and the fetus. Some therapies, however, may be detrimental to temperature, and electrolyte balance benefit both the mother of the fetus. Optimal maternal oxygenation, blood pressure, of such therapies on the fetus must be considered. Many treatment (2).

oxygen can be given to the mother while awaiting fetal evalu-
the woman is visibly pregnant), maternal evaluation should not occur. If the gestational age is 23 weeks or more (or if 
ing fetal oxygenation or effecting delivery if improvement does 
indirect assessments of the fetal condition (discussed later in 
sician to potential hypoxia and/or fetal compromise. These 
assessment and monitoring is determination of the adequacy 
activity is sufficient. At later gestational ages, the goal of fetal 
are appropriate to maximize treatment to the woman without 
place the fetus at unnecessary risk.

Finally, fetal assessment is dependent on gestational age. At early gestational ages, simply documenting fetal cardiac activity is sufficient. At later gestational ages, the goal of fetal assessment and monitoring is determination of the adequacy of fetal oxygenation, and, when necessary, alerting the physi-

more likely at later gestational ages. Even prior to the 
contact with maternal blood and 
flow temporarily stops. However, the 
flow into and across the intervillous space. During uterine con-
vascular resistance of the maternal arteries governs the rate of 
exist and ensures adequate circulation; the maternal arteriolar pressure exceeds the pressure in the intervillous space, which 
pressure exceeds the pressure in the maternal veins. The placenta itself is a low-resistance organ, and, accordingly, the pressure differen-
tional across the intervillous space is small. Therefore, the 
the vascular resistance of the maternal arteries governs the rate of 
flow into and across the intervillous space. During uterine con-
tracts, the intrauterine pressure exceeds the pressure in the 
temorary stops. However, the 
ivillous space becomes somewhat dilated during a contraction, allowing for continued contact with maternal blood and 
exchange, although with reduced efficiency.

The placenta has a high rate of oxygen consumption. It 
serves as the main organ of gas and nutrient exchange for the 
(5). However, oxygen extracted at the fetal–maternal interface serves not only the fetus, but the placenta as well. 
This highly metabolic organ uses as much, and possibly more, of the total oxygen and nutrients as the fetus in order to 
maintain its own growth and metabolism (6).

**Fetal–Placental Respiration**

Fetal oxygen consumption remains constant over a wide range of changes in oxygen delivery and will decrease only when extraction is maximal and delivery is further reduced (7). A 50% reduction in uterine blood flow is compensated by an increase in umbilical blood flow and an increase in oxygen extraction to maintain oxygen delivery (8). This compensatory mechanism remains adequate only with short-term reductions in uterine blood flow. A critical point exists below
which oxygen uptake becomes dependent on oxygen delivery (9). Long-term reductions result in decreased consumption secondary to the decrease in delivery. Below this threshold, tissue hypoxia occurs, there is an inability to maintain oxidative metabolism, and fetal acidemia results (10). A chronic decrease in oxygen consumption also will lead to decreases in both fetal growth and fetal activity in an effort to conserve oxygen for cellular homeostasis (11).

Oxygenated blood is carried to the fetus via the umbilical vein, while deoxygenated blood is carried back to the placenta via the two umbilical arteries. The human fetal umbilical venous PO$_2$ is low compared to postnatal standards—around 30 mmHg. Despite this low PO$_2$, adequate amounts of oxygen can be delivered to the fetal tissues. This delivery is facilitated by the high fetal cardiac output, relative to body size, and the affinity of fetal hemoglobin for oxygen (12). Fetal hemoglobin’s high affinity for oxygen ensures that virtually all of the fetal hemoglobin is maximally saturated, even at the low PO$_2$ of the fetal umbilical venous blood. This affinity can be altered by factors such as acidosis and temperature. An increase in pH or a decrease in temperature causes a shift of the hemoglobin oxygen dissociation curve to the left, indicating a higher oxygen affinity.

At baseline, a healthy fetus has twice as much placental respiratory function as is needed to maintain its normal oxygen consumption (13). However, if placental function, even temporarily, deteriorates beyond that threshold, the fetus has very few adaptations available to deal with acute hypoxia. There are no short-term homeostatic mechanisms to alter placental respiratory gas exchange (13–17). The fetus is also unable to further increase its combined ventricular output from its high baseline level. However, the fetus does respond to hypoxia by preferentially redistributing blood to the brain, adrenal glands, and heart, and decreasing blood flow to other organs in an attempt to limit the adverse effects of hypoxia to the most vital organs.

Once the reduction in O$_2$ content in the peripheral tissues reaches a level where anaerobic metabolism is required, the fetus quickly becomes acidic, because it has difficulty metabolizing lactic acid. Accumulation of lactic acid decreases the oxygen tension in the umbilical vein further by shifting the fetal oxyhemoglobin dissociation curve to the right and therefore decreasing hemoglobin saturation and total oxygen uptake in the placenta. Hypoxemia results in decreases in fetal breathing movements, rapid eye movements, general muscle tone and activity, and baseline heart rate (18). These changes minimize the fetus’s consumption of oxygen and allow a greater proportion of the cardiac output to be used for maintaining the oxygen supply to the brain (19). Resumption of these activities will occur after several hours, even in the presence of continued hypoxia, as the fetus begins to adapt to a chronic hypoxic condition (20). Therefore, fetal heart rate monitoring or ultrasound observation of the fetus is limited in its ability to predict poor outcome in cases when significant hypoxic episodes occurred prior to the monitoring period. For example, the fetal heart rate has been noted to return to baseline 12 to 16 hours after a hypoxic event that was not associated with prolonged acidosis (21).

Maternal hypercarbia can also contribute to fetal acidosis. Usually transfer of carbon dioxide from the fetal to maternal circulation occurs readily, because the placenta is highly permeable to carbon dioxide. In fact, CO$_2$ transfer across the chorionic villi is accomplished faster than the transfer of oxygen. Also, favoring the transfer of carbon dioxide is the higher affinity that maternal blood has for carbon dioxide compared to fetal blood. Finally, the mild respiratory alkalosis that is normally present in pregnant women results in a lower PCO$_2$, further enhancing the transfer of carbon dioxide from the fetus to the maternal blood. However, if maternal PCO$_2$ is abnormally elevated, fetal transfer is hindered and will result in elevations of fetal PCO$_2$ and fetal acidosis (22).

**DIAGNOSIS**

Regulation of the fetal heart rate is governed by a complex interplay of the sympathetic and parasympathetic nervous systems (23). The sympathetic nervous system exerts influence through the release of norepinephrine, which accelerates the heart rate and increases inotropy; the parasympathetic nervous system decreases the heart rate. Fetal heart rate variability results from the constant “push–pull” of these two systems. Gestational age has some effect on the fetal heart rate, with a general decrease in the baseline heart rate occurring with advancing gestation.

Electronic fetal monitoring, introduced in the 1960s, has become ubiquitous in labor and delivery units in developed countries. This type of fetal monitoring typically is used at any gestational age at which ex utero survival is possible, that is when cesarean delivery would be considered for the indication of an abnormal fetal heart rate tracing. Electronic fetal monitoring requires very little in preparation or maintenance but does require an experienced interpreter. This technique results in a continuous tracing of the fetal heart rate, coupled with a tracing of uterine activity. Monitoring may be used during labor or may be used as a way to evaluate for fetal well-being during the antepartum period. The goal for electronic fetal monitoring is twofold: to avoid stillbirth, and to avoid neonatal encephalopathy and cerebral palsy. The efficacy of the currently available techniques is controversial and further research is needed in this area. Continuous fetal monitoring during labor does decrease the likelihood of stillbirth but has not impacted the rate of neonatal encephalopathy or cerebral palsy (24–26).

**Antenatal Fetal Testing**

Given the limited evidence of efficacy, antenatal testing is only recommended in high-risk patients. Conditions that might indicate antenatal fetal testing include but are not limited to diabetes, hypertension, fetal growth restriction, multiple gestations, oligohydramnios, prior stillbirths, preterm premature rupture of membranes, and decreased fetal movement.

**Nonstress Test**

The most commonly used antenatal test is the nonstress test (NST), which utilizes the same cardiotocography technology as continuous fetal monitoring during labor. An NST involves fetal heart rate monitoring for a period of 20 to 40 minutes. The underlying premise for this test relates to the fact that a nonacidotic, neurologically intact fetus will have fetal heart rate accelerations in response to fetal movement. The NST is
described as reactive or nonreactive. The presence within a 20-minute window of two fetal heart rate accelerations lasting at least 15 seconds, that peak at least 15 beats per minute (bpm) above the baseline, characterizes a “reactive” NST (Fig. 81.1). A reactive test is highly reassuring, with a false-negative (i.e., fetal death within the next week) rate of 1.9 per 1,000 (27). However, the normal fetus periodically has episodes of decreased heart rate variability and no accelerations for 30 to 40 minutes due to sleep, and this is the most common reason for a “nonreactive” test result. A nonreactive NST often only requires further testing. Due to immaturity of the sympathetic and parasympathetic nervous systems, fetuses at less than 32 weeks gestational age may not have a reactive NST despite the absence of compromise (28).

Fetal heart rate decelerations can also be appreciated on an NST or during continuous fetal monitoring during labor. Decelerations occur when the fetal heart rate falls below the baseline heart rate; they are classified according to their appearance and location in relation to uterine contractions. Different types of decelerations are caused by different mechanisms. Therefore, each type of deceleration has different implications for fetal status.

- **Early decelerations**: Early decelerations begin at the onset of uterine contractions and appear to mirror the contraction (Fig. 81.2) (29). They are believed to be caused by pressure on the fetal head. This pressure results in an alteration in cerebral blood flow and stimulation of the vagal center, causing parasympathetic stimulation and a subsequent decrease in the fetal heart rate. Early decelerations are thought to be benign and generally are not associated with fetal hypoxia, acidosis, or low Apgar scores.

- **Variable decelerations**: Variable decelerations are abrupt decreases in the fetal heart rate at least 15 bpm below baseline with the onset to nadir lasting less than 30 seconds. Variable decelerations do not necessarily correlate with contractions (Fig. 81.3) (29). They are thought to be caused by intermittent umbilical cord compression. “Shoulders” can be seen both preceding and following these variable decelerations, and should not be considered accelerations, as they are a manifestation of the increase in sympathetic nervous system stimulation during fetal heart rate decelerations. Mild or isolated variable decelerations are benign. Repetitive moderate or severe variable decelerations may indicate fetal compromise.

- **Late decelerations**: Late decelerations occur late in relation to the uterine contraction. Their onset begins after the contraction begins, and they resolve after the resolution of the contraction (29) (Fig. 81.4). These decelerations occur as a result of decreased uteroplacental oxygen delivery to the fetus, but they may not necessarily signify poor placental function—late decelerations may be caused by maternal hypotension or decreased uterine blood flow. Persistence of late decelerations, especially in the absence of baseline fetal heart rate variability, is an ominous sign of fetal compromise.

- **Prolonged decelerations**: A prolonged deceleration is any deceleration at least 15 bpm below the baseline that lasts for 2 to 10 minutes (29). Repetitive prolonged decelerations are cause for concern for fetal compromise. Any deceleration lasting longer than 10 minutes is considered a change in baseline if the new rate is greater than 110 bpm, or bradycardia if the new heart rate is less than 110 bpm.

The last aspect of the fetal heart rate tracing analyzed on an NST is heart rate variability, or the fluctuations in the fetal heart rate seen outside of accelerations or decelerations (29). Variability is described as absent (no fluctuation), minimal (<5 bpm), moderate (5 to 15 bpm), or marked (>25 bpm) (29). In addition to causing the NST to be nonreactive, fetal sleep can be a reason for decreased variability, but persistently minimal or absent fetal heart rate variability is the most significant sign of fetal compromise (30). When evaluating all of these aspects of an NST, a normal result is a reactive NST with a
baseline between 110 and 160 bpm, no decelerations, and moderate variability.

The other (bottom panel) line on the NST represents the tocodynamometer, a strain gauge placed on the maternal abdomen used to measure uterine contractions. Though frequency and duration of contractions can be assessed with a tocodynamometer, the strength of a contraction cannot be assessed without an intrauterine pressure catheter (IUPC), placement of which requires ruptured amniotic membranes.

**Contraction Stress Test**

The contraction stress test (CST) is cardiotocography evaluated during spontaneous or induced contractions. It may be used as a follow-up to a nonreactive NST. The test requires at least three contractions during a 10-minute window and evaluates the fetal heart rate response to these contractions. Because of the necessity of uterine contractions, this test is contraindicated in various situations, including significantly
preterm gestations and those in whom labor is contraindi
cated. The underlying premise for this test involves the idea
that fetal oxygenation will transiently worsen in the presence
of uterine contractions. In the already-compromised fetus,
this will result in late decelerations. The CST is interpreted
based on the presence or absence of late decelerations. A posi-
tive CST is one in which late decelerations occur with at least
50% of contractions and generally indicates that delivery is
warranted. A negative test result (with no late decelerations)
is highly reassuring, with a false-negative rate of only 0.3 in
1,000 (27) (Fig. 81.5). In practice, CST is infrequently utilized
today, because the test is resource intensive and has more fre-
cent contraindications than other forms of antenatal testing.

**Biophysical Profile**

The biophysical profile (BPP) consists of an NST and an
ultrasound examination. This test can be performed as a

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**FIGURE 81.4** Late decelerations occur late in relation to the contraction and may be a sign of fetal compromise.

**FIGURE 81.5** Negative contraction stress test. This contraction stress test is negative, indicating good fetal reserve.
follow-up test to a nonreactive NST or can be used as a primary form of surveillance. Ultrasound is used to evaluate fetal tone, gross body movements, breathing movements, and amount of amniotic fluid present. The score is derived from these various assessments (Table 81.1). The false-negative rate of a BPP of 8/10 or 10/10 is 0.8 per 1,000 (27). However, as with all antenatal testing, many factors may alter the results of the BPP including maternal sedation, drug use, or hypoglycemia.

**Doppler**

As an adjunct to antenatal testing with NST, CST, or BPP, Doppler ultrasound is used to address specific concerns in fetuses at high risk for stillbirth. In growth-restricted fetuses, Doppler evaluation of flow in the umbilical artery is utilized. Diminished diastolic flow signifies placental insufficiency and increased resistance to flow in the placenta. Absent end-diastolic flow indicates the need for delivery if the gestational age is 34 weeks or more. Reversed end-diastolic flow signifies even more critical resistance to flow and is an indication for delivery even earlier (31).

The other clinical situation in which Doppler is routinely used is for evaluation of fetuses at high risk for anemia. In this situation, elevated peak systolic velocity in the middle cerebral artery correlates with fetal anemia, and assessment of middle cerebral artery Doppler is used to help determine the need for percutaneous umbilical blood sampling. Umbilical blood sampling directly assesses fetal hematocrit and, if anemia is confirmed, intrauterine fetal blood transfusion can be done (32,33). Current recommendations are to plan for fetal blood sampling and possible transfusion when the peak systolic velocity is more than 1.55 multiples of the median for the given gestational age (34). Amnioncentesis, to assess indirectly the bilirubin concentration in amniotic fluid, previously was the mainstay for monitoring fetuses at risk for anemia; middle cerebral artery Doppler interrogation has almost completely replaced amnioncentesis for that indication.

**Intrapartum Fetal Surveillance**

Intrapartum fetal surveillance is cardiotocography during labor. The same parameters evaluated on an NST are used in an ongoing fashion for intrapartum fetal surveillance. However, fetal heart rate tracings in the intrapartum period are described according to three categories (35):

- **Category I:** Baseline fetal heart rate 110 to 160 bpm; moderate fetal heart rate variability accelerations may be present or absent; no late or variable decelerations; may have early decelerations
- **Category II:** All fetal heart rate patterns that are not category I or category III
- **Category III:** Absent variability with recurrent late decelerations, absent variability with recurrent variable decelerations, or bradycardia

These categories were developed to help determine the need for treatment. However, because most tracings are category II, utility of this classification system is limited, and some authors have advocated further stratification of category II (36,37). Consideration should also be given to the tocodynamometer (or IUPC tracing) during labor. Tachysystole is the term for more than five contractions in 10 minutes, averaged over 30 minutes. Tachysystole may occur as a result of oxytocin administered to stimulate contractions during labor inductions or augmentation of spontaneous labor. If tachysystole occurs spontaneously, it may signify placental abruption.

**TREATMENT**

**Abnormal Antenatal Testing**

All tests used for antenatal testing (NST, CST, BPP) have low positive predictive values and high negative predictive values. That is, they all have a low rate of false-negative results but a high rate of false-positive results. Therefore, if antenatal testing is concerning, repeat testing is appropriate. Generally, an alternative test is used as the method of repeat testing. For example, if the NST was nonreactive, a BPP would be performed (Table 81.2). If the second test is reassuring, the likelihood of fetal acidemia is low, and repeat testing would be scheduled within the next few days to a week. However, if repeat testing also is nonreassuring, the approach generally would be to proceed with delivery. The mode of delivery would be determined by the degree of concern caused by antenatal testing results and obstetric factors. However, in some cases, fetal testing will be abnormal due to maternal disease, and these fetal testing abnormalities may resolve with maternal stabilization. Some specific examples of maternal situations where special consideration is required in interpreting fetal testing are addressed below:

- **Maternal fever:** Maternal fever, regardless of cause, generally causes fetal tachycardia. In this situation, fetal tachycardia may not be an indication for delivery but rather treatment of maternal infection and ongoing fetal monitoring.
- **Sickle cell crisis:** Sickle cell crisis can cause fetal hypoxia due to the decreased maternal oxygen–carrying capacity, and maternal treatment with oxygen, intravenous fluid resuscitation and blood transfusion will frequently improve fetal testing.
- **Maternal hypoxia:** Once maternal hypoxia is corrected, the fetus will frequently resuscitate in utero with return to reassuring fetal testing over an hour or two.

**TABLE 81.1 Components and Scoring of the Biophysical Profile**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Score = 2</th>
<th>Score = 0</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonstress test</td>
<td>Reactive</td>
<td>Nonreactive</td>
</tr>
<tr>
<td>Fetal breathing</td>
<td>≥1 episode of fetal breathing for ≥30 s</td>
<td>&lt;30 s of breathing</td>
</tr>
<tr>
<td>Fetal tone</td>
<td>≥1 episode of active extension and flexion</td>
<td>No flexion/extension</td>
</tr>
<tr>
<td>Gross movement</td>
<td>≥3 movements</td>
<td>≤3 movements</td>
</tr>
<tr>
<td>Amniotic fluid</td>
<td>Single pocket ≥2 cm</td>
<td>Largest pocket &lt;2 cm</td>
</tr>
</tbody>
</table>

*Score is 2 or 0 for each parameter. The maximum duration of the test is 30 minutes.

**TABLE 81.2 Biophysical Score and Recommended Interventions**

<table>
<thead>
<tr>
<th>BPP Score</th>
<th>Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>8/10 or 10/10</td>
<td>Normal; continue care as indicated</td>
</tr>
<tr>
<td>6/10 or 8/10 with low amniotic fluid</td>
<td>Repeat within 24 hr</td>
</tr>
<tr>
<td>0/10, 2/10, 4/10</td>
<td>Delivery is usually indicated</td>
</tr>
</tbody>
</table>
• Maternal hypotension: Maternal hypotension will frequently cause uteroplacental insufficiency and late decelerations on the fetal heart rate tracing. However, upon resolution of maternal hypotension, the fetal tracing usually improves.

• Maternal acidosis without hypoxemia: Metabolic acidosis (seen in diabetic ketoacidosis and some drug toxicities) will take longer than maternal hypoxia to affect the fetus. However, in utero fetal resuscitation after maternal stabilization will take longer as well.

• Seizures: During a grand mal seizure, the pregnant woman becomes hypoxic. A resultant fetal bradycardia most often occurs. In cases of short-duration seizures such as is seen in patients with eclampsia, this bradycardia will usually resolve when maternal respiration resumes and should not be an indication for emergent delivery. Maternal stabilization should be achieved prior to consideration of fetal delivery.

• Cardiac arrest: The presence of an enlarged uterus, especially in the third trimester, compromises maternal cardiopulmonary resuscitation. Therefore, left lateral displacement of the uterus is essential from the beginning of any cardiopulmonary resuscitation of a pregnant woman in the second half of pregnancy. In addition, perimortem cesarean delivery should be begun by 4 minutes into an unsuccessful resuscitation or the likelihood that the infant will be alive and neurologically intact will be low (38).

Abnormal Intrapartum Testing

A category I tracing is considered normal, and no treatment is required. A category III tracing is considered to represent a high probability of abnormal fetal acid-base status, and efforts should be made to expediently resolve the underlying cause or deliver the fetus. A category II tracing requires continued surveillance, reevaluation, and actions to try to resolve the underlying cause (29).

The response to category II or category III tracings initially should consist of maneuvers aimed at “intrauterine resuscitation.” These maneuvers include turning the pregnant woman to left lateral position, providing supplemental oxygen, stopping any exogenous augmentation of uterine contractions, and increasing the rate of intravenous fluid delivery. The left lateral (and also right lateral) position decreases the pressure exerted on the IVC by the gravid uterus and allows for improved maternal venous return (1). Minimizing contractions allows for more continuous flow of oxygenated maternal blood to the intervillous space. The administration of supplemental oxygen has no effect on uterine or umbilical blood flow but may increase fetal venous PO2.

CONTROVERSIES

As discussed previously, though the use of electronic fetal monitoring has been associated with a decrease in intrapartum stillbirth rates, hopes that it would decrease the likelihood of cerebral palsy have not been realized. Furthermore, the low positive predictive value of intrapartum monitoring is thought to have contributed significantly to an increase in the cesarean delivery rate in the United States from 5% in 1970 to 32% today (39). Likewise, the low positive predictive value of tests used for antenatal fetal testing can lead to iatrogenic preterm delivery, an argument for testing only high-risk patients.

The currently advocated intrapartum fetal heart rate categories were created because intrainterpreter and interinterpreter variations in fetal heart rate tracing interpretation are high, and therefore, responses to the same fetal heart rate tracing varied widely (29). The current three-tier category system was an effort to more objectively stratify tracings. However, there are continued concerns that the current categories do not sufficiently stratify patients, and we agree with others that it is likely that subcategories of category II tracings will emerge (40). Such technologies as fetal pulse oximetry and analysis of the ST segment of the fetal electrocardiogram have been evaluated (41–43). However, these technologies have not been shown to improve on current monitoring.

Key Points

• Whether or not the gestational age is compatible with the possibility of extraterine survival significantly affects how to care for a critically ill pregnant woman;

• Diagnostic testing and treatment should not be withheld due to concerns about fetal effects. Consultation with an obstetrician or maternal–fetal medicine specialist can facilitate tailoring treatment for pregnant women;

• Physiologic changes occur in pregnancy that maximize oxygen delivery to the fetus and carbon dioxide transfer back to the maternal circulation;

• Antenatal fetal testing is indicated to evaluate a potentially viable fetus when there is concern for fetal hypoxia or death;

• The negative predictive value of all tests used for fetal surveillance is high, but the positive predictive value of all tests of fetal well-being is low.

References


41. Saade G. I: Fetal ECG analysis of the ST segment as an adjunct to intrapartum fetal heart rate monitoring. JOGN. 2010;17:1664–1665.

