Antepartum testing entails the evaluation of fetal health through a variety of modalities, including fetal heart rate monitoring and ultrasound, occurring at points in pregnancy that are remote from delivery, as opposed to intrapartum testing, which is performed in the patient experiencing labor. The goal for antepartum testing should be, as with all medical tests, an optimization of health outcomes for the test’s recipients, in this case the maternal-fetal dyad. A false-negative test will be one that fails to identify a fetus at risk of death or major morbidity, which could have been prevented by delivery. False-positive results, however, can lead to iatrogenic preterm birth, which itself can be associated with significant morbidity. Even if testing does not lead to delivery, positive results can also generate significant maternal anxiety and stress, as well as cost. The optimal antepartum fetal testing strategy would appropriately identify an at-risk fetus prior to an irreversible event while minimizing maternal anxiety, cost, and iatrogenic prematurity. Such optimization, however, is difficult to achieve.

Antepartum Fetal Surveillance

INDICATIONS FOR SURVEILLANCE

Intrauterine fetal death may result from a wide range of potential etiologies including congenital structural malformations, genetic abnormalities, fetomaternal hemorrhage, infection (TORCH and chorioamnionitis), umbilical cord obstruction, placental abruption, and uteroplacental insufficiency.\(^25\) The methods commonly used for antenatal fetal surveillance rely on fetal biophysical parameters that are sensitive to hypoxemia and acidemia, such as heart rate and movement. Thus it is primarily useful in a fetus at risk for hypoxemia specifically because of chronic uteroplacental insufficiency. Intrauterine demise from sudden catastrophic events, such as abruption secondary to maternal trauma or cord compression at the time of membrane rupture, are likely not predictable by antepartum monitoring.

The indications for antenatal testing are those that increase the risk of uteroplacental insufficiency, many of which are listed in Table 13-1. The optimal antenatal testing strategy for each of these would be beyond the scope of a single chapter, and additionally in many circumstances the exact strategy is controversial because there is often little or no prospective or randomized data from which to determine an optimal approach. Many conditions for which testing has been suggested are those for which epidemiological studies have identified an increased risk of intrauterine demise. However, in some circumstances the risk of stillbirth, although achieving statistical significance in large studies, may remain small in actual magnitude. Additionally, an association between a particular risk factor and stillbirth alone does NOT necessarily demonstrate that there is a benefit from antenatal surveillance, because that would require a specific study of antepartum testing for the given risk factor. For example, a history of a prior unexplained stillbirth is associated with an increased risk of stillbirth,\(^12\) though because there are few or no prospective interventional studies, monitoring for these conditions is primarily based upon expert opinion.\(^1\)

Of note, uterine contraction monitoring (tocometry) is performed simultaneously with electronic fetal cardiac monitoring as part of the nonstress and contraction stress test. This is primarily to allow for the interpretation of fetal heart rate decelerations relative to uterine contractile activity. Uterine contraction monitoring alone as a method of identifying patients at increased risk of preterm birth is of low clinical utility.\(^13\)

PHYSIOLOGIC BASIS FOR ANTENATAL SURVEILLANCE

The application and interpretation of antepartum fetal monitoring necessitates an understanding of the progressive fetal changes that can occur secondary to increasing placental insufficiency progressing to intrauterine demise. In experiments involving animal and human fetuses, hypoxemia and acidosis have been shown consistently to alter fetal biophysical parameters such as heart rate, movement, breathing, and tone.\(^4,19,21\) The fetal heart rate (FHR) is normally controlled by the fetal central nervous system (CNS) and mediated by sympathetic or parasympathetic nerve impulses originating in the fetal brainstem. The presence of intermittent FHR accelerations associated with fetal movement is believed to be an indicator of an intact fetal autonomic nervous system. In a
study of fetal blood sampling of pregnancies resulting in healthy neonates, Weiner and colleagues established a range of normal fetal venous pH measurements. In this population, the lower 2.5 percentile of fetal venous pH was 7.37. Manning and colleagues, showed that fetuses without heart rate accelerations had a mean umbilical vein pH of 7.28 (± 0.11), and fetuses with abnormal movement had a mean pH of 7.16 (± 0.08). These and similar observations were the basis for the development of antenatal fetal testing modalities that are currently in use.

**PATIENT ASSESSMENT OF FETAL MOVEMENT**

The patient’s own subjective assessment of the activity of her fetus is perhaps the simplest and most universal of antepartum surveillance methods, although its subjective nature leads to difficulty in quantification and empiric evaluation. As described above, fetal movement decreases with increasing hypoxia, which serves as the physiologic basis of the biophysical profile as well as subjective fetal movement monitoring. As a result, a perceived decrease

<table>
<thead>
<tr>
<th>Maternal Conditions</th>
<th>Pregnancy-Related Conditions</th>
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<tr>
<td>Antiphospholipid syndrome</td>
<td>Pregnancy-induced hypertension</td>
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<tr>
<td>Hyperthyroidism (poorly controlled)</td>
<td>Decreased fetal movement</td>
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<td>Diabetes mellitus</td>
<td>Oligohydramnios</td>
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<td>Cyanotic heart disease</td>
<td>Polyhydramnios</td>
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<td>Systemic lupus erythematosus</td>
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<td>Hypertensive disorders</td>
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<td>Chronic renal disease</td>
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<td>Hemoglobinopathies</td>
<td>Previous fetal demise</td>
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<td>(hemoglobin SS, SC, or S-thalassemia)</td>
<td>(unexplained or recurrent risk)</td>
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<td></td>
<td>Isoimmunization (moderate to severe)</td>
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<td>Preterm premature rupture of membranes</td>
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<td>Unexplained third-trimester bleeding</td>
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**TABLE 13-1** Indications for Antenatal Surveillance

**NONSTRESS TEST**

In most institutions, the first-line assessment tool for fetal surveillance is the nonstress test (NST). In the outpatient setting the patient typically rests in a reclining chair with a lateral tilt. Ideally she should have not recently smoked. Although commonly provided in antepartum testing units, the maternal ingestion of juice or food has not been demonstrated to increase the probability of a reactive nonstress test. The FHR is monitored with an external transducer for up to 40 minutes and observed for the presence of accelerations above the baseline. A reactive test is one in which there are at least two accelerations that peak 15 beats/min above the baseline and last (not at the peak) for at least 15 seconds before returning to baseline (Figure 13-1), colloquially referred to as “15 × 15.” Most NSTs are reactive within the first 20 minutes. For tests that are not, possibly because of a fetal sleep cycle, an additional 20 minutes of monitoring may be needed. A nonreactive NST is one in which two such accelerations do not occur within 40 minutes.

The optimal gestational age at which to begin antenatal surveillance depends on the clinical condition. In making this decision, the physician must weigh the risk of intervention at a premature gestational age against the risk of intrauterine fetal death. The American College of Obstetricians and Gynecologists recommends initiating testing at 32 to 34 weeks’ gestation for most at-risk patients, with the acknowledgment that some situations may warrant testing earlier at 26 to 28 weeks of gestation. FHR variability and reactivity vary with gestational age. Before 28 weeks of gestation, 50% of all NSTs may not be reactive. From 28 to 32 weeks of gestation, approximately 15% of normal fetuses have nonreactive NSTs. Thus prior to 32 weeks nonstress tests are often
considered reactive if there are two accelerations that peak 10 beats/min above baseline and last for at least 10 seconds ("10 × 10") rather than the traditional cutoff ("15 × 15"). Of note, the magnitude of accelerations in fetuses less than 32 weeks can vary normally over time, thus a fetus at less than 32 weeks is reactive by 10 × 10 criteria even if it had previously demonstrated 15 × 15 accelerations.9

Although the NST is noninvasive and easy to perform, it is limited by a high false-positive rate. Normal fetuses often have periods of nonreactivity because of benign variations such as sleep cycles. Vibroacoustic stimulation may be used safely in the setting of a nonreactive NST to elicit FHR accelerations without compromising the sensitivity of the NST.29 In this situation the operator places an artificial larynx on the maternal abdomen and activates the device for 1 to 3 seconds. This technique is often useful in situations in which the FHR has normal beat-to-beat variability and no decelerations, but does not show any accelerations. If the test remains nonreactive, further evaluation with a biophysical profile or contraction stress test (CST) is warranted as long as the FHR is otherwise reassuring. However, if the tracing is overtly concerning (see Category III later in this chapter), additional testing may need to be deferred in favor of delivery depending upon the exact gestational age and clinical circumstance.

**CONTRACTION STRESS TEST**

The CST is designed to evaluate FHR response to maternal uterine contractions. The principles that are applied to the evaluation of intrapartum FHR monitoring (see FHR Monitoring) are used here. In response to the stress of the contraction, a hypoxemic fetus shows FHR patterns of concern, such as late decelerations.

Similar to the NST, for the CST the patient is placed in a recumbent tilted position, and FHR is monitored with an external fetal monitor. The FHR pattern is evaluated while the patient experiences at least three contractions lasting 40 seconds within a 10-minute period. If the patient is not contracting spontaneously, contractions may be induced with nipple stimulation or intravenous oxytocin. Nipple stimulation can be self-administered by the patient or a breast pump can be used. If no late or significant variable decelerations are noted on FHR tracing, CST is considered to be negative. If there are late decelerations after at least 50% of the contractions, CST is positive. If late decelerations are present less than 50% of the time, or if significant variable decelerations are present, the test is considered to be equivocal. Contraindications to the performance of CST include clinical situations in which labor would be undesirable (e.g., placenta previa or previous classic cesarean section).

**AMNIOTIC FLUID VOLUME ASSESSMENT**

Amniotic fluid volume is commonly estimated by ultrasound via one of two primary methods (see Chapter 25). The amniotic fluid index (AFI) is calculated by measuring and adding the maximal vertical pockets of fluid (without loops of umbilical cord) in each of the four quadrants of the maternal abdomen (Figure 13-2). Alternatively, the single deepest vertical pocket of fluid alone can be measured. Decreased amniotic fluid volume, or oligohydramnios, is typically defined as either an AFI of 5 cm or less or no single measurable vertical pocket of fluid greater than 2 cm, although gestational age–specific norms can be used as well. Neither method is perfectly sensitive or specific for the detection of oligohydramnios.18 Oligohydramnios can occur secondary to a range of causes including rupture of the fetal membranes and congenital fetal anomalies of the urinary tract. In the absence of membrane rupture or congenital anomalies, however, the most concerning etiology would be decreased fetal urine production secondary to the shunting of blood flow away from the fetal kidneys in the context of uteroplacental insufficiency.

When oligohydramnios is diagnosed, the first step is to rule out membrane rupture and congenital anomalies and, if not present, assess the fetus for other evidence of uteroplacental insufficiency, including fetal biometric

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**BOX 13-1 BIOPHYSICAL PROFILE SCORING SYSTEM**

1. Fetal breathing movements (one or more episodes lasting at least 30 seconds)
2. Fetal movement (three or more discrete body or limb movements)
3. Fetal tone (one or more episodes of active extension with return to flexion of a limb or trunk, or the opening and closing of a fetal hand)
4. Amniotic fluid volume (originally described as a single vertical pocket of ≥1 cm, subsequently modified to ≥2 cm) (see Figure 13-2)
5. Reactive NST

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**AMNIOTIC FLUID VOLUME ASSESSMENT**

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When oligohydramnios is diagnosed, the first step is to rule out membrane rupture and congenital anomalies and, if not present, assess the fetus for other evidence of uteroplacental insufficiency, including fetal biometric
measurements to assess growth restriction. Delivery is usually performed for oligohydramnios at term, although at preterm gestations delivery decisions will involve multiple factors including the exact gestational age and presumed etiology of the decreased fluid, with conservative management being reasonable in many circumstances.

**DOPPLER FLOW VELOCIMETRY**

Qualitative and quantitative evaluation of maternal and fetal blood vessels by Doppler sonography has been the focus of intense research over the last several years and is continuing to evolve rapidly. The list of clinical scenarios in which it has been utilized includes the evaluation of the fetal middle cerebral artery in cases of red blood cell isoimmunization, monochorionic twins with twin-twin transfusion syndrome, the screening and diagnosis of congenital cardiac anomalies, and the diagnosis of congenital vascular anomalies. These indications will likely continue to expand. However, the primary utility of Doppler sonography is in the evaluation of a fetus with possible intrauterine growth restriction. In normal pregnancies or when the fetus has demonstrated normal growth, there is no current role for Doppler sonography of fetal vessels because they have not been found to convey benefit in a low-risk population.

For the fetus in which measurements demonstrate potential growth restriction, Doppler sonography has both diagnostic and predictive benefit. Although more extreme biometric deviations are usually pathologic, many fetuses with ultrasound weight estimations at the fifth to tenth percentile will be small but healthy. In these cases, either the ultrasound weight estimation is incorrect or the true birth weight is less than 10% but the fetus is just an otherwise healthy outlier of the normal weight distribution. Doppler sonography of fetal vessels in these circumstances can potentially identify the fetuses that are healthy, thus avoiding iatrogenic prematurity and additional antenatal testing. Abnormal results, however, can identify a fetus that is at true risk. In cases of suspected growth restriction, abnormal blood flow in the umbilical artery is associated with increased risk of perinatal morbidity and mortality. A Cochrane review of 11 randomized trials showed a trend toward decreased perinatal mortality with the use of Doppler assessment of the umbilical artery in high-risk pregnancies. The use of umbilical artery Doppler flow velocimetry as a primary testing method results in fewer antenatal tests and less intervention with similar neonatal outcome compared with pregnancies monitored by NST alone.

Although there are many potential causes of intrauterine growth restriction, most cases that are not secondary to intrinsic fetal etiology (e.g., TORCH, skeletal dysplasias, genetic anomalies) arise from uteroplacental insufficiency, which itself can be detected through the Doppler evaluation of the umbilical artery carrying blood from the fetus to the placenta. Pathologic placental processes such as thrombosis and infarction decrease the relative size of the placental vascular bed and increase placental vascular resistance. As placental vascular resistance increases, there is a progressive diminution of blood flow during diastole, resulting in an altered ratio of systolic (S) and diastolic (D) blood flow that can be detected with Doppler sonography (Figure 13-3). Numerically, this can be quantified as either the systolic/diastolic (S/D) ratio, resistance index ([S-D]/S), or pulsatility index ([S-D]/average blood flow). In more extreme circumstances, blood flow during diastole may be absent (absent end diastolic flow or AEDF) or even reversed (REDF), as demonstrated in Figure 13-3.

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**Figure 13-2** Ultrasound image of the maximal vertical pocket measured as part of either the biophysical profile or the amniotic fluid index. Note the absence of umbilical cord in the measured pocket.

**Figure 13-3** Composite images of three studies of fetal umbilical arterial velocimetry, ranging from normal to markedly abnormal. A, Normal velocimetry pattern. B, Absent diastolic flow, indicating increased placental resistance. C, Reversal of diastolic flow, indicating worsening placental function. D, diastolic velocity; S, systolic velocity.
Even venous pulsations can be a sign of heart failure in an older adult with heart disease, myocardial decompensation in the fetus can generate venous pulsations that are then reflected back through the ductus venosus and umbilical vein. When present these can be an ominous finding, with delivery often being necessitated within a few days.

While normal Doppler results in a fetus with concerning biometric measurements signal that a pregnancy can safely continue, the optimal management of a fetus with abnormal Doppler studies is far from clear. If other tests of fetal well-being (nonstress test, biophysical profile) are not reassuring, then delivery is usually indicated. Likewise, abnormal Doppler results can be used to guide the frequency of antepartum testing. Other than additional monitoring, however, few actual treatment options are available.

Abnormal umbilical artery Doppler S/D ratios imply placental vascular pathology, although the test does not otherwise specifically address the immediate state of fetal oxygenation or health. Many individuals with mild elevations of the S/D ratio will deliver healthy babies at term, which is why Doppler sonography is discouraged in low-risk patients or those with normal fetal biometric evaluations. For a fetus with both concerning biometric measures and abnormal umbilical artery Doppler results, the immediate fetal health can be assessed by the NST, CST, or BPP as described previously. Additionally, the fetal status can be evaluated through Doppler sonography of additional fetal vessels beyond the umbilical artery. Turan and colleagues serially evaluated 104 fetuses with uteroplacental insufficiency and growth restriction and performed sonography on the middle cerebral artery, umbilical artery and vein, and ductus venosus until the patient was delivered. A sequential pattern of worsening and multivessel Doppler anomalies can often be identified prior to a deceleration of fetal health necessitating delivery (Figure 13-4). In response to increasing hypoxia, blood flow is diverted away from nonvital organs such as the kidney (resulting in oligohydramnios) and preferentially toward vital organs such as the brain, a process referred to as cephalization. The increased cerebral blood flow can be reflected in a decreasing pulsatility index in the fetal middle cerebral artery (Figure 13-5). The absence of cephalization can be reassuring, although it should be noted that it can sometimes be absent in critically ill fetuses that have lost the ability to preferentially direct their blood flow. More severe degrees of hypoxia eventually generate myocardial decompensation, which can be evaluated through the Doppler evaluation of the ductus venosus and umbilical artery. Just as jugular venous pulsations can be a sign of heart failure in an older adult with heart disease, myocardial decompensation in the fetus can generate venous pulsations that are then reflected back through the ductus venosus and umbilical vein. When present these can be an ominous finding, with delivery often being necessitated within a few days.

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exist to improve the fetal health while in utero, and thus the question often becomes whether or not to deliver prematurely. For a fetus with growth restriction and abnormal multivessel Doppler evaluations but a reassuring NST, CST, or BPP, further decompensation at some point in the future is probable, and ideally one would want to deliver prior to an irreversible injury. Thus one might consider iatrogenic premature delivery despite the reassuring NST or BPP. On the other hand, many fetuses with absent or reverse umbilical artery diastolic flow can safely remain in utero for even several weeks. The Growth Restriction Intervention Trial (GRIT) attempted to provide guidance in this regard by randomizing 348 women with “compromise” in a premature fetus to either immediate or delayed delivery. No significant differences in overall mortality were identified between the two groups, since the increase in stillbirth in the expectant management group was balanced by neonatal losses secondary to prematurity in the group that underwent immediate delivery. The subjects included in GRIT were very heterogeneous with regard to growth and Doppler results found that in 61% of patients with positive tests, the increase in stillbirth in the tested population (after antepartum testing with normal results) has been reported to be approximately 1.9 per 1000 for NST, 0.3 per 1000 for CST, 0.8 per 1000 for biophysical profile, and 0.8 per 1000 for modified biophysical profile. These rates are comparable to the rates for the risk of fetal death in a low-risk population.

The false-positive rate is more difficult to ascertain because a positive test usually results in obstetric intervention, significantly decreasing the likelihood of intrauterine death. One study showed, however, that 90% of nonreactive NSTs are followed by a negative CST result, consistent with a high false-positive rate for NST. A study of CSTs in which physicians were blinded to the results found that in 61% of patients with positive tests, there were no fetal late decelerations in labor, no low Apgar scores, and no significant neonatal morbidity. Manning and colleagues reported on a cohort of 913 infants delivered after biophysical profile score of 6 or less. Nearly 40% of infants with scores of 6 showed no markers of fetal compromise at delivery, as defined by fetal distress in labor, admission to the neonatal intensive care unit, a 5-minute Apgar score of 7 or less, or an umbilical cord pH less than or equal to 7.20. There was a significant inverse linear association, however, between biophysical profile score and these markers, and all fetuses with scores of 0 had at least one of these markers at delivery.

In a clinically stable situation, reassuring tests (reactive NST, negative CST, and biophysical profile of 8 or 10) are considered reliable for 1 week, and so testing is usually performed on a weekly basis. Labile conditions may merit more frequent testing; the frequency is left to the discretion of the physician. If the indication for testing is not a persistent one (e.g., maternal perception of decreased fetal movement), there is no evidence to support the continuation of antenatal testing. In certain high-risk populations, the false-negative rate of NST may be unacceptably high. The stillbirth rate within 1 week of a reactive NST is markedly higher for patients with diabetes mellitus (14 per 1000) and fetal growth restriction (20 per 1000). Boehm and co-workers found that the stillbirth rate decreased from 6.1 per 1000 to 1.9 per 1000 in their high-risk population when the frequency of testing was changed from once weekly to twice weekly. For this reason, testing twice weekly may be appropriate in certain populations, such as those described.

Clinically, one should always give consideration to maternal illness as a cause of nonreassuring fetal status. For example, if the mother is acidemic from any etiology, placental equilibration will eventually lead to acidemia in an otherwise healthy fetus, which in turn can lead to abnormal antenatal testing results. A classic example is maternal diabetic ketoacidosis. In such circumstances, the appropriate course of action is to correct the maternal condition first and not to necessarily directly intervene on behalf of the fetus despite the nonreassuring antenatal testing. The fetal status will improve as the maternal status is improved, thus avoiding iatrogenic delivery, and cesarean sections or other efforts to deliver the fetus may not be safe if the mother is critically ill.

**Evaluation of the Intrapartum Fetus**

The process of labor and delivery is a period of significant metabolic stress for both the laboring mother and her baby, although in the great majority of cases these stressors are easily tolerated and labor results in a perfectly healthy mother and child. In some cases, however, the process is tolerated poorly and the fetus develops a degree of acidosis that places it at risk of multiorgan dysfunction or even death. Thus in the 1970s, FHR monitoring during labor was introduced, with the hope of identifying heart rate patterns that were predictive of adverse outcomes and would allow for intervention prior to irreversible events. Subsequently, continuous electronic FHR monitoring during labor has been used in the majority of laboring patients in developed countries for the last several decades. The initial hopes and promises of continuous FHR monitoring, however, have not necessarily been fulfilled during this time. The incidence of cerebral palsy has remained stable, whereas the cesarean section rate has progressively increased, in part because of operative deliveries being performed for FHR patterns which may or may not be sufficiently predictive of adverse outcomes. The contemporary practitioner utilizing FHR monitoring during labor must thus be cautious to both intervene prior to an irreversible event and to avoid iatrogenic operative delivery. This has proved to be a difficult balance.
INTRAPARTUM OXYGENATION AND NEUROLOGIC MORBIDITY

The strongest correlate to adverse outcomes is decreased tissue concentrations of oxygen (hypoxia) and decreased tissue pH (acidosis). What is more easily and often evaluated, however, is decreased oxygenation or pH in the peripheral blood, referred to respectively as hypoxemia and acidemia. These are what is measured when the blood from the umbilical artery and vein is sampled after delivery, and thus serve as common surrogate outcomes in clinical research. Although significant hypoxemia will eventuallylead to tissue-level hypoxia and acidosis, the presence of the former does not necessarily guarantee the latter. Although hypoxemia and acidemia can be easily evaluated by laboratory methods, determining the presence or absence of hypoxia or acidosis is more complex and often involves physical and clinical more so than laboratory assessments.

Another important differentiation is between respiratory and metabolic acidosis in the fetus and neonate. The concept of a "respiratory" acidosis in a fetus may seem unusual, because they are not literally using their lungs to exchange air, although the same concepts that are useful outside of the uterus can be applied to the transplacental exchange of oxygen and carbon dioxide. A respiratory acidosis occurs when carbon dioxide accumulates secondary to impaired clearance by the lungs or, in the case of a fetus, the placenta. A fetal metabolic acidosis will be the result of a prolonged or severe deprivation of oxygen, triggering lactate production in fetal tissue. A respiratory or metabolic acidosis, although often occurring in combination, can be differentiated from one another by the measurement of base deficit, with a high base deficit indicating a metabolic process. Metabolic processes are more concerning than respiratory ones for several reasons. First, an umbilical artery acidemia with an increased base deficit strongly implies excess tissue lactate generation. Thus there is probable acidosis and not just acidemia. Additionally, a respiratory acidemia can rapidly correct itself once normal ventilation is established and excessive carbon dioxide is cleared, whereas the correction of a metabolic acidosis requires the cessation of lactate generation at a tissue level and is thus delayed relative to the onset of appropriate oxygenation. Clinically, the newborn with an isolated respiratory acidosis (or acidemia) will have a low umbilical cord pH at birth and low 1-minute Apgar score, although once ventilation is established, will enjoy rapid clinical improvement and a subsequent uneventful newborn period. By contrast, the neonate who remains clinically depressed through the first several minutes of life despite adequate ventilation is more likely to have a metabolic acidosis and an increased umbilical artery base deficit.

As discussed previously, various findings in the FHR patterns have been correlated with fetal hypoxemia and acidemia. The outcomes of hypoxemia and acidemia, however, are in and of themselves surrogate outcomes. Although fetal risks increase with increasing metabolic derangements, many fetuses with hypoxemia and acidemia, especially if it is a purely respiratory process, will subsequently have a normal newborn course. If an operative delivery is performed in a fetus that has hypoxemia or acidemia but that would have, if left alone, delivered vaginally without permanent neurologic injury, then the intervention has not been clearly beneficial. The most meaningful clinical question then is if continuous FHR monitoring can predict the fetus that will not survive the labor process or survive with permanent neurologic injury secondary to intrapartum events. Given the multitude of complex factors that contribute to neurologic injury, it is perhaps not unsurprising that an evaluation of FHR alone has not proved to be sufficiently predictive in this regard. Using cerebral palsy as an endpoint, one study found the positive predictive value of FHR monitoring to be 0.14%.

The overall determination of neonatal health following the stress of delivery is not determined by a single laboratory value, but a combination of laboratory and clinical evidence of metabolic acidemia with a clinical course that is consistent with a hypoxic event. For the child who is subsequently diagnosed as having cerebral palsy, a multitude of other potential etiologies (anatomic, infectious, genetic, thrombotic, and metabolic, among others) needs to be ruled out as well, because intrapartum hypoxic events account for only a small percentage of cases of neonatal neurologic injury. To provide guidance in these complicated issues, the American College of Obstetricians and Gynecologists and American Academy of Pediatricians convened a joint task force that in 2003 established criteria to define an acute intrapartum event as having been sufficient to cause cerebral palsy. These criteria are outlined in Box 13-2. In the absence of these explicit criteria, it is not optimal for a provider to ascribe neurologic outcomes to a potential intrapartum event, and appropriate caution should always be applied to the potentially inappropriate use of the expressions asphyxia, newborn encephalopathy and hypoxic-ischemic encephalopathy in the medical records.

CONTINUOUS VERSUS INTERMITTENT FETAL HEART RATE MONITORING

Two options for fetal monitoring are available for laboring women: continuous and intermittent fetal monitoring. Provider and patient opinions vary regarding which is optimal, although for low-risk patients guidelines exist for both, and both options are considered acceptable and within usual standards of care. A 2008 meta-analysis by the Cochrane Collaborative identified 12 studies that compared continuous electronic FHR monitoring to either no or intermittent monitoring during labor. The use of continuous FHR monitoring was not associated with a decreased risk of perinatal mortality or cerebral palsy. There was, however, a significant reduction in neonatal seizures (relative risk of 0.5). This benefit was balanced against a significant increase in the risk of undergoing either cesarean section or operative vaginal delivery. Thus the optimal mode of fetal monitoring is unclear. The occurrence of neonatal seizures, low Apgar scores, and hypoxemia at the time of birth can all cause significant emotional distress for new parents even in the absence of clear differences in long-term neurologic outcomes or survival, and many parents would potentially be willing to accept an increased risk of operative delivery.
to prevent neonatal seizures. On the other hand, many patients are highly motivated to have a spontaneous vaginal delivery and would be willing to accept a risk of transient neonatal seizures if there are no significant differences in longer-term neurologic outcomes. For low-risk patients who would be candidates for either approach, the most optimal approach may be to discuss the relative advantages and disadvantages of either early in pregnancy and allow the patients to then make the choice that works best for them.

Continuous electronic FHR monitoring may be performed externally or internally. Telemetry units are available so that a patient need not be confined to a bed to be monitored. Additionally, many modern units are waterproof and would thus allow for continuous monitoring while a patient is laboring in a bath or tub. Most external monitors use a Doppler device with computerized logic to interpret and count the Doppler signals. Internal FHR monitoring may be performed in circumstances in which external monitoring is technically difficult, such as maternal obesity. Internal monitoring uses an electrode that is attached to the fetal scalp, and contraindications to fetal scalp puncture (transmittable maternal infections such as hepatitis or HIV) are likewise relative contraindications for internal fetal monitoring.

Internal monitoring also necessitates rupture of the fetal membranes. If the membranes are intact and internal monitoring is determined to be necessary, they can be artificially ruptured to facilitate monitoring, although this can carry a risk of umbilical cord prolapse if the fetal presenting part is not engaged.

**INTERPRETATION OF CONTINUOUS FHR RECORDINGS**

**Baseline Fetal Heart Rate**

Baseline FHR is the average FHR rounded to increments of 5 beats/min during a 10-minute segment, excluding periodic or episodic changes, periods of marked variability, or baseline segments that differ by more than 25 beats/min. In any given 10-minute window, the minimal baseline duration must be at least 2 minutes or the baseline is considered indeterminate. A normal FHR baseline rate ranges from 110 to 160 beats/min. If the baseline FHR is less than 110 beats/min, it is termed bradycardia. If the baseline FHR is greater than 160 beats/min, it is termed tachycardia (Figure 13-6). Of note, although rates of 110 and 160 beats/min are the accepted cutoff values for FHR, many fetuses with heart rates above or below these values will be otherwise healthy outliers. For example, a fetus that is experiencing otherwise uncomplicated labor with a fetal heart rate tracing that is perfectly reassuring other than a baseline in the 100s is likely normal.

As described following, events that can be associated with hypoxemia or the later development of hypoxemia, such as umbilical cord compression, produce decelerations of the fetal heart rate. However, it is worth noting that the primary response to hypoxemia is not bradycardia but tachycardia secondary to sympathetic discharges. A progressive increase in the heart rate baseline during labor can raise concern for hypoxemia if other FHR anomalies, such as decreasing variability, become progressively apparent over the same time period. Tachycardia may also be associated with conditions other than hypoxia, such as maternal fever, intra-amniotic infection, thyroid disease, the presence of medication, and cardiac arrhythmia.

**Fetal Heart Rate Variability**

The presence of baseline FHR variability is a useful indicator of fetal CNS integrity. Variability is defined as fluctuations in FHR baseline of two cycles per minute.
or greater, with irregular amplitude and inconstant frequency. Of note, although the fetus will normally have beat-to-beat heart rate variation, the actual PQRS complexes themselves are normal, differentiating this healthy phenomenon from atrial fibrillation or other pathologic arrhythmias. The variation represents alternating responses to sympathetic and parasympathetic inputs. If either the sympathetic or parasympathetic system instead predominates, such as would be the case of vagal nerve stimulation or increased sympathetic activity owing to hypoxia, this variability would be lost (Figure 13-7). Thus, moderate FHR variability is strongly associated (98%) with an umbilical pH greater than 7.15, and in combination with a normal FHR baseline, generates a high degree of reassurance regardless of the presence or absence of accelerations or decelerations. Accelerations are periodic elevations above the baseline, and they are usually associated with fetal movement. The presence of FHR accelerations during labor is always reassuring, as it is in a nonlaboring patient. Their absence during labor, however, is not necessarily concerning if other aspects of the FHR pattern are reassuring (see category I). Unlike a nonstress test in an antepartum patient, a “reactive” FHR tracing with “15 × 15” accelerations is not required for reassurance in the laboring patient.

### Decelerations

Decelerations in FHR are episodic decreases below the baseline. Most decelerations are mediated through parasympathetic stimulation from the vagal nerve. These in turn are triggered by a variety of stimuli, including transient increases in intracranial pressure (“early” decelerations), increased systemic vascular resistance (“variable” decelerations) and hypoxemia (some “late” decelerations). Thus most decelerations do not specifically signify the presence of fetal acidosis, and in fact many are simply interesting demonstrations of human physiologic reflexes. A portion of “late” decelerations, however, occurs secondary to the suppression of myocardial function by tissue-level hypoxia, which is clinically concerning. Clinically differentiating these from other deceleration patterns, however, is often imprecise.

Decelerations are classified by their morphology and then by whether they are recurrent or prolonged. Decelerations are defined as “recurrent” if they occur with at least 50% of the contractions. A “prolonged” deceleration is one that lasts for more than 2 minutes. With regard to morphology, three types of decelerations were initially described by Hon and colleagues in 1967. Early decelerations are shallow and symmetric, gradual in onset and recovery, and associated with a contraction such that the nadir of the deceleration occurs at the same time as the peak of the contraction (Figure 13-8). Physiologically, early decelerations are a demonstration of Cushing reflex, in which increased intracranial pressure generates bradycardia through stimulation of the vagal nerve. Because of the unfused cranial fontanelles, pressure applied to the fetal cranium, such as when the head is pressed against

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**Figure 13-7** Abnormal (absent) fetal heart rate (FHR) variability. Because there are no decelerations present, this would qualify as a category II FHR tracing. (From Freeman R, Garite T, Fetal heart rate monitoring. Baltimore: Williams & Wilkins; 1981:138.)
maternal tissue during a contraction, is translated into increased intracranial pressure and can trigger activation of the vagal nerve. Like most reflexes, the response is virtually instantaneous and the magnitude of vagal nerve stimulation correlates with the magnitude of pressure applied against the fetal head. This is why “early” decelerations appear as mirror images of the contractions. This entire process is unrelated to fetal oxygenation and acid-base balance, which is why early decelerations, although conceptually interesting, are not of clinical importance.

Variable decelerations are typically associated with an abrupt onset and abrupt return to baseline. They vary in shape, depth, and duration and in the occurrence of contractions. They are also frequently preceded and followed by small accelerations in FHR (Figure 13-9 and Figure 13-10). Variable decelerations are usually associated with compression of the umbilical cord and represent physiologic changes in response to alterations in vascular resistance and preload. The umbilical cord contains a single large, thin-walled vein and two smaller, muscular arteries. When the umbilical cord is initially compressed, the umbilical vein is thus occluded first. This causes a decrease in venous blood returning to the fetal heart and thus a decrease in preload, which in turn triggers tachycardia. This is why variable decelerations are often preceded and followed by small increases in FHR, referred to colloquially as “shoulders.” As increasing compressive force is applied to the umbilical cord, the muscular arteries are eventually compressed as well. This then leads to a significant increase in vascular resistance, which in turn generates bradycardia via vagal nerve stimulation via baroreceptors. Although variable decelerations can
Sometimes occur normally during the labor process or even antenatal testing, their presence should alert the practitioner to the potential presence of umbilical cord compression, causes for which could include low amniotic fluid (oligohydramnios) or prolapse of the umbilical cord through the cervix. Overall, variable decelerations represent anticipated physiologic reflexes to umbilical cord compression and not the presence of hypoxemia or acidemia per se. However, severe and repetitive compression will eventually compromise oxygenation and overall health, and thus interventions (which can be as simple as maternal positional changes) would be warranted in this circumstance. Additionally, some fetuses can develop hypoxemia during periods of umbilical cord compression, which then normalizes after the compression is released. This can present as a period of tachycardia that follows resolution of the variable deceleration owing to a sympathetic response to the hypoxemia. These are referred to as “overshoots” (see Figure 13-10).

Late decelerations, by contrast, have a more gradual onset and return to baseline—typically 30 seconds or more from onset to nadir. The onset, nadir, and recovery of the deceleration occur after the onset, peak, and end of the contraction (Figure 13-11). During a uterine contraction, placental perfusion is temporarily impaired secondary to myometrial compression of the spiral arteries, which lose their muscularis in early pregnancy and are thus compressible. In a fetus that is undergoing the labor process normally, however, this transient event is well tolerated without clinically meaningful hypoxemia. For fetuses that are experiencing a decreasing oxygen reserve, however, the perfusion reduction during a contraction can have more significant effects, albeit not always tissue acidosis or multiorgan dysfunction. These effects and their resolution will always be delayed relative to the contraction itself, because the impact of decreased perfusion will be progressive and then require time to resolve once the contraction is complete. This is why “late” decelerations have their characteristic appearance relative to uterine contractions.

The actual mechanism of late decelerations occurs secondary to two separate although interrelated processes, one of which is related to hypoxemia and the other to tissue-level hypoxia. Although hypoxemia generates a sympathetic response that leads to tachycardia and thus an increasing FHR baseline, it can also generate bradycardia through a more indirect pathway: the decrease in peripheral oxygen concentration triggers a sympathetic output, which in turn transiently increases the blood pressure, which in turn triggers baroreceptors, which in turn stimulate the vagal nerve, which then decreases the heart rate. Thus the presence of late decelerations can signify transient hypoxemia during and resolving after uterine contractions. In this situation it would be optimal to resolve the transient hypoxemia, and interventional measures, such as positional changes and supplemental oxygenation, are usually undertaken. The fetus in this scenario, however, is not necessarily acidotic (or even has tissue-level hypoxia) and, presuming it can recover appropriately between contractions, can still possibly proceed with a normal labor course and uncomplicated vaginal delivery. The other potential mechanism for late decelerations, however, involves direct suppression of myocardial activity secondary to tissue-level changes in which the bradycardia reflects the inability of the myocardium to function properly in the setting of hypoxia. Secondary to differences between respiratory and metabolic acidosis and all of the other complex variables involved in organ function, many of the fetuses in this scenario will still have normal long-term outcomes, although this is the scenario of greatest clinical concern and the one in which consideration could be given to expedition through operative delivery. For the patient experiencing late decelerations, however, it can be difficult to determine if they are occurring secondary to which of these two underlying pathways. Context is important in this regard such that in the case of a persistently hypoxic fetus, other FHR changes, such as progressive tachycardia and loss of variability, should accompany the late decelerations. The fetus who is experiencing late decelerations but for whom the FHR has a normal rate and variability between contractions is more likely to be experiencing only transient hypoxemia.

A sinusoidal heart rate pattern consists of a regular oscillation of the baseline variability, in a smooth undulating pattern. This pattern typically lasts at least 10 minutes, has a relatively fixed period of three to five cycles per minute, and an amplitude of 5 to 15 beats/min above and below the baseline (Figure 13-12). This pattern is quite rare, but can be associated with severe chronic anemia or severe hypoxia and acidosis, although rarely can also be an incidental finding. 

Figure 13-11 Late decelerations. Note the timing of the onset, nadir, and recovery of the deceleration, which occur after the onset, peak, and end of the contraction. (From Freeman R, Garite T. Fetal heart rate monitoring. Baltimore: Williams & Wilkins; 1981:201.)
any tracing that does not meet the more specified criteria for category I or III. Except for patterns that are sinusoidal, category III tracings require the combination of absent variability plus one more additional finding as described below. Thus a tracing that has appropriate variability but has other findings, such as late or variable decelerations that would exclude it from category I, would fall into category II.

**Category III**

These are abnormal tracings that are potentially predictive of an abnormal fetal acid-base status at the time of observation. As such, they require prompt evaluation and initiation of attempts at correction. These tracings include either:

1. Absent baseline FHR variability along with any of the following:
   a. Recurrent late decelerations
   b. Recurrent variable decelerations
   c. Bradycardia
2. Sinusoidal pattern

**MANAGEMENT OF NON–CATEGORY I FHR PATTERNS DURING LABOR**

The evaluation of a category II or III pattern begins with a search for an underlying etiology that would itself require immediate delivery. For example, an acute change in the FHR pattern could have occurred secondary to umbilical cord prolapse (Figure 13-13), placental abruption, or uterine rupture. In the absence of such events, one should search for a remediable cause of the concerning FHR tracing. Uterine perfusion is very sensitive to maternal blood pressure, and even relative redistributions of maternal blood flow, such as can occur after the initiation of regional anesthesia, can impact the FHR pattern. Abnormal FHR patterns may also result from contractions that are too frequent in timing to allow for recovery between them. The occurrence of uterine contractions more frequently than 5 in 10 minutes is generally referred to as tachysystole (Figure 13-14) and can occur as a consequence of labor induction agents. When the tachysystole is resolved, either by discontinuing or decreasing an oxytocin infusion or administering a tocolytic, the fetal...
status typically improves. Maternal position in labor can affect the FHR tracing because the supine position decreases uterine blood flow and placental perfusion. Repositioning a patient to the lateral recumbent position can improve a concerning FHR pattern with no other intervention. Supplemental oxygen therapy for the mother should also be administered.

When recurrent variable decelerations are present, amnioinfusion, in which fluid is infused into the uterine cavity, has been shown to decrease the rate of variable decelerations and cesarean delivery for nonreassuring fetal status.

When faced with a concerning FHR tracing with no clear secondary cause and which persists despite attempts at conservative management, some options do exist for additional reassurance, because many of these fetuses will still have a normal acid-base status secondary to the imprecision of continuous FHR monitoring. An acceleration in FHR after vibroacoustic stimulation or fetal scalp stimulation with a digital examination provides reliable reassurance of a normal fetal pH and allows labor to continue.

Blood sampling from the fetal scalp can be used to assess the fetal pH or lactate directly, although this is invasive and requires technical expertise, ruptured membranes, and adequate access to the fetal skin. Of note, fetal breathing motions are not generally present during active labor, and thus there is only a limited role in BPPs for additional reassurance in patients who are in active labor.

Because of these limitations, newer approaches are needed to identify correctly those fetuses with concerning FHR patterns that are actually at risk of neurologic injury and could benefit from intervention. Two of the best-studied approaches have been fetal pulse oximetry and ST segment analysis. Unfortunately, a multicenter randomized trial of fetal pulse oximetry, in addition to standard continuous FHR monitoring, showed no significant difference in cesarean delivery rates or neonatal outcomes. One of the interesting aspects of this study was that fetal pulse oximetry did result in a reduction in the number of women undergoing cesarean sections for the indication of “nonreassuring fetal status,” although this was balanced by an increase in the number of patients undergoing cesarean delivery for failure to progress. One possible explanation for this finding is that some abnormal FHR patterns in which the fetus is not actually hypoxic may be associated with labor progression abnormalities. A recent meta-analysis of randomized controlled trials of ST segment analysis during labor demonstrated a reduction in operative delivery compared with conventional monitoring, holding significant promise for this technology. ST segment evaluation requires the use of a fetal scalp electrode, and thus may have some of the limitations of internal fetal monitoring, including the need for ruptured fetal membranes and the absence of maternal infectious diseases that could be transmitted by fetal skin puncture. A multicenter trial of ST analysis by the NICHD is ongoing.

**Summary**

In high-risk populations at increased risk of perinatal mortality, antenatal fetal surveillance plays a large role in prenatal care. Pregnancies at risk for progressive deterioration of placental function leading to fetal hypoxemia

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**Figure 13-13** Prolonged deceleration associated with acute prolapse of the umbilical cord. Note the reassuring fetal heart rate tracing leading up to the deceleration, indicating an acute event. (From Freeman R, Garite T. Fetal heart rate monitoring. Baltimore: Williams & Wilkins; 1981:83.)

**Figure 13-14** Prolonged deceleration associated with uterine hyperstimulation. Note the recovery of the deceleration after discontinuation of the oxytocin infusion. (From Freeman R, Garite T. Fetal heart rate monitoring. Baltimore: Williams & Wilkins; 1981:85.)
and acidosis are most likely to benefit from the methods currently in use. The various modalities, including NST, CST, biophysical profile, and Doppler velocimetry, rely on fetal biophysical parameters that are significantly associated with the presence or absence of fetal hypoxemia. Because all tests are associated with a false-positive rate, each test result should be interpreted within the clinical context presented by the patient.

Despite the inherent stress of labor, most fetuses are able to tolerate the transient episodes of hypoxemia without harm. Rarely the process of labor and delivery places a fetus in jeopardy of long-term neurologic damage or death as a result of profound hypoxemia and metabolic acidosis. Since the 1970s, electronic FHR monitoring has emerged as the most common technology to monitor fetuses during labor, in the hopes of identifying at-risk fetuses to effect delivery before permanent harm is done. Although not without benefit, monitoring has also likely resulted in a dramatic increase in cesarean deliveries without reducing the rate of cerebral palsy. It is hoped that future research and technology development will refine and improve this technology and result in benefit to mothers and infants.

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