Nonstress test and contraction stress test

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INTRODUCTION

Fetal health is evaluated, in part, by assessment of fetal heart rate (FHR) patterns. The primary goal is to identify fetuses at risk of intrauterine death or neonatal complications and intervene (often by delivery) to prevent these adverse outcomes, if possible.

The nonstress test (NST) and the contraction stress test (CST) are used for antepartum FHR testing. An abnormal test (nonreactive NST, positive CST) is sometimes associated with adverse fetal or neonatal outcomes, while a normal test (reactive NST, negative CST) is usually associated with a neurologically intact and adequately oxygenated fetus. When interpreting these tests, the clinician needs to account for gestational age, prior results of fetal assessment, maternal conditions (including medications), and fetal condition (eg, growth restriction, anemia, arrhythmia).

The NST and CST will be reviewed here. Intrapartum fetal evaluation and additional techniques for assessing fetal health are discussed separately.

- (See "Intrapartum fetal heart rate assessment".)
- (See "The fetal biophysical profile".)
- (See "Decreased fetal movement: Diagnosis, evaluation, and management".)
- (See "Doppler ultrasound of the umbilical artery for fetal surveillance".)

PHYSIOLOGIC BASIS OF FETAL HEART RATE CHANGES

Physiologic development of the fetal heart occurs across gestation and affects fetal heart rate (FHR) patterns.

The parasympathetic and sympathetic nervous systems comprise the autonomic nervous system, which contributes to the regulation of the FHR. FHR changes result from moment-to-moment autonomic modulation by medullary cardiorespiratory centers in response to inputs from chemoreceptors, baroreceptors, central nervous system activities (eg, arousal, sleep), catecholamines, and blood volume [1].

The parasympathetic innervation of the heart is primarily mediated by the vagus nerve, which influences the sinoatrial (SA) and atrioventricular (AV) nodes. Parasympathetic stimulation slows the FHR and medications (eg, atropine) that block the release of acetylcholine from the vagus nerve increase SA node firing and may accelerate the baseline FHR by up to 20 beats per minute at term, depending on the dose [2].

Sympathetic nerves are distributed throughout the myocardium of the term fetus. Sympathetic stimulation results in release of norepinephrine, which accelerates the FHR, increases baseline variability, and improves inotropy. Blockade of sympathetic activity slows the FHR and blunts accelerations.

FHR variability is not consistently observed before 24 weeks of gestation, while the absence of variability is abnormal after 28 weeks of gestation since the parasympathetic nervous system is consistently developed by the third trimester. However, once a fetus has demonstrated normal FHR variability, persistent loss of variability is an abnormal finding at any gestational age.

Effect of gestational age on fetal heart rate — The parasympathetic nervous system exerts a progressively greater influence on the FHR as gestational age advances: Advancing gestational age is associated with slowing of the baseline heart rate within the normal range of 110 to 160 beats per minute (figure 1).

FHR variability is rarely present before 24 weeks of gestation, while the absence of variability is abnormal after 28 weeks of gestation since the parasympathetic nervous system is consistently developed by the third trimester. However, once a fetus has demonstrated normal FHR variability, persistent loss of variability is an abnormal finding at any gestational age.

Advancing gestational age is also associated with increased frequency and amplitude of FHR accelerations, which are modulated by the sympathetic nervous system [3,4]. Fifty percent of normal fetuses demonstrate accelerations with fetal movements at 24 weeks; this proportion rises to over 95 percent at 30 weeks of gestation [5]. Before 32 weeks, however, accelerations are typically only 10 beats per minute for 10 seconds rather than the 15 beats per minute sustained for 15 seconds noted after 30 weeks [6].

Cardiovascular response to hypoxemia — Fetal oxygenation depends upon adequate maternal oxygenation, uteroplacental and fetoplacental blood flow, and distribution of oxygenated blood to fetal tissues. Therefore, fetal hypoxemia may result from a variety of sources including maternal disorders (eg, respiratory insufficiency, hypotension, vasoconstrictors, alkalosis), acute or chronic

placental dysfunction (eg, abruptio placentae, infarction, confined placental mosaicism), uterine factors (eg, rupture, tachysystole), and fetal factors (eg, arrhythmia, fetal hydrops, umbilical cord compression).

Transient, mild hypoxemia associated with contractions stimulates chemoreceptors in the fetal carotid arteries and aortic arch. These receptors signal the fetal brainstem to improve perfusion of major fetal organs: the brain, heart, adrenals, and placenta. The brainstem responds by stimulating the sympathetic system, which releases catecholamines and, in turn, increases the FHR and variability. If hypoxemia persists or worsens, peripheral arterial beds constrict, resulting in systemic hypertension [7]. Baroreceptors then respond by signaling the brainstem, leading to vagal stimulation, which slows the FHR. This sequence is the probable mechanism for a positive CST and was derived from fetal sheep studies (see 'Contraction stress test' below). The FHR response also has a nonadrenergic component, apparently mediated by humoral substances, such as vasoconstrictor hormones, released from the adrenal as a direct result of hypoxemia.

As hypoxemia worsens, the normal efferent sympathetic response to fetal movement is abolished, so FHR accelerations disappear. This stage is reflected by nonreactivity of the NST. (See 'Nonstress test' below.)

Prolonged and/or severe interruption of fetal oxygenation can lead to fetal heart rate decelerations and a loss of baseline variability. If oxygen deprivation progresses to the stage of fetal metabolic acidemia, the fetus may exhibit late decelerations due to direct myocardial depression.

The cause of interrupted fetal oxygenation also affects the type of FHR decelerations. Uterine contractions reduce placentofetal blood flow, which can lead to late decelerations, and compression of the umbilical cord reduces blood flow in the umbilical vein and arteries, which can result in variable decelerations [8]. Prolonged decelerations can result from acute interruption of fetal oxygenation at the level of the maternal lungs (eg, maternal hypoxemia), maternal heart (eg, acute reduction in cardiac output), maternal vasculature (eg, maternal hypotension), uterus (eg, excessive contractions or uterine rupture), placenta (eg, placental abruption), or umbilical cord (eg, cord prolapse).

Fetal head compression causes increased intracranial pressure and reflex slowing of the FHR, which results in early decelerations. Early decelerations are not related to decreased fetal oxygenation and are not associated with adverse outcome.

EQUIPMENT AND NOMENCLATURE

Equipment — Electronic fetal monitors are used to continuously record the fetal heart rate (FHR) in graphical form. In most external FHR monitoring systems, the FHR is measured by focusing an ultrasound beam on the fetal heart from a small Doppler ultrasound device placed on the maternal abdomen. A bedside monitor interprets the Doppler signals, which reflect the heart valve

movements as well as atrial and ventricular systole. The complex wave generated is analyzed and the peak detected and used for calculations. An internal computer then calculates the FHR by averaging several consecutive peak to peak frequencies (to minimize artifact). This process of averaging is called "autocorrelation." It produces a FHR waveform closely resembling that derived from a fetal electrocardiogram (ECG), although there is more baseline variability inherent with the Doppler technique.

The most recent generation of fetal monitors calculates FHR, FHR variability, and fetal QT intervals from information obtained from multiple electrodes placed on the maternal abdomen (eg, Monica AN24 monitor, MindChild Medical monitor). The maternal ECG signal and ambient noise are filtered out by algorithms run by microprocessors. This software has resulted in noninvasive fetal ECGs as accurate as the information obtained from a fetal scalp electrode [9-11]. It is most commonly used in obese women in whom continuous Doppler ultrasound monitoring is technically difficult and does not provide an adequate quality FHR tracing for interpretation.

Nomenclature — The most commonly used American system for describing FHR findings was developed by a workshop convened by the National Institute of Child Health and Human Development (table 1) [12].

ANTEPARTUM FETAL HEART RATE TESTING

The NST and CST are used for antepartum fetal heart rate (FHR) testing. There is no conclusive evidence from randomized trials that use of NSTs and CSTs leads to a reduction in fetal death or neurologic injury. Nevertheless, performing NSTs or CSTs in pregnancies deemed to be at high risk of adverse fetal outcome is a standard obstetric practice in the United States and elsewhere, based upon circumstantial evidence and longstanding convention (see 'Test performance' below). Because of medical liability precedents, it is impossible to abandon use of these tests in the United States while awaiting data from well-designed randomized trials, which may never be performed.

Indications — Antepartum FHR testing is performed in pregnancies in which the clinician believes the risk of fetal hypoxic injury or demise is increased [13]. These pregnancies are complicated by maternal medical or obstetrical conditions potentially associated with placental dysfunction or poor uteroplacental perfusion. They also include fetal disorders and other conditions potentially associated with an increased risk of fetal death. (See "Overview of antepartum fetal surveillance", section on 'Indications for fetal surveillance'.)

Test performance — The use of NSTs and CSTs became widespread in the early 1980s when a seminal observational study reported stillbirth rates (corrected for lethal congenital anomalies and unpredictable causes of demise) after reactive and nonreactive NSTs were 1.9 and 26 per 1000 births, respectively [14]. The stillbirth rates after a negative CST, reactive positive CST, or

nonreactive positive CST were 0.3, 0, and 88 per 1000 births, respectively. These findings supported use of these tests.

In 2015, a Cochrane review attempted to determine whether using the NST can improve maternal or perinatal outcome by identifying high-risk pregnancies requiring prompt induction of labor or immediate delivery by cesarean [15]. Six randomized trials involving 2105 women were included. Tested patients were compared with controls who did not undergo NSTs or their test results were concealed. No significant differences between groups were noted in maternal outcomes, such as frequency of induction of labor or cesarean delivery, or in infant outcomes, including perinatal mortality, low five-minute Apgar, neonatal intensive care unit admission, and neonatal seizures. Perinatal mortality (adjusted for lethal anomalies) in the testing group was higher than in controls (2.3 versus 1.1 percent, four studies, n = 1627; odds ratio [OR] 2.05, 95% CI 0.95-4.42). In contrast, when the authors compared computerized cardiotocography (automated evaluation of the FHR) versus traditional cardiotocography, computerized cardiotocography was associated with a significant reduction in perinatal mortality (relative risk [RR] 0.20, 95% CI 0.04-0.88; two trials, 0.9 versus 4.2 percent, 469 women). There was no significant difference in potentially preventable deaths (RR 0.23, 95% CI 0.04-1.29; two trials, n = 469); however, the meta-analysis was underpowered to assess this outcome.

There are many limitations to the trials in this meta-analysis, which prevent making an assessment of the value of the NST in current practice. Randomization methods were suboptimal, which could have created bias. The total number of adverse events, such as perinatal mortality and abnormal neurologic signs, was too low to determine whether small, but statistically significant, differences in outcome might result from testing. Furthermore, the trials were conducted in the early 1980s, when these tests were first introduced into clinical practice and clinicians were inexperienced in test interpretation and subsequent pregnancy management. It is possible that the tests would be interpreted and acted upon differently today, with different outcomes. Lastly, fetal assessment and neonatal care have changed since the 1980s; the combined use of ultrasound and FHR testing may be more predictive of fetal status and need for intervention than either test alone.

Choice of test — There is no evidence from randomized trials on which to base a recommendation for use of the CST versus the NST for antepartum fetal monitoring. Although some observational studies suggest that the CST is more predictive of adverse outcome than the NST [14,16], others have not demonstrated any improvement in predicting perinatal morbidity compared with the NST, even when both tests are combined [17]. Given comparable efficacy, NSTs have become the standard of care because CSTs generally require uterine stimulation and are potentially risky since contractions are induced.

The only randomized trial of antepartum fetal testing compared the biophysical profile (BPP, which includes an NST) with the NST alone in management of 652 high-risk pregnancies and was inconclusive [18]. Costs were not considered.

Management of pregnancies with nonreassuring test results — Management of the fetus with a nonreactive NST or positive CST depends upon the clinical setting. At term, delivery, rather than further evaluation, is usually warranted since fetal hypoxemia cannot be definitively excluded when these tests are nonreassuring. Induction of labor with continuous FHR and contraction monitoring are reasonable in patients planning a vaginal birth, but repetitive late decelerations or severe variable decelerations generally indicate a 20 to 40 percent risk of abnormal FHR tracings in labor. Consequently, expeditious delivery by cesarean is frequently indicated.

Nonreassuring antepartum FHR test results in a preterm gestation pose a greater dilemma. Preterm delivery may be indicated for persistent nonreactive NSTs that have been confirmed by additional tests of fetal status (eg, CST, BPP, Doppler studies), particularly when uteroplacental insufficiency is strongly suspected, such as fetal growth restriction, or there is evidence of fetal anemia. In such cases the underlying maternal diagnosis often clarifies the timing of delivery and the most appropriate management.

Tests

Nonstress test — The NST is the most common cardiotocographic method of antepartum fetal assessment. It is noninvasive and can be performed in any setting with an electronic fetal monitor. There is no direct risk of maternal or fetal injury associated with NSTs.

NSTs are initiated when fetal neurologic maturity enables FHR acceleration (typically at 26 to 28 weeks) and the fetus is believed to be at increased risk of death. There is no high-quality evidence defining the optimal frequency of testing [19]. Testing is performed at daily to weekly intervals as long as the indication for testing persists. The frequency of testing is based on clinical judgment, taking into consideration whether the test is being performed to screen for fetal hypoxemia in a high-risk pregnancy or to monitor a fetus with suspected, but compensated, fetal hypoxemia.

Maternal or fetal deterioration requires reevaluation despite recent reassuring test results. A pattern of accelerations, moderate variability, and no significant decelerations during the NST indicates no fetal hypoxemia during the test, but is not predictive of the future, especially with changes in the clinical setting over time.

Reactive tests — The NST is reactive from 32 weeks to term if there are two or more fetal heart rate accelerations reaching a peak of at least 15 beats per minute (bpm) above the baseline rate and lasting at least 15 seconds from onset to return to baseline (15 x 15) in a 20-minute period (figure 2 and table 1) [6,13,20]. A reactive test provides reliable evidence of normal fetal oxygenation, regardless of the length of observation time needed to demonstrate reactivity [21].

The optimal duration of the NST has not been established. Some sources recommend that the NST should be continued for at least 20 minutes, even if two qualifying accelerations have been observed before that time [22]. However, large studies evaluating the predictive value of the NST combined with amniotic fluid volume assessment have not included this requirement [23-25].

There are no published data to confirm a benefit of extending the duration of NST once two qualifying accelerations have been observed.

Prior to 32 weeks of gestation — Different criteria have been suggested for gestational ages less than 32 weeks, given the physiological immaturity of the fetal heart at this stage. (see 'Effect of gestational age on fetal heart rate' above).

Before 32 weeks of gestation, a reactive NST may be defined as two accelerations that rise at least 10 bpm above baseline and have a duration of at least 10 seconds (10 X 10) [12]. Whether clinicians choose to apply the conventional 15 x 15 criteria or adopt the 10 x 10 criteria when performing NSTs before 32 weeks of gestation, they should use the same approach in all patients. However, once a fetus has demonstrated the maturity to generate accelerations of 15 bpm for 15 seconds, an acceleration of 10 bpm for 10 seconds may not have the same ability to confirm fetal well-being, even before 32 weeks of gestation. Available evidence has not resolved this issue definitively

The American College of Obstetricians and Gynecologists have concluded that “the predictive value of NSTs based on a lower threshold for accelerations (at least 10 beats per minute above the baseline and at least 10 seconds from baseline to baseline) has been evaluated in pregnancies less than 32 weeks of gestation and has been found to sufficiently predict fetal wellbeing" [13], based on the following two studies:

- In one study of 143 women who underwent antepartum testing before 32 weeks, the frequency of adverse perinatal outcomes was similar when 10 x 10 criteria were used to define a normal test rather than the conventional 15 X 15 criteria, and the time required to achieve a reactive NST was shorter with the less-stringent criteria [26]. However, the study was not powered to detect differences in adverse outcome or to confirm the safety of applying the 10 x 10 criteria before 32 weeks.

- In another study including 488 women undergoing antepartum testing before 32 weeks, perinatal outcome was similar whether the last NST before delivery was reactive using 10 x 10 or 15 x 15 criteria [27]. After controlling for known confounding factors, such as gestational age and birthweight, there was a trend toward more perinatal deaths following a reactive 10 x 10 NST than following a reactive 15 x 15 NST.

Reactive with decelerations — The significance of a reactive NST with FHR decelerations is unclear. Multiple observational studies have described an increased frequency of intrapartum FHR decelerations and operative delivery when this combination occurs [28-33], except when the decelerations are brief [34]; however, outcomes are usually good. The majority of FHR decelerations during NST are variable decelerations, reflecting transient episodes of umbilical cord compression. Prolonged decelerations, late decelerations, or variable decelerations

without a clear explanation require further evaluation. Management is guided by the results of additional fetal assessment.

If decelerations are observed during the NST, it is reasonable to undertake sonographic evaluation to look for factors associated with FHR decelerations, such as oligohydramnios, nuchal cord or other compromise of the umbilical cord, or intrauterine growth restriction. Other causes, such as intermittent fetal cardiac arrhythmias, may also be diagnosed by sonography. (See "Fetal arrhythmias".)

Additional assessment by BPP can provide further reassurance of fetal well-being, thus supporting nonintervention. In the absence of a clinical cause explaining the deceleration(s), one approach is to follow these pregnancies with at least twice weekly BPPs and/or NST until the test becomes abnormal or fetal pulmonary maturity is attained. Pulmonary maturity may be assumed if the gestational age is 39 weeks by appropriate criteria or confirmed by amniocentesis if in doubt. If the NST is reactive and the BPP is reassuring, induction with continuous FHR monitoring is reasonable. As discussed above, cesarean or instrumental delivery is common because of the increased frequency of category II and III FHR tracings during labor. (See "Management of intrapartum category I, II, and III fetal heart rate tracings".)

Doppler ultrasound of the umbilical artery is a useful ancillary test of fetal assessment when intrauterine growth restriction is identified. (See "Doppler ultrasound of the umbilical artery for fetal surveillance" and "Fetal growth restriction: Evaluation and management", section on 'Doppler velocimetry'.)

Nonreactive tests — An NST is nonreactive if it does not meet acceleration criteria for a reactive NST (see 'Reactive tests' above). The fetal heart rate should be monitored for at least 40 minutes before interpreting the test as nonreactive. Nonreactivity may be a sign of interrupted fetal oxygenation to the point of metabolic acidemia. The mean umbilical vein pH associated with a nonreactive NST is 7.28±0.11, which is higher than the pH associated with a low BPP score 7.16±0.08 [35]. The normal range of antepartum fetal umbilical vein pH has been established by cordocentesis (table 2).

Other causes of nonreactivity include fetal immaturity, quiet fetal sleep, maternal smoking (women who smoke should not smoke proximate to an NST), fetal neurologic or cardiac anomalies, sepsis, or maternal ingestion of drugs with cardiac effects [36]. Sleep is a common and benign cause of nonreactivity. Sleep cycles may last up to 40 minutes [5]. In a study that observed late preterm fetuses from uncomplicated pregnancies for 100 minutes, quiet sleep (no eye movements, no somatic movements except for the occasional startle, and a FHR pattern with little baseline variability) occurred at least once in 30 percent of fetuses, but 96 percent of the fetuses cycled between quiet sleep and active states during the period of observation [37].

Although commonly practiced, meta-analyses have reported that neither maternal glucose administration [38] nor transabdominal manual fetal manipulation [39] significantly decrease the incidence of nonreactive test results related to quiet fetal sleep. The meta-analysis of manual fetal manipulation included only two trials and reported a non-statistical reduction in nonreactive NSTs compared with no or mock stimulation (OR 0.31, 95% CI 0.02-6.20) [39]. Differences in study design may have resulted in the discordant results and, in turn, the wide confidence interval. Further analysis of the value of manual stimulation is needed. Changing maternal position does not increase reactivity as long as she is tested in a position that does not lead to hypotension from uterine compression of the great vessels [40]. Cocoa and caffeine consumption may affect fetal movement, but the dose, timing, and effect on NST reactivity have not been evaluated [41-43]. No randomized trials or observational studies have assessed the effect of maternal hydration (oral or intravenous) on fetal heart rate reactivity. Maternal hydration (oral or intravenous) may increase the amniotic fluid index [44-46] and decrease the baseline fetal heart rate [47,48], but there is no evidence that it increases fetal movement or heart rate reactivity.

Up to 50 to 60 percent of nonreactive NSTs are false positives (defined as a nonreactive NST that is followed by labor with no FHR changes necessitating intervention). For this reason, ancillary tests are used to distinguish the hypoxemic fetus from nonreactivity due to other entities. Some options include:

- Repeat the test in 30 minutes
- Perform vibroacoustic stimulation to elicit accelerations (see 'Vibroacoustic stimulation' below)
- Perform a BPP to evaluate other parameters of fetal well-being (see "The fetal biophysical profile")
- If possible, modify factors potentially causing nonreactive results (eg, smoking proximate to the test)

Vibroacoustic stimulation — Vibroacoustic stimulation can decrease the number of nonreactive NSTs related to quiet fetal sleep cycles and shorten performance time, without reducing the predictive value of a reactive NST. A vibroauditory source, typically an artificial larynx, placed on or just above the maternal abdomen, is used to stimulate fetal movement. In a 2013 systematic review, vibroacoustic stimulation decreased mean overall testing time by almost seven minutes, compared with mock or no stimulation (-6.93, 95% CI -12.09 to -1.76), and reduced the frequency of nonreactive NSTs by 40 percent (OR 0.62, 95% CI 0.48-0.81) [49].

There are no evidence-based standards for performing this procedure. It has been performed as soon as five minutes after initiation of the NST. The stimulus is applied for one to five seconds and may be repeated. The optimal placement and number of applications of the stimulus have not been evaluated. The American College of Obstetricians and Gynecologists suggests positioning the device on the maternal abdomen and applying a stimulus for one to two seconds [13]. If no fetal response occurs, the stimulus may be repeated up to three times for progressively longer durations of up to three seconds.

Halogen light — Transabdominal light stimulation with a halogen light for 10 seconds appears to stimulate the fetus and may be as effective as vibroacoustic stimulation [50-52]. More safety and efficacy data are needed before this method can be recommended in place of vibroacoustic stimulation.

Music — One study found that fetal exposure to Mozart's "Turkish March," and to slightly a lesser extent Strauss' "Trisch-Tratsch Polka," was associated with increased fetal movement, increased number of accelerations, and improved variability [53]. Musical intervention may be a noninvasive and inexpensive tool for shortening the time to reactivity.

Contraction stress test — For the CST, either a dilute oxytocin solution is infused or nipple stimulation is performed until three contractions occur within 10 minutes. There is no standard technique for nipple stimulation. The patient gently massages the nipple of one breast through her clothes for two minutes, stopping with onset of contractions; stimulation is resumed if contractions are too infrequent for CST interpretation. Both nipples can be stimulated if no contractions occur. In women who are having spontaneous contractions of adequate frequency, oxytocin or nipple stimulation is unnecessary. Relative contraindications to stimulating contractions for a CST are conditions that are also contraindications to labor and vaginal delivery, such as placenta previa, vasa previa, and previous classical cesarean delivery or extensive uterine surgery. Preterm labor, patients at high risk for preterm delivery, and preterm premature rupture of membranes are also relative contraindications.

The CST is interpreted as follows [13]:

- **Positive** – A positive (nonreassuring) test has late decelerations following ≥50 percent of contractions. The test is positive even if the contraction frequency is less than three in 10 minutes.
- **Negative** – A negative (reassuring) test has no late decelerations or significant variable decelerations.
- **Equivocal** – An equivocal-suspicious test has intermittent late decelerations or significant variable decelerations, while an equivocal-tachysystolic has decelerations with contractions occurring more frequently than every two minutes or lasting longer than 90 seconds.
- **Unsatisfactory** – An unsatisfactory test is uninterpretable or fewer than three contractions in 10 minutes.

The presence or absence of accelerations is also generally noted. For example, a reactive positive CST is a FHR tracing that meets criteria for both a reactive NST and a positive CST.

Positive contraction stress test — A positive (nonreassuring) CST may indicate fetal hypoxemia and correlates with a 20 to 40 percent incidence of intrapartum category II or III FHR patterns (true positive). An equivocal-suspicious test with repetitive variable decelerations is also

associated with intrapartum category II or III FHR patterns, which are often related to cord compression due to oligohydramnios, especially with postterm pregnancy. [28].

Because of the CST's high false-positive rate (>60 percent), FHR reactivity during the test is used to differentiate false-positive tests (an intrapartum FHR not requiring intervention) from true-positive tests (an abnormal intrapartum FHR requiring intervention) [54,55]. In one study, 50 percent of reactive positive CSTs were false positives, whereas 100 percent of nonreactive positive CSTs were true positives [54].

SUMMARY AND RECOMMENDATIONS

- Fetal health is evaluated, in part, by assessment of fetal heart rate (FHR) patterns. The primary goal is to identify fetuses at risk for intrauterine death or other deficiencies of intrauterine oxygenation and intervene (by measures directed at improving fetal oxygenation, or by delivery) to prevent these adverse outcomes, if possible. (See 'Introduction' above.)

- FHR patterns reflect the fetal response to input from chemoreceptors, baroreceptors, central nervous system activities, hormonal regulation, and blood volume. Gestational age is a factor in interpretation of FHR patterns. (See 'Physiologic basis of fetal heart rate changes' above.)

- Antepartum FHR testing is performed in pregnancies in which the clinician believes the risk of fetal hypoxic injury or demise is increased. (See 'Indications' above.)

- The NST (with or without amniotic fluid assessment) has become the preferred antepartum test of fetal oxygenation status because the CST, which usually requires stimulation of uterine contractions, is more time-consuming, more invasive, and more limited by contraindications than the NST. The biophysical profile (BPP) is a reasonable alternative. (See 'Choice of test' above.)

- The frequency of testing depends upon maternal and fetal status. (See 'Nonstress test' above.)

- The NST is considered reactive if there are two or more fetal heart rate accelerations reaching a peak of at least 15 beats per minute (bpm) above the baseline rate and lasting at least 15 seconds from onset to return to baseline in a 20-minute period (figure 2). There are no published data to confirm a benefit of extending the duration of NST once two qualifying accelerations have been observed. A reactive test is reassuring of fetal well-being and is reassuring regardless of the length of observation time needed to demonstrate reactivity. (See 'Reactive tests' above.)

- Before 32 weeks, given the physiological immaturity of the fetal heart at this stage, reactivity can be based on two accelerations of at least 10 bpm, lasting for at least 10 seconds, and over a 20-minute interval. However, once a fetus has demonstrated the maturity to have

accelerations of 15 bpm for 15 seconds, then an acceleration of 10 bpm for 10 seconds is probably no longer sufficient to demonstrate fetal well-being, even if less than 32 weeks of gestation. (See 'Reactive tests' above.)

- The CST is interpreted as follows (see 'Contraction stress test' above):
  - Positive – Late decelerations following ≥50 percent of contractions, even if the contraction frequency is less than three in 10 minutes.
  - Negative – No late decelerations or significant variable decelerations with contraction frequency of three in 10 minutes.
  - Equivocal – Intermittent late decelerations or significant variable decelerations, while an equivocal-tachysystolic has decelerations with contractions occurring more frequently than every two minutes or lasting longer than 90 seconds.
  - Unsatisfactory – Uninterpretable or fewer than three contractions in 10 minutes.
- The fetal heart rate should be monitored for at least 40 minutes before interpreting the test as nonreactive. Abnormal test results may be due to interruption of fetal oxygenation, but should be interpreted within the context of the clinical situation, given the high false-positive rate. Vibroacoustic stimulation is useful for decreasing the number of nonreactive NSTs related to quiet fetal sleep cycles. (See 'Nonreactive tests' above and 'Vibroacoustic stimulation' above.)
- Hospitalization for prolonged fetal monitoring or delivery usually is warranted for persistent nonreactive NSTs that have been confirmed by additional tests of fetal status (eg, CST, BPP, particularly when abnormal fetal oxygenation is strongly suspected, such as in cases of fetal growth restriction. (See 'Management of pregnancies with nonreassuring test results' above.)

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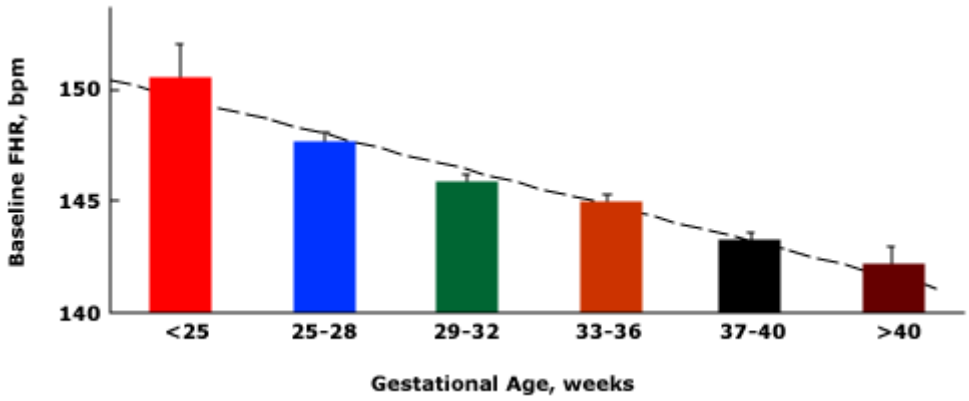
Topic 409 Version 28.0

GRAPHICS

Graph of baseline FHR according to gestational age, weeks



Bpm: beats per minute; FHR: fetal heart rate.

Adapted from: Park MI, Hwang JG, Cha KJ, et al. Computerized analysis of fetal heart rate parameters by gestational age. Int J Gynaecol Obstet 2001; 74:157.

Graphic 62797 Version 2.0

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.

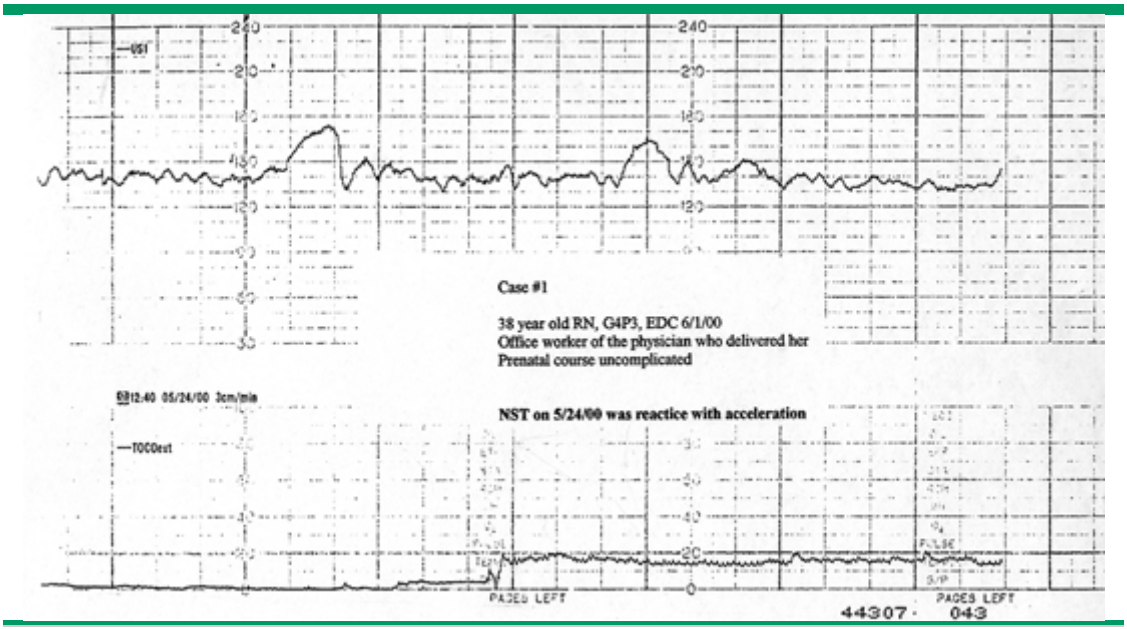
NICHD: National Institute of Child Health and Human Development; bpm: beats per minute; sec: seconds; FHR: fetal heart rate.

*"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.

Adapted from: National Institue of Child Health and Human Development Research Planning Workshop. Am J Obstet Gynecol 1997; 177:1385 and Macones GA, Hankins GD, Spong CY, et al. The 2008 National Institute of Child Health and Human Development Workshop Report on Electronic Fetal Monitoring: Update on Definitions, Interpretation, and Research Guidelines. Obstet Gynecol 2008; 112:661.

Graphic 65859 Version 7.0

Reactive nonstress test performed eight days before the patient's estimated delivery date



Reactive nonstress test. The baseline fetal heart rate is between 130 and 140 beats per minute. There are two accelerations >15 beats per minute; one peaks at approximately 170 beats per minute and the other peaks at approximately 160 beats per minute. The duration of each acceleration exceeds 20 seconds. Variability is moderate (6 to 25 beats per minute). The top tracing is the fetal heart rate. The y-axis reflects the fetal heart rate measured in beats per minute. The x-axis reflects time; each of the smallest divisions represents 10 seconds with one minute between bold vertical lines.
The bottom tracing shows the frequency and duration of uterine contractions.

Graphic 72845 Version 6.0

Antepartum umbilical vein and artery pH

GA	Umbilical vein pH			Umbilical artery pH		
	5th percentile	Mean	95th percentile	5th percentile	Mean	95th percentile
18	7.385	7.423	7.461	7.360	7.398	7.435
20	7.382	7.420	7.458	7.357	7.394	7.431
22	7.379	7.417	7.454	7.353	7.390	7.427
24	7.376	7.414	7.451	7.350	7.386	7.423
26	7.373	7.410	7.448	7.346	7.383	7.419
28	7.370	7.407	7.445	7.342	7.379	7.416
30	7.366	7.404	7.442	7.338	7.375	7.412
32	7.363	7.401	7.439	7.334	7.371	7.409
34	7.360	7.398	7.436	7.330	7.368	7.406
36	7.357	7.395	7.433	7.325	7.364	7.403
38	7.353	7.392	7.430	7.321	7.360	7.399
40	7.350	7.388	7.427	7.317	7.357	7.396

pH values were determined by cordocentesis in fetuses whose mothers were not in labor.

GA: gestational age.

Graphic 76880 Version 3.0

Contributor Disclosures

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

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