated with continuous electronic FHR monitoring is usually considered a reasonable trade-off for a possible reduction in risk of adverse fetal/neonatal outcome and the reduction in personnel costs.

- American College of Obstetricians and Gynecologists [8]:
  - Either continuous electronic FHR monitoring or intermittent auscultation is acceptable in uncomplicated patients.
  - High-risk pregnancies (eg, preeclampsia, suspected growth restriction, type 1 diabetes mellitus) should be monitored continuously during labor.

- National Institute for Health and Care Excellence [9]:
  - In all birth settings, offer intermittent auscultation to low-risk women in the first stage of labor. Do not perform cardiotocography in low-risk women.
  - Advise continuous cardiotocography if any of the following risk factors occur during labor:
    - Suspected chorioamnionitis, sepsis, or temperature ≥38°C
    - Severe hypertension (≥160/110 mmHg)
    - Oxytocin use
    - Significant meconium
    - Fresh vaginal bleeding
  - If continuous cardiotocography was used because of concerns arising from intermittent auscultation but the tracing is normal after 20 minutes of observation, remove the cardiotocograph and return to intermittent auscultation.
Equipment — External FHR monitoring is as reliable as internal FHR monitoring in most cases [10]. Internal FHR monitoring is preferable when the externally-derived tracing is difficult to obtain or interpret because of poor technical quality. This may result from a number of conditions, including prematurity, maternal obesity, frequent maternal or fetal movement, uterine myomata, polyhydramnios, or multiple gestation.

Noninvasive (external) electronic monitoring — Noninvasive (external) FHR monitoring can be performed continuously or intermittently with a Doppler ultrasound device on the maternal abdomen. This device detects the motion of the fetal heart and uses this information to create a complex wave form. The computer then detects the peaks of successive waves to calculate a mechanical R wave-to-R wave (RR) interval. To minimize artifact caused by the inherent variation in the Doppler ultrasound signal, the computer calculates the FHR by averaging several consecutive RR intervals. This technology, called autocorrelation, reduces signal variation and produces a processed sound, which is not the actual fetal heartbeat. The FHR tracing derived from Doppler ultrasound using the autocorrelation technique closely resembles that derived from a fetal electrocardiogram (ECG), although baseline variability may be more apparent with the Doppler technique. An alternative technology (eg, Monica AN24) uses several electrodes placed on the maternal abdomen to detect the electrical fetal RR interval. Wireless monitors enable FHR monitoring while the women is ambulating [11].

Invasive (internal) electronic monitoring — A fetal ECG can be obtained by placing a bipolar spiral electrode into the fetal scalp transcervically; a second reference electrode is placed on the maternal thigh to eliminate electrical interference from maternal cardiac activity. A computer calculates the FHR based on the RR interval (waveform 1). Artifact is minimal so the signal is clear and provides accurate measurement of the variability between successive heartbeats without the need for autocorrelation.

Application of artificial intelligence computer programs to fetal ECG signal processing has led to development of devices to overcome the limitations of FHR pattern interpretation by human observers [12,13]. This has been made possible by technical improvements in signal acquisition and processing, and by algorithms for pattern interpretation based on standardization of visual pattern analysis and correlation with fetal scalp blood and umbilical artery pH determinations. As an example, the STAN monitor analyzes the fetal T wave and ST segment. However, this technology has not been widely adopted in the United States and remains investigational. (See ‘ST analysis’ below.)

Frequency and duration of monitoring — Upon admission to the labor unit, the standard of care in most hospitals is to assess the FHR for a minimum of 20 to 30 minutes, along with uterine activity and maternal vital signs. The goal is to identify fetuses who have or are at increased risk for abnormal intrapartum FHR patterns and who might benefit from continuous rather than intermittent intrapartum FHR monitoring [14]. This initial assessment and additional assessments during labor may also inform decisions regarding the use of uterine stimulants, such as oxytocin or prostaglandins. However, a 2017 systematic review of randomized trials found that the "labor admission test" did not reliably predict fetal ability to tolerate labor over time in low-risk pregnancies and did not reduce neonatal morbidity compared with intermittent auscultation of the FHR [15]. There were insufficient data to draw conclusions about the value of the admission test in women with high-risk pregnancies.

A common practice in low-risk pregnancies with a normal initial FHR pattern is to continuously monitor the FHR when possible (ie, patient is not ambulating, bathing, moving around, etc) and review the FHR 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 [8]. High-risk pregnancies are monitored continuously and the FHR is usually reviewed at least every 15 minutes in the active phase of the first stage of labor, and at least every 5 minutes in the second stage [8]. The interpretation of the FHR should be documented in the intrapartum record.

As discussed above, intermittent auscultation is rarely used intrapartum in the United States (see 'Continuous monitoring versus intermittent auscultation: Recommendations of selected national organizations' above). If intermittent auscultation is used in low-risk pregnancies, a standard approach is to determine, evaluate and record the FHR during and immediately after a uterine contraction at least every 30 minutes during the active phase of the first stage of labor and at least every 15 minutes during the second stage [16]. If risk factors are present, the FHR is determined, evaluated and recorded preferably before, during, and after a uterine contraction at least every 15 minutes during the active phase of the first stage of labor and at least every 5 minutes during the second stage.

PHYSIOLOGIC SIGNIFICANCE OF SELECTED FHR CHARACTERISTICS

Baseline FHR — A normal baseline FHR is 110 to 160 bpm, and reflects lack of pathology or pathologic effects from factors that regulate FHR. These factors include intrinsic cardiac pacemakers (sinoatrial node, atrioventricular node), cardiac conduction pathways, autonomic innervation (sympathetic, parasympathetic), intrinsic humoral factors (catecholamines), extrinsic factors (medications), and local factors (calcium, potassium), which are discussed in detail separately. (See "Nonstress test and contraction stress test", section on 'Physiologic basis of fetal heart rate changes'.)

Baseline bradycardia may be associated with maternal beta-blocker therapy, hypothermia, hypoglycemia, hypothyroidism, or fetal heart block or interruption of fetal oxygenation. Fetal tachycardia may be associated with maternal fever, infection, medications, hyperthyroidism, elevated catecholamines, or fetal anemia, arrhythmia, or interruption of fetal oxygenation.

Variability — FHR variability is the result of integrated activity between the sympathetic and parasympathetic branches of the autonomic nervous system. Moderate baseline variability reflects the oxygenation of the central nervous system and reliably predicts the absence of damaging degrees of hypoxia-induced metabolic acidemia at the time it is observed [17-19].
However, the converse is not true: Minimal or absent variability alone is a poor predictor of fetal metabolic acidemia or hypoxic injury at the time it is observed [17]. Other conditions potentially associated with minimal or absent variability include fetal sleep cycle, arrhythmia, medications, extreme prematurity, congenital anomalies, or preexisting neurologic injury. Most of the literature regarding decreased variability does not differentiate between absent variability (amplitude range undetectable) and minimal variability (amplitude range detectable but ≤5 bpm). Therefore, it is not possible to make definitive conclusions about the clinical significance of absent versus minimal variability.

The significance of marked variability is unknown. It may be a normal variant or an exaggerated autonomic response to transient interruption of fetal oxygenation.

**Accelerations** — FHR accelerations are frequently associated with fetal movement, possibly as a result of stimulation of peripheral proprioceptors, increased catecholamine release, and autonomic stimulation of the heart. As with moderate baseline variability, FHR accelerations reliably predict the absence of damaging degrees of fetal hypoxia and fetal metabolic acidemia at the time they are observed [17-19]. (See ‘FHR response to stimulation’ below.)

However, the converse is not true. The absence of accelerations is a poor predictor of fetal metabolic acidemia or hypoxic injury [17]. Other conditions potentially associated with the absence of accelerations include fetal sleep cycle, arrhythmia, medications, extreme prematurity, congenital anomalies, fetal anemia, and preexisting neurologic injury.

**Decelerations unrelated to fetal oxygenation**

**Early deceleration** — An early deceleration likely represents an autonomic response to changes in intracranial pressure and/or cerebral blood flow caused by intrapartum compression of the fetal head during a uterine contraction and maternal expulsive efforts, although the precise physiologic mechanism is not known. Early decelerations are clinically benign: They are not associated with an interruption of fetal oxygenation, metabolic acidemia, or hypoxic-ischemic neurologic injury. (See “Management of intrapartum category I, II, and III fetal heart rate tracings”, section on ‘Category I pattern’.)

Well-designed case-control studies have failed to identify any measure of uterine activity as an independent risk factor for cerebral palsy [20-25]. In a large cohort study comparing neonatal outcomes of more than 380,000 spontaneous vaginal deliveries with those of more than 33,000 cesarean deliveries without labor, neonates exposed to uterine contractions of sufficient duration, frequency and intensity to result in vaginal delivery had no higher rates of mechanical brain injury (intracranial hemorrhage) than those with no exposure to labor [26].

**Decelerations related to interruption of fetal oxygenation**

**Late deceleration** — In most cases, a late deceleration is a reflex fetal response to transient hypoxemia during a uterine contraction (table 1 and waveform 2 and waveform 3 and waveform 4 and waveform 5 and waveform 6) [27]. When uterine contractions compress maternal blood vessels traversing the uterine wall, maternal perfusion of the intervillous space of the placenta is reduced. Reduced delivery of oxygenated blood to the intervillous space can reduce diffusion of oxygen into the fetal capillary blood in the chorionic villi, leading to a decline in fetal PO2. When fetal PO2 falls below the normal range (approximately 15 to 25 mmHg in the umbilical artery), chemoreceptors initiate an autonomic reflex response. Initially, sympathetic outflow causes peripheral vasconstriction, shunting oxygenated blood flow away from non-vital vascular beds and toward vital organs such as the brain, heart and adrenal glands. The resulting increase in fetal blood pressure is detected by baroreceptors, which trigger a parasympathetic reflex and slow the heart rate, reduce cardiac output, and return blood pressure to normal. After the contraction, fetal oxygenation is restored, autonomic reflexes subside, and the FHR gradually returns to baseline. This combined sympathetic-parasympathetic reflex response to transient interruption of fetal oxygenation has been confirmed in animal studies [27-36]. Interruption of the oxygen pathway to the fetus can occur at multiple maternal levels in addition to uterine contractions, such as the lungs (eg, maternal hypoxemia), heart (eg, poor cardiac output) or vasculature (eg, hypotension).

Rarely, fetal oxygenation is interrupted sufficiently to result in both severe hypoxemia and metabolic acidemia and, in turn, direct myocardial depression and late decelerations [27]. Late decelerations related to severe hypoxemia, metabolic acidemia, and myocardial depression are predictive of an increased risk for adverse neonatal outcome. The more benign late decelerations resulting from a reflex response to transient hypoxemia can be distinguished from these more concerning late decelerations by the observation of moderate baseline variability or accelerations, which reliably exclude the presence of damaging degrees of hypoxia-induced metabolic acidemia [17-19]. However, recurrent late decelerations with absent/minimal variability and no accelerations require prompt attention because, in such cases, ongoing hypoxic injury cannot be excluded by the FHR tracing alone [17-19,27]. (See “Management of intrapartum category I, II, and III fetal heart rate tracings”, section on ‘Category III pattern’ and “Management of intrapartum category I, II, and III fetal heart rate tracings”, section on ‘Late decelerations without loss of variability or accelerations’.)

**Variable deceleration** — A variable deceleration reflects the fetal autonomic reflex response to transient mechanical compression of the umbilical cord (table 1 and waveform 7 and waveform 8) [32,37-45]. Initially, compression of the umbilical cord occludes the thin-walled, compliant umbilical vein, decreasing fetal venous return and triggering a baroreceptor-mediated reflex rise in FHR (sometimes referred to as a “shoulder”). Further compression occludes the umbilical arteries, causing an abrupt increase in fetal peripheral resistance and blood pressure. Baroreceptors detect the abrupt rise in blood pressure, triggering an increase in parasympathetic outflow and an abrupt decrease in heart rate. As the cord is decompressed, this sequence of events occurs in reverse.
Cord compression with or without other sources of disruption of fetal oxygenation may result in recurrent variable decelerations with absent/minimal variability and no accelerations. Prompt attention is required because ongoing hypoxic injury cannot be excluded [17-19]. (See "Management of intrapartum category I, II, and III fetal heart rate tracings", section on 'Category III pattern' and "Management of intrapartum category I, II, and III fetal heart rate tracings", section on 'Variable decelerations without loss of variability or accelerations'.) Classification of variable decelerations as mild, moderate, or severe does not correlate with outcome and is not recommended by the National Institute of Child Health and Human Development [17]. Similarly, the clinical significance of "atypical" features of variable decelerations is unclear (atypical features include a slow return of the FHR to baseline after the end of the contraction (sometimes called a "variable with a late component"), biphasic decelerations, tachycardia after the variable deceleration (sometimes referred to as "overshoot"), accelerations preceding and/or following the variable deceleration (sometimes called "shoulders"), or reduction in post-deceleration baseline) [17]. Terms such as "variable with a late component," "shoulders," "overshoots," "Hon pattern," and "conversion pattern" have been used in some descriptive reports to identify FHR patterns that have not been demonstrated in appropriately controlled studies to impact fetal condition or neonatal outcome. Such terms are not included in standard fetal monitoring terminology. In the absence of appropriate scientific confirmation of clinical significance, the use of such terms should be avoided.

Prolonged deceleration — By definition, a prolonged deceleration reflects a fall in FHR baseline by ≥15 bpm and lasting ≥2 minutes but <10 minutes [17,46]. It is caused by the same physiologic mechanisms responsible for late or variable decelerations, but interruption of fetal oxygenation occurs for a prolonged period of time. As discussed above, absent/minimal variability and no accelerations require prompt attention because ongoing hypoxic injury cannot be excluded. (See "Management of intrapartum category I, II, and III fetal heart rate tracings", section on 'Category III pattern' and "Management of intrapartum category I, II, and III fetal heart rate tracings", section on 'Fetal bradycardia/prolonged deceleration without loss of variability'.) If the fall in FHR baseline lasts ≥10 minutes, it is defined as a baseline change [17]. A baseline change with absent/minimal variability and no accelerations requires prompt attention because ongoing hypoxic injury cannot be excluded.

Sinusoidal pattern — The sinusoidal pattern is characterized by fluctuations in the FHR baseline with regular amplitude and frequency. This pattern is associated with severe fetal anemia, although the pathophysiologic mechanism has not been definitively proven [47-49]. Variations of the pattern have been described in association with administration of narcotic analgesics and with chorioamnionitis. (See "Management of intrapartum category I, II, and III fetal heart rate tracings").

**NICHD CLASSIFICATION AND INTERPRETATION OF FHR PATTERNS**

Interpretation of a FHR tracing includes qualitative and quantitative description of baseline rate and variability; presence/absence of accelerations, decelerations, or sinusoidal pattern; and changes or trends of the FHR over time, as well as assessment of uterine activity. Standard definitions of FHR baseline, variability, accelerations, decelerations, and sinusoidal pattern (table 1) were proposed by the National Institute of Child Health and Human Development (NICHD) in 1997 and reaffirmed in 2008 [17,46]. They are used clinically throughout the United States, and have been endorsed by ACOG [8]. The International Federation of Gynecology and Obstetrics (FIGO) published a similar consensus guideline in 2015 (FIGO2015) (table 2), which is used in many other countries [50].

In 2008, the NICHD also introduced a three-tier FHR classification system (table 3), in which category I represents a normal tracing (predictive of normal fetal acid-base status at the time of observation), category II represents an indeterminate tracing, and category III represents an abnormal tracing (associated with an increased risk of abnormal fetal acid-base status at the time of observation) [17]. Some evidence suggests that neonatal outcomes can be improved with use of this standardized approach to pattern recognition coupled with a standardized package of therapeutic interventions (table 4) [51].

**Category I FHR pattern** — A category I pattern is normal: it indicates minimal likelihood of significant metabolic acidemia and ongoing fetal hypoxic injury at that point in time. The fetal status and FHR pattern may remain stable over time, or the fetal status may change, resulting in a category II or category III pattern.

A category I pattern has all of the following components (table 3 and waveform 9) [17]:

- A baseline FHR of 110 to 160 bpm
- Moderate FHR variability (6 to 25 bpm)
- Absence of late or variable FHR decelerations
- Early decelerations may or may not be present (waveform 10)
- Accelerations may or may not be present

In a study of the intrapartum FHR characteristics of over 48,000 patients with a singleton, non-anomalous fetus in term labor at 10 hospitals, category I patterns were observed at some point during labor in over 99 percent of tracings [52].

**Category III FHR pattern** — A category III pattern is abnormal: it is associated with an increased likelihood of severe hypoxia and metabolic acidemia at that point in time.

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https://www.uptodate.com/contents/intrapartum-fetal-heart-rate-assessment/print?search=%E8%83%8E%E5%BF%83%E7%9B%91%E6%8A%...
A category III tracing has at least one of the following components (table 3) [17]:
- Absent variability with recurrent late decelerations (waveform 2 and waveform 3)
- Absent variability with recurrent variable decelerations (waveform 7)
- Absent variability with bradycardia
- A sinusoidal pattern (waveform 11)

Late decelerations and variable decelerations are considered recurrent when they occur with at least 50 percent of uterine contractions in a 20-minute window [17, 46].

In a study of the intrapartum FHR characteristics of over 48,000 patients with a singleton, non-anomalous fetus in term labor at 10 hospitals, category III patterns were observed at some point in 0.1 percent of tracings [52].

Prompt evaluation, expeditious use of conservative measures to improve fetal oxygenation, and/or expeditious delivery are indicated when a category III pattern is observed because fetal/neonatal morbidity or mortality may occur if the pattern persists. (See "Management of intrapartum category I, II, and III fetal heart rate tracings", section on 'Category III pattern'.)

However, category III patterns may also be caused by conditions unrelated to hypoxemia. (See 'Pitfalls in attributing category II and category III patterns to fetal hypoxia' below.)

**Category II FHR pattern** — Category II FHR patterns include all FHR patterns that are not classified as category I (normal) or category III (abnormal) (table 3). The Society of Obstetricians and Gynaecologists of Canada classifies these tracings as "atypical" [53]. The potential for development of fetal acidosis and acidemia varies widely across the different types of category II tracings. (See 'Pitfalls in attributing category II and category III patterns to fetal hypoxia' below.)

Because category II tracings may remain stable for a prolonged period of time, have an uncertain prognosis, and are common (observed at some point in 84 percent of tracings [52]), pregnancies with this pattern are the most difficult to evaluate and manage. (See "Management of intrapartum category I, II, and III fetal heart rate tracings", section on 'Category II pattern'.)

**Pitfalls in attributing category II and category III patterns to fetal hypoxia** — Category II and category III patterns can be related to a number of conditions other than hypoxia:

- **Fetal sleep cycle** — Fetal sleep is associated with decreased variability and reduced frequency of accelerations. Sleep cycles may last up to 40 minutes [54].

- **Technical factors** — Technical factors include a faulty leg plate, electrode, or monitor; setting the recording rate at 1 cm/min instead of the standard 3 cm/min; and the computer algorithm used by the monitor, which may double very slow fetal heart rates and halve fast rates (>240 bpm).

- **Maternal heart rate artifact** — Maternal artifact refers to an electronic fetal monitor record that shows the maternal, rather than the fetal, heart rate [55]. It may occur in several scenarios, which can be detected if the clinician is aware of the phenomenon, and evaluates suspicious tracings. Evaluation for maternal artifact involves using more reliable methods for documenting the maternal heart rate (eg, checking the radial pulse, applying a pulse oximetry or electrocardiographic monitor) and FHR (eg, ultrasound of fetal heart, internal scalp electrode).

  - If the fetus is dead, an internal fetal scalp electrode may detect the maternal electrocardiogram (ECG) and record the maternal heart rate instead of the FHR [56].
  - The external Doppler device may record the maternal heart rate (even if the fetus is alive) from a nearby artery (eg, uterine artery). The maternal heart rate pattern can appear deceptively similar to a normal FHR pattern, including normal-appearing baseline rate and variability, when the mother is tachycardic.
  - If the maternal heart rate is recorded, maternal heart rate accelerations during uterine contractions can be mistaken for FHR accelerations [57-59]. Heart rate accelerations that coincide with uterine contractions should prompt further evaluation to exclude this phenomenon, especially during maternal pushing in the second stage.
  - The external Doppler device can alternate between recording the fetal and the maternal heart rate. When switching from one to the other, the tracing will not necessarily show visual discontinuity; therefore, visual continuity does not reliably exclude this phenomenon. Some newer electronic monitors will alert the user to "signal coincidence" or "signal ambiguity" when the monitor's computer logic determines that the maternal heart rate derived from a maternal ECG or pulse oximetry transducer is the same as the presumed FHR derived from the Doppler transducer (or scalp electrode).

Suspected signal coincidence or ambiguity should prompt further evaluation to confirm the source of the heart rate signal, since the maternal heart rate is not informative. If there is any question, other methods should be employed as needed, including ultrasound of the fetal heart, palpation of the maternal pulse, fetal scalp electrode, or maternal pulse oximetry.
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**Drug effects** – Transplacental passage of maternal medication can affect the FHR. For example, opioids and magnesium sulfate can decrease variability, butorphanol can cause a sinusoidal pattern, and beta-blockers and atropine can increase FHR [8].

**Maternal fever** – Maternal fever is associated with increased baseline FHR and decreased variability.

**Fetal cardiac arrhythmias** – Electronic FHR monitoring patterns may suggest the presence of a fetal arrhythmia, which may be benign or may seriously affect cardiac function. Other sonographic and non-sonographic modalities are needed to make a definitive diagnosis and treatment plan. (See “Fetal arrhythmias”.)

The following are clues that an arrhythmia is present; however, any FHR less than 110 bpm requires thorough evaluation before it can be attributed to a benign condition.

- Sharp downward spikes that nadir at approximately half of the baseline rate suggest dropped beats.
- A sharp upward spike followed immediately by a downward spike suggests a premature beat with a compensatory pause.
- A persistent or intermittent baseline rate that is approximately half of the normal rate may be due to 2:1 heart block.
- A baseline rate less than 110 bpm but higher than half of the normal rate suggests sinus bradycardia. Structural cardiac abnormalities may be associated with bradyarrhythmias, such as complete heart block.
- A FHR >240 bpm suggests a tachyarrhythmia; however, because the upper limit of the FHR graph on standard paper is 240 bpm, the monitor may not record any heart rate or it may record half of the FHR.

**Preexisting fetal neurologic injury** – Abnormal FHR patterns observed from the moment monitoring is initiated have been attributed to antepartum neurologic injury. The most commonly described pattern is a persistent nonreactive heart rate and a persistent fixed baseline with minimal or absent variability [6,60].

However, a normal intrapartum FHR monitoring does not exclude the possibility of antepartum neurologic injury, since antepartum injury does not always manifest in the intrapartum FHR tracing [61].

Studies of infants with congenital brain lesions suggest that damage to the medulla oblongata and mid brain may be responsible for the loss of FHR variability [62].

**CLINICAL APPROACH**

To improve neonatal outcome, it is crucial that clinicians correctly define and interpret FHR tracings, communicate effectively with other labor and delivery providers when the pattern is not normal, and initiate appropriate and timely interventions [51].

**Key principles** — Key principles when monitoring the FHR pattern include:

- Confirm that the monitor is recording the FHR and uterine activity adequately to permit appropriately-informed management decisions. Ensure that the maternal heart rate is not being recorded. (See ‘Pitfalls in attributing category II and category III patterns to fetal hypoxia’ above.)

- Assess uterine activity along with baseline FHR, variability, accelerations, decelerations, and sinusoidal pattern, and place the tracing in a category (I, II, or III).

- If the tracing is category I and the patient is low-risk, initiate routine intrapartum fetal surveillance.

- If the tracing is not category I, evaluate the integrity of the fetal oxygen pathway (maternal lungs, heart, and vasculature, as well as the uterus, placenta, and umbilical cord).

  Attempt to correct the problem, if possible, by initiating measures to improve fetal oxygenation, such as reduction in dose or discontinuation of oxytocin, maternal repositioning, and intravenous fluid bolus. The management of category II and III tracings is summarized in the table (table 4) and discussed in detail separately. (See “Management of intrapartum category I, II, and III fetal heart rate tracings”.)

  Also employ ancillary tests to further assess the fetal condition. (See ‘Useful ancillary tests for intrapartum fetal evaluation’ below.)

- If the FHR pattern does not improve within a reasonable period of time, begin planning for the possible need for rapid delivery. This may include availability of an operating room and specialized equipment, notification of anesthesia and pediatrics, consent forms, and laboratory tests.

- Determine whether operative intervention (cesarean or instrumental vaginal delivery) is needed, and the urgency of this intervention. Prepare for rapid intervention, if needed.

**Useful ancillary tests for intrapartum fetal evaluation**
Fetal scalp blood sampling — Fetal scalp blood sampling is an intrapartum procedure intended to assess the presence and degree of fetal acidemia by analyzing fetal capillary blood. An amnioscope with a light source is used to expose the fetal scalp, which is cleansed of blood, and a no. 11 blade. The blood is collected in long, heparinized capillary tubes. The test requires that the cervix is dilated at least 2 to 3 cm, can be difficult to perform, is not uncomfortable. The examiner stimulates the fetal vertex by prodding it with the examining finger or an instrument, such as an Allis clamp, during a vaginal examination or by vibroacoustic stimulation at the maternal abdominal wall overlying the uterus [65]. This should be performed when the FHR is at its baseline rate. Performance during a deceleration is not likely to terminate the deceleration, is not predictive of fetal acid-base status, and might exacerbate fetal compromise if parasympathetic tone increases in response to the stimulus. When accelerations are induced in this setting, the fetal pH is >7.20 in over 90 percent of cases, and when no accelerations occur, pH is <7.20 in approximately 50 percent of cases [65-69]. Accelerations elicited by fetal scalp stimulation have the same ability to exclude on-going hypoxic injury as spontaneous accelerations [17].

A 2002 meta-analysis that assessed performance of various stimulation tests (vibroacoustic stimulation, digital scalp stimulation, fetal scalp puncture, Allis clamp scalp stimulation) for the prediction of intrapartum fetal acidemia found them to be similarly effective and more useful for predicting the absence, rather than the presence, of acidemia [67]. Less traumatic techniques (vibroacoustic stimulation, digital scalp stimulation) are preferable to more traumatic techniques (fetal scalp puncture, Allis clamp scalp stimulation). For digital stimulation, the pooled likelihood ratio (LR) for acidemia with a negative test (ie, acceleration elicited) was 0.06 (95% CI 0.01-0.31). Failure to elicit an acceleration was not a definitive sign of acidemia. For digital stimulation, the pooled LR of acidemia with a positive test (ie, no acceleration) was 15 (95% CI 3-76).

In low-resource and nonobstetric settings where a change in FHR in response to acoustic stimulation cannot be assessed electronically, maternal perception of sound-provoked fetal movement appears to be predictive of fetal well-being [70-74].

Less useful ancillary tests for intrapartum fetal evaluation — Use of ST-analysis does not appear to result in meaningful improvements in pregnancy outcome, but is common practice in some institutions.

The STAN S31 fetal heart monitor, monitors the fetal electrocardiogram (ECG) during labor. Use of this device is based on the principle that fetal hypoxemia can result in elevation or depression of the ST segment. The monitor's software automatically identifies and analyzes changes in the T wave and the ST segment of the fetal ECG, which is obtained via a spiral electrode attached to the fetal scalp. The analysis is displayed in the lower section of the monitor's screen as a series of data points ("T/QRS crosses") and event markers. A visual alert ("ST event") appears when ST changes occur. Studies have reported the STAN computerized interpretation of FHR monitoring system has 38 to 90 percent sensitivity and 83 to 100 percent specificity for detecting fetal acidemia [75-77].

Clinicians who choose to use ST analysis should be trained and credentialed in its use and interpretation. The STAN S31 fetal heart monitor has been approved by the US Food and Drug Administration as an adjunct to assessment of abnormal FHR tracings in pregnancies over 36 weeks of gestation, in labor, with vertex presentation and ruptured fetal membranes. It is not indicated for monitoring initiated in the second stage of labor, since there may not be enough time to establish the baseline fetal ECG data required for automatic ST event signals. Transcutaneous Electrical Nerve Stimulation (TENS) for analgesia during labor is another contraindication because TENS may interfere with acquisition of the fetal ECG signal.

Three meta-analyses concluded use of ST-analysis did not result in meaningful improvements in pregnancy outcome [78-80]. As an example, in a 2016 systematic review and meta-analysis including seven randomized trials with a total of over 27,000 pregnancies, intrapartum fetal electrocardiogram analysis (PR to RR interval, ST segment) did not improve any neonatal outcome or decrease cesarean delivery rates compared with continuous electronic FHR monitoring alone, but fewer fetal scalp sampling procedures were performed [80]. The largest randomized trial in the review assigned over 11,000 women to "open" or "masked" monitoring with fetal ST-segment analysis and reported no significant between-group differences in the rate of cesarean delivery, any operative delivery, or the primary composite outcome (stillbirth, neonatal death, five-minute Apgar score ≤3, cord artery pH ≤7.05 and base deficit in extracellular fluid ≥12, intubation in the delivery room, seizures, neonatal encephalopathy) [65,77].

These conclusions have been challenged in an editorial in which the authors asserted that, while the meta-analyses of ST-analysis did not confirm a significant reduction in perinatal death, neonatal seizures, or neonatal encephalopathy, these outcomes are so uncommon that it is unlikely a study large enough to prove no benefit statistically will ever be performed [81]. They also argued that reductions in the rates of fetal blood sampling, operative vaginal delivery and metabolic acidemia are not irrelevant, despite the lack of reduction in hard outcomes.

Fetal scalp blood sampling — Fetal scalp blood sampling is an intrapartum procedure intended to assess the presence and degree of fetal acidemia by analyzing fetal capillary blood. An amnioscope with a light source is used to expose the fetal scalp, which is cleansed of blood, mucous, and amniotic fluid. The scalp is smeared with silicone gel so that a droplet of blood forms when the scalp is punctured with a 2-mm blade. The blood is collected in long, heparinized capillary tubes. The test requires that the cervix be dilated at least 2 to 3 cm, can be difficult to perform, and can be uncomfortable for the parturient. It is contraindicated when the mother is known to have a serious transmissible infection.
such as HIV or hepatitis, and in fetuses at increased risk of a bleeding diathesis. Rare complications described in case reports include infection, hemorrhage, and leakage of cerebrospinal fluid [82-84].

Both pH and lactate measurements require the same laboratory facilities for microsample analysis. Less blood is needed for measurement of lactate than pH, otherwise one test does not clearly perform better than the other [85]. Intrapartum fetal scalp blood sampling to measure pH or lactate has not been clearly proven to reduce emergency cesarean deliveries or operative vaginal births or to improve long-term perinatal outcome [86,87]. For this reason and many others, including quality control issues, cost, patient discomfort, sample failure rates up to 10 percent, and unavailability of sampling kits, fetal scalp blood sampling is performed rarely in the United States and elsewhere. (See "Umbilical cord blood acid-base analysis at delivery").

**Fetal pulse oximetry** — Although intuitively a promising technique for fetal evaluation, fetal pulse oximetry has not been useful clinically.

Data from human and animal studies suggest that fetal arterial oxygen saturation (SaO2 by blood gas co-oximetry) >30 percent is usually associated with pH >7.13 [75,88]. In humans, the mean fetal oxygen saturation (SpO2 by fetal pulse oximetry) during the first and second stages of labor is 59±10 percent and 53±10 percent, respectively [76,89]. In the setting of an abnormal FHR pattern, fetal SpO2 <30 percent for greater than 10 minutes has been associated with an increased risk of fetal acidemia [77,78,90-93].

However, in a 2014 systematic review and meta-analysis of randomized trials comparing the outcome of pregnancies in which both fetal pulse oximetry and cardiotocography results were available for intrapartum clinical management with the outcome of controls in which only cardiotocography results were available (n = seven trials, 8013 women), fetal pulse oximetry had no statistical effect on the overall rate of cesarean delivery or the rate of maternal or infant outcomes evaluated; the rates were similar in both groups [69,94].

**Use of decision aids** — Although recognition and management of category I and III tracings is relatively straightforward, the potential for development of progressive fetal hypoxia, metabolic acidosis, and metabolic acidemia varies widely across the different types of category II tracings. In part for this reason, investigators have proposed various algorithmic approaches to recognition, interpretation, and management of FHR tracings beyond category I. These approaches have had some success in achieving earlier recognition of tracings associated with metabolic acidemia [95]. However, their ability to improve neonatal outcome and/or reduce unnecessary interventions remains unproven, thus a change in the standard clinical approach described above is not warranted [96,97].

The following are examples of some decision aids:

- A five-tier FHR classification system has been proposed to identify fetuses at risk of developing acidosis [98-100]. The system focuses on baseline FHR, variability, and decelerations to stratify the risk of evolution to acidemia. Depending on risk level, the system suggests different interventions, such as conservative measures or delivery. The five-tier has not been validated in a large prospective or randomized trial and no data are available to indicate that it improves neonatal outcome or reduces operative intervention.

- An online risk assessment calculator is available for management of category II tracings [101]. It takes into account factors such as labor stage (latent or active first stage or second stage), labor progress (normal or abnormal), assessment of variability (presence/absence of moderate variability or accelerations), and assessment of decelerations (frequency and duration of recurrent decelerations). Based on information entered by the clinician, the calculator suggests observation or delivery. The algorithm on which the calculator is based has been reported to facilitate earlier recognition of some, but not all, FHR tracings associated with metabolic acidemia without increasing the rate of operative intervention [18,95]. The performance of the calculator has not been assessed in a clinical trial.

- Two randomized trials (FM-ALERT [96] and INFANT [97]) have evaluated the use of continuous intrapartum fetal monitoring with computerized interpretation and real-time alerts. Neither trial reported a benefit in any maternal or neonatal outcome compared with usual care (intrapartum fetal monitoring with clinician interpretation).

The larger INFANT trial included over 47,000 pregnancies at or near term and followed offspring to two years of age [97]. Compared with usual care, the intervention did not increase recognition of abnormal FHR patterns, did not increase the rate of spontaneous vaginal delivery, and did not improve neonatal outcome (composite or individual endpoints such as pH <7.05, metabolic acidosis, seizures, neonatal encephalopathy, hospital stay). Developmental assessment at age two years was similar for both groups.

### CONTROVERSIAL THEORIES

There is clear consensus that normal intrapartum FHR monitoring, when accompanied by normal Apgar scores, normal umbilical artery blood gas results, or both, is inconsistent with an acute intrapartum hypoxic-ischemic event sufficient to cause hypoxic-ischemic encephalopathy (HIE) and cerebral palsy (CP) [19,102]. Intrapartum HIE that is sufficient to cause CP requires significant interruption of fetal oxygenation, usually manifested by significant FHR abnormalities (eg, category III tracing), metabolic acidemia (reflected by low umbilical artery pH and elevated base deficit), and immediate newborn depression (usually reflected by low Apgar scores). (See "Etiology and pathogenesis of neonatal encephalopathy").

Contrary to this consensus, one group has proposed that the standard markers of intrapartum fetal hypoxia described above need not be present in order to establish that an intrapartum event was the cause of later neurologic impairment and that impairment can be due to the
effects of mechanical forces of labor on the fetal head [103]. Although mechanical forces of labor cause pressure on the fetal head, injury by this hypothetical mechanism has no scientific basis: It has never been reported, and a 2017 systematic review concluded that fetal intracranial pressure is well protected from extracranial forces and available data do not support intrapartum extracranial pressure as a cause of fetal brain injury [104].

Another group has proposed a risk-scoring system, dubbed the "Fetal Reserve Index (FRI)," for interpretation and management of FHR patterns [105-107]. The FRI is a weighted calculation of eight risk categories, including 10 maternal risk factors (eg, chronic debilitating disease, short stature), 9 obstetric risk factors (eg, placental abruption, malpresentation), and 12 fetal risk factors (eg, meconium passage, chorioamnionitis). Their criteria for abnormal FHR patterns and uterine activity are defined differently from standard National Institute of Child Health and Human Development and international definitions. This system has never been validated; furthermore, the authors' lack of standard criteria for diagnosing cases of HIE, lack of appropriate controls, and multiple obvious sources of potential bias preclude reasonably founded conclusions regarding safety or utility of the FRI.

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Labor".)

SUMMARY AND RECOMMENDATIONS

- The primary goal of intrapartum fetal heart rate (FHR) monitoring is to assess the adequacy of fetal oxygenation and presence of fetal metabolic acidemia during labor so that timely intervention can be undertaken to reduce the likelihood of neurologic injury or death. There is some evidence that intrapartum fetal monitoring is associated with a reduction in intrapartum death. However, conclusive evidence of a reduction in long-term neurologic impairment is lacking. (See "Does intrapartum FHR monitoring improve outcome?" above.)

- Electronic fetal monitoring has a high false-positive rate for predicting adverse neonatal outcomes and may increase the rate of operative delivery. (See "Does intrapartum FHR monitoring improve outcome?" above.)

- Contemporary evidence does not confirm earlier assumptions that electronic FHR monitoring is associated independently with a significant increase in the rate of cesarean delivery. (See "Does intrapartum FHR monitoring improve outcome?" above.)

- Two commonly used modalities for intrapartum FHR monitoring are continuous electronic FHR monitoring and intermittent auscultation. Contemporary evidence indicates that neither test performs better than the other, provided that intermittent auscultation is performed as prescribed in randomized trials. (See "Does intrapartum FHR monitoring improve outcome?" above and "Continuous monitoring versus intermittent auscultation: Recommendations of selected national organizations" above.)

- Medicolegal precedent in the United States mandates some form of intrapartum FHR monitoring. For most high-risk pregnant women, continuous electronic FHR monitoring is recommended. For low-risk women, either intermittent or continuous electronic FHR monitoring is reasonable, but frequent intermittent monitoring is cumbersome and difficult for most institutional nursing services to achieve. (See "Candidates for intrapartum fetal monitoring" above and "Continuous monitoring versus intermittent auscultation: Recommendations of selected national organizations" above.)

- Intrapartum FHR monitoring is usually not performed when detection of a FHR abnormality would not prompt any intervention (eg, conservative maneuvers to improve fetal oxygenation and/or cesarean delivery). For example, pregnancies at a gestational age below the limit of viability and pregnancies in which the fetus has an untreatable anomaly lethal in the newborn. (See "Candidates for intrapartum fetal monitoring" above.)

- Standardized FHR definitions proposed by the National Institute of Child Health and Human Development (NICHD) and endorsed by the American College of Obstetricians and Gynecologist are summarized in the table (table 1). (See "NICHD classification and interpretation of FHR patterns" above.)

- The three-tier system of FHR classification proposed by the NICHD is summarized in the table (table 3). General concepts of intrapartum FHR management are summarized in the table (table 4). Use of this standardized approach to pattern recognition coupled with a standardized package of therapeutic interventions may improve neonatal outcome. (See "NICHD classification and interpretation of FHR patterns" above and "Management of intrapartum category I, II, and III fetal heart rate tracings".)

- The appearance of the FHR tracing can be affected by factors other than fetal oxygenation (eg, fetal sleep and arrhythmias, maternal artifact and medications). (See "Pitfalls in attributing category II and category III patterns to fetal hypoxia" above.)

- Normal human labor is characterized by regular uterine contractions and repeated episodes of transient interruption of fetal oxygenation. Most fetuses tolerate this process well, but some do not. A major goal of intrapartum fetal monitoring is to distinguish the former from the latter. (See "Introduction" above and "Clinical approach" above.)
Intrapartum fetal heart rate assessment - UpToDate

Key principles for evaluation of the FHR tracing and management of FHR patterns include (see ‘Key principles’ above):

- Confirm that the monitor is correctly recording the FHR and uterine activity.
- Assess uterine activity and classify the tracing in a category (I, II or III).
- If the tracing is category I and the patient is low-risk, initiate routine intrapartum fetal surveillance.
- If the tracing is not category I, evaluate the fetal oxygenation pathway.
- Attempt to correct oxygenation problems, if possible. The management of category II and III tracings is summarized in the table (table 4) and discussed in detail separately. (See “Management of intrapartum category I, II, and III fetal heart rate tracings”.)
- Employ ancillary tests to further assess the fetal condition. (See ‘Useful ancillary tests for intrapartum fetal evaluation’ above.)
- If the FHR pattern does not improve within a reasonable period of time, begin planning for the possible need for rapid delivery.

Moderate baseline variability and/or FHR accelerations reflect the oxygenation of the central nervous system and reliably predict the absence of ongoing hypoxic injury and metabolic acidemia at the time it is observed. (See ‘Variability’ above and ‘Accelerations’ above.)

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The interval, in fractions of a second, between these beats is used to calculate the instantaneous beat to beat interval and then a rolling average is taken and recorded of the fetal heart rate tracing.
**NICHD definitions of FHR characteristics and patterns**

### Variability
- Fluctuations in baseline that are irregular in amplitude and frequency
- Absent = amplitude undetectable
- Minimal = amplitude 0 to 5 bpm
- Moderate = amplitude 6 to 25 bpm
- Marked = amplitude over 25 bpm

Measured in a 10-minute window. The amplitude is measured peak to trough. There is no distinction between short-term and long-term variability.

### Baseline rate
- Bradycardia = below 110 bpm
- Normal = 110 to 160 bpm
- Tachycardia = over 160 bpm

The baseline rate is the mean bpm (rounded to 0 or 5) over a 10-minute interval, excluding periodic changes, periods of marked variability, and segments that differ by more than 25 bpm. The baseline must be identifiable for two minutes during the interval (but not necessarily a contiguous two minutes); otherwise, it is considered indeterminate.

### Acceleration
- An abrupt* increase in the FHR. Before 32 weeks of gestation, accelerations should last ≥10 sec and peak ≥10 bpm above baseline. As of 32 weeks gestation, accelerations should last ≥15 sec and peak ≥15 bpm above baseline.
- A prolonged acceleration is ≥2 minutes but less than 10 minutes. An acceleration of 10 minutes or more is considered a change in baseline.

### Late deceleration
- A gradual* decrease and return to baseline of the FHR associated with a uterine contraction. The deceleration is delayed in timing, with the nadir of the deceleration occurring after the peak of the contraction. The onset, nadir, and recovery usually occur after the onset, peak, and termination of a contraction.

### Early deceleration
- A gradual* decrease and return to baseline of the FHR associated with a uterine contraction. The nadir of the FHR and the peak of the contraction occur at the same time. The deceleration's onset, nadir, and termination are usually coincident with the onset, peak, and termination of the contraction.

### Variable deceleration
- An abrupt* decrease in FHR below the baseline. The decrease is ≥15 bpm, lasting ≥15 secs and <2 minutes from onset to return to baseline. The onset, depth, and duration of variable decelerations commonly vary with successive uterine contractions.

### Prolonged deceleration
- A decrease in FHR below the baseline of 15 bpm or more, lasting at least 2 minutes but <10 minutes from onset to return to baseline. A prolonged deceleration of 10 minutes or more is considered a change in baseline.

NICHD: National Institute of Child Health and Human Development; bpm: beats per minute; sec: seconds; FHR: fetal heart rate.

*Gradual* and *abrupt* changes are defined as taking ≥30 seconds or <30 seconds, respectively, from the onset of the deceleration/acceleration to its nadir/peak.


Graphic 65859 Version 7.0
Late decelerations are characterized by a gradual decrease and return to baseline of the fetal heart rate associated with uterine contractions. The deceleration is delayed in timing, with the nadir of the deceleration occurring after the peak of the contraction. The onset, nadir, and recovery usually occur after the onset, peak, and termination of a contraction. In this example, variability is minimal.

Courtesy of Robert L Barbieri, MD.

Graphic 67464 Version 3.0
Late decelerations 2

Late decelerations are characterized by gradual decrease and return to baseline of the fetal heart rate associated with a uterine contraction. The deceleration is delayed in timing, with the nadir of the deceleration occurring after the peak of the contraction. The onset, nadir, and recovery usually occur after the onset, peak, and termination of a contraction. In this tracing, late decelerations have occurred after the first two contractions.

Graphic 96432 Version 1.0
Late deceleration

Courtesy of Dr. Christina Davidson.

Graphic 96977 Version 1.0
Recurrent late decelerations

Yellow tracing in the upper panel represents the fetal heart rate. Arrow points to the nadir of a late deceleration.

Courtesy of Dr. Christina Davidson.

Graphic 96980 Version 1.0
Deep late deceleration

Yellow tracing in the upper panel represents the fetal heart rate. Arrow points to the nadir of the late deceleration.

Courtesy of Dr. Christina Davidson.

Graphic 96978 Version 1.0
Variable decelerations

Recurrent variable decelerations with absent to minimal variability.

*Courtesy of Bruce K Young, MD.*

Graphic 86060 Version 3.0
Variable decelerations with minimal to absent variability

Variability is minimal to absent.

Courtesy of Bruce K Young, MD.

Graphic 86062 Version 3.0
<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Suspicious</th>
<th>Pathological</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Baseline</strong></td>
<td>110 to 160 bpm</td>
<td>Lacking at least one characteristic of normality, but with no pathological features</td>
<td>&lt;100 bpm</td>
</tr>
<tr>
<td><strong>Variability</strong></td>
<td>5 to 25 bpm</td>
<td>Lacking at least one characteristic of normality, but with no pathological features</td>
<td>Reduced variability, increased variability, or sinusoidal pattern</td>
</tr>
<tr>
<td><strong>Decelerations</strong></td>
<td>No repetitive(^\text{¶}) decelerations</td>
<td>Lacking at least one characteristic of normality, but with no pathological features</td>
<td>Repetitive (^\text{¶}) late or prolonged decelerations during &gt;30 minutes or 20 minutes if reduced variability, or one prolonged deceleration with &gt;5 minutes</td>
</tr>
<tr>
<td><strong>Interpretation</strong></td>
<td>Fetus with no hypoxia/acidosis</td>
<td>Fetus with a low probability of having hypoxia/acidosis</td>
<td>Fetus with a high probability of having hypoxia/acidosis</td>
</tr>
<tr>
<td><strong>Clinical management</strong></td>
<td>No intervention necessary to improve fetal oxygenation state</td>
<td>Action to correct reversible causes if identified, close monitoring or additional methods to evaluate fetal oxygenation(^{[1]})</td>
<td>Immediate action to correct reversible causes, additional methods to evaluate fetal oxygenation(^{[1]}), or if this is not possible expedite delivery. In acute situations (cord prolapse, uterine rupture, or placental abruption) immediate delivery should be accomplished</td>
</tr>
</tbody>
</table>

FIGO: International Federation of Gynaecology and Obstetrics; bpm: beats per minute.

\(^*\) The presence of accelerations denotes a fetus that does not have hypoxia/acidosis, but their absence during labor is of uncertain significance.

\(^{¶}\) Decelerations are repetitive in nature when they are associated with more than 50% of uterine contractions\(^{[2]}\).

References:

From: Ayres-de-Campos D, Spong CY, Candaharan E. FIGO consensus guidelines on intrapartum fetal monitoring: Cardiotocography. Int J Gynaecol Obstet 2015; 131(1):13-24. [https://obgyn.onlinelibrary.wiley.com/doi/abs/10.1016/j.ijgo.2015.06.020](https://obgyn.onlinelibrary.wiley.com/doi/abs/10.1016/j.ijgo.2015.06.020). Copyright © 2015 International Federation of Gynecology and Obstetrics. Reproduced with permission of John Wiley & Sons Inc. This image has been provided by or is owned by Wiley. Further permission is needed before it can be downloaded to PowerPoint, printed, shared or emailed. Please contact Wiley’s permissions department either via email: permissions@wiley.com or use the RightsLink service by clicking on the ‘Request Permission’ link accompanying this article on Wiley Online Library ([http://onlinelibrary.wiley.com](http://onlinelibrary.wiley.com)).
### NICHD criteria for category I, II, and III FHR tracings

#### Category I

All of the following criteria must be present. Tracings meeting these criteria are predictive of normal fetal acid-base balance at the time of observation.

- Baseline rate: 110 to 160 bpm
- Moderate baseline FHR variability
- No late or variable decelerations
- Early decelerations may be present or absent
- Accelerations may be present or absent

#### Category III

Category III tracings are predictive of abnormal fetal acid-base status at the time of observation. Prompt evaluation is indicated and most parturients will require expeditious intervention, such as provision of supplemental oxygen, change in position, treatment of hypotension, and discontinuation of any uterotonic drugs being administered. Category III tracings include either (1) or (2) below.

1. Absent baseline FHR variability and any of the following:
   - Recurrent late decelerations
   - Recurrent variable decelerations
   - Bradycardia
2. Sinusoidal pattern

#### Category II

FHR tracing does not meet criteria for either category I or III and is considered indeterminate.

NICHD: National Institute of Child Health and Human Development; FHR: fetal heart rate; bpm: beats per minute.


Graphic 57583 Version 9.0
## Management of Intrapartum Fetal Heart Rate Tracings

<table>
<thead>
<tr>
<th>Fetal Heart Rate Tracing</th>
<th>Possible Etiologies and Interpretation</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Category I</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline 110 to 160 beats per minute with moderate variability and no late or variable decelerations. Accelerations and early decelerations may be present or absent.</td>
<td>This is a normal tracing.</td>
<td>Intermittent or continuous fetal monitoring based on clinical status and underlying risk factors. Review every 30 minutes in the first stage and every 15 minutes in the second stage of labor.</td>
</tr>
<tr>
<td><strong>Category II</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intermittent variable decelerations (&lt;50 percent of contractions)</td>
<td>Common finding usually associated with normal outcome.</td>
<td>No intervention required.</td>
</tr>
<tr>
<td>Recurrent variable decelerations (&gt;50 percent of contractions)</td>
<td>Umbilical cord compression. May be associated with impending acidemia, especially if progressive increase in depth, duration, and frequency. Moderate variability and/or accelerations suggest fetus is not currently acidemic.</td>
<td>Reposition mother to left or right lateral. Amnioinfusion is an option. Adjunctive measures to promote fetal oxygenation (oxygen supplementation, intravenous fluid bolus, reducing uterine contraction frequency) may be useful. Initiate scalp stimulation to provoke fetal heart rate acceleration, which is a sign that the fetus is not acidic. Delivery is indicated if tracing does not improve and acidemia suspected.</td>
</tr>
<tr>
<td>Recurrent late decelerations</td>
<td>Transient or chronic uteroplacental insufficiency, such as from hypotension, tachysystole, or maternal hypoxia. Accelerations and/or moderate variability suggest fetus is not currently acidemic.</td>
<td>Reposition mother to left or right lateral. Adjunctive measures to promote fetal oxygenation include oxygen supplementation, intravenous fluid bolus, reduce uterine contraction frequency. Persistent late decelerations with minimal variability and absent accelerations suggest fetal acidemia; this is even more likely if variability is absent (category III). Initiate scalp stimulation to provoke fetal heart rate acceleration, which is a sign that the fetus is not acidic.</td>
</tr>
<tr>
<td>Fetal tachycardia (baseline heart rate greater than 160 beats per minute for at least 10 minutes)</td>
<td>Infection, medication, maternal medical disorders, obstetric complications, fetal tachycardia (typically rate over 200 beats per minute). Fetal acidemic more likely when associated with minimal or absent variability, absent accelerations, and/or recurrent decelerations.</td>
<td>Treat underlying cause, if known. Initiate scalp stimulation to provoke fetal heart rate acceleration, which is a sign that the fetus is not acidic.</td>
</tr>
<tr>
<td>Bradycardia (baseline heart rate less than 110 beats per minute for at least 10 minutes)</td>
<td>Acute onset may be due to hypotension, umbilical cord occlusion, rapid fetal descent, tachysystole, abruptio, uterine rupture. Fetal acidemic more likely when associated with minimal or absent variability and absent accelerations during baseline periods.</td>
<td>Treat underlying cause, if known. Initiate scalp stimulation to provoke fetal heart rate acceleration, which is a sign that the fetus is not acidic. Delivery is indicated if tracing does not improve and acidemia suspected.</td>
</tr>
<tr>
<td>Prolonged decelerations (15 beats per minute drop below baseline for more than 2 and less than 10 minutes)</td>
<td>Fetal sleep, medication, fetal acidemia. If due to fetal sleep, should recover in 20 to 60 minutes. If due to maternal medication, should recover as medication wears off.</td>
<td>If decreased fetal oxygenation suspected, reposition mother to left or right lateral. Adjunctive measures to promote fetal oxygenation include oxygen supplementation, intravenous fluid bolus, reduce uterine contraction frequency. Initiate scalp stimulation to provoke fetal heart rate acceleration, which is a sign that the fetus is not acidic. If no improvement and no accelerations, delivery is indicated if acidemia suspected or confirmed by scalp pH.</td>
</tr>
<tr>
<td>Minimal variability</td>
<td>Fetal acidemia. If due to fetal sleep, should recover in 20 to 60 minutes. If due to maternal medication, should recover as medication wears off.</td>
<td>If decreased fetal oxygenation suspected, reposition mother to left or right lateral. Adjunctive measures to promote fetal oxygenation include oxygen supplementation, intravenous fluid bolus, reduce uterine contraction frequency. Initiate scalp stimulation to provoke fetal heart rate acceleration, which is a sign that the fetus is not acidic. If no improvement and no accelerations, delivery is indicated if acidemia suspected or confirmed by scalp pH.</td>
</tr>
<tr>
<td>Tachysystole (more than 5 contractions in 10 minutes, averaged over 30 minutes) with fetal heart rate changes. Tachysystole that is spontaneous and associated with a normal fetal heart rate pattern does not require treatment, but the possibility of placental abruption as the underlying etiology should be considered.</td>
<td>Spontaneous labor: Tachysystole may be associated with fetal acidemia if accompanied by recurrent fetal heart rate decelerations.</td>
<td>Reposition mother to left or right lateral, oxygen supplementation, intravenous fluid bolus. If ineffective, reduce uterine contraction frequency with a tocolytic. Initiate scalp stimulation to provoke fetal heart rate acceleration, which is a sign that the fetus is not acidic.</td>
</tr>
<tr>
<td><strong>Category III</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absent baseline variability and recurrent late decelerations, recurrent variable decelerations, or bradycardia</td>
<td>Increased risk of fetal acidemia.</td>
<td>Prepare for delivery and reposition mother to left or right lateral, oxygen supplementation, intravenous fluid bolus. Initiate scalp stimulation to provoke fetal heart rate acceleration, which is a sign that the fetus is not acidic. If no improvement after conservative measures and scalp stimulation does not result in acceleration, delivery is advisable.</td>
</tr>
<tr>
<td>Sinusoidal</td>
<td>Increased risk of hypoxemia. Risk of acidemia increased if prolonged or amplitude of 15 beats per minute or more.</td>
<td>Prepare for delivery and reposition mother to left or right lateral, oxygen supplementation, intravenous fluid bolus. Initiate scalp stimulation to provoke fetal heart rate acceleration, which is a sign that the fetus is not acidic. If no improvement after conservative measures and scalp stimulation does not result in acceleration, delivery is advisable.</td>
</tr>
</tbody>
</table>
This chart represents a suggested approach to the interpretation and management of fetal heart rate patterns. It is not intended as a standard of care. Patient-specific factors need to be considered in the evaluation and management of individual patients.


Graphic 71679 Version 6.0
Normal variability and accelerations

Courtesy of Bruce K Young, MD.

Graphic 86064 Version 1.0
Early deceleration

Early decelerations are uniform, mirror the contractions, and decelerate only 10 to 20 beats per minute.


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Graphic 94981 Version 4.0
Sinusoidal FHR pattern

Sinusoidal heart rate pattern in a patient presenting with spontaneous fetomaternal hemorrhage near term. The patient reported decreased fetal movement.

FHR: fetal heart rate.

Graphic 61189 Version 4.0
Contributor Disclosures

David A Miller, MD  Consultant/Advisory Boards: CCSI [Fetal monitoring (Fetal monitor)]. Other Financial Interest: GE Healthcare [Fetal monitoring (Online education program)]. Vincenzo Berghella, MD  Nothing to disclose  Vanessa A Barss, MD, FACOG  Nothing to disclose

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