CHAPTER 16

Intrapartum Fetal Evaluation

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KEY TERMS

Acidemia	Increased hydrogen ion concentration in blood
Acidosis	Increased hydrogen ion concentration in tissue
Asphyxia	Hypoxia with metabolic acidosis
Base deficit	Buffer base content below normal (this is calculated from a normogram using pH and Pco_2)
Base excess	Buffer base content above normal
Hypoxemia	Decreased oxygen concentration in blood
Hypoxia	Decreased oxygen concentration in tissue
pH	The negative log of hydrogen ion concentration ($7.0 = 1 \times 10^{-7}$)

KEY ABBREVIATIONS

American Congress of Obstetricians and Gynecologists	ACOG
Association of Women's Health, Obstetric and Neonatal Nurses	AWHONN
Cardiotocography	CTG
Central Nervous System	CNS
Electrocardiogram	ECG
Electronic Fetal Heart Rate Monitoring	EFM
Federal Drug Administration	FDA
Fetal Heart Rate	FHR
Human Immunodeficiency Virus	HIV
Magnesium Sulfate	$MgSO_4$
Non-reassuring Fetal Status	NRFS
National Institute of Child Health and Human Development	NICHD
Premature Rupture of the Membranes	PROM

The question being asked by the clinician evaluating the fetus in labor is quite simple: What is the status of fetal oxygenation? If hypoxia is severe enough and lasts long enough, fetal tissue and organ damage will result, which may result in long-term injuries or death. Hypoxia severe enough to cause tissue damage virtually always occurs only in the face of a significant metabolic acidosis, and the term *asphyxia* is used in this situation (Figure 16-1). To clarify the terminology used in these situations, see the Key Terms.

Although there are other, less frequent causes of fetal injury and death in labor (e.g., infection, hemorrhage), hypoxia is by far the most common etiology and the one for which medical and surgical interventions have the potential for preventing injury and death. Before intensive intrapartum fetal heart rate (FHR) monitoring, relatively uniform intrapartum fetal death rates of 3 to 4 per 1000 were reported.[1] Thus, on an obstetrical service of 200 to 300 monthly deliveries, 1 intrapartum death would occur each month; but now such events are extremely rare in monitored fetuses. Fetal hypoxia that is severe and associated with metabolic acidosis, but not sufficient to result in death, may alternatively cause asphyxial injury to the fetus and newborn. The fetal central nervous system (CNS) is the organ system most vulnerable to long-term injury. **However, the fetus destined to have permanent neurologic damage will virtually always have multiorgan dysfunction**

in the newborn period. Usually, complications such as seizures, respiratory distress, pulmonary hypertension with persistent fetal circulation, renal failure, bowel dysfunction, and pulmonary hemorrhage are seen in the baby who will ultimately have permanent neurologic injury.[2] Babies who recover from these complications and survive may be normal or may develop cerebral palsy. Cerebral palsy is defined as a movement disorder, usually spastic in nature, that is present at birth, nonprogressive and often, but not always, associated with varying degrees of mental retardation.[3] Seizures are often seen in children with cerebral palsy. However, mental retardation or seizures, in the absence of spasticity, are rarely the result of peripartum asphyxia. It is still unclear whether other neurologic dysfunction in children, such as learning and behavioral disorders, can be the result of perinatal asphyxia. Cerebral palsy will develop in 0.5% of all births and is prevalent in about 0.1% of all school-aged children.[3,4] Prematurity remains the leading cause of cerebral palsy. It is estimated that peripartum events contribute to no more than 25% of the overall rate of this disease.[5]

Thus, the goal of intrapartum monitoring is to detect hypoxia in labor and allow the clinician to implement nonoperative interventions such as positioning and oxygen (O_2) administration to correct or ameliorate the oxygen deficiency. If this is unsuccessful, the monitor should help the clinician to determine the severity and duration of the hypoxia and whether there is a metabolic acidosis. And finally, if there is sufficient hypoxia and metabolic acidosis is present or developing, the monitor should give adequate warning and time to permit the clinician to deliver the baby expeditiously, whether by operative vaginal or cesarean delivery, to prevent damage or death from occurring. Unfortunately, the fetus is quite inaccessible, and until recently we have had crude and limited tools available to determine all the above information necessary to make correct and timely decisions to accomplish these goals.

HISTORY OF FETAL MONITORING

Because of the inaccessible location of the fetus, evaluating fetal well-being, or more specifically, fetal oxygen status, has been an ongoing and difficult challenge. In the 1600s, Kilian first proposed that the FHR might be used to diagnose fetal distress and to indicate when the clinician should intervene on behalf of the fetus. The sound of the fetal heart had first been detected by Marsac of France in the 1600s and described in a poem by his colleague, Phillipe LeGaust. This observation went unnoticed until 1818, when Mayor, and subsequently Kergaradec, described the fetal heart sounds by placing an ear on the maternal abdomen. Kergaradec suggested that auscultation of the fetal heart could be used to determine fetal viability and fetal lie. In 1893, Von Winckel described the criteria for fetal distress that were to remain essentially unchanged until the arrival of electronic FHR monitoring. These included tachycardia (FHR >160 beats/minute), bradycardia (FHR <100 beats/minute), irregular heart rate, passage of meconium, and gross alteration of fetal movement.[1]

These criteria went unquestioned until 1968, when Benson and colleagues published the results of the Collaborative Project.[6] These authors reviewed the benefits of auscultation in more than 24,000 deliveries and concluded, "there was no reliable indicator of fetal distress in terms of FHR save in extreme degree." Thus, it became apparent that other, more sophisticated means of intrapartum fetal evaluation were required. In 1906, Cremer described the use of the fetal electrocardiogram (ECG) using abdominal and intravaginal electrical leads.[7] Several investigators made attempts using ECG waveforms to detect fetal hypoxia, but ultimately concluded that there was no consistent fetal electrocardiographic changes with fetal distress.[8] The subsequent history of electronic FHR monitoring (EFM) is a story of technologic development and empirical observations of alterations in FHR associated with various causes of fetal hypoxia and acidosis.

In 1958, Edward Hon (the "father of EFM" in the United States) reported on the instantaneous recording of the fetal ECG from the maternal abdomen.[9] He and his colleagues manually measured R-R intervals from a continuous ECG tracing and mathematically converted these to rate, in beats per minute, and then hand-recorded each interval on graph paper. From these efforts Hon, Caldeyro-Barcia in Uruguay, and Hammacher in Germany began to describe various FHR patterns associated with fetal distress.[10-12] Despite attempts by these and subsequent

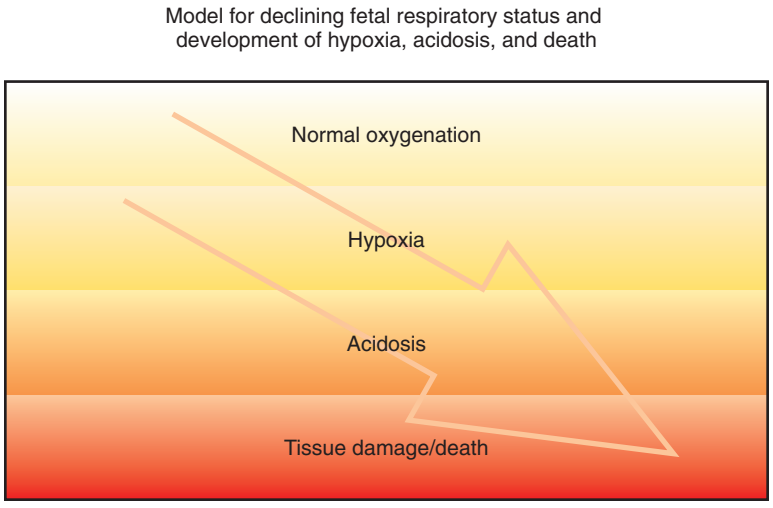

FIGURE 16-1. The purpose of fetal heart rate monitoring is to detect fetal hypoxia and metabolic acidosis. Many intrapartum fetuses develop hypoxia intermittently but never progress to metabolic acidosis. The idea is to avoid intervention for hypoxia, but to intervene in the presence of early metabolic acidosis before it can result in tissue damage or fetal death.

leaders in the field, universal standards for monitoring and terminology were never really established. For example, Europeans tend to refer to electronic fetal heart rate monitoring (EFM in the United States) as "cardiotocography" (CTG) and run their tracings at a paper speed of 1 cm/minute, compared with 3 cm/minute in the United States. **However, in recent years efforts by organizations such as the National Institute of Child Health and Human Development (NICHD) and American College of Obstetricians and Gynecologists (ACOG), *and similar societies in other countries,* have finally set standards for terminology that appear to be gaining widespread acceptance.** The first commercially available electronic fetal monitor was produced in the late 1960s, and by the mid-1970s, EFM was in use in most labor and delivery units in the United States. Today, most women giving birth in the United States have electronic FHR monitoring during labor.

INSTRUMENTATION FOR ELECTRONIC FETAL HEART RATE MONITORING

Many technologic advances have been made since the first monitors were produced. External FHR monitoring using electrocardiography did not work in labor, and phonocardiography was subject to fetal and maternal movement and other external noise. Doppler became the dominant modality for external monitoring. Initially, this modality was difficult to use because the complex Doppler signal made it difficult to determine which point within that signal the computer should use to measure the interval from beat to beat to convert to rate (Figure 16-2). Logic, or computer processing formulas, were used to get apparently good continuous signals, but this process introduced artifact, and the apparent variability and other aspects of the FHR were often inaccurate. Ultimately, better Doppler devices, coupled with autocorrelation formulas for processing the signal, have resulted in excellent external FHR signals that can be relied on clinically. **External monitoring is necessary at all times when the membranes are intact and cannot or should not be ruptured** (Figure 16-3). In addition, certain clinical situations make it unwise to puncture the skin with a fetal electrode for fear of vertical transmission of infection to the fetus. Such conditions include maternal infection with human immunodeficiency virus (HIV), hepatitis C, and herpes simplex.

It is often necessary to apply an internal electrode to obtain a high-quality, accurate, continuous FHR tracing. This is especially true in patients who are obese, in those with a premature fetus, or when the mother or fetus is moving too much to obtain an adequate signal. The original internal electrode was made from a modified skin clip that required a special instrument to place on the fetal scalp. In the mid-1970s, an easier to insert and less traumatic spiral electrode was introduced (Figure 16-4). This is applied to the fetal scalp manually without additional instruments and without the requirement for a speculum to visualize the scalp. The electrical circuit for this electrode includes the spiral electrode for one pole and a small

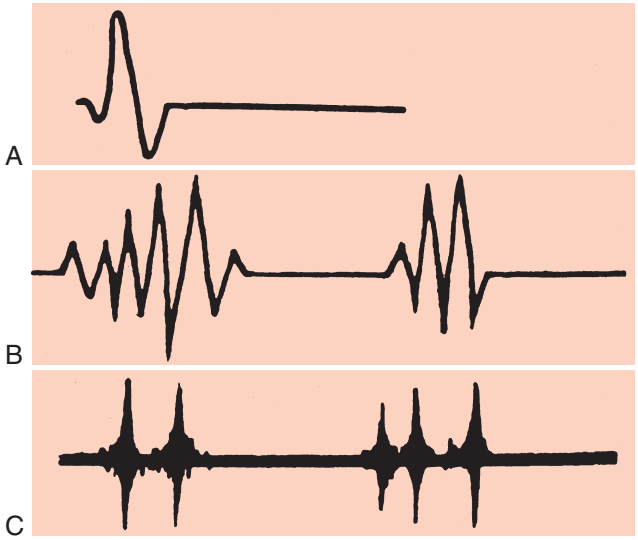

FIGURE 16-2. These complexes represent the types of signals that the fetal heart rate may be required to count. **A,** Electrocardiogram. **B,** Doppler. **C,** Phonocardiogram. Note the complexity of the Doppler signal. To consistently count the same place in the signal complex and avoid artifactually increasing variability, complex signal processing formulas are required.

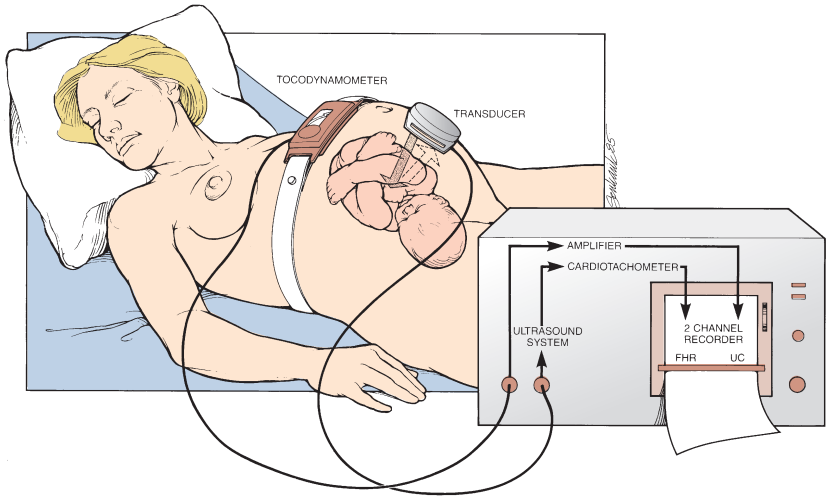

FIGURE 16-3. Instrumentation for external monitoring. Contractions are detected by the pressure-sensitive tocodynamometer, amplified, and then recorded. Fetal heart rate is monitored using the Doppler ultrasound transducer, which both emits and receives the reflected ultrasound signal that is then counted and recorded.

metal bar at the base of the plastic, which, bathed in vaginal secretions, completes the circuit through the mother's body. The spiral electrode has remained in use without substantial change since its introduction. **The FHR tracing results from the signal processor, which counts every R-R interval of the ECG from the scalp electrode, converts this interval to rate, and displays every interval (in rate as beats/minute) on the top channel of the two-channel fetal monitor recording paper.** The signal is amplified by an automatic gain amplifier, which increases the amplitude (gain) until an adequate signal is available to count (Figure 16-5). It must be remembered that when the fetus is dead, the amplifier may increase the gain of the small maternal ECG transmitted through the dead fetus, and this may be easily misinterpreted as a fetal bradycardia (Figure 16-6).

It is clear that the term *electronic fetal monitoring*, unlike the European version *cardiotocography*, undervalues the lower channel of the fetal monitor tracing, which provides information about the uterine contractions in labor. Contractions can also be monitored externally or internally. The external monitoring device, or tocodynamometer, is basically a ring-style pressure transducer attached to the maternal abdomen by a belt that maintains tight continuous contact. When the uterus contracts, the change in shape and rigidity depresses the plunger of the sensor, which changes the voltage of the electrical current. The change in voltage is proportional to the strength of the uterine contractions. **The tocodynamometer depicts the frequency of the contractions accurately, but the strength of the contractions only relatively, because it cannot measure actual intrauterine pressure.** In addition, the apparent duration of the contraction varies with the sensitivity of the monitor, which is negatively affected by variables such as maternal obesity and premature gestational age (Figure 16-7). The advantage of the external monitor is that it can be used when membranes are intact, and it is noninvasive. Its disadvantages, in addition to its inherently limited accuracy, is that it is more uncomfortable for the mother and limits her mobility. Contractions can be more accurately monitored using an intrauterine pressure catheter. The catheters require that the membranes be ruptured and are inserted transcervically beyond and above the fetal presenting part to rest within the uterine cavity. The original pressure catheters were open water-filled systems attached to a pressure transducer adjacent to the fetal monitor. These systems, while accurate, required frequent adjustments and flushing. Newer catheters have closed systems with the strain gauges in the tips or with sensors that relay the signal to a strain gauge at the base of the catheter. Although more expensive, they are easier to use

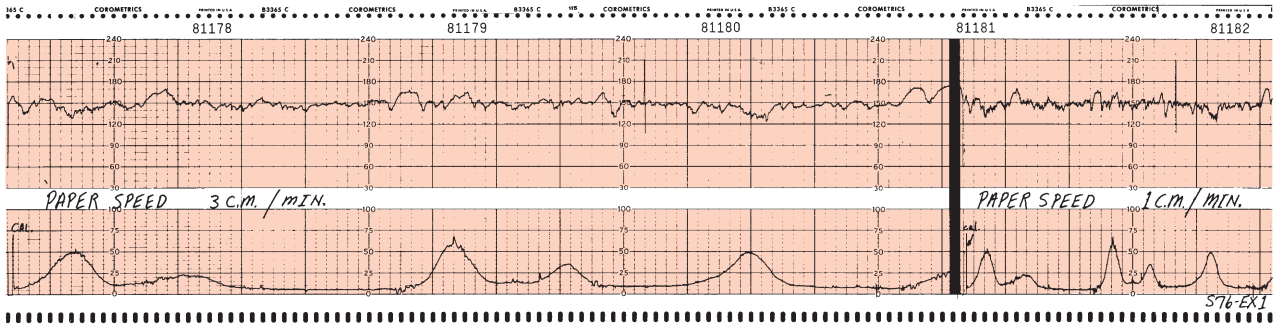

FIGURE 16-4. Internal fetal heart rate data gathered at the standard recording speed of 3 cm/minute for the first portion. The same data are being recorded at a speed of 1 cm/minute in the last segment. Normal long-term and short-term variabilities are present. Note that the uterine activity channel has been calibrated so that the intrauterine pressure readings can be measured correctly.

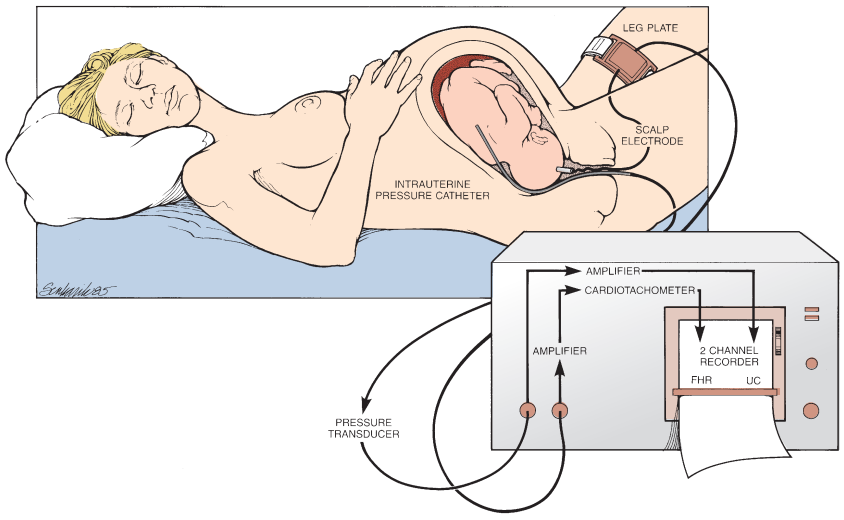

FIGURE 16-5. Techniques used for direct monitoring of fetal heart rate and uterine contractions. Uterine contractions are assessed with an intrauterine pressure catheter connected to a pressure transducer. This signal is then amplified and recorded. The fetal electrocardiogram is obtained by direct application of the scalp electrode, which is then attached to a leg plate on the mother's thigh. The signal is transmitted to the monitor, where it is amplified, counted by the cardiotachometer, and then recorded.

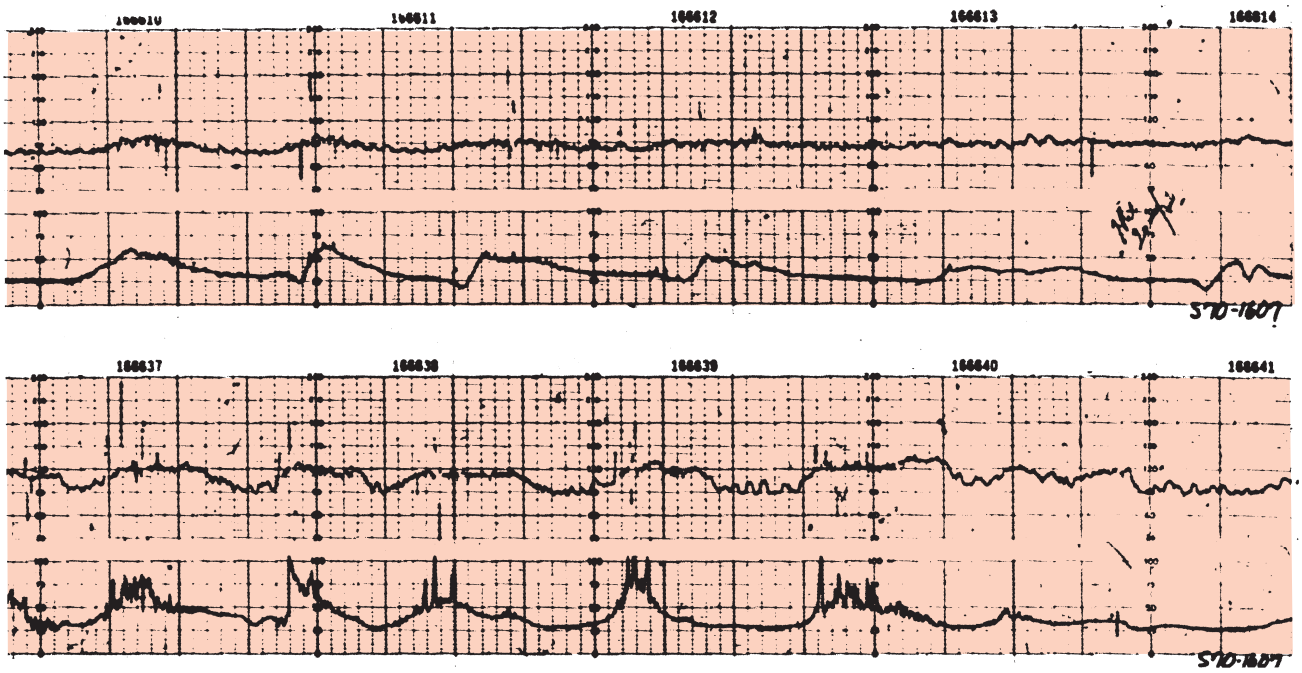

FIGURE 16-6. This is a tracing from an internal electrode demonstrating an apparent bradycardia with a rate of about 90 beats/minute. In actuality, this tracing is from a dead fetus, and the automatic gain amplifier increases the amplitude of the maternal electrocardiogram signal, allowing the monitor to count and display maternal heart rate. Note the accelerations of the maternal heart rate with contractions, typical of a heart rate response to the pain from contractions.

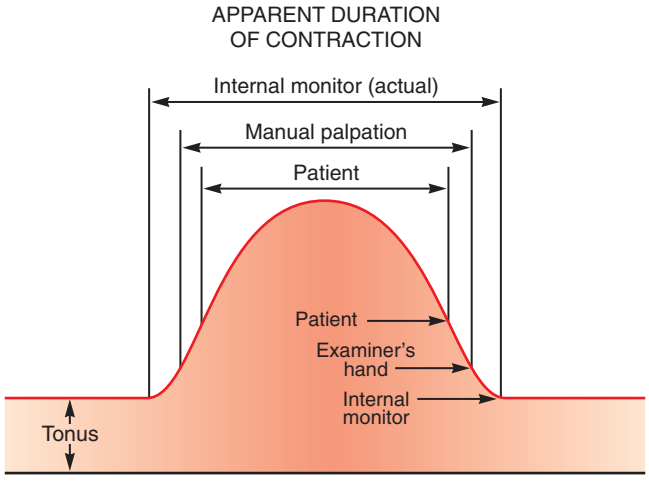

FIGURE 16-7. The sensitivity of the device used to monitor a uterine contraction can affect not only the apparent strength of the contraction but also the apparent duration of the contraction.

and require less nursing attention. Once the catheter is electronically "zeroed" (calibrated), the contractions are accurately recorded in terms of frequency, duration, and intensity on the lower channel of the two-channel recording paper or television monitor. This channel is conveniently calibrated at 0 to 100 mm Hg on its vertical scale, from which contraction amplitude can be read. These catheters also are often made with a second port through which saline can be infused for amnioinfusion (see later).

The goal of monitoring is to maintain adequate, high-quality, continuous FHR and contraction tracings while maintaining maximal maternal comfort and avoiding the risk for trauma and infection in the fetus and mother. External devices minimize risk but often give less accurate information and are more uncomfortable for the mother. In general, when the FHR is reassuring and there is an adequate tracing and when the progress of labor is adequate, the external devices are fine. When better-quality FHR monitoring is required or it becomes important to accurately assess uterine contraction duration and intensity, internal devices may be necessary.

PHYSIOLOGIC BASIS OF FETAL HEART RATE MONITORING

The basis of FHR monitoring is, in a real sense, fetal brain monitoring. The fetal brain is constantly responding to stimuli, both peripheral and central, with signals to the fetal heart that alter the heart rate on a moment-to-moment basis. Such stimuli to which the brain responds include chemoreceptors, baroreceptors, and direct effects of metabolic changes within the brain itself. The benefit for the brain to modulate the FHR is derived from its goal of maintaining optimal perfusion to the brain without compromising blood flow to other organs any more than is necessary. It should be intuitively obvious, therefore, that the use of FHR to monitor fetal oxygenation is inherently crude and nonspecific because many stimuli other than oxygen either cause the brain to alter the FHR or may have a direct effect on the fetal heart. **This really explains the most important basic premise of EFM: when the FHR is normal in appearance, one can be assured with high reliability that the fetus is well oxygenated, but when the FHR is not entirely normal, it may be the result of hypoxia or of other variables that may also affect FHR.** In the past when the FHR became abnormal and the clinician decided intervention was necessary because of concern over fetal

hypoxia, the term *fetal distress* was used. More often than not, however, such intervention results in the delivery of a well-oxygenated, nonacidotic, vigorous newborn. This understanding led to an attempt at more accurately reflecting the limitations of interpreting an abnormal FHR. **The term fetal distress was abandoned in favor of the more intellectually honest term, *non-reassuring fetal status* (NRFS).**[13] Even more recently, a further refinement has been recommended as a result of a second workshop sponsored by the NICHD and subsequently adopted by both ACOG and the Association of Women's Health, Obstetric and Neonatal Nurses (AWHONN), **which placed FHR patterns into three categories based on the likelihood of adequate or inadequate oxygenation and acidosis.**[14]

Fetal oxygenation is determined by many factors. **The placenta functions as the fetal lung.** Oxygen transfer across the placenta, as in the lung or any membrane, is proportional to the difference between partial pressures of oxygen between the mother and the fetus, the blood flow to the placenta, a coefficient of diffusion for the gas, and the surface area of the placenta. Transfer is inversely proportional to the thickness of the membrane (placenta). Thus, under normal circumstances during labor, the only variable that alters fetal oxygenation is the temporary interruption in blood flow to the placenta that occurs as a result of the compression of the spiral arteries by the wall of the uterus at the peak of the contraction. The duration that the spiral arteries will be compressed will thus depend on the duration and strength of the contraction (Figure 16-8). Under normal circumstances, the fetus tolerates these periods of stasis well without a significant change in its oxygen content. Contractions that are unusually long or unusually strong may, however, result in transient periods of fetal hypoxemia.

Other variables that have the potential for altering fetal oxygenation most commonly include those that affect uterine perfusion. A laboring woman in the supine position can develop hypotension as a result of vena caval compression from the uterus. Maternal hypotension with redistribution of blood flow away from the placenta occurs not infrequently with regional anesthesia. Maternal hemorrhage, such as in placenta previa or abruptio placentae, may have similar effects. There are several forms of microvascular disease that can impair fetal oxygenation from poor perfusion within the uteroplacental vascular bed. Examples include hypertension, preeclampsia or eclampsia, collagen vascular disease, diabetic vasculopathy, and postmaturity. Abruptio placentae may compromise fetal oxygenation in several ways. These include maternal hypotension, as previously mentioned; a decrease in the surface area of the placenta; and uterine hyperactivity.

Although the placenta functions as the fetal lung, the umbilical cord functions as its trachea, leading oxygen to the baby and carbon dioxide (CO_2) away. Alteration in umbilical cord blood flow is a very common occurrence during labor, either from direct compression or from stretch. Direct compression may occur when the cord becomes impinged between any part of the fetal body and the uterine wall, either with contractions or with fetal movement. This is especially more common when there is oligohydramnios because there is less amniotic fluid to provide a cushion for the cord.[15] Alternatively, cord stretch may occur as the fetus descends into the pelvis. Typically, this is seen just before complete dilation, when descent of the vertex normally occurs. There are three potent stimuli that produce spasm of the umbilical vessels that have evolved to allow cessation of fetal umbilical cord blood flow following birth. These include a lower ambient temperature, a higher oxygen tension as the baby begins breathing, and the stretch of the umbilical cord as the baby falls from the birth canal.[16] Thus, it should not be surprising that transient cessation of cord blood flow will occur with stretching of the cord during descent if the cord is looped around the baby's neck and descent of the vertex occurs.

It becomes important, therefore, to understand the physiologic mechanisms that control the FHR. This is so not only because the FHR may be used to determine the severity of the hypoxia and whether a metabolic acidosis is ensuing but also because the FHR pattern can elucidate the mechanism of the reduction in fetal oxygenation. Thus, by knowing the cause of any hypoxia, the treatment, when possible, can be more specifically directed at the cause. Finally, an understanding of the mechanism and progression of the FHR pattern can often also provide an opportunity to predict how fetal oxygenation will progress over time.

The FHR has many characteristics that we are able to use to accomplish this interpretation. These include the *baseline rate;* the *variability* of the FHR from beat to beat; transient alterations below the baseline, termed *decelerations;* and transient alterations above the baseline, termed *accelerations*. Rate and variability are generally included as *characteristics of the baseline* FHR, and decelerations and accelerations as *periodic changes*. In 1997, the NICHD convened a workshop to standardize terminology in FHR monitoring subsequently accepted and endorsed by most obstetrical organizations, and the subsequent descriptions and definitions in this chapter are consistent with those recommendations, except where explanations are provided to explain potential limitations of this newer system.[14]

Tachycardia

The baseline FHR is typically between 120 and 160 beats/minute. In very early gestation (15 to 20 weeks), the FHR is significantly higher than in the term fetus. The decline in FHR represents a maturation of fetal vagal tone with progressing gestation.[18] If atropine or other vagolytic drugs are administered, the FHR will regress to the higher baseline of 160 beats/minute. Thus, the baseline heart rate is largely a function of vagal activity. Many factors have the potential to alter the fetal baseline. For interpretation of the baseline rate, the minimal duration must be at least 2 minutes of adequate signal in a 10-minute window, or the baseline for that period is indeterminate. Rates above 160 beats/minute are called *tachycardia*. Tachycardia may have great clinical significance. **The two most common causes of tachycardia are maternal fever and drugs that directly raise the FHR.** Maternal fever raises the core temperature of the fetus, which is always about 1° F higher than the maternal temperature. With maternal fever, virtually all fetuses have tachycardia (Figure 16-9). The FHR rises approximately 10 beats/minute for each 1° F increase in

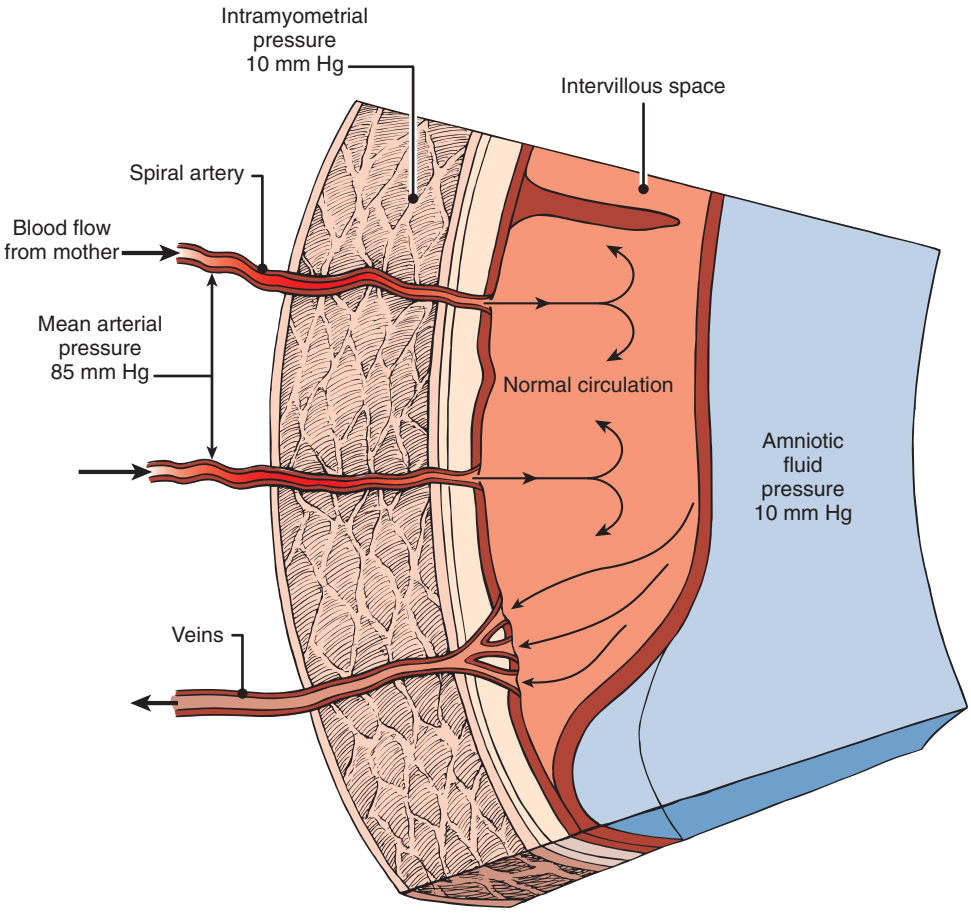

FIGURE 16-8. In the resting state between contractions **(A),** the intraluminal pressure within the spiral arteries exceeds the intramyometrial pressure. Thus, uteroplacental blood flow is sustained. However, at the peak of a uterine contraction **(B),** the myometrial pressure can exceed the arterial pressure, and uterine blood flow will be transiently interrupted, temporarily halting oxygen delivery to the placenta.

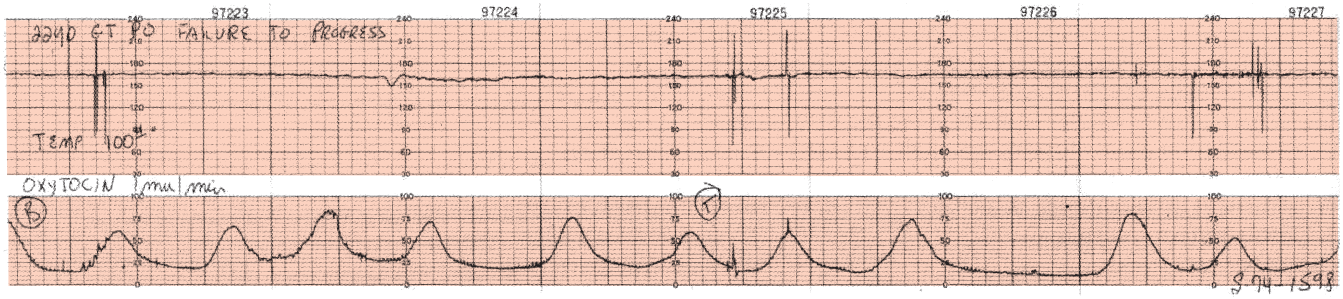

FIGURE 16-9. A tachycardia with a fetal heart rate of 170 beats/minute seen in association with a maternal fever of 38° C (100.4° F).

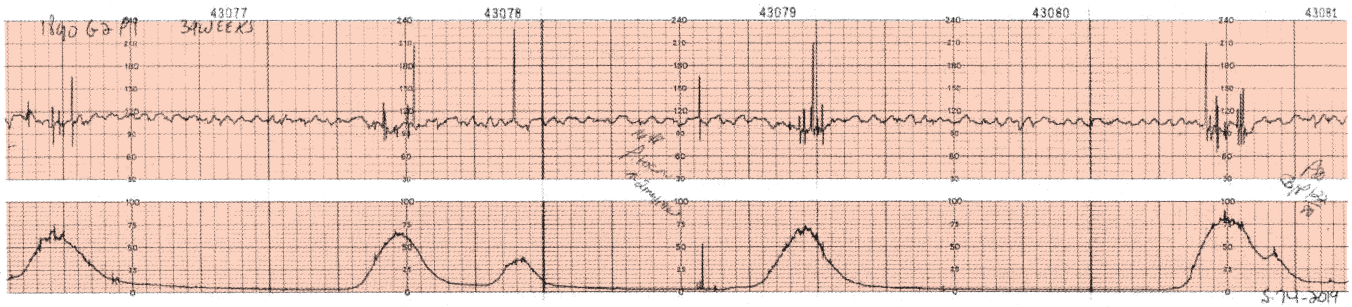

FIGURE 16-10. A bradycardia with a fetal heart rate of 110 beats/minute. This patient is in premature labor at 34 weeks' gestation and is being treated with magnesium sulfate. The fetal bradycardia is probably because of maternal hypothermia, which can be seen with vasodilation caused by the magnesium sulfate.

maternal temperature. Because at term with chorioamnionitis only 1% to 2% of fetuses are septic, the tachycardia is unlikely to indicate fetal sepsis, but rather is probably caused by an increase in fetal metabolic rate associated with the elevated temperature. Drugs that elevate the FHR fall into one of two categories: vagolytic and β-sympathomimetic. Commonly used drugs that are vagolytic include scopolamine, atropine and phenothiazines, and hydroxyzine. These drugs, however, rarely raise the FHR above 160 beats/minute. β-Sympathomimetics include terbutaline and ritodrine, used for preterm labor, and terbutaline and epinephrine, used for bronchospasm. Other, less common causes of fetal tachycardia include fetal hyperthyroidism, fetal anemia, fetal heart failure, and fetal tachyarrhythmias. As fetal hypoxia becomes progressively worse and persists over time, fetal tachycardia often develops. However, when contractions are present, tachycardia is not the first physiologic response to hypoxia and, in the absence of decelerations, in the laboring patient, is rarely if ever caused by hypoxia.[19]

Bradycardia

An FHR less than 110 beats/minute is termed a *bradycardia*. One must distinguish between a baseline FHR less than 110 beats/minute that is an established baseline and one that follows a prolonged deceleration. This is an important issue because an established baseline bradycardia is often innocuous (Figure 16-10), whereas a prolonged deceleration to less than 110 beats/minute lasting more than 60 to 90 seconds may often indicate significant fetal hypoxia (Figure 16-11). When a prolonged deceleration lasts more than 10 minutes it becomes a bradycardia according to the newer NICHD terminology.[17] True established fetal bradycardias occur infrequently and can be due to several possible causes. In the range of 90 to 110 beats/minute, a bradycardia may often be a normal variant, and these fetuses are usually bradycardic after birth, but are otherwise well oxygenated and normal. **As with tachycardia and fever, maternal hypothermia may cause fetal bradycardia.** This is commonly seen with patients on magnesium sulfate ($MgSO_4$) who are vasodilated and has also been described with maternal hypoglycemia and hypothermia.[20,21] Drugs, such as propranolol, may also result in fetal bradycardia. A fetal baseline heart rate in the range of 80 beats/minute or less, especially with minimal or absent variability, may be caused by a complete heart block. Complete heart block may be caused by antibodies associated with maternal lupus erythematosus, may be seen with congenital cardiac anomalies, or is idiopathic.[22] Whereas a heart block is not associated with hypoxia, it makes the FHR uninformative in monitoring fetal oxygenation because the fetal brain is no longer communicating with the ventricle of the heart that is being monitored. When a patient is admitted with a baseline bradycardia, one should also consider the possibility of maternal heart rate being recorded with a dead fetus (whether internal or external monitor) (see Figure 16-6). Real-time ultrasound is used to verify that the bradycardia is fetal in origin.

Variability

The fetal cardiotachometer is unique compared to adult monitors in that it records the interval-to-interval difference in rate for every heart beat. Thus, differences in heart rate from beat to beat are recorded as "variability" reflected visually as a line that fluctuates above and below the baseline. **This variability is a reflection of neuromodulation of the FHR by an intact and active CNS and also reflects normal cardiac responsiveness.** Generally, the variability of

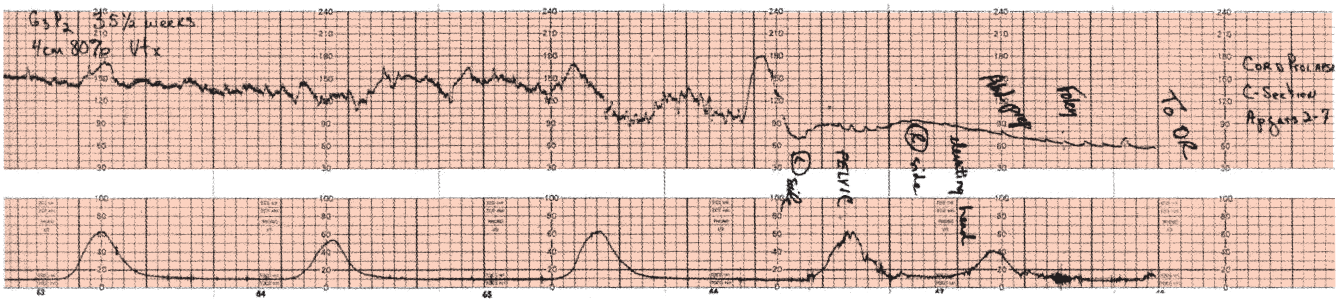

FIGURE 16-11. The prolonged deceleration to 60 beats/minute, in this case caused by umbilical cord prolapse, evolves following a prolonged deceleration from a normal baseline rate. When the prolonged deceleration lasts longer than 10 minutes, it then becomes a bradycardia according to the new NICHD terminology.[17]

the FHR is described as having two components: *short-term* and *long-term variability*. Short-term variability is the beat-to-beat irregularity in the FHR and is caused by the difference in rates between successive beats of the FHR. It is caused by the push-pull effect of sympathetic and parasympathetic nerve input, but the vagus nerve has the dominant role in affecting variability. Long-term variability is the waviness of the FHR tracing, and is generally seen in three to five cycles per minute. Previous texts spent considerable effort in distinguishing between the significance of short- and long-term variability, but in general they are reduced or increased together, and there is no clear evidence that distinguishing between the two is helpful clinically. **The NICHD Research Planning Workshop also concluded that no distinction should be made between short- and long-term variability.**[17]

This characteristic of the fetal baseline can be one of the most useful single parameters in determining the severity of fetal hypoxia if understood correctly. The simplest way to describe the causes of alterations, especially reductions, in FHR variability is to say that this parameter reflects the activity of the fetal brain. When the fetus is alert and active, the FHR variability is normal or increased. When the fetus is obtunded, by whatever cause, the variability is reduced. Because severe hypoxia, especially when it reaches the level of metabolic acidosis, will always depress the CNS, normal variability reliably indicates the absence of severe hypoxia and acidosis (Figure 16-12*A*). Unfortunately, the converse is not true because there are many things that cause the CNS to be depressed (see box, Potential Causes of Decreased Variability of the Fetal Heart Rate); thus, reduced (minimal or absent) variability is a very nonspecific finding and must be interpreted in the context of other indicators of hypoxia, and other causes of reduced variability must be considered (Figure 16-12*B*). **In general, anything that is associated with depressed or reduced brain function will diminish variability.** This includes fetal sleep cycles; drugs, especially CNS depressants; fetal anomalies, especially of the CNS; and previous insults that have damaged the fetal brain. FHR variability is also affected by gestational age, and very immature fetuses of less than 26 weeks' gestation often have reduced variability from an immature CNS, although this varies from fetus to fetus. In addition, as heart rate increases with fetal tachycardia, variability is often reduced from the rate alone because sympathetic dominance overrides the natural influence of the vagus. Increased FHR variability, original referred to as a "saltatory" FHR pattern, in nonlaboring animals has been shown to be associated with very early or minimal hypoxia.[23] This is a rare pattern and difficult to interpret. In labor, late, variable, or prolonged decelerations will virtually always be present with early hypoxia, and increased variability is not consistent with an acidotic fetus; therefore, this finding can be interpreted in context with the entire FHR pattern.

POTENTIAL CAUSES OF DECREASED VARIABILITY OF THE FETAL HEART RATE

- Depression due to hypoxia and acidosis
- Fetal anomalies, especially of the central nervous system
- Fetal sepsis
- Tumors of the central nervous system
- Fetal heart block
- Tachycardia
- Extreme prematurity
- Previous neurologic insult
- Fetal sleep cycles
- Drugs, medications
  - Narcotics
  - Barbiturates
  - Tranquilizers
  - Phenothiazines
  - Parasympatholytics
  - General anesthetics

Another problem with variability besides its nonspecificity is that the interpretation of variability is quite subjective. Variability is usually described quantitatively, The NICHD Research Planning Workshop[17] suggested that FHR variability be defined as follows:
Absent: amplitude undetectable
Minimal: amplitude > undetectable and ≤5 beats/minute
Moderate: amplitude 6 to 25 beats/minute
Marked: >25 beats/minute.

Experts given tracings to interpret often disagree on the quantification of variability even using just these four categories. Trying to categorize variability any further is fraught with even more potential for disagreement and does not appear to have predictive value.

PERIODIC CHANGES

Variability, tachycardia, and bradycardia are characteristic alterations of the baseline heart rate. Periodic changes of the FHR include decelerations and accelerations. These

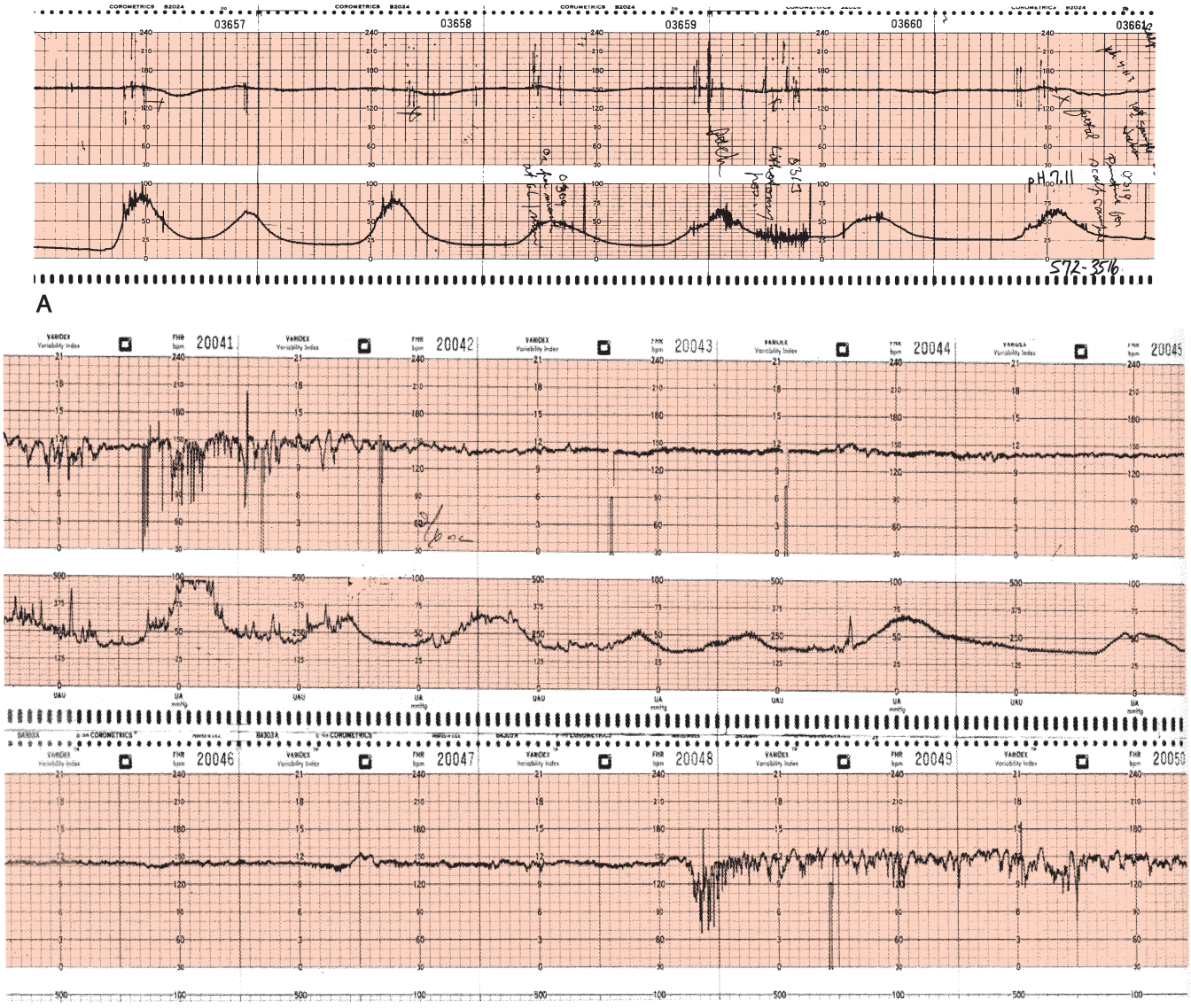

FIGURE 16-12. **A,** The markedly decreased variability seen in this case is in association with persistent late decelerations. A scalp pH of 7.11 confirms that the loss of variability is because of central nervous system depression caused by acidosis. **B,** The abrupt decrease in variability seen here, in contrast to **A,** is not seen in association with any decelerations that might suggest hypoxia. Thus, the decreased variability must be because of another reason, and in this case, given the equally abrupt return to normal variability in **B,** is probably caused by a fetal sleep cycle.

are transient changes in the FHR of relatively brief duration with return to the original baseline FHR. In labor, these usually occur in response to uterine contractions but may also occur with fetal movement.

Decelerations

There are four principal types of decelerations: *early*, *late*, *variable*, and *prolonged*. These are named for their timing, relationship to contractions, duration, and shape, but are important distinctions more because they describe the cause of the decelerations.

Early Decelerations

Early decelerations are shallow, symmetrical, uniform decelerations with onset and return that are gradual, resulting in a U-shaped deceleration (Figure 16-13). They begin early in the contraction, have their nadir coincident with the peak of the contraction, and return to the baseline by the time the contraction is over. Early decelerations are not associated with accelerations that precede or follow the deceleration. These decelerations typically do not descend more than 30 to 40 beats/minute below the baseline rate. **They are thought to be caused by compression of the fetal head by the uterine cervix as it overrides the anterior fontanel of the cranium.**[24] This results in altered cerebral blood flow, precipitating a vagal reflex with the resultant slowing of the FHR. More nonspecific head compression can result in decelerations that are indistinguishable from variable decelerations. Because of the similar cause, these latter decelerations have often been called *early decelerations*, but are by definition not so. Because the cervix creates the pressure, typically early decelerations are usually seen

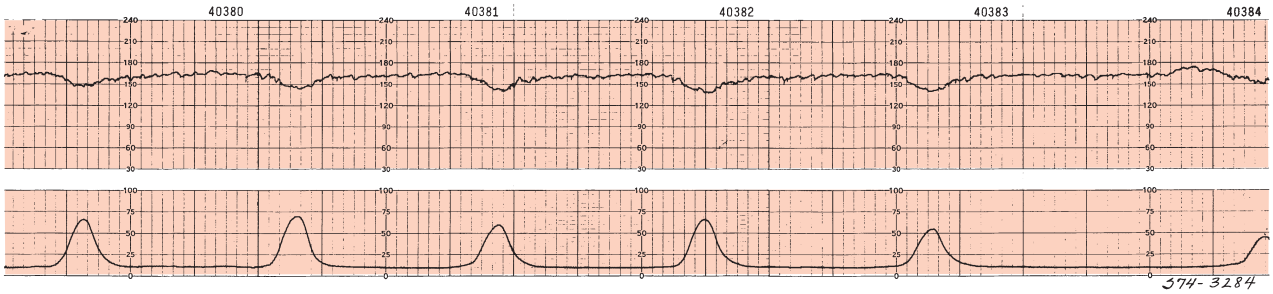

FIGURE 16-13. Early decelerations.

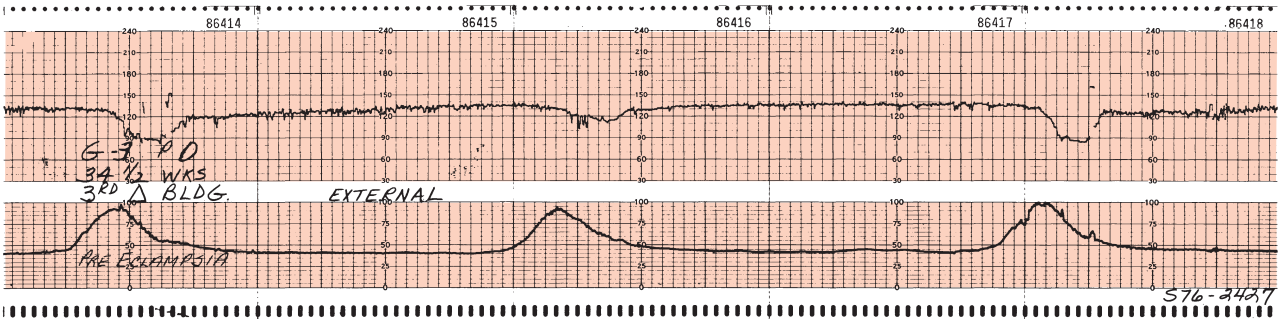

FIGURE 16-14. A case complicated by third-trimester bleeding in which the external heart rate and uterine activity data are collected. Note the presence of persistent late decelerations with only three contractions in 20 minutes as well as the apparent loss of variability of the fetal heart rate. The rise in baseline tone of the uterine activity channel cannot be evaluated with the external system.

between 4 and 6 cm of dilation (E.H. Hon, personal communication). They do not indicate fetal hypoxia and are only significant in that they may be easily confused with late decelerations because of their similar shape and depth. They are the most infrequent of decelerations, occurring in about 5% to 10% of all fetuses in labor.

Late Decelerations

Late decelerations are similar in appearance to early decelerations. They, too, are of gradual onset and return, are U-shaped, and generally descend below the baseline no more than 30 to 40 beats/minute, although there are exceptions. **However, in contrast to early decelerations, late decelerations are delayed in timing relative to the contraction.** They begin usually about 30 seconds after the onset of the contraction or even at or after its peak. Their nadir is after the peak of the contraction. In most cases, the onset, nadir, and recovery of the deceleration occur after the beginning, peak, and ending of the contraction, respectively. FHR variability may be unchanged or even increased during the decelerations. These decelerations are not associated with accelerations immediately preceding or following their onset and return (Figure 16-14).

The physiology of late decelerations is quite complex, but an understanding of the physiology pays dividends in terms of interpreting and managing these important FHR changes. **Late decelerations are generally said to be caused by "uteroplacental insufficiency."** This implies that uteroplacental perfusion is temporarily interrupted during the peak of strong contractions. The fetus that normally will not become hypoxic with this temporary halt in blood flow may do so if there is insufficient perfusion or oxygen exchange, as with, for example, hypotension or microvascular diseases within the uterus or placenta. Whereas this may be a correct idealized description, in reality any compromise of delivery, exchange, or uptake in fetal oxygen, other than by umbilical cord compression, can result in a late deceleration if the insult is sufficient. Physiologically, oxygen sensors within the fetal brain detect a relative drop in fetal oxygen tension in association with the uterine contraction. This change initially results in an increase in sympathetic neuronal response, causing an elevation in fetal blood pressure that, when detected by baroreceptors, produces a protective slowing in the FHR in response to the increase in peripheral vascular resistance. This has been referred to as the "reflex" type of late deceleration. This complex double reflex is probably the reason the deceleration is delayed.[25] During this type of reflex, the depth of the deceleration is proportional to the severity of the hypoxia, and the deceleration moves closer to the contraction as the hypoxia becomes more severe. However, there is also a second type of late deceleration, caused by "myocardial depression." As the hypoxia continues and becomes more severe, late decelerations are no longer vagally mediated and are seen even with interruption of the vagus nerve; thus, they are directly myocardial in origin. These decelerations are *not* proportional in their depth to the severity of the hypoxia and actually may become more shallow as the hypoxia becomes quite severe. Because of this latter type of deceleration, the depth of the late deceleration cannot always be used to judge the severity of the hypoxia. **Because of the mechanisms causing these changes, *late decelerations virtually always indicate fetal hypoxia.*** Only the severity of the hypoxia and the overall duration of the hypoxia will determine whether a metabolic acidosis will occur, and this is highly unpredictable.

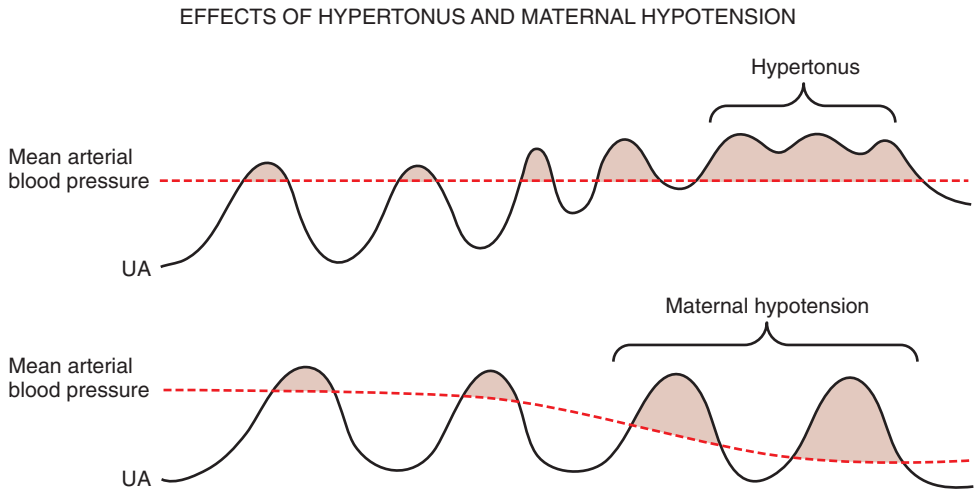

FIGURE 16-15. The two most common causes of late decelerations in labor are excessive uterine contractions (usually caused by oxytocin) and maternal hypotension. Both result in decrease in uteroplacental perfusion, hypertonus by interrupting the transmyometrial perfusion for a prolonged period, and hypotension by dropping the perfusion pressure, thus increasing the amount of time perfusion is interrupted even with a normal contraction. *UA,* Uterine artery.

One reason this may be so is found in recent data suggesting that the oxygen threshold that triggers the brain to slow the fetal heart in this characteristic way may be more related to the relative drop from baseline oxygenation rather than an absolute number.[26] Thus, the fetus accustomed to higher than average oxygen saturation may have a drop in oxygen at or only slightly below the normal range—a level deep enough to signal a late deceleration, but not low enough to require anaerobic metabolism. Another important point in understanding the results of hypoxia associated with late decelerations is that the placenta's capacity for exchanging oxygen is substantially less than its capacity for exchanging CO_2. In situations in which there are persistent late decelerations and the fetus becomes sufficiently hypoxic to develop a metabolic acidosis, there may be no retention of CO_2. This is quite analogous to the adult with lung but no airway disease in whom hypoxia is often seen without difficulty in eliminating CO_2. Thus, the metabolic acidosis is usually not mixed with a respiratory acidosis. The only common exception to this is with abruptio placentae, in which CO_2 retention is seen with late decelerations.[27]

Causes of late decelerations include any factor that can alter delivery, exchange, or uptake of oxygen at the fetal-maternal interface within the placenta. Most commonly, late decelerations are observed in patients without inherent pathology. Excessive uterine contractions, usually seen with oxytocin, are the single most common cause of late decelerations. In these situations, the duration of interruption of uterine blood flow is prolonged, the hypoxia is more than the normal fetus can endure, and late decelerations are expressed. Conduction anesthesia (spinal or epidural) can cause either systemic or local hypoperfusion or hypotension, and thus the level of contractions required to interrupt uterine blood flow is lower, and again the duration of interruption of uterine blood flow is prolonged (Figure 16-15). The most common pathologic conditions of the placenta associated with late decelerations are those characterized by either microvascular disease in the placenta or local vasospasm compromising blood flow and thus exchange. Common causes include postmaturity, maternal hypertension (chronic hypertension or preeclampsia), collagen vascular diseases, and diabetes mellitus in its more advanced stages. Besides altering perfusion, abruptio placentae is an example of altered placental exchange caused by a combination of reduced placental surface area and increased contractions that typically result in late decelerations when the separation is sufficient to cause fetal hypoxia. Severe maternal anemia or maternal hypoxemia may compromise oxygen delivery and result in late decelerations. Conversely, chronic fetal anemia may diminish fetal oxygen uptake and be associated with late decelerations.

Variable Decelerations

The most common type of decelerations seen in the laboring patient are variable decelerations. Variable decelerations are, in general, synonymous with umbilical cord compression, and anything that results in the interruption of blood flow within the umbilical cord will result in a variable deceleration. The variable deceleration is the most difficult pattern to describe, but the easiest to recognize visually. First and foremost, the term *variable* is by far the best single word to describe this type of deceleration. It is variable in all ways: size, shape, depth, duration, and timing relative to the contraction. The onset is usually abrupt and sharp. The return is similarly abrupt in most situations. The depth and duration are proportional to the severity and duration of interruption of cord blood flow. Variable decelerations are usually seen with accelerations immediately preceding the onset of the deceleration and immediately following the return to baseline. The definitions from the NICHD Research Planning Workshop also include that variable decelerations are those lasting a minimum of 15 seconds and descending 15 beats or more below the baseline and that the duration of a variable deceleration should be limited to 2 minutes, and that beyond 2 minutes, it should be called a prolonged

deceleration.[17] More than 50 years ago, Barcroft first described the variable deceleration when he ligated the umbilical cord of a fetal goat[28] (Figure 16-16). In 1975, Lee and Hon externalized the human umbilical cord before cesarean delivery and demonstrated that the reflex involved in the complex pattern of the variable deceleration is one that is caused primarily by changes in systemic blood pressure in the fetus and is mediated through baroreceptors (Figure 16-17).[29] When the umbilical cord is gradually compressed, the thinner-walled umbilical vein collapses first, and blood flow returning to the fetus is interrupted. This results in decreased cardiac return, fetal hypotension, and a baroreceptor reflex that leads the brain to accelerate the heart rate in order to maintain cardiac output. This increase in heart rate is the acceleration that precedes the variable deceleration. With continuing compression, the umbilical artery is compressed, and the fetus detects an increase in systemic vascular resistance because the previously low-resistance placental bed, to which 50% of fetal cardiac output normally flows, is occluded. The baroreceptors detect the increase in resistance, and the heart slows as a protective mechanism. As the cord vessels gradually open, the arteries open first, and the heart rate returns to baseline; but if the flow in the vein is still blocked, an acceleration of the same mechanism of the one that preceded the deceleration occurs. Although this model is idealized, one might surmise that the orderly occlusion of vein, vein and artery, vein does not always occur. This is probably the reason that, with variable decelerations, any combination of deceleration with acceleration preceding, preceding and following, following only, neither, or even acceleration alone may be seen with cord compression, as in Figure 16-18.

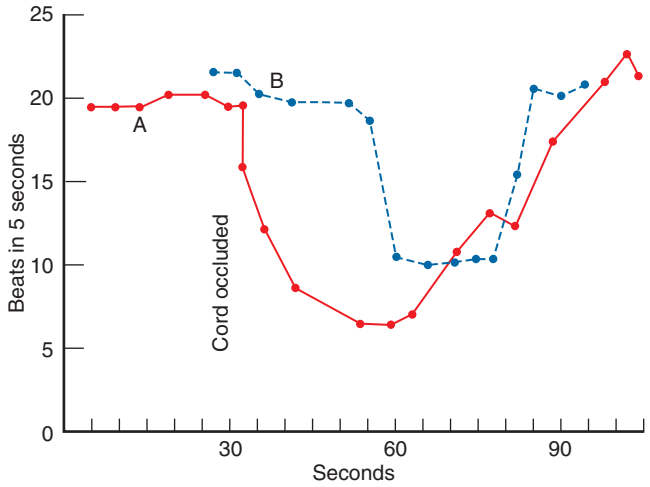

FIGURE 16-16. This is the original description of a variable deceleration in a fetal goat by Barcroft. The *solid line A* represents the fetal heart rate with temporary umbilical cord occlusion and the *dotted line B* is the fetal heart rate with temporary cord occlusion after the vagal nerve has been severed.

In reality, although we refer to umbilical cord compression as the single mechanism for interruption of cord blood flow, there are probably several different mechanisms that may have the same end result. Compression may be the mechanism that occurs when the cord is impinged between a fetal body part and the uterine wall during contractions or with fetal movement. As previously mentioned, cord stretch may be the reason the flow is compromised with

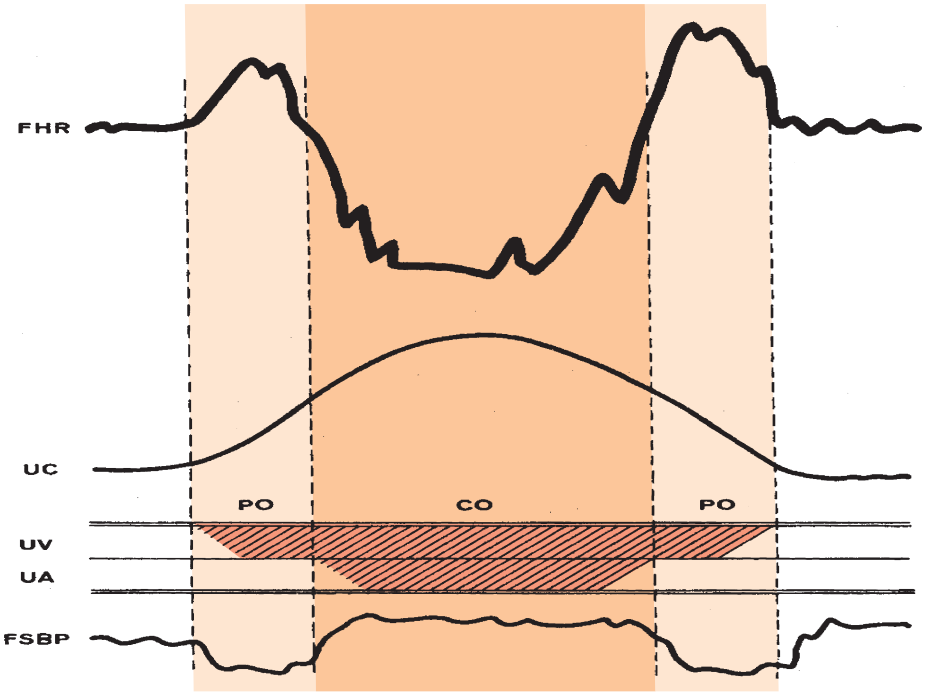

FIGURE 16-17. This figure represents fetal heart rate *(FHR)* and fetal systemic blood pressure *(FSBP)* occurring during compression of the umbilical vein *(UV)* and umbilical artery *(UA)*. Note the acceleration of the FHR as the FSBP is decreased, marking a baroreceptor response to decreased cardiac return, and the deceleration of the FHR when the FSBP is increased, the baroreceptor response to increased peripheral resistance. *UC,* Uterine contraction.

nuchal cords and seen as the baby descends through the pelvis. If cold saline is infused too rapidly with amnioinfusion, the FHR may slow, presumably as a result of cord spasm, the natural fetal reflex to cold stimulus. **Whatever the mechanism, it is most important to realize that variable decelerations are initially caused by a reflex in response to changes in pressure and not hypoxia.** Thus, variable decelerations (even deep and prolonged) can be seen in fetuses with no change in oxygen saturation (Figure 16-19).

Variable decelerations are seen in most labors, and most often these decelerations occur without fetal hypoxemia. It is apparent that additional criteria are needed to separate those benign variable decelerations not likely to be associated with hypoxia from those that are. Kubli and coworkers described a category of mild, moderate, and severe variable decelerations based on depth and duration (see box, Classifications of the Severity of Variable Decelerations).[30] Although there is indeed a correlation between the severity of these decelerations and the likelihood of hypoxia, one can see from Figure 16-19 that it is difficult to pick a specific depth and duration that always predicts oxygen compromise. Therefore, in addition, characteristics of the fetal baseline are also used, including the development of tachycardia and loss of variability (Figure 16-20). When cord compression occurs with each contraction and is sustained for a prolonged period of time, there can be a change from the usual abrupt return to baseline to a slow or delayed return to baseline (Figure 16-21). This is also often called a *late component*, although a combined pattern of late and variable decelerations (Figure 16-22) should be distinguished from progressive, severe variable decelerations that result in slow return to baseline because the etiologies and thus the potential treatments will differ. This particular discriminator of variable decelerations can be one of the most confusing aspects in all of fetal monitoring. Because a slow return to baseline can represent fetal hypoxia from either progressive cord compression or from a coincident late deceleration, the question is, Does this finding always represent hypoxia? Many times this sign appears without significant cord compression preceding its onset; therefore, it is unlikely that a substantial oxygen deficit has developed. Thus, many times these are benign findings and may represent more slow release of the cord or some other unexplained phenomenon. Finally, in extreme situations in which there is profound fetal hypoxia and acidosis, the variable decelerations will appear smoother and rounded or "blunted," rather than having the usual abrupt changes seen with the more common

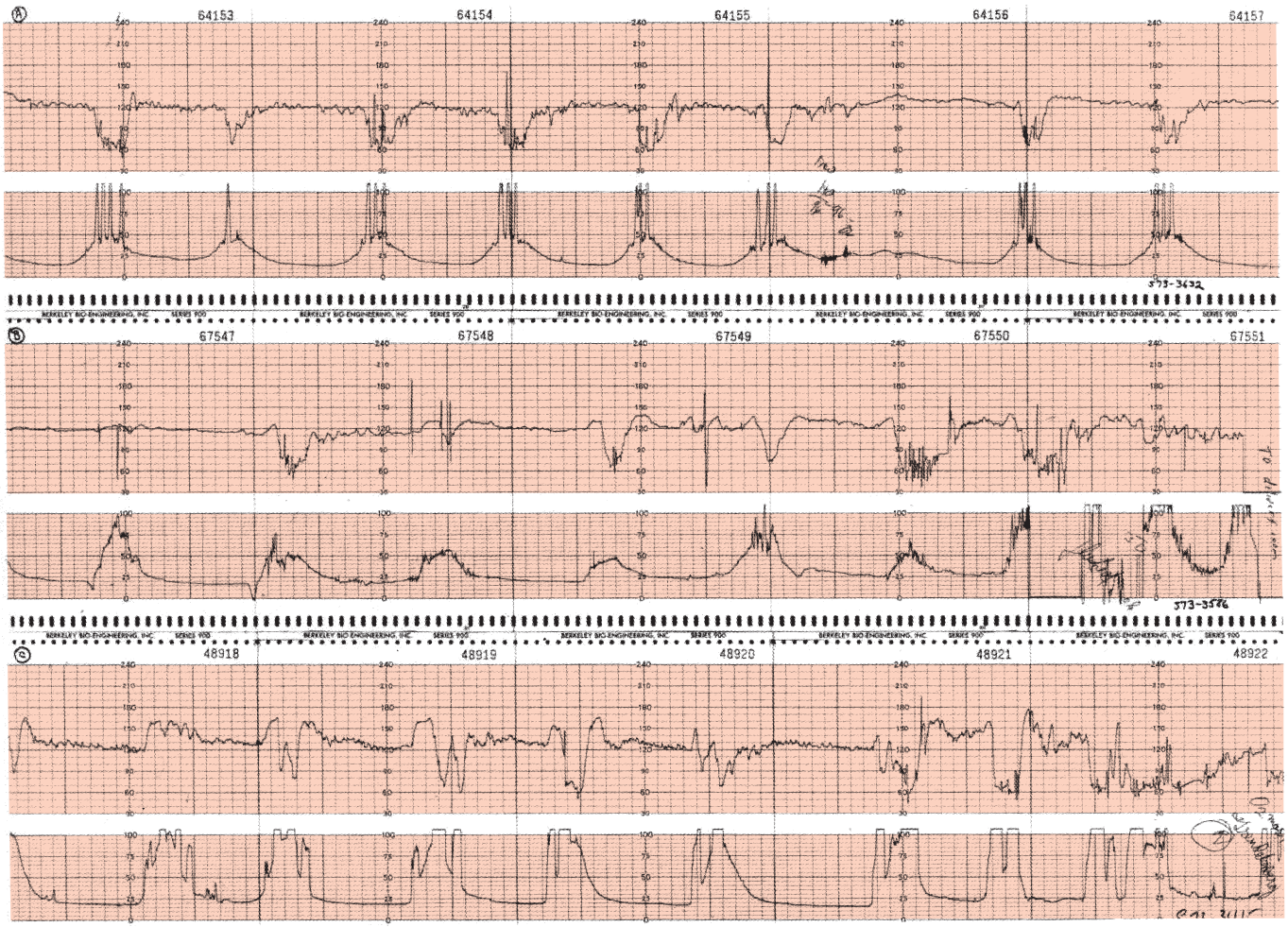

FIGURE 16-18. These are typical variable decelerations. Note that such decelerations are often recognized by the accelerations that precede and follow the decelerations.

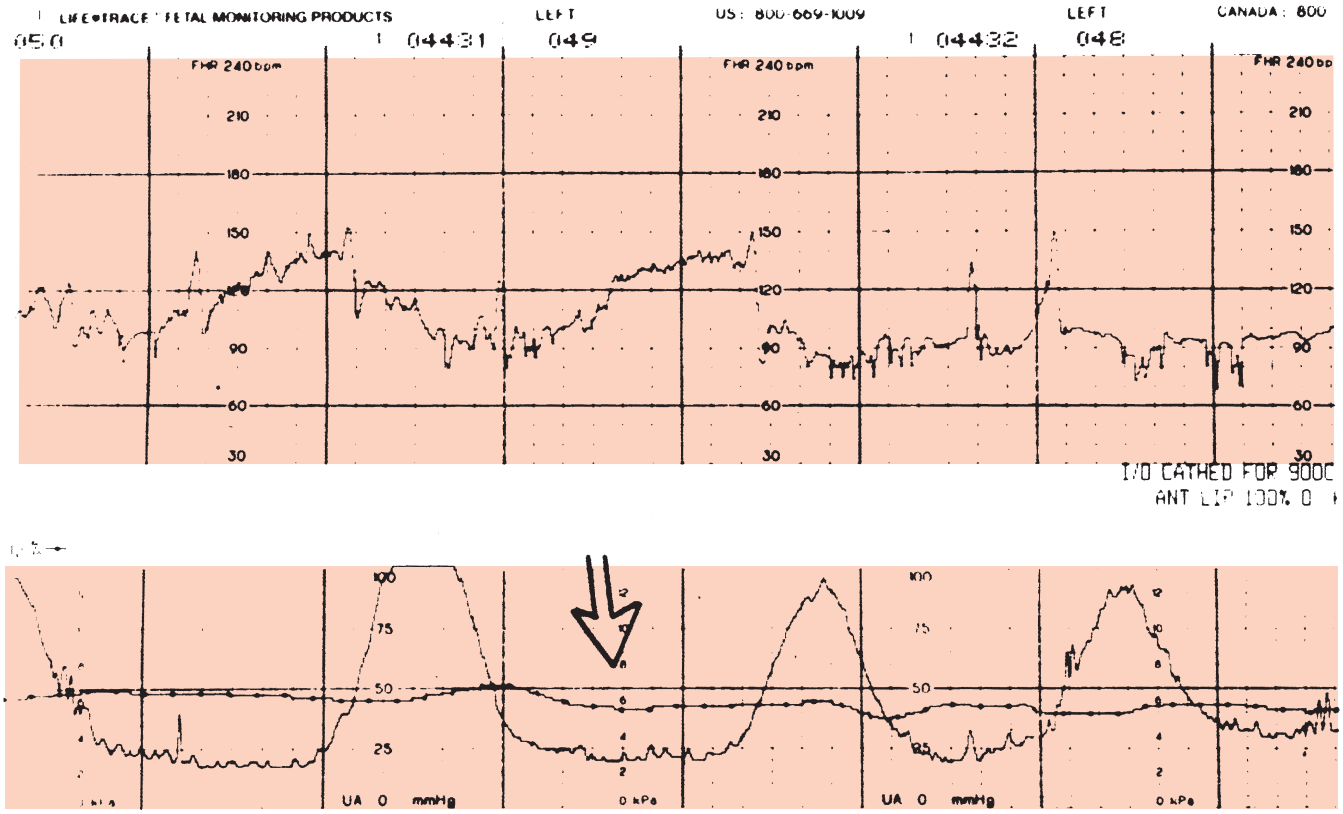

FIGURE 16-19. Superimposed on the contraction monitor tracing is a continuous tracing using a fetal pulse oximeter. The tracing shows an fetal oxygen saturation value ranging from 50% to 40% (normal, 35% to 60%). Note the consistently normal saturation values despite the prolonged fetal heart rate decelerations to 80 beats/minute.

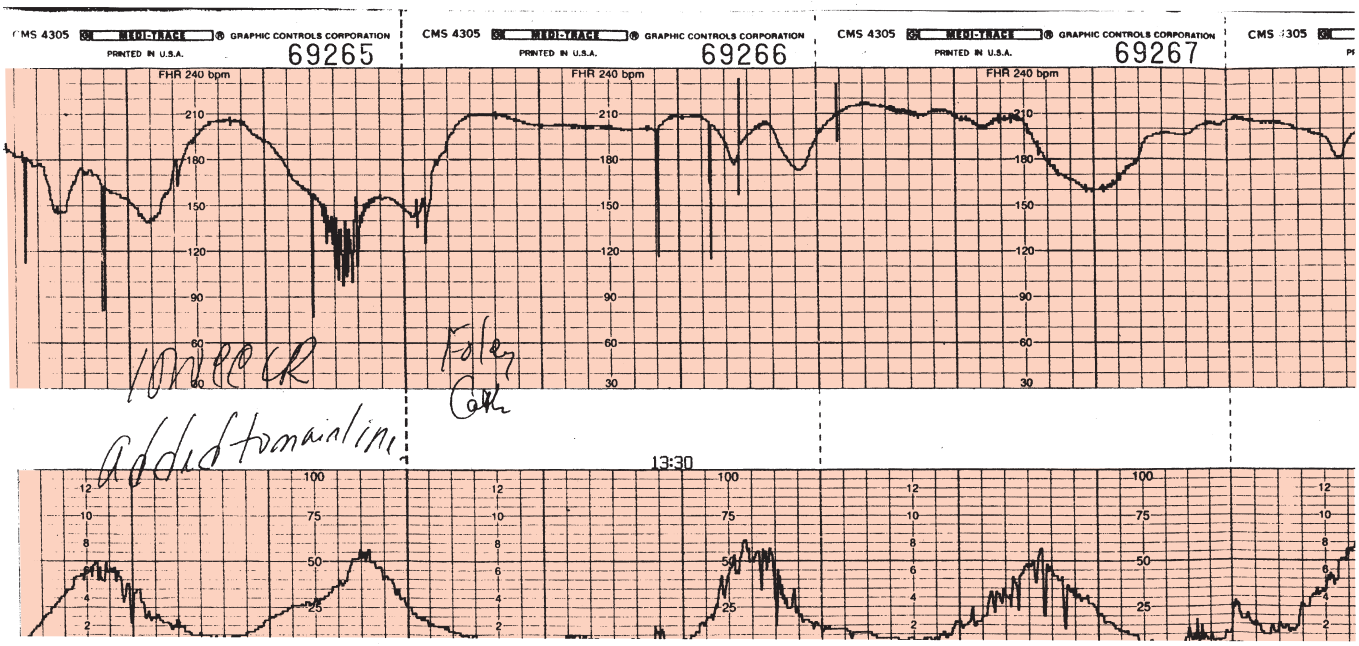

FIGURE 16-20. The loss of variability and tachycardia in association with these variable decelerations makes this a non-reassuring fetal heart rate pattern (NICHD Category III).[17]

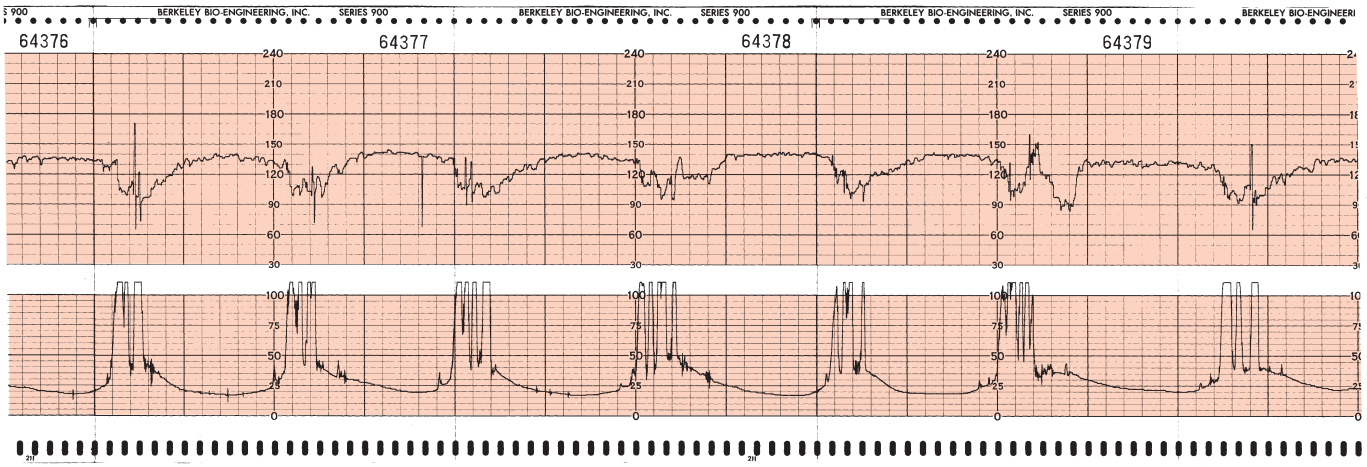

FIGURE 16-21. These variable decelerations, although mild in depth and duration, are associated with a slow, rather than abrupt, return to baseline. This may be a sign of developing hypoxia as a result of repetitive umbilical cord compression with inadequate opportunity to reoxygenate between events. Generally, this finding makes the variable decelerations non-reassuring.

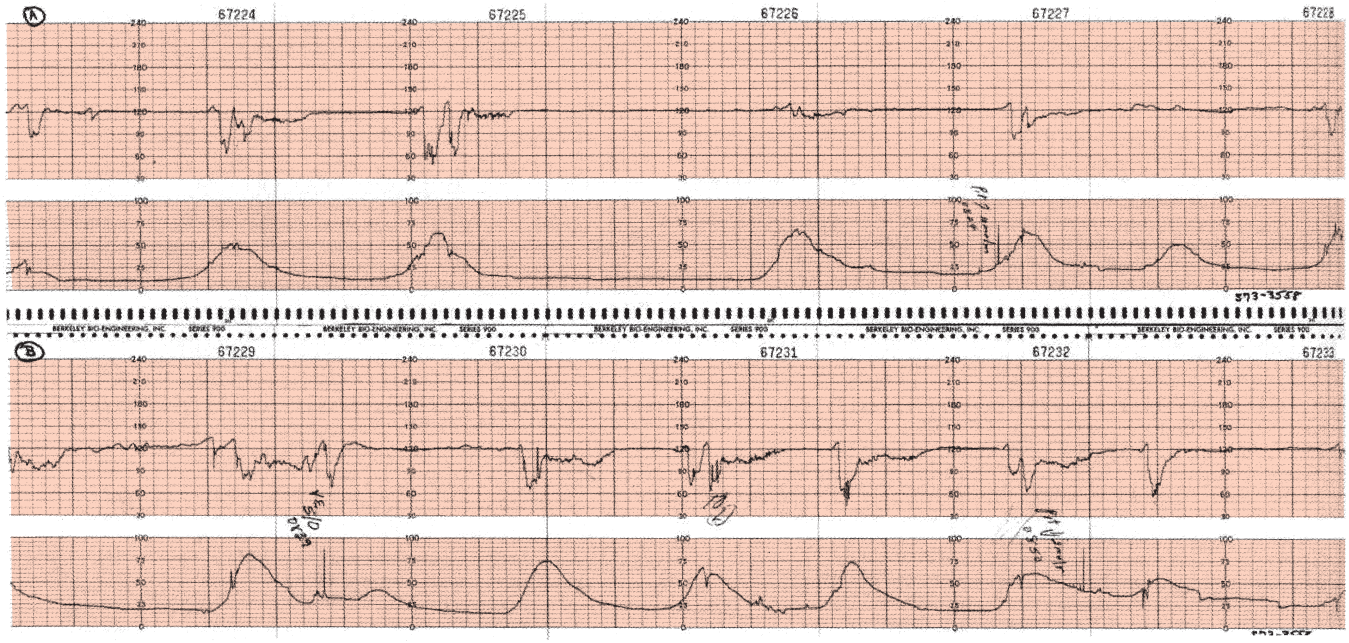

FIGURE 16-22. Here are repetitive variable decelerations with a slow return to baseline. However, the minimal depth and duration of the variable decelerations and the fact that one can see an independent late deceleration with the third contraction would suggest that this pattern is actually a combined one of mild variable and persistent late decelerations.

CLASSIFICATIONS OF THE SEVERITY OF VARIABLE DECELERATIONS

Mild
- Deceleration of a duration of less than 30 seconds, regardless of depth
- Decelerations not below 80 beats/minute, regardless of duration

Moderate
- Deceleration with a level below 80 beats/minute

Severe
- Deceleration to a level below 70 beats/minute or more than 70 beats/minute below the baseline for more than 60 seconds

Data from Kubli FW, Hon EH, Khazin AE, et al: Observations on heart rate and pH in the human fetus during labor. Am J Obstet Gynecol 104:1190, 1969.

benign decelerations. Such cases are virtually always seen in association with absent FHR variability, and they can also be followed by a blunted acceleration following the return to baseline described by Goodlin and Lowe as "overshoot" (Figure 16-23).[31] This is a rare situation and is only seen when all criteria are met, including absent variability, blunted variable decelerations, no acceleration preceding the variable deceleration, and no other spontaneous accelerations of the FHR.

There are four categories of causes of cord compression patterns that are useful to consider from a management standpoint. Variable decelerations appearing early in labor are often caused by oligohydramnios. Other variable decelerations often first appear when the patient reaches 8 to 9 cm of dilation, the time in labor when the curve for descent of the presenting part becomes steep. This is

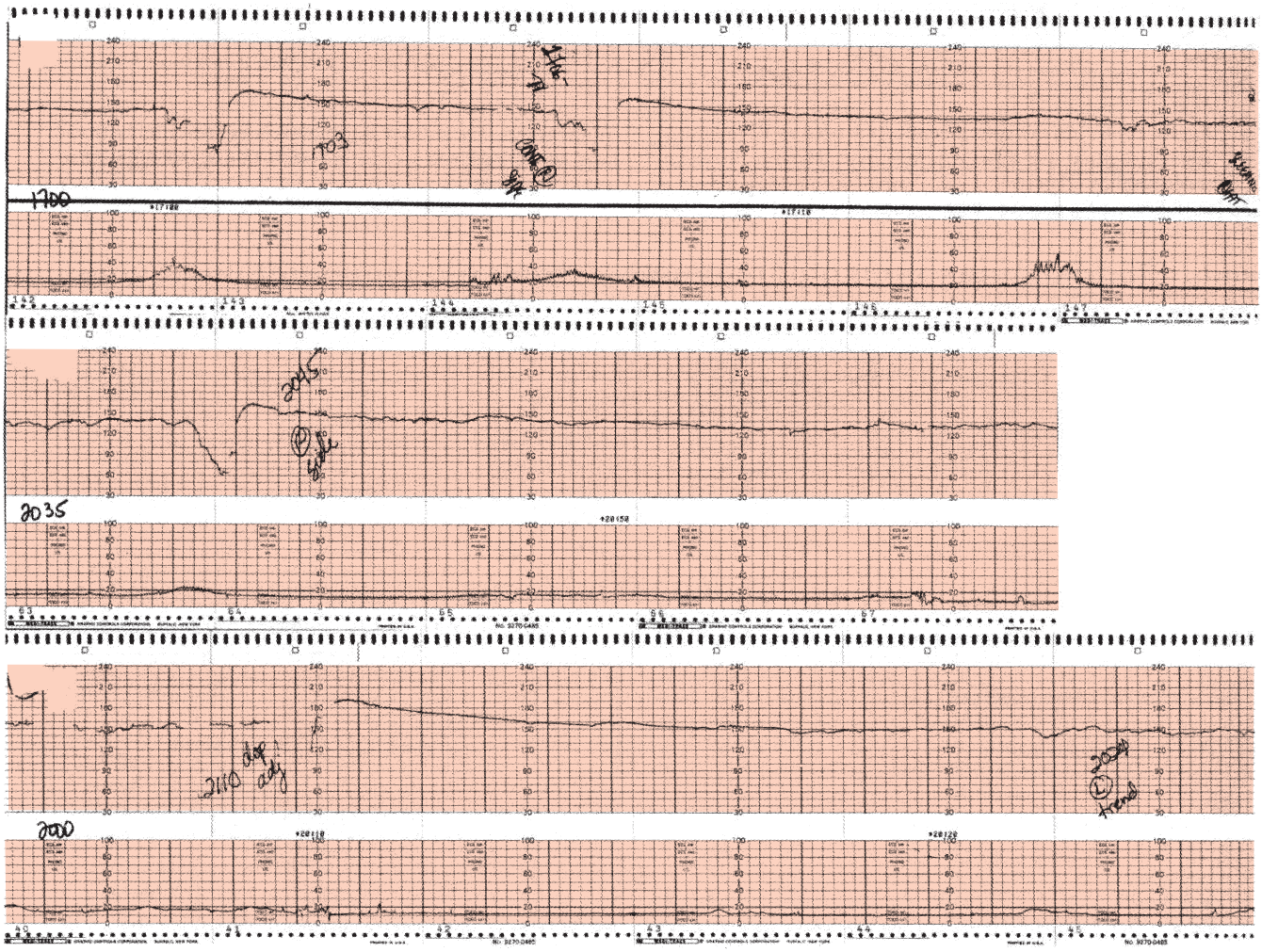

FIGURE 16-23. The accelerations following these variable decelerations, without any acceleration preceding, in association with absent variability fulfill the criteria to describe the accelerations as "overshoot." Such a finding can be ominous and is often associated with marked metabolic acidosis.

probably most often because of nuchal cords, wherein the cord becomes stretched with descent of the fetal head, and as previously described, cord stretch is a profound stimulus for vasospasm in the cord vessels.[16] Unusual types of abnormal umbilical cords, such as short cords, true knots, velamentous cord insertion, cord looped around the extremities, and occult cord prolapse, will produce variable decelerations. And finally, the rarest form of cord compression, and one that usually requires rapid cesarean delivery, is true umbilical cord prolapse.

Because the umbilical cord is most analogous to the adult trachea, interruption in cord flow results in both retention of CO_2 and cessation of O_2 delivery. When this becomes progressive, the intermittent compression often first leads to a progressive increase in fetal CO_2, which results in a respiratory acidosis. If the cord compression continues and also is sufficiently severe to cause insufficient delivery of oxygen, then a metabolic acidosis can also develop. Thus, in a fetal acidosis resulting from cord compression, the acidosis can be respiratory or combined respiratory and metabolic, but should not be metabolic alone.

Prolonged Decelerations

Prolonged decelerations are sporadically occurring decelerations lasting 90 to 120 seconds or more. The NICHD Research Planning Workshop proposed that prolonged decelerations be defined as those lasting 2 to 10 minutes and that beyond 10 minutes this is a baseline change.[17] In most cases, the sudden drop in FHR is the result of some adverse afferent stimulus caused by the brain detecting changes in oxygenation, blood pressure, or even intracranial pressure, as with sustained head compression. Unlike the other three decelerations, in which the type of deceleration defines the pathophysiologic mechanism, prolonged decelerations may be caused by virtually any of the mechanisms previously described, but usually are of a more profound and sustained nature. Prolonged umbilical cord compression, profound placental insufficiency, or even sustained head compression may lead to prolonged decelerations.

The presence of and severity of hypoxia are thought to correlate with the following variables with a prolonged deceleration: the depth and duration of the deceleration, how abruptly it returns to baseline, how much variability is

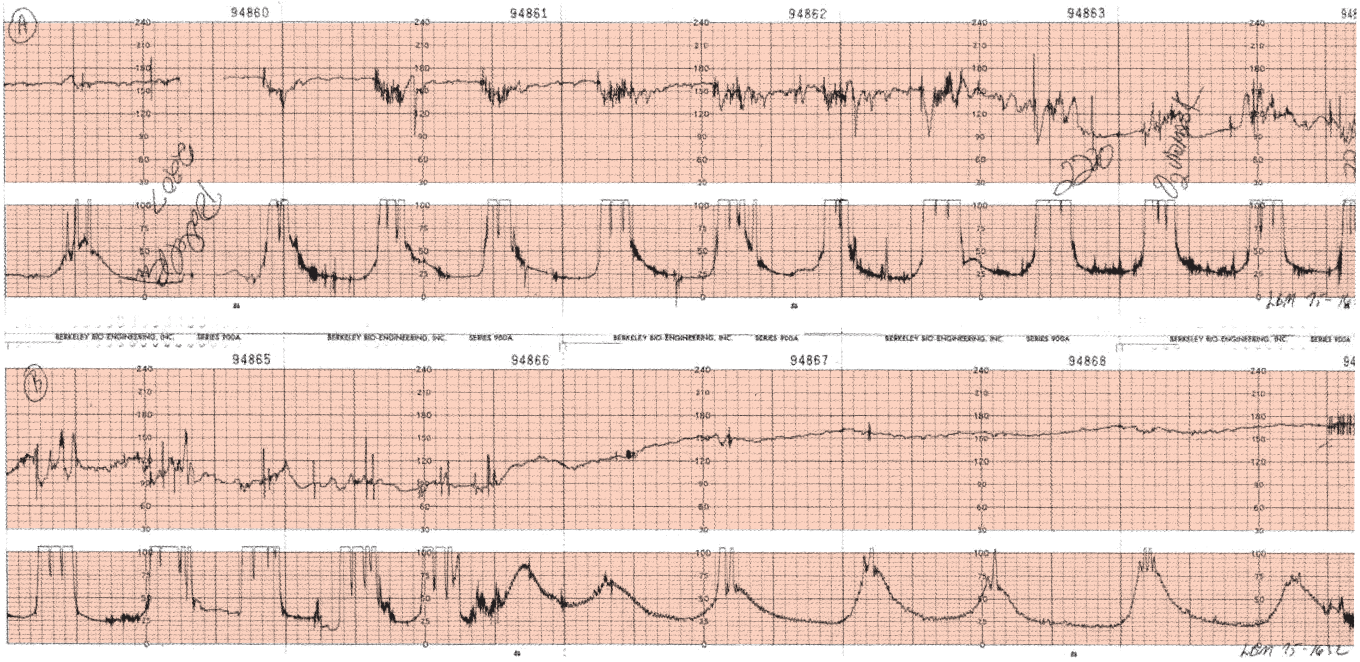

FIGURE 16-24. Often following a prolonged deceleration, as with the one shown in this figure, there is a temporary period of tachycardia and loss of variability. Such a response would suggest that this was a significant hypoxic event. The etiology of this deceleration is not clear but may have been a result of excessive uterine contractions compounded by maternal pushing.

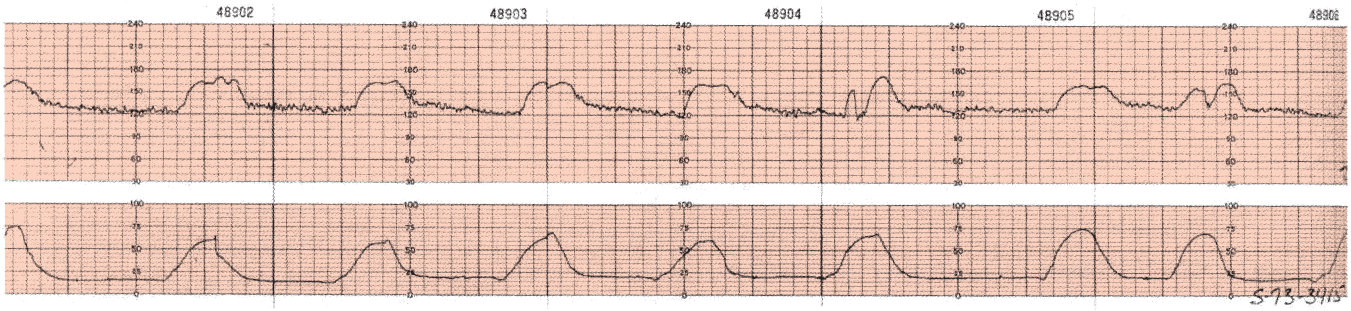

FIGURE 16-25. These are accelerations of the fetal heart. They are usually seen with fetal movement and are often coincident with uterine contractions as well, as in this patient.

lost during the deceleration, and whether there is a rebound tachycardia and loss of variability following the deceleration (Figure 16-24). Examples of the more profound stimuli that may result in this type of deceleration include prolapsed umbilical cord or other forms of prolonged cord compression, prolonged uterine hyperstimulation, hypotension following conduction anesthesia, severe degrees of abruptio placentae, uterine rupture, paracervical anesthesia, an eclamptic seizure, and rapid descent through the birth canal. Occasionally, less severe stimuli such as examination of the fetal head, Valsalva maneuvers, or application of a scalp electrode may cause milder forms of prolonged decelerations.

Accelerations

Accelerations are periodic changes of the FHR above the baseline (Figure 16-25) **defined as an transient increase of the FHR lasting at least 15 seconds and rising 15 beats or more above the baseline.** Because decelerations are of lower amplitude in more premature gestations, the definition of accelerations in the fetus before 32 weeks are those of an amplitude of 10 beats/minute or more lasting 10 or more seconds. They are not classified by type. Except for those accelerations previously described that are associated with variable decelerations, virtually all accelerations are a physiologic response to fetal movement.[32] Accelerations are usually short in duration, lasting no more than 30 to 90 seconds, but in an unusually active fetus, they can be sustained as long as 30 minutes or more. Again, the NICHD Research Planning Workshop disagrees somewhat on this definition, in that they propose that accelerations of more than 10 minutes be defined as a change in baseline.[17] This definition may not always be consistent with the actual fetal physiology because one can on occasion see sustained accelerations (Figure 16-26) that are associated with an actively moving fetus, and when the fetus becomes quiet, the FHR will return to baseline. It is important that this acceleration not be confused with a

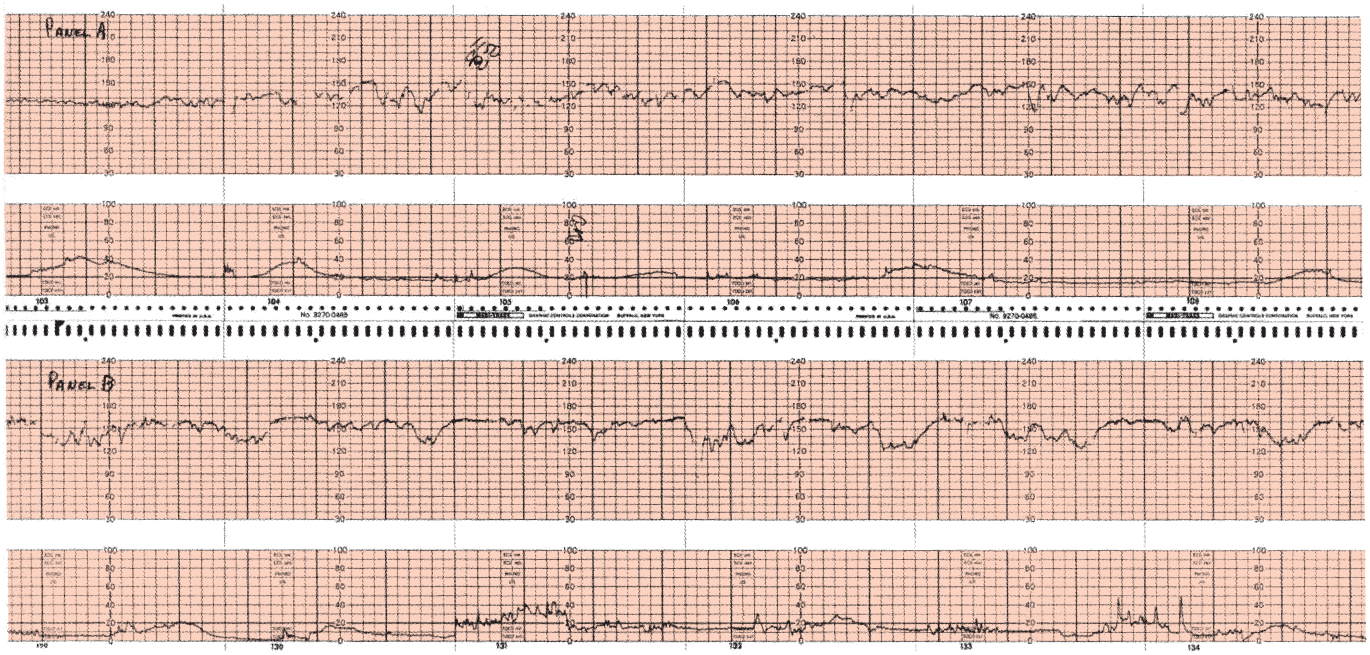

FIGURE 16-26. This figure shows prolonged and repetitive accelerations, especially in the lower panel. Accelerations that are sustained or confluent can be easily confused with a tachycardia, and the return to baseline can be confused with decelerations.

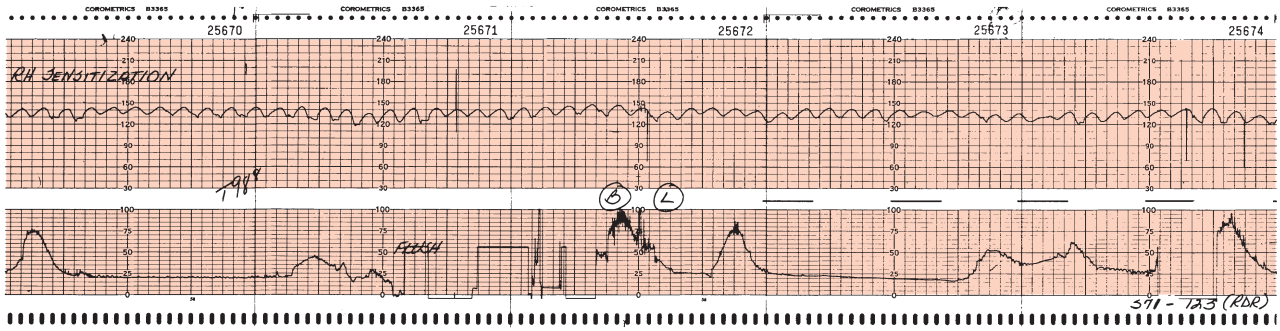

FIGURE 16-27. The sinusoidal heart rate pattern with its even undulations is demonstrated. Internal monitoring shows the absence of beat-to-beat variability characteristic of true sinusoidal patterns.

baseline change because sustained accelerations, which are consistent with a well-oxygenated, vigorous fetus, can be confused visually with fetal tachycardia, and the return of the FHR to the original baseline can be confused with decelerations.

The presence of accelerations has virtually the same meaning as normal FHR variability, but the absence of accelerations means only that the baby is not moving. Because accelerations can be quantified in beats per minute above the baseline and duration, their presence is less subjective than quantifying FHR variability. Clark and coworkers made the observation that in fetuses having otherwise non-reassuring FHR patterns, the presence of accelerations virtually always ruled out a pH less than 7.20 on scalp sampling.[33] Subsequently, these authors and others have confirmed that the presence of spontaneous accelerations or accelerations induced by stimulation of the fetal scalp or acoustic stimulation with a vibroacoustic stimulator has the same reliability.[34,35] If there is no acceleration in the face of an otherwise concerning (category II) FHR, most studies have shown that about 50% of these fetuses have an acidotic pH value on scalp sampling. However, the absence of accelerations in a fetus with a category I or otherwise reassuring pattern rarely indicates fetal acidosis.

Sinusoidal Patterns

This pattern was originally described by Kubli and colleagues in 1972[36] and Shenker in 1973[37] and is rare, but significant. This pattern is strongly associated with fetal hypoxia, most often seen in the presence of severe fetal anemia. Using strict criteria for this pattern, defined by Modanlou and associates, there will be a high correlation with significant fetal acidosis and/or severe anemia.[38] These criteria for identifying a sinusoidal FHR include (1) a stable baseline FHR of 120 to 160 beats/minute with regular sine wave–like oscillations, (2) an amplitude of 5 to 15 beats/minute, (3) a frequency of 2 to 5 cycles/minute, (4) fixed or absent short-term variability, (5) oscillation of the sine wave above and below the baseline, and (6) absence of accelerations (Figure 16-27). **The NICHD has defined the sinusoidal pattern more simply as one "having**

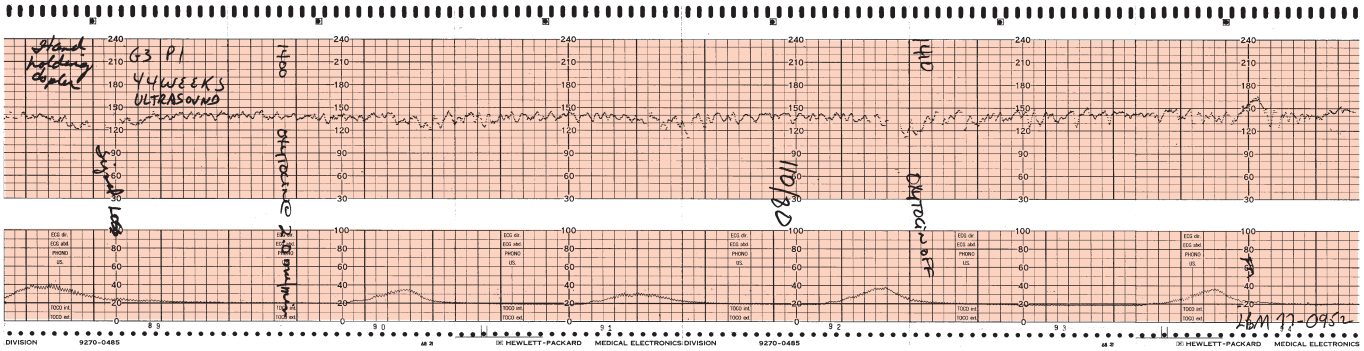

FIGURE 16-28. More common than sinusoidal tracings are those that are actually normal but can easily be confused with a sinusoidal pattern. There are often small accelerations or above-average variability that mimics the sine wave–like pattern. It is important to use the strict criteria defined in the text before interpreting a pattern as sinusoidal.

a visually apparent, smooth, sine wave-like undulating pattern in FHR baseline with a cycle frequency of 3-5 per minute lasting for 20 minutes or more." The pathophysiology of the sinusoidal pattern has been elucidated by Murata and colleagues, who correlated the pattern with levels of fetal arginine vasopressin and subsequently reproduced the pattern with vagotomy and injection of arginine vasopressin.[39] Arginine vasopressin is elevated with hemorrhage or acidosis, and it appears that in such situations with a severely compromised fetus and little vagal activity, the hormone directly affects the fetal heart, and this FHR pattern results. The sinusoidal pattern has also been described after injection of certain narcotics such as butorphanol (Stadol)[40] and meperidine (Demerol).[41] Unfortunately, there are relatively commonly seen FHR patterns that mimic sinusoidal patterns (pseudosinusoidal) and are associated with well-oxygenated and nonanemic fetuses.[38] These can easily be confused with sinusoidal FHR patterns (Figure 16-28); therefore, it is quite important to strictly apply all six criteria before calling a pattern sinusoidal.

Additional Terminology from the NICHD Workshop

In addition to those definitions provided previously, there are some additional points made in the accepted terminology for FHR patterns that are important to include.[17] When describing decelerations, they are termed *recurrent* if they occur with 50% or more of contractions and *intermittent* if they occur with less than 50% of contractions in any 20-minute segment of the tracing.

Uterine contractions are quantified as the number of contractions present in a 10-minute window averaged over 30 minutes. Normal contraction frequency is five or fewer per 10 minutes, and tachysystole is six or more. The Workshop advised abandonment of the terms *hyperstimulation* and *hypercontractility*.

TABLE 16-1 THREE-TIERED FETAL HEART RATE INTERPRETATION SYSTEM

Category I

Category I fetal heart rate (FHR) tracings include all the following:

- Baseline rate: 110-160 beats/min
- Baseline FHR variability: moderate
- Late or variable decelerations: absent
- Early decelerations: present or absent
- Accelerations: present or absent

Category II

Category II FHR tracings include all FHR tracings not categorized as category I or category III. Category II tracings may represent an appreciable fraction of those encountered in clinical care. Examples of category II FHR tracings include any of the following:

Baseline Rate

- Bradycardia not accompanied by absent baseline variability
- Tachycardia

Baseline FHR Variability

- Minimal baseline variability
- Absent baseline variability not accompanied by recurrent decelerations
- Marked baseline variability

Accelerations

- Absence of induced accelerations after fetal stimulation

Periodic or Episodic Decelerations

- Recurrent variable decelerations accompanied by minimal or moderate baseline variability
- Prolonged deceleration: >2 minutes but <10 minutes
- Recurrent late decelerations with moderate baseline variability
- Variable decelerations with other characteristics, such as slow return to baseline, "overshoots," or "shoulders"

Category III

Category III FHR tracings include either:

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

From Macones GA, Hankins GD, Spong CY, et al: The 2008 National Institute of Child Health and Human Development Workshop Report on Electronic Fetal Monitoring. Obstet Gynecol 112:661-666, 2008.

Evolution of Fetal Heart Rate Patterns

One of the sources of greatest confusion regarding FHR pattern interpretation and management is that the patterns have very poor specificity in terms of predicting fetal hypoxia and acidosis, newborn depression, or need for resuscitation (Table 16-1). **When patterns are normal or "reassuring"** (category I), there is almost always normal oxygenation, and the baby is born vigorous, with normal pH and Apgar scores. However, when the pattern is non-reassuring (category II), the baby is more often normal than depressed or acidotic.

In addition to the inherent problem we have in trying to use EFM, a nonspecific modality, to determine fetal oxygenation, there is another reason why many studies have demonstrated such poor correlation with adverse

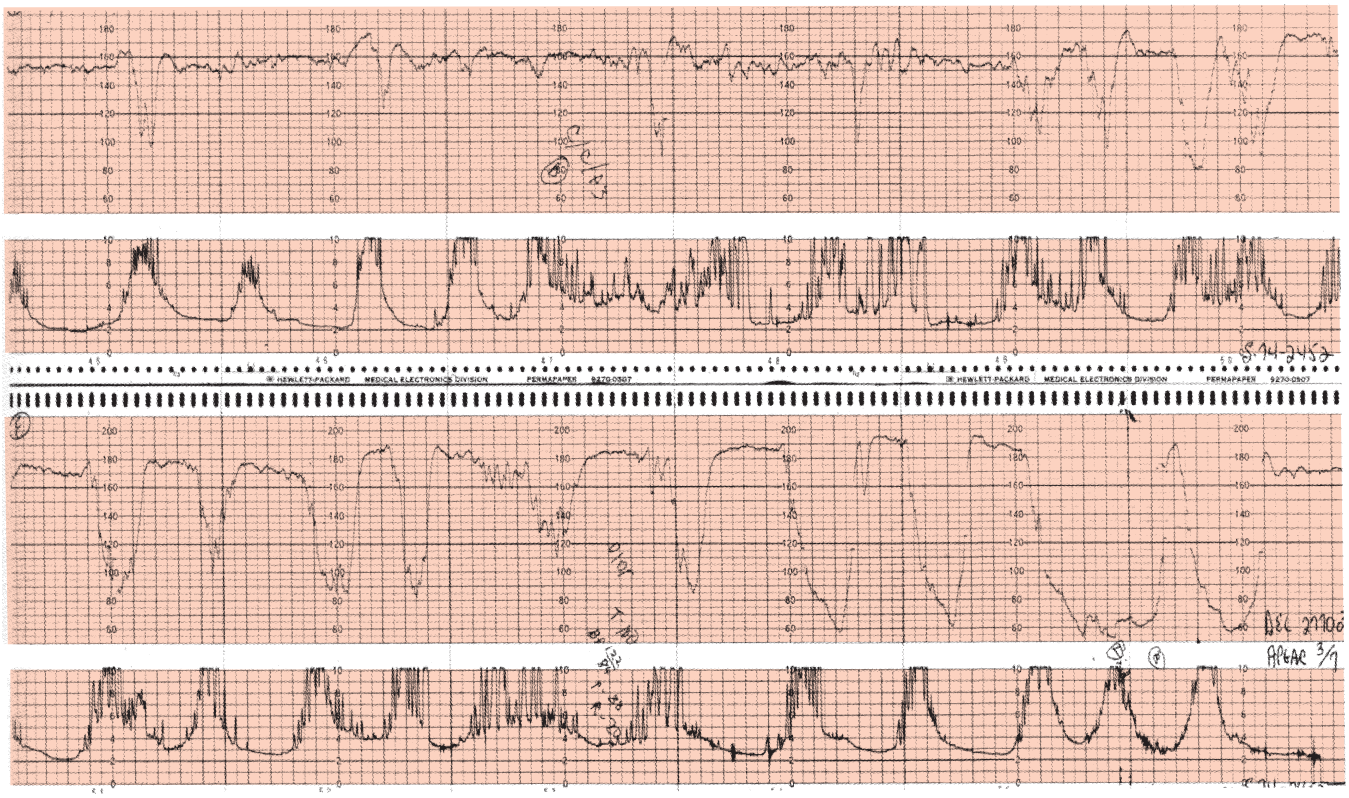

FIGURE 16-29. Loss of variability and tachycardia should only be interpreted as indicative of hypoxia and developing acidosis when they are associated with decelerations that suggest progressive hypoxia as with these variable decelerations, which are becoming deeper and more prolonged.

perinatal outcome. Investigators have tried to correlate specific FHR findings in isolation without taking into account the expected evolution of the FHR patterns.[19,30] For example, if one attempts to correlate FHR variability with acidosis or depression, normal variability will correlate well with a normal pH and normal Apgar scores, but reduced or absent variability will correlate poorly with acidosis or depression. This is partly because of the multiple causes of reduced variability. However, in the fetus with persistent late decelerations who then loses FHR variability, the correlation with acidosis and depression should improve substantially because there was evidence that *hypoxia led to the acidosis*. This is a critical concept in understanding FHR monitoring. Murata and coworkers demonstrated that, in the fetal monkey whose oxygenation was progressively reduced, late decelerations consistently *preceded* the loss of variability and accelerations.[42] **Decelerations are the indicators of hypoxia. If hypoxia is the cause of the reduced variability, then those decelerations indicative of hypoxia should *precede* the development of tachycardia, loss of variability, or disappearance of accelerations** (Figure 16-29). Furthermore, the duration and appearance of the decelerations should be of sufficient magnitude to suggest that CNS depression could have resulted.

Therefore, the fetus who develops one of the latter variables (e.g., loss of accelerations) in the absence of decelerations is not likely to have hypoxia and another cause should be considered (e.g., drugs, sleep cycle). It must be remembered that when the fetus demonstrates one of these baseline changes or absence of accelerations on admission, it is not possible to know whether evidence of hypoxia preceded these changes. In addition, this approach does not apply antepartum, when the nonstress test is used. Contractions are not present, and there will not be an opportunity to assess for the presence or absence of decelerations in response to contractions.

MANAGEMENT OF NON-REASSURING FETAL HEART RATE PATTERNS

Traditionally, a FHR pattern that suggested fetal hypoxia would be called fetal distress if it was sufficiently concerning to warrant immediate operative intervention. But, as previously mentioned, in most cases, when a cesarean or forceps delivery was done for "fetal distress," the fetus was delivered without evidence of significant hypoxia or acidosis. This has led to the recommendation from the ACOG to use the term *NRFS*[13] and subsequently to the *three-tiered system*. This is also descriptive of the approach to the management of the fetus with concerning FHR patterns. That is, when the FHR pattern is suggestive of hypoxia, and therefore non-reassuring, other means of reassurance should be used when possible.

Interventions for Non-reassuring Fetal Status

The ideal intervention for fetal hypoxia is a cause-specific, noninvasive one that permanently reverses the problem. Although not always possible, this should certainly be the goal. Obviously, the first step in achieving this goal is to

recognize the cause of the abnormal FHR pattern. A thorough knowledge of the pathophysiology of FHR changes, coupled with a careful clinical patient evaluation and a knowledge of common causes of specific FHR changes, will maximize the opportunity for this goal to succeed. In addition to cause-specific types of interventions, virtually all cases of hypoxia should theoretically also benefit by more generic interventions that have the potential to maximize oxygen delivery and placental exchange.

Nonsurgical Interventions

Oxygen Administration

One of the most obvious ways to maximize oxygen delivery to the fetus is to give additional oxygen to the mother. Whereas diffusion across a membrane is driven by P_{O_2} as opposed to oxygen content, and whereas maternal P_{O_2} can be raised substantially with mask O_2, it is not well established that fetal P_{O_2} is raised substantially by routine maternal oxygen administration. In the classic study by Khazin, Hon, and Hehre,[43] late decelerations were alleviated within a few minutes by administration of oxygen to the mother. Later studies showed beneficial effects with decreased FHR variability and tachycardia.[44,45] In 2003, Fawole and Hofmer[46] reviewed the available studies for the *Cochrane Database of Systematic Reviews* and concluded that there was not sufficient evidence that maternal oxygen administration improved fetal oxygenation and that maternal oxygen administration could not be supported at the time. Recent evidence, from fetuses being monitored with pulse oximetry, demonstrated that fetal oxygen did increase significantly in patients with both a regular face mask and even more so with a non-rebreathing face mask and that fetuses with the lower initial oxygen saturation increased the most.[47,48] Simpson and James[49] found similar results with oxygen administration, showing a 15% increase with normal baseline oxygen saturations and a 26% increase with a baseline oxygen saturation of less than 40%. These studies are physiologically plausible because the larger the difference in oxygen tension between the mother and the fetus, the larger the effect that would be expected to be seen in the fetal oxygen tension and saturation. **Routine oxygen administration by face mask has become such standard practice with non-reassuring FHR patterns that it is difficult to recommend otherwise until further studies are available to substantiate or refute current knowledge.**

Lateral Positioning

Ideally, all patients should labor in the lateral recumbent position, at least from the standpoint of maximizing uterine perfusion. The reasons for this are, at least theoretically, twofold: (1) in being inactive and recumbent, the body is required to deliver the least amount of blood flow to other muscles; and (2) in the lateral position, there is no compression by the uterus on the vena cava or aorta, thus maximizing cardiac return and cardiac output. Several studies have confirmed the beneficial effects of lateral maternal positioning.[50,51] In the study by Simpson and James,[49] patients were randomized to six positions, and fetal oxygen saturation was highest in both right and left lateral recumbent positions compared with the supine position by an average of 29%.

Hydration

Most patients in labor are either restricted or prohibited from taking oral fluids for fear of requiring an urgent operative delivery in the presence of a full stomach. If not fluid restricted, individuals involved in sustained exercise, and possibly by inference in active labor, do not voluntarily ingest adequate amounts of fluid because of a phenomenon called *autodehydration*.[52] In addition, recent evidence would suggest that the usual amount of intravenous fluid of 125 mL/hour is a gross underestimate of the replacement required in labor.[53] Thus, by increasing fluid administration, there is the potential to maximize intravascular volume and thus uterine perfusion.

Oxytocin

In a patient with a non-reassuring pattern, the more time there is between contractions, the more time there is to maximally perfuse the placenta and deliver oxygen. **In patients receiving oxytocin, there is potential to improve oxygenation by decreasing or discontinuing oxytocin.** Often, however, this becomes a difficult situation because many patients will stop progressing in labor in terms of continued dilation and descent if the oxytocin is discontinued. It is often necessary to restart the oxytocin, and this may be appropriate, especially if there are accelerations or other means to document the absence of acidosis. Written documentation explaining the necessity and appropriateness of continuing oxytocin in this situation is especially important. The situation with patients who develop persistently non-reassuring patterns, especially with loss of accelerations or absence of other reassurance, and who require discontinuation of oxytocin, but then fail to progress because adequate contractions cannot be sustained, is often referred to as *fetal intolerance to labor*; although this is not an endorsed term in the newer terminology.

Tocolytics

There are numerous references in the obstetrical literature to the use of tocolytics to maximize oxygen delivery, by essentially the same mechanism described in the earlier paragraph on discontinuing oxytocin.[54-57] Tocolytics are appropriate when patients are having spontaneous excessive contractions leading to non-reassuring FHR patterns, especially prolonged decelerations (Figure 16-30). Different tocolytics have been described, but the most commonly used one, and perhaps the one that provides the most rapid response, is subcutaneous terbutaline, 0.25 mg. Tocolytics have also been used for intrauterine resuscitation after the decision is made to perform an operative delivery while waiting for preparations to be made. Subcutaneous terbutaline in this latter setting has been demonstrated to improve Apgar scores and cord pH values without apparent complications such as postpartum hemorrhage.[58]

Amnioinfusion

In situations in which variable decelerations appear to be caused by oligohydramnios, reestablishing intrauterine fluid volume by a process called *amnioinfusion* has been demonstrated in numerous randomized studies to ameliorate the variable decelerations, improve Apgar scores and

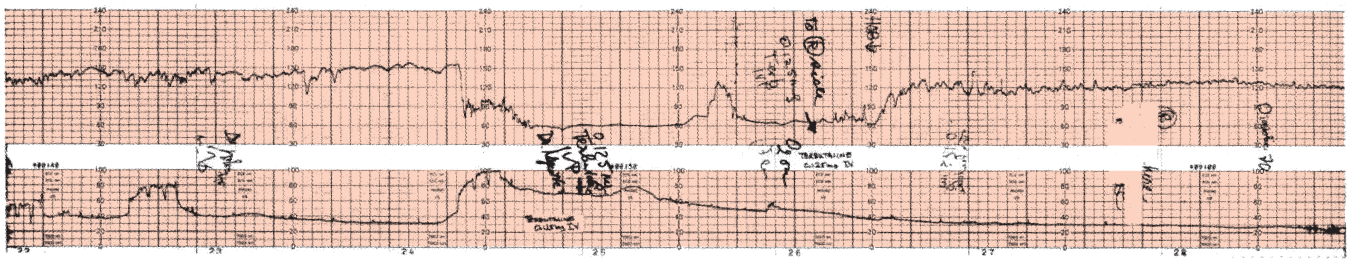

FIGURE 16-30. This prolonged deceleration in a patient with a spontaneous prolonged contraction is treated with subcutaneous terbutaline with apparent resolution of both the contraction and the deceleration.

cord pH values, and even reduce the need for cesarean delivery for NRFS.[59-61] Reference to this idea can be found as far back as 1925,[62] but it was rediscovered and proposed by Miyazaki and Taylor in 1983.[63] Intrauterine pressure catheters are now made with a port that allows the simultaneous administration of saline to accomplish this goal. Thus, in the patient with variable decelerations that suggest progression to more non-reassuring types, and in whom the likely cause is oligohydramnios, the implementation of amnioinfusion is warranted. What has not yet been established is whether in some situations amnioinfusion should be started prophylactically when there is an unusually high risk for the development of variable decelerations from oligohydramnios, such as preterm premature rupture of membranes (PROM).

Theoretically, using amnioinfusion before the onset of the decelerations in certain fetuses, such as very premature ones or those with intrauterine growth restriction that will progress to acidosis and depression much more rapidly with cord compression, will prevent the rapid evolution of hypoxia and acidosis. No studies are available as of yet to compare therapeutic and prophylactic amnioinfusion. Amnioinfusion also has been proposed, in several prospective randomized trials, to be used to avoid the fetal and neonatal pulmonary problems in the presence of meconium.[64] The theory behind this use of amnioinfusion is that (1) it dilutes the meconium by increasing fluid volume, and (2) by avoiding fetal gasping, which can occur with significant hypoxic episodes (i.e., sustained cord compression), the likelihood of meconium aspiration before delivery is reduced. Although initial studies of the benefits of amnioinfusion in the setting of meconium were promising and for a time its use for this indication gained widespread implementation,[65] recent studies have been less supportive. A review of this subject for the Cochrane database[66] including 13 studies of more than 4000 women concluded there was no reduction in meconium aspiration, perinatal death, or severe morbidity, except in an unexplained subgroup of studies done in populations in which "limited peripartum surveillance" was available.

MECONIUM

The presence of meconium is an extremely confusing issue when evaluating the fetus in labor. The quandary arises from the fact that although a hypoxic insult eliciting a significant vagal response from the fetus often results in the passage of meconium from the fetal gut, passage of meconium can also occur in the absence of any significant or sustained hypoxia. Meconium is not only a potential sign of fetal hypoxia but is also a potential toxin if the fetus aspirates this particulate matter with a gasping breath in utero or when it takes its first breaths following birth. **The thickness of the meconium is also a reflection of the amount of amniotic fluid, and thick meconium virtually always reflects some degree of oligohydramnios.** Thus, there may be a vicious cycle in such a situation. Oligohydramnios often leads to cord compression; the vagal response to cord compression may also lead to further passage of meconium, but also when sustained or prolonged, it may lead to fetal gasping, increasing the likelihood that meconium aspiration can occur before birth. Furthermore, because oligohydramnios may be an indicator of failing placental function, meconium may also indicate that the fetus is at risk for placental insufficiency. **In general, meconium should alert the clinician to the potential for oligohydramnios, umbilical cord compression, placental insufficiency, and meconium aspiration.** Fortunately, a reassuring FHR tracing is generally reliable, and patients with meconium can be managed expectantly. But in the presence of meconium, especially thick meconium, the risk factors associated with meconium should be entered in the equation when managing relatively non-reassuring patterns, as should all clinical variables.

Alternatives for Evaluating the Fetus with a Non-reassuring Fetal Heart Rate Pattern

In the fetus with a persistently non-reassuring FHR pattern, when nonsurgical efforts at reversing or improving the pattern fail, the next step is to attempt to find out whether the hypoxia has progressed to metabolic acidosis.

FETAL SCALP pH

Determination of fetal scalp pH is historically the oldest and most well-tested method for determining whether the fetus is acidotic. Technically, a plastic cone is inserted transvaginally against the fetal vertex. The cervix needs to be at least 4 to 5 cm dilated and the vertex at a –1 station or below to accomplish this. Mineral oil or another lubricant is applied to the scalp so that blood will bead, and then using a lancet, the scalp is pricked and blood is then collected in a long capillary tube (Figure 16-31). The tube will hold about 100 µL of blood, and about 30 µL is needed to perform a pH test alone and 70 µL to determine P_{CO_2} as well. To determine whether an acidosis is metabolic or respiratory, the P_{CO_2} is needed. This is an important distinction because the question being asked is whether there

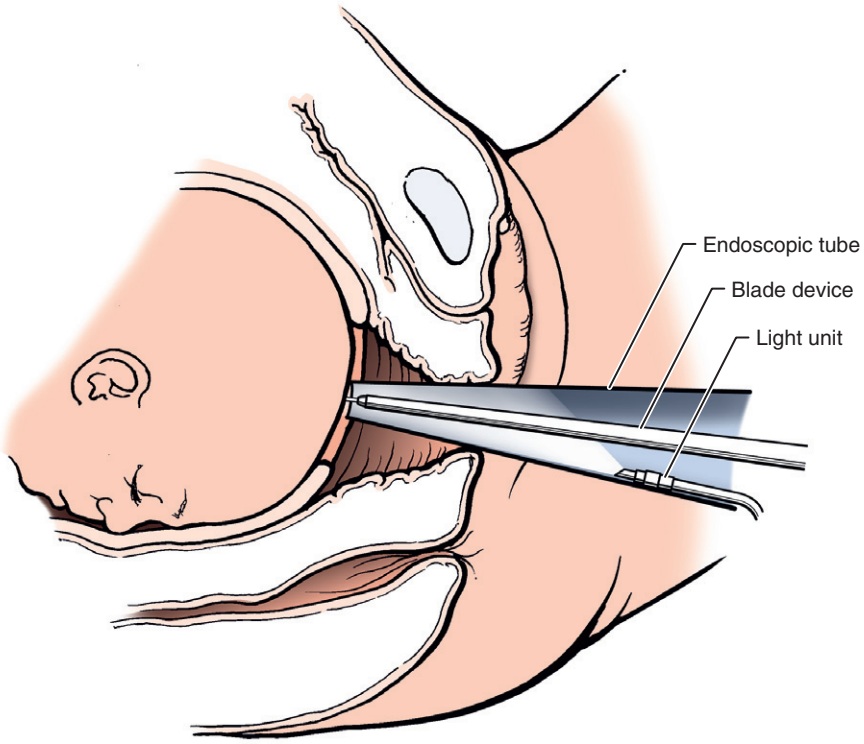

FIGURE 16-31. Technique of fetal scalp blood sampling.

has been sufficient hypoxia to lead to metabolic acidosis. Respiratory acidosis is far less concerning, but without determining PCO_2, this cannot be sorted out. Unfortunately, it is difficult to obtain enough blood for both pH and PCO_2 in most instances. PCO_2 determination is especially important when doing scalp pH for variable decelerations because most acidosis is respiratory in this situation. **A scalp pH less than 7.20 is consistent with fetal acidosis and a pH of 7.20 to 7.25 is borderline and should be repeated immediately.** A reassuring value higher than 7.25 must be repeated every 20 to 30 minutes as long as the pattern persists, and the fetus is not acidotic. In practice, because this technique is cumbersome, fraught with technical inaccuracy, uncomfortable for the patient, and with the requirement to perform repeated samples, scalp pH assessment is used very infrequently.[67] Even in large teaching services accustomed to using this technique, its abandonment, coupled with an appreciation for the utility of accelerations for predicting presence or absence of acidosis, does not appreciably increase the need for operative intervention.[68]

Accelerations and Variability

In the fetus with a non-reassuring pattern, spontaneous accelerations have the same significance as those elicited by scalp or acoustic stimulation. **Thus, any acceleration—spontaneous or induced—indicates the absence of acidosis.** It should be emphasized that the absence of accelerations as well when there is an absence of decelerations or other patterns suggesting hypoxia should not elicit concern. The fetus is often not moving in labor; and from a pathophysiologic perspective, without evidence of hypoxia, the fetus cannot develop a metabolic acidosis. Thus, the application of the interpretation of accelerations should generally be restricted to the fetus with an otherwise non-reassuring FHR pattern, when the question is being asked: Hypoxia is present. Is the fetus now developing a metabolic acidosis? Although moderate variability similarly can also be used to rule out the acidotic fetus, the problem with this choice is that interpretation of variability is not nearly as reliable as it is with accelerations. And although one can reliably wait on a pattern with non-reassuring decelerations such as recurrent late or recurrent severe variable decelerations based on moderate variability alone, one should be sure that there is no question that the FHR variability is at least moderate and that there is consistent agreement of this among the clinicians providing care to the patient.

Fetal Pulse Oximetry

One of the most potentially exciting yet currently disappointing developments in obstetrics was the introduction of fetal pulse oximetry. Because FHR monitoring is specifically intended to monitor fetal oxygenation and because this modality is so nonspecific, it stands to reason that what we should be using ideally is a device that directly monitors fetal oxygenation, pH, or both. Since the mid-1980s, the use of pulse oximetry has revolutionized monitoring of "air-breathing" adults, children, and neonates. Animal studies of this technology validated its accuracy.[69,70] Fetal pulse oximetry when first introduced was approved by the U.S. Food and Drug Administration (FDA) for clinical use. Its value for more accurately assessing fetal oxygenation in labor was promising in initial studies,[71] but not supported in later investigations[72] and ultimately not endorsed by ACOG. As a result, the manufacturer removed the product from the market, and it is no longer in use.

ST Analysis, the STAN Technology

One other promising new technology for improving interpretation of non-reassuring, especially category II, tracings is the introduction of fetal ECG waveform analysis using computerized algorithms detecting changes in the ST waveform which are indicative of fetal metabolic acidosis. Trials in Europe, including several randomized controlled trials usually coupled with scalp pH monitoring, have generally shown a reduction in operative deliveries for concerning FHR patterns and a reduction in the incidence of fetal acidosis.[73-75] An ongoing trial of this technology in the United States by the NICHD Maternal-Fetal Medicine Units Network is currently underway.

Computerized Interpretation of Fetal Heart Rate Patterns

One of the problems with FHR monitoring has been the dismal record of agreement on interpretation of FHR patterns. Intraobserver and interobserver comparisons of interpretation usually achieves only about 30% to 70% agreement.[76] The idea of computer interpretation of FHR patterns has been a goal attempted by many over the history of EFM. Recently, the first FDA-approved EFM computer-based interpretation system has come onto the market. Termed the *PeriCALM EFM system*, this product shows promise in accuracy of assessing which tracings are and are not associated with metabolic acidosis[77] and compares favorably with the agreement of interpretation of FHR patterns with expert interpreters.[78]

Operative Intervention for Non-reassuring Fetal Status

When the fetus is determined to have a persistently non-reassuring FHR pattern and backup methods (e.g., scalp pH, moderate variability, accelerations, ST waveform analysis) cannot provide reassurance that the fetus is not acidotic, operative intervention is indicated to expeditiously deliver the baby to avoid further deterioration. Because the interpretation of FHR patterns has been so inconsistent and the predictability of most non-reassuring patterns has been so poor, the NICHD convened a second Workshop on the Intrapartum Interpretation of EFM.[14] Their proposal, which has since been accepted by ACOG and AWHONN and many hospitals throughout the country, is that FHR patterns be divided into three categories (see Table 16-1). **Category I is strongly predictive of the absence of hypoxia and normal fetal acid-base status, and these patterns require no intervention. Category III patterns are predictive of a fetal metabolic acidosis and require "prompt evaluation."** Moreover, it can easily be stated that if measures at improving category III patterns, such as position change, oxygen administration, fluid boluses, and discontinuing oxytocin, do not improve the pattern, immediate operative delivery is warranted. **Category II includes more than 80% of FHR patterns seen in labor[79] and is said by the Workshop to "require evaluation and continued surveillance and reevaluation taking into account the entire clinical circumstances."** However, there are some substantial limitations of this new system for most patterns in category II. As Parer points out in his editorial on the limitations of this three-tiered system,[79] category II contains a mixture of patterns, some of which are well established to have no association with hypoxia or acidosis (e.g., mild or moderate variable decelerations with moderate variability) and others that have a high association with, or if left unchanged are likely to develop into, a fetus with hypoxia and acidosis (e.g., persistent late decelerations with minimal variability). Furthermore the Workshop recommendations and those adopted by ACOG give us little guidance on management of these FHR patterns in category II beyond "continued surveillance and reevaluation." Therefore, the following issues are raised and suggestions are made in an effort to provide more clearcut guidance on management of FHR patterns, acknowledging fully that the evidence to support these statements may be limited and that other experts may approach these situations differently.

Several questions arise when the decision has been made for intervention for delivery for a concerning FHR pattern. What is the best choice, operative vaginal or cesarean delivery? How much time do we have to perform the delivery? What anesthetic should be used? What is the prognosis of the baby? And, finally, are there situations in which the baby is already damaged or otherwise not likely to benefit from this intervention?

Choosing operative vaginal over cesarean delivery is not difficult if the patient is in early labor. For the patient near or at complete dilation, this becomes a question of judgment. Which route is more likely to create the more rapid delivery while at the same time result in the least complications for mother and baby? When the clinician is unsure whether an attempt at operative vaginal delivery will succeed, the question is even more difficult. This decision will depend not only on the variables that predict success for an operative vaginal delivery (e.g., station, clinical pelvimetry, size of the baby, skill of the clinician), but also on the severity of the FHR pattern and whether there is time to find out whether an operative delivery will succeed. The time for intervention also is a question of judgment. Except for the situation of a prolonged deceleration to less than 70 beats/minute with loss of variability that will not recover and requires the most rapid intervention safely possible, most other situations require judgment and integration of the entire clinical picture of mother and baby.

The question of how much time is available to perform an operative intervention in the face of a non-reassuring FHR pattern is a complex one, muddied not only by the unpredictability of the non-reassuring pattern but also by the medicolegal pressures that have arisen as a result of EFM. **The ACOG recommends that "all hospitals have the capability of performing a cesarean delivery within 30 minutes of the decision to operate,"[80] but that "not all indications for a cesarean delivery will require a 30-minute response time."** The examples given that mandate an expeditious delivery include hemorrhage from placenta previa, abruptio placentae, prolapse of the umbilical cord, and ruptured uterus. In some situations (e.g., sustained prolonged deceleration to <70 beats/minute with loss of variability), 30 minutes may be too long to avoid damage; in others, this may be too restrictive and may result in suboptimal anesthetic choices and compromised preoperative preparation. Thus, a judgment made on the basis of

the severity of the FHR pattern and the overall clinical status of mother and baby must be integrated into this difficult decision.

MANAGEMENT OF NON-REASSURING FETAL HEART RATE PATTERNS: A PROPOSED PROTOCOL

The following algorithm for management of non-reassuring (categories II and III) FHR patterns is proposed:

1. When the pattern suggests the beginning development of hypoxia or is already non-reassuring:
   a. Identify, when possible, the cause of the problem (e.g., hypotension from an epidural).
   b. Correct the cause when possible (e.g., fluids and ephedrine to correct the hypotension).
   c. Give measures to maximize placental oxygen delivery and exchange: oxygen by face mask, lateral positioning, hydration, consider decreasing or discontinuing oxytocin.
2. If the pattern becomes or remains non-reassuring and the above measures have been completed:
   a. Attempt to provide other measures to rule out metabolic acidosis.
      - Accelerations—spontaneous or elicited
      - Moderate variability
      - Scalp pH (if used)
   b. If reassurance using one of the above methods can be provided, and the pattern persists, continuous or intermittent (every 30 minutes) evidence of absence of acidosis must be ascertained.
   c. If reassurance of the absence of acidosis cannot be provided, deliver expeditiously by the safest and most reasonable means (operative vaginal or cesarean delivery).

Category II patterns that qualify as non-reassuring and cannot be corrected and therefore warrant evidence of the absence of metabolic acidosis include the following:

1. Recurrent late decelerations (≥50% of contractions)
2. Non-reassuring variable decelerations
   a. Progressively severe in depth and duration
   b. With developing tachycardia and loss of variability
3. Recurrent prolonged decelerations
4. The confusing pattern
   a. A pattern of absent variability but without explanatory decelerations at the time of initiating monitoring
   b. An unusual pattern that does not fit into one of the categories defined previously but does not have elements of a reassuring pattern.

Several of the previous presentations and others warrant additional discussion. Prolonged decelerations proceeding to bradycardia that will not return to baseline are a potentially ominous situation. Causes of these patterns include virtually any substantial insult that can cause severe hypoxia, especially when the FHR goes below 80 beats/minute. Examples include abruptio placentae, ruptured uterus, cord prolapse or sustained cord compression, profound hypotension, maternal seizure or respiratory arrest, and rapid descent and impending delivery of the fetus. Generally, the following approach to these bradycardias should be taken. First, be patient. Often these are noticed on the central monitor, and caregivers unnecessarily run to the room and frighten the patient and family. Because most of these decelerations will spontaneously resolve in 1 to 3 minutes, such action is usually unwarranted. If the deceleration does not resolve, the patient should be examined to rule out cord prolapse and sudden descent. If these are not present, determine whether there is an apparent cause that can be specifically corrected. If the cause cannot be found, the general explanation by process of elimination is sustained cord compression, and repositioning the patient, oxygen administration, and discontinuing oxytocin are done. Should none of these work, operative delivery will be required unless spontaneous delivery is imminent. How long one should wait for the corrective measures or spontaneous recovery to occur is somewhat variable. This will be determined by the depth of the deceleration, the loss of variability during the deceleration, whether evidence of hypoxia preceded the deceleration, and whether the heart rate is intermittently returning toward baseline or is just staying down. Evidence to recommend a precise amount of time wherein intervention must occur is difficult to integrate because the problem is usually relative hypoxia rather than complete anoxia. Complete anoxia in real life probably occurs only with severe degrees of uterine rupture and complete abruption. Even with cord prolapse there is usually some cord blood flow. Windle performed the classic experiment using complete anoxia in fetal monkeys.[81] Monkeys allowed to breathe in 6 minutes or less showed no clinical or pathologic ill effects. Asphyxiation for 7 to 12 minutes resulted in transient motor and behavioral changes, with some scarring in certain specific areas of the brain in some animals. Those anoxic for 12 to 17 minutes, if death did not occur, had the most severe neurologic and clinical effects. Therefore, in the worst case of all, if delivery occurs in less than 12 minutes from the onset of the deceleration, damage will be unlikely, unless there was some hypoxia before the deceleration.

The most difficult pattern to manage is recurrent prolonged decelerations with or without bradycardia that do recover (Figure 16-32). Generally, these can be managed using the same algorithm for non-reassuring patterns described previously. However, even if one can provide reassurance that acidosis does not exist following any of the decelerations, there is a concern that this pattern portends a deceleration that will recur and will not recover, and one will be placed in the situation described in the previous paragraph. Therefore, there will be occasions when the recurrent prolonged decelerations are concerning enough that operative intervention is warranted even if there is no concern about acidosis at the present moment. One must integrate the frequency, severity, and duration of the deceleration; the fetal response in terms of tachycardia and loss of variability; presence or absence of meconium, and how much time is expected before spontaneous delivery will occur to make this difficult decision. Therefore, for example, in the nulliparous patient at 4 cm, having decelerations lasting 4 minutes to 70 beats/minute every 10 minutes, operative delivery may be warranted. However, in the multipara at 8 cm making normal progress with similar or less frequent decelerations, it may be justified to manage these expectantly, but with all preparations

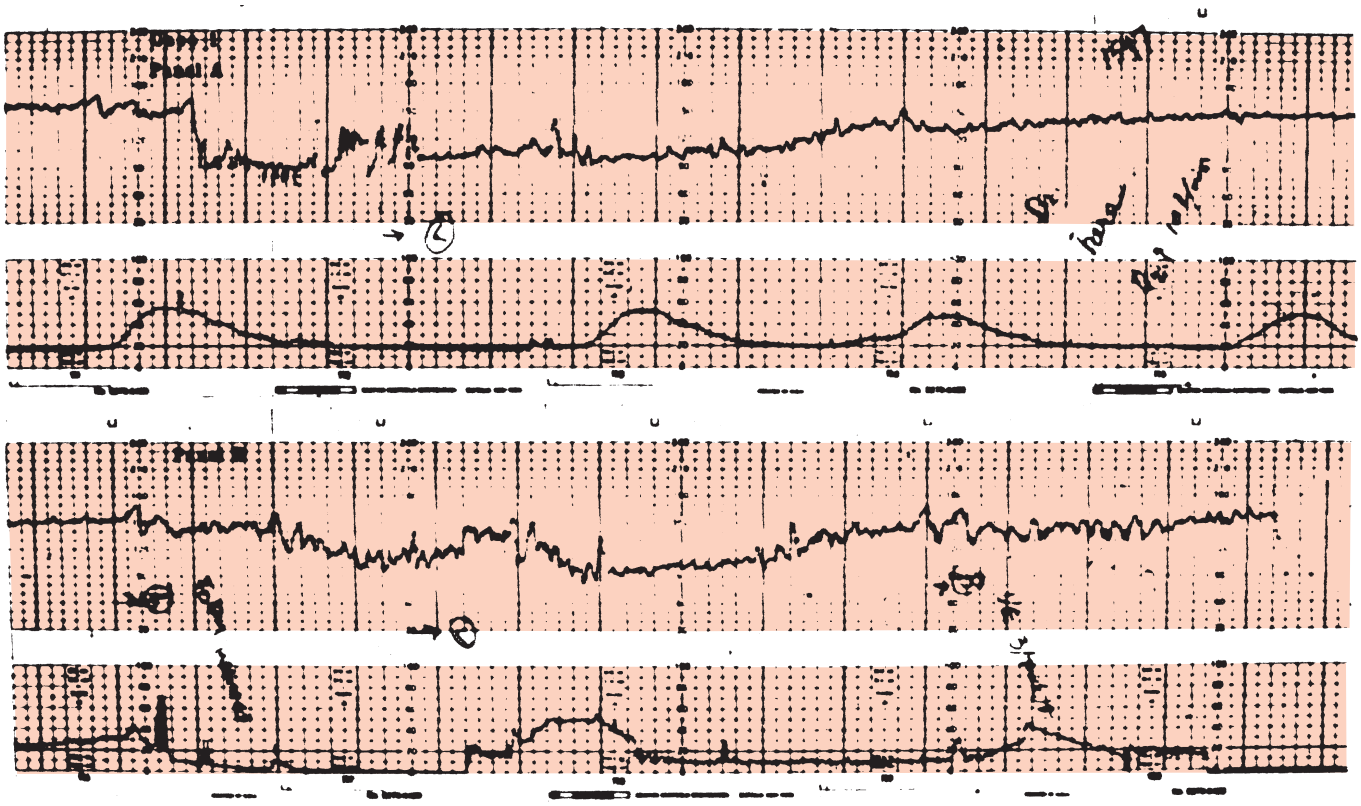

FIGURE 16-32. Recurrent unexplained prolonged decelerations.

made for immediate delivery should one of the decelerations not recover.

Auscultation as an Alternative

Almost all randomized controlled trials have demonstrated that intermittent auscultation is as effective as EFM in detecting fetal hypoxia in labor. There are some limitations to this statement, however. Virtually all these trials compared EFM to intermittent auscultation with one-on-one nursing, in which the auscultation was performed every 15 minutes in the first stage and every 5 minutes in the second stage, and the auscultation was performed for a period of 60 seconds through and following an entire uterine contraction. This is a situation that is difficult to duplicate in everyday practice because of lower nurse-to-patient ratios and because emergencies with other patients often take a nurse away from the bedside for long periods of time. Second, in most of the studies, fetuses who entered labor may have been monitored electronically before randomization, and often very-high-risk patients were excluded from study. Ingemarsson and associates have shown that 50% of patients who develop non-reassuring FHR patterns in labor had a non-reassuring FHR pattern on admission.[82] Finally, in virtually all the studies comparing EFM with auscultation, when the auscultated FHR was abnormal, the patient was then monitored electronically. Furthermore, there are non-reassuring FHR tracings that are quite indicative of hypoxia and acidosis and not likely to be detected with auscultation (Figure 16-33).

Therefore, it is reasonable to conclude that auscultation is an acceptable option for monitoring the fetus in labor when certain conditions are in place. The fetus should have a reassuring FHR on admission monitored electronically. The patient should have one-on-one nursing. The standards for frequency from the ACOG for auscultation are at least every 30 minutes in the first stage and every 15 minutes in the second stage for the low-risk patient and every 15 minutes in the first stage and every 5 minutes in the second stage for the high-risk patient.[83] Fetuses with abnormal FHR patterns on auscultation should have electronic monitoring to define the pattern and monitor for progression to worsening or non-reassuring patterns.

ASSESSMENT OF FETAL CONDITION AT BIRTH

The Apgar score was originally introduced as a tool to be used in guiding the need for neonatal resuscitation. Subsequently, this means of fetal assessment became used routinely for all births. However, the Apgar score has been expected to predict far more than was originally intended. Such expectations have included evaluating acid-base status at birth (i.e., the presence or absence of perinatal asphyxia) and even predicting long-term prognosis. **Unfortunately, the Apgar is a nonspecific measure of these parameters because many other causes of fetal depression may mimic that seen with asphyxia, such as drugs, anomalies, prematurity, suctioning for meconium, and so forth.** In situations in which FHR patterns have been concerning and other backup methods for evaluating fetal oxygenation or fetal acid-base status have been used, or in cases in which the baby is unexpectedly depressed, it is important to

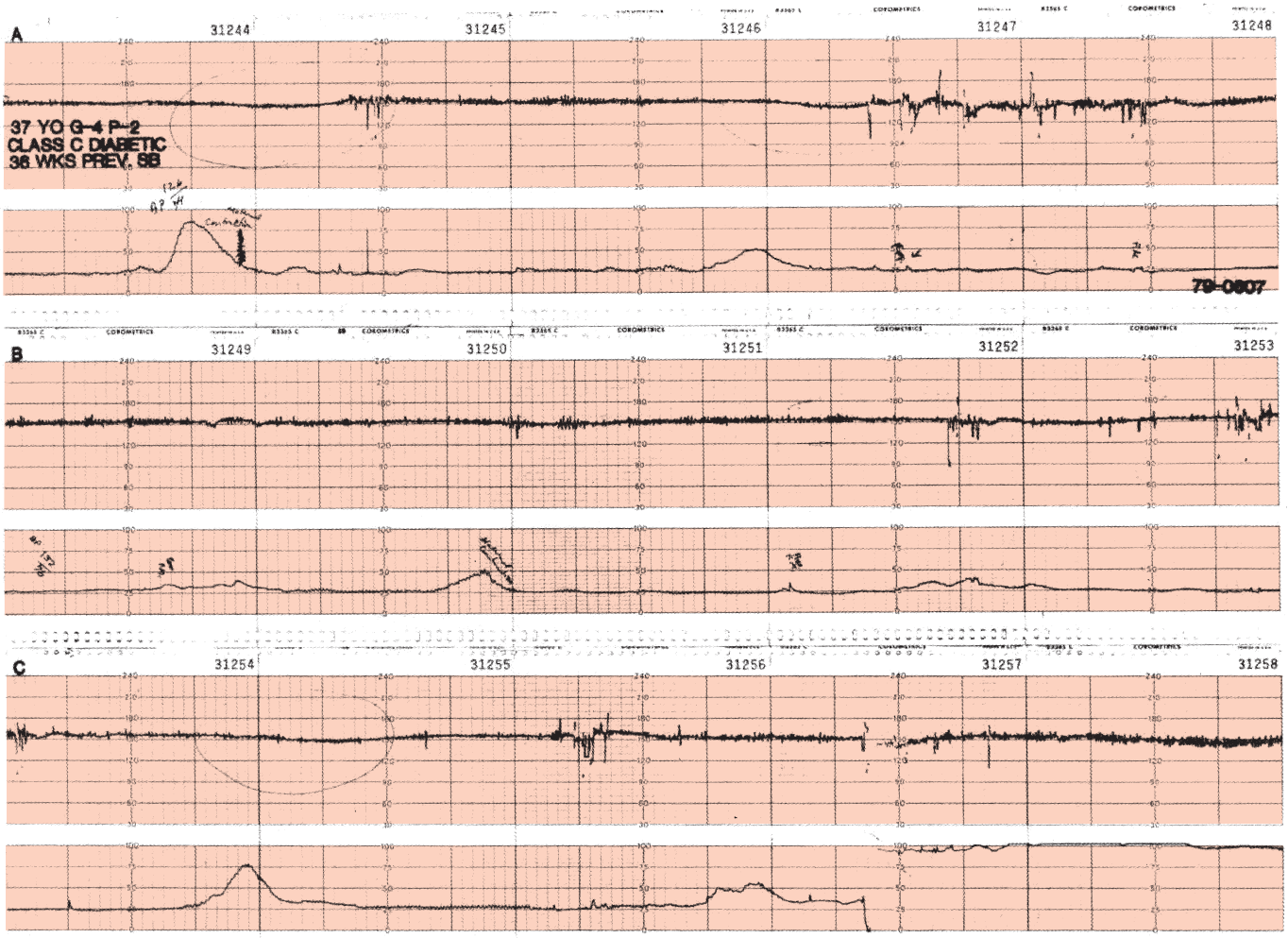

FIGURE 16-33. These persistent late decelerations associated with absent variability are difficult to recognize even with an internal electrode. This is a non-reassuring fetal heart rate pattern, which is often ominous. It is unlikely that such decelerations, associated with a normal baseline rate, would be recognized with intermittent auscultation.

specifically evaluate these parameters at birth, using umbilical cord blood gases. To accomplish this, a doubly clamped 10- to 30-cm section of umbilical cord is taken after the original cord clamping and separation of the baby from the cord. Using heparinized syringes, samples of umbilical artery and vein are separately obtained. These samples are evaluated for respiratory gases. Normal ranges for umbilical cord gases are shown in Table 16-2. Cord blood P_{O_2} or O_2 saturation is not useful because many normal newborns are initially hypoxemic until normal extrauterine respiration is established. Although cord pH, especially arterial, is the essential value, P_{CO_2} is also very important because if the pH is low, the P_{CO_2} is used to determine whether the acidosis is respiratory or metabolic. Respiratory acidosis is not predictive of newborn or long-term injury and should correlate with little or no need for resuscitation. In addition, the cord gases should be used to correlate interpretation of the FHR patterns, as previously described in the sections on their pathophysiology, and to determine the appropriateness of operative intervention or lack of it. These gases can help the pediatrician in determining the etiology of immediate complications and the need for more intense observation of the baby. In addition, these values are often

TABLE 16-2 NORMAL BLOOD GAS VALUES OF UMBILICAL ARTERY AND VEIN

	MEAN VALUE	NORMAL RANGE*
Artery		
pH	7.27	7.15 to 7.38
P_{CO_2}	50	–.35 to 70
Bicarbonate	–3.34	–.17 to 28
Base excess	–3.6	–2.0 to –9.0
Vein		
pH	7.34	7.20 to 7.41
P_{CO_2}	40	33 to 50
Bicarbonate	21	15 to 26
Base excess	–2.6	–1.0 to –8.0

Data from Nijland R, Jongsma HW, Nijhuis JG, et al: Arterial oxygen saturation in relation to metabolic acidosis in fetal lambs. Am J Obstet Gynecol 172:810-819, 1994.

*Values are ±2 SD and represent a composite of multiple studies.

useful if the baby develops any long-term neurologic injury in determining whether any such injury may have been related to peripartum asphyxia.[84] Studies have shown that if such asphyxia is present, in order for it to result in long-term injury, it must have been severe (metabolic acidosis

with a pH <7.00 to 7.05) and be associated with multiple organ dysfunction in the newborn period.[2]

RISKS AND BENEFITS OF ELECTRONIC FETAL HEART RATE MONITORING

Electronic FHR monitoring was introduced with the hope that this modality would reduce or eliminate the devastating consequences of asphyxia. Enthusiasm for this new technology established the role of continuous FHR monitoring in labor before studies demonstrated its accuracy. Initial retrospective studies evaluated more than 135,000 patients and showed more than a three-fold improvement in the intrapartum fetal death rate for patients monitored electronically.[1] However, most subsequent prospective, randomized, controlled trials have failed to demonstrate an improvement in the intrapartum fetal death rate using EFM.[85-91] In these studies, however, electronic FHR monitoring was compared with frequent intermittent auscultation with one-on-one nursing, a standard that is difficult to maintain. Many patients with abnormal FHR patterns on admission were not randomized, and virtually always, patients with an abnormal FHR on auscultation were ultimately monitored electronically. A randomized controlled trial of EFM versus intermittent auscultation conducted by Vintzileos and colleagues did demonstrate a significant improvement in perinatal mortality in the electronically monitored group.[92] The past three decades have not shown a change in the 2 per 1000 incidence of cerebral palsy, suggesting that the widespread use of EFM has not affected this problem. However, these data are somewhat difficult to analyze because of other changes occurring simultaneously. There has been a dramatic improvement in the survival of very-low-birthweight premature babies during this time period, and prematurity accounts for most cases of cerebral palsy. Second, term babies with asphyxia have had an increase in survival during this time period, allowing for a potential increase in surviving children with brain damage. Any of these factors may obscure an effect that FHR monitoring may have had in reducing the incidence of cerebral palsy. EFM has other potential benefits. These include an ability to understand the mechanism of developing hypoxia and to treat it more specifically. **It provides the ability to accurately monitor uterine contractions so that we can better understand progress or lack of progress in labor as well as monitor the effects of oxytocin-stimulated contractions on fetal oxygenation.** The monitor is ultimately, like all other monitors in intensive care situations, a labor-saving device that allows nurses to perform other tasks simultaneously.

EFM has several disadvantages, however. During the period in which FHR monitoring has risen in popularity, there was a parallel increase in the cesarean delivery rate. Certainly, this was not all caused by EFM because there were many other changes in obstetrical practice during this time period. In virtually all the randomized controlled trials, EFM resulted in an increase in the cesarean delivery rate over intermittent auscultation without a concomitant improvement in outcome.[85-89] There is also, however, a desire to deliver babies before significant hypoxia has any potential to damage the baby, and the nonspecific changes in FHR only fuel this concern, setting up the current environment of excessive intervention. **In reality, metabolic acidosis occurs in only about 2% of all labors, and even allowing for a reasonable amount of latitude in early intervention, cesarean delivery rates should not exceed 4% to 5% for this indication.** Unfortunately, rates of 10% for NRFS are common. Thus, the need for better, more specific modalities to allow us to evaluate hypoxia and acidosis in the fetus with a non-reassuring FHR pattern are still being sought. It was hoped that fetal pulse oximetry would meet this need, but that has not been realized.

The second major problem associated with EFM is the fear of a lawsuit should the child be compromised in any way. The monitor has created an expectation of perfect outcome. The interpretation of abnormal FHR tracings is highly subjective and variable, and "experts" often give diametrically opposite interpretations of the same tracing. The modality itself is nonspecific, and babies with anomalies or preexisting brain damage will often have abnormal FHR tracings easily confused with ongoing hypoxia. Finally, a jury cannot help but be sympathetic to a family and baby with disfiguring and debilitating cerebral palsy, and large financial awards seem to be the only way at present to compensate these unfortunate victims. However, these outcomes are not consistent with what we know about asphyxia. More than 75% of brain-damaged children have causes that are *not* related to perinatal asphyxia. Many cases of asphyxia occur *before* labor or early in labor before the patient arrives in the hospital. Few of these cases are truly preventable. One can only hope that once these pressures are removed from the labor and delivery suite by new technology or other solutions, the opportunity to do what is best for the fetus and mother will be enhanced, and this will be the only motivating force.

SUMMARY

EFM has become the standard means for evaluating fetal oxygenation in labor. Because of fetal inaccessibility and the lack of alternatives to more specifically evaluate fetal oxygenation, this modality has been the only alternative. EFM is highly reliable when reassuring (category I) and most often unreliable with equivocal tracings (category II), except in the extreme (category III), when there is high likelihood of fetal acidosis. Despite or even because of these limitations, it is imperative that the clinician understand as much as possible about the underlying physiologic explanations of normal and abnormal FHR patterns because this allows the only reasonable opportunity to appropriately evaluate and manage these changes. The new terminology proposed by the NICHD Workshop and endorsed by ACOG and AWHONN should be used consistently. The goals of FHR monitoring should be to carefully and thoroughly monitor all patients in active labor; avoid unnecessary operative and nonoperative intervention for benign and innocuous FHR patterns; correct non-reassuring FHR patterns with noninvasive, etiology-specific therapies when possible, or if not possible use appropriate means such as scalp pH, accelerations, or moderate variability to rule out acidosis; and finally, if acidosis cannot be ruled out, operatively intervene in an expeditious manner appropriate for the entire clinical situation.

KEY POINTS

- The goals of intrapartum fetal evaluation by electronic FHR monitoring and available back-up methods are to detect fetal hypoxia, reverse the hypoxia with nonsurgical means, or if unsuccessful, determine whether the hypoxia has progressed to metabolic acidosis, and if so deliver the baby expeditiously to avoid the hypoxia and acidosis from resulting in any damage to the baby.
- New terminology has been proposed by the NICHD Workshop on intrapartum EFM and should be consistently employed in practice.
- EFM is an inherently suboptimal method of determining fetal hypoxia and acidosis because many factors besides these variables may alter the FHR and mimic changes caused by hypoxia and acidosis. When the FHR is normal, its reliability for predicting the absence of fetal compromise is high, but when the FHR is abnormal, its reliability for predicting the presence of asphyxia is poor.
- The three-tiered classification of FHR patterns proposed by the NICHD Workshop on EFM and endorsed by ACOG and AWHONN should be used as a template for evaluation and management of FHR patterns, but the limitations of the guidelines for the frequently occurring category II patterns should be realized and plans made to deal more specifically with different types of FHR patterns within this group.
- Late decelerations are always indicative of relative fetal hypoxia and are caused by inadequate oxygen delivery, exchange, or uptake that is aggravated by the additional hypoperfusion of the placenta caused by contractions. Variable decelerations are caused by a decrease in umbilical cord flow resulting from cord compression or cord stretch. Prolonged decelerations may be caused by any mechanism that decreases fetal oxygenation.
- In labor, loss of variability, loss of accelerations, and tachycardia should only be interpreted as indicative of fetal compromise in the presence of non-reassuring decelerations (late, non-reassuring variable or prolonged decelerations) because signs of hypoxia should always precede signs of neurologic depression secondary to hypoxia.
- In the presence of oligohydramnios and variable decelerations, intrapartum amnioinfusion has been shown to decrease rates of cesarean delivery for NRFS.
- In the presence of an otherwise non-reassuring FHR pattern, the presence of accelerations of the FHR, either spontaneous or elicited by scalp stimulation or vibroacoustic stimulation, indicates the absence of fetal acidosis. The absence of accelerations is associated with a 50% chance of fetal acidosis, but only in the setting of a non-reassuring FHR.
- Umbilical cord blood gases should be obtained and documented in situations in which there is a non-reassuring or confusing FHR pattern during labor, neonatal depression following birth, prematurity, or suctioning for meconium. These values will help clarify the reasons for abnormal FHR patterns or for neonatal depression.
- Although there is correlation between a non-reassuring FHR pattern and neonatal depression, the FHR is a poor predictor of long-term neurologic sequelae. Furthermore, fetuses with previous neurologic insults may have significantly abnormal FHR patterns even when they are well oxygenated in labor.

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