CHAPTER 24: Intrapartum Assessment

To study the forces exerted by labour, a rubber bag was inserted into the uterus which was connected with a manometer. In this way it was found that the intra-uterine pressure, in the intervals between the contractions, was represented by a column of mercury 20 millimeters high, 5 of which were due to the tonicity of the walls and 15 to its contents. During the pains, however, the mercury rose considerably, reaching a height of from 80 to 250 millimeters.

—J. Whitridge Williams (1903)

INTRODUCTION

Little is written in the first edition of this textbook concerning monitoring of the fetus during labor. Much later, periodic auscultation of the fetal heartbeat with a fetoscope was adopted. These practices were eclipsed in the late 1960s and early 1970s by the development of electronic fetal monitoring (Hon, 1958). It was hoped that the continuous graph-paper portrayal of the fetal heart rate was potentially diagnostic in assessing pathophysiological events affecting the fetus.

When first introduced, electronic fetal heart rate monitoring was used primarily in complicated pregnancies but gradually became used in most pregnancies. Now, more than 85 percent of all live births in the United States undergo electronic fetal monitoring (Ananth, 2013).

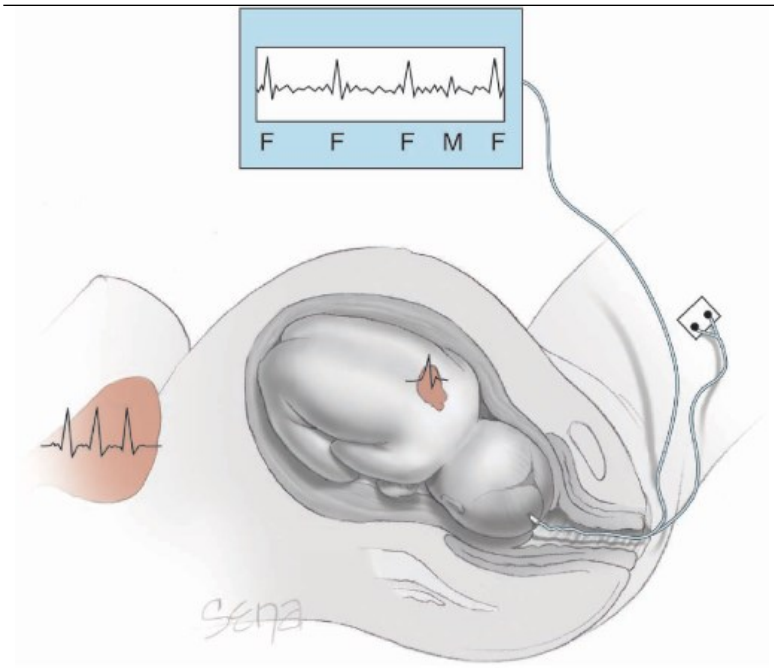
ELECTRONIC FETAL MONITORING

Internal (Direct) Electronic Monitoring

Direct fetal heart measurement is accomplished by attaching a bipolar spiral electrode directly to the fetus (Fig. 24-1). The wire electrode penetrates the fetal scalp, and the second pole is a metal wing on the electrode. The electrical fetal cardiac signal—P wave, QRS complex, and T wave—is amplified and fed into a cardiotachometer for heart rate calculation. The peak R-wave voltage is the portion of the fetal electrocardiogram (ECG) most reliably detected.

FIGURE 24-1

Internal electronic fetal monitoring. Schematic representation of a bipolar electrode attached to the fetal scalp for detection of fetal QRS complexes (F). Also shown is the maternal heart and corresponding electrical complex (M) that is detected.



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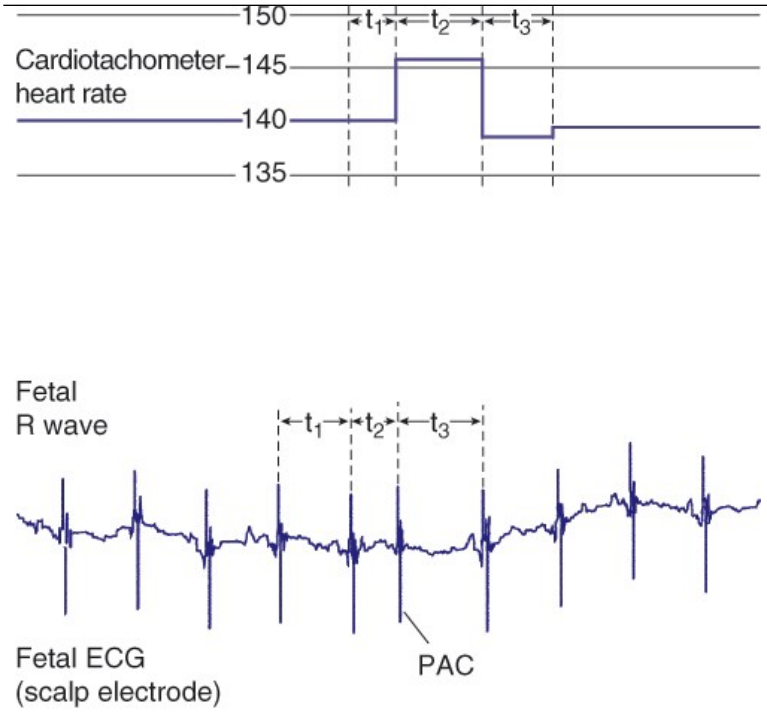
An example of the method of fetal heart rate processing employed when a scalp electrode is used is shown in Figure 24-2. Time (t) in milliseconds between fetal R waves is fed into a cardiotachometer, where a new fetal heart rate is set with the arrival of each new R wave. As also shown in Figure 24-2, a premature atrial contraction is computed as a heart rate acceleration because the interval (t_2) is shorter than the preceding one (t_1). The phenomenon of continuous R-to-R wave fetal heart rate computation is known as *beat-to-beat variability.*

FIGURE 24-2

Schematic representation of fetal electrocardiographic signals used to compute continuing beat-to-beat heart rate with scalp electrodes. Time intervals (t_1, t_2, t_3) in milliseconds between successive fetal R waves are used by a cardiotachometer to compute instantaneous fetal heart rate. ECG = electrocardiogram; PAC = premature atrial contraction.



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Electrical cardiac complexes detected by the electrode include those generated by the mother. However, the amplitude of the maternal ECG signal is diminished when recorded through the fetal scalp electrode and is masked by the fetal ECG. Shown in Figure 24-3 are simultaneous recordings of maternal chest wall ECG signals and fetal scalp electrode ECG signals. This fetus is experiencing premature atrial contractions, which cause the cardiotachometer to rapidly and erratically seek new heart rates, resulting in the "spiking" shown in the standard fetal monitor tracing. Importantly, when the fetus is dead, the maternal R waves are still detected by the scalp electrode as the next best signal and are counted by the cardiotachometer (Fig. 24-4).

FIGURE 24-3

The top tracing shows standard fetal monitor tracing of heart rate using a fetal scalp electrode. Spiking of the fetal rate in the monitor tracing is due to premature atrial contractions. The second panel displays accompanying contractions. The bottom two tracings represent cardiac electrical complexes detected from fetal scalp and maternal chest wall electrodes. ECG = electrocardiogram; F = fetus; M = mother; PAC = fetal premature atrial contraction.

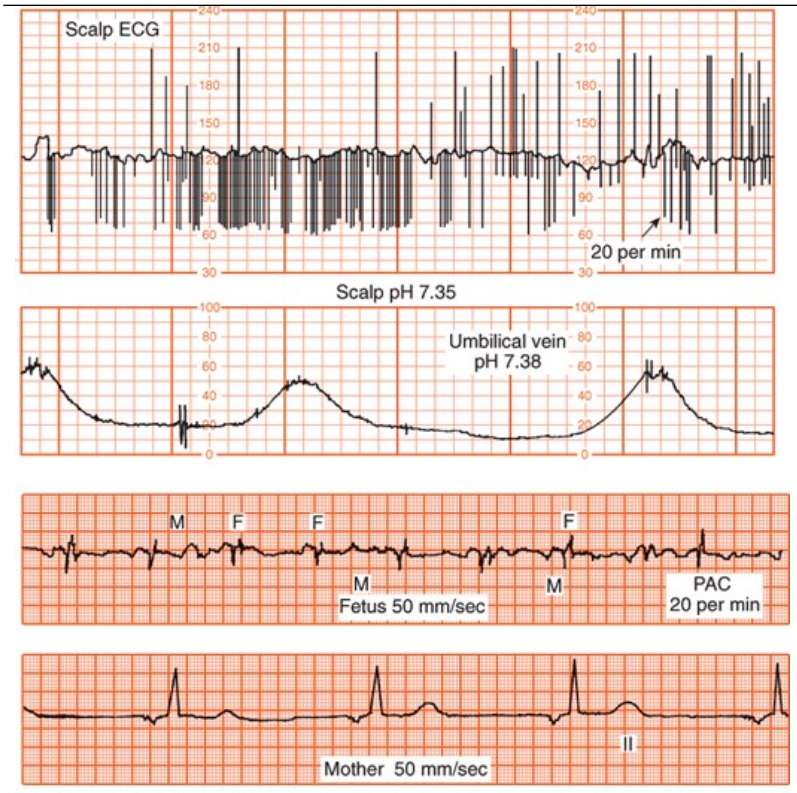

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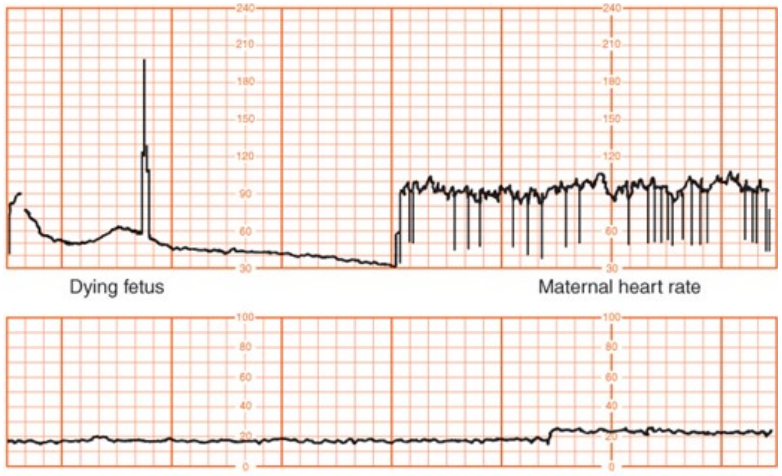
FIGURE 24-4

Placental abruption. In the upper panel, the fetal scalp electrode first detected the heart rate of the dying fetus. After fetal death, the maternal electrocardiogram complex is detected and recorded. The second panel displays an absence of uterine contractions.



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External (Indirect) Electronic Monitoring

Although membrane rupture may be avoided, external monitoring does not provide the precision of fetal heart rate measurement afforded by internal monitoring (Nunes, 2014). In some women—for example, those who are obese—external monitoring may be difficult (Brocato, 2017).

With external monitoring, the fetal heart rate is detected through the maternal abdominal wall using the *ultrasound Doppler principle*. Ultrasound

waves undergo a shift in frequency as they are reflected from moving fetal heart valves and from pulsatile blood ejected during systole (Chap. 10, Doppler). The unit consists of a transducer that emits ultrasound and a sensor to detect a shift in frequency of the reflected sound. The transducer is placed on the maternal abdomen at a site where fetal heart action is best detected. A coupling gel must be applied because air conducts ultrasound waves poorly. The device is held in position by an elastic belt. Correct positioning enhances differentiation of fetal cardiac motion from maternal arterial pulsations (Neilson, 2008).

Ultrasound Doppler signals are edited electronically before fetal heart rate data are printed onto monitor paper. Reflected ultrasound signals from moving fetal heart valves are analyzed through a microprocessor that compares incoming signals with the most recent previous signal. This process, called *autocorrelation*, is based on the premise that the fetal heart rate has regularity, whereas "noise" is random and without regularity. Several fetal heart motions must be deemed electronically acceptable by the microprocessor before the fetal heart rate is printed. Such electronic editing has greatly improved the tracing quality of the externally recorded fetal heart rate. Other features of current fetal monitors include the capability to monitor twin fetuses, monitor concurrent maternal heart rate, display the fetal ECG, and record maternal pulse oximetry values. Many fetal monitors are capable of interfacing with archival storage systems, which obviates maintaining actual paper tracings.

Technological advances now allow fetal heart rate monitoring from a remote, centralized location. Theoretically, the ability to monitor several patients simultaneously was hoped to improve neonatal outcomes. That said, only one study on centralized fetal monitoring has been reported. Anderson and colleagues (2011) measured the ability of 12 individuals to detect critical signals in fetal heart rate tracings on one, two, or four monitors. The results showed that detection accuracy declined as the number of displays increased.

Fetal Heart Rate Patterns

The interpretation of fetal heart rate patterns can be problematic without definitions and nomenclature. In one example, Blackwell and colleagues (2011) asked three Maternal-Fetal Medicine specialists to independently interpret 154 fetal heart rate tracings. Interobserver agreement was poor for the most ominous tracings and moderate for less severe patterns. The National Institute of Child Health and Human Development (NICHD) Research Planning Workshop (1997) brought together investigators with expertise in the field to propose standardized, unambiguous definitions for interpretation of fetal heart rate patterns during labor. This workshop reconvened in 2008. The definitions proposed as a result of this second workshop are used in this chapter and have been adopted by the American College of Obstetricians and Gynecologists (2017a) (Table 24-1). Importantly, interpretation of electronic fetal heart rate data is based on the visual pattern of the heart rate as portrayed on chart recorder graph paper. Thus, the choice of vertical and horizontal scaling greatly affects the appearance of the fetal heart rate. Scaling factors recommended by the NICHD Workshop are 30 beats per minute (beats/min or bpm) per vertical cm (range, 30 to 240 bpm) and 3 cm/min chart recorder paper speed. Fetal heart rate variation is falsely displayed at the slower 1 cm/min paper speed compared with that of the smoother baseline recorded at 3 cm/min. Thus, pattern recognition can be considerably distorted depending on the scaling factors used.

TABLE 24-1

Electronic Fetal Monitoring Definitions

Pattern	Definition
Baseline	• The mean FHR rounded to increments of 5 bpm during a 10-min segment, excluding: —Periodic or episodic changes —Periods of marked FHR variability —Segments of baseline that differ by more than 25 bpm • The baseline must be for a minimum of 2 min in any 10-min segment or the baseline for that time period is indeterminate. In this case, one may refer to the prior 10-min window for determination of baseline. • Normal FHR baseline: 110–160 bpm • Tachycardia: FHR baselines is greater than 160 beats per minute • Bradycardia: FHR baseline is less than 110 beats per minute
Baseline	• Fluctuations in the baseline FHR that are irregular in amplitude and frequency

variability	• Variability is visually quantified as the amplitude of peak-to-trough in beats per minute —Absent: amplitude range undetectable —Minimal: amplitude range detectable but 5 beats per minutes or fewer —Moderate (normal): amplitude range 6–25 beats per minute —Marked: amplitude range greater than 25 beats per minute
Acceleration	• A visually apparent abrupt increase (onset to peak in less than 30 seconds) in the FHR • At 32 weeks of gestation and beyond, an acceleration has a peak of 15 bpm or more above baseline, with a duration of 15 sec or more but less than 2 minutes from onset to return • Before 32 weeks, an acceleration has a peak of 10 bpm or more above baseline, with a duration of 10 seconds or more but less than 2 minutes from onset to return • Prolonged acceleration lasts 2 minutes or more but less than 10 minutes in duration • If an acceleration lasts 10 minutes or longer, it is a baseline change
Early deceleration	• Visually apparent usually symmetrical gradual decrease and return of the FHR associated with a uterine contraction • A gradual FHR decrease is defined as from the onset to the FHR nadir of 30 seconds or more • The decrease in FHR is calculated from the onset to the nadir of the deceleration • The nadir of the deceleration occurs at the same time as the peak of the contraction • In most cases the onset, nadir, and recovery of the deceleration are coincident with the beginning, peak, and ending of the contraction, respectively
Late deceleration	• Visually apparent usually symmetrical gradual decrease and return of the FHR associated with a uterine contraction • A gradual FHR decrease is defined as from the onset to the FHR nadir of 30 seconds or more • The decrease in FHR is calculated from the onset to the nadir of the deceleration • The deceleration is delayed in timing, with the nadir of the deceleration occurring 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
Variable deceleration	• Visually apparent abrupt decrease in FHR • An abrupt FHR decrease is defined as from the onset of the deceleration to the beginning of the FHR nadir of less than 30 seconds • The decrease in FHR is calculated from the onset to the nadir of the deceleration • The decrease in FHR is 15 beats per minute or greater, lasting 15 seconds or greater, and less than 2 minutes in duration • When variable decelerations are associated with uterine contraction, their onset, depth, and duration commonly vary with successive uterine contractions
Prolonged deceleration	• Visually apparent decrease in the FHR below the baseline • Decrease in FHR from the baseline that is 15 beats per minute or more, and less than 2 minutes in duration • If a deceleration last 10 minutes or longer, it is a baseline change
Sinusoidal pattern	• Visually apparent, smooth, sine wave-line undulating pattern in FHR baseline with a cycle frequency of 3–5 per minute which persists for 20 minutes or more

FHR = fetal heart rate.

Data from Macones, 2008.

Baseline Fetal Heart Activity

This refers to the modal characteristics that prevail apart from periodic accelerations or decelerations associated with uterine contractions. Descriptive characteristics of baseline fetal heart activity include *rate, beat-to-beat variability, fetal arrhythmia,* and distinct patterns such as *sinusoidal* or *saltatory* fetal heart rates.

Rate

With increasing fetal maturation, the heart rate decreases. This continues postnatally such that the average rate is 85 bpm by age 8 years (Tintinalli, 2016). Pillai and James (1990) reported that the baseline fetal heart rate declined an average of 24 bpm between 16 weeks' gestation and term, or approximately 1 bpm per week. This normal gradual slowing of the fetal heart rate is thought to correspond to maturation of parasympathetic (vagal) heart control (Renou, 1969).

The baseline fetal heart rate is the approximate mean rate rounded to increments of 5 bpm during a 10-minute tracing segment. In any 10-minute window, the minimum interpretable baseline duration must be at least 2 minutes. If the baseline fetal heart rate is less than 110 bpm, it is termed *bradycardia.* If the baseline rate is greater than 160 bpm, it is called *tachycardia.* The average fetal heart rate is considered the result of tonic balance between *accelerator* and *decelerator* influences on pacemaker cells. In this concept, the sympathetic system is the accelerator influence, and the parasympathetic system is the decelerator factor mediated by vagal slowing of heart rate (Dawes, 1985). Heart rate also is under the control of arterial chemoreceptors such that both hypoxia and hypercapnia can modulate rate. More severe and prolonged hypoxia, with a rising blood lactate level and severe metabolic acidemia, induces a prolonged fall in heart rate (Thakor, 2009).

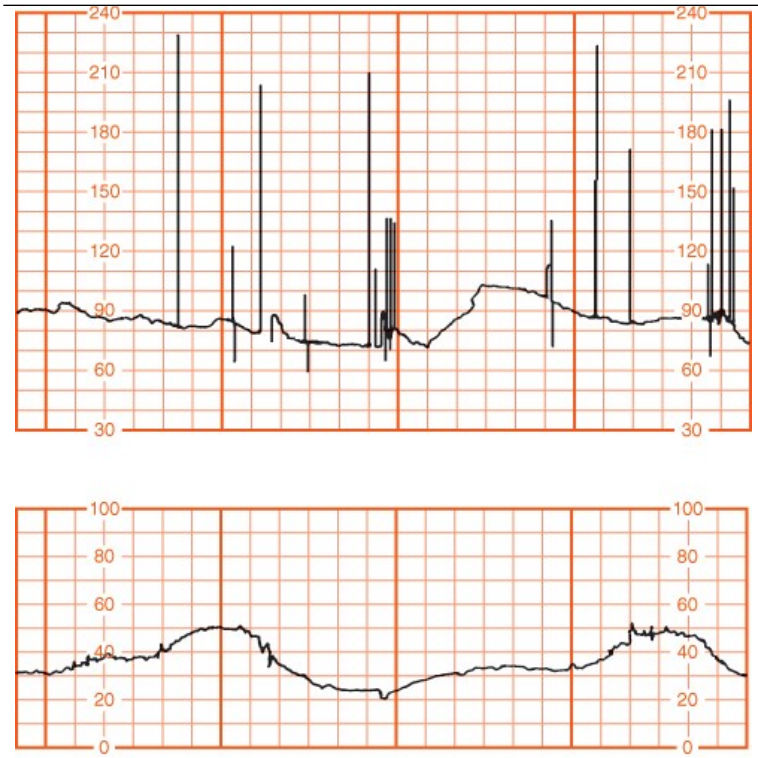
Bradycardia

In the third trimester, the normal mean baseline fetal heart rate has generally been accepted to range between 120 and 160 bpm. But, pragmatically, a rate between 100 and 119 bpm, in the absence of other changes, usually is not considered to represent fetal compromise. Such low but potentially normal baseline heart rates also have been attributed to head compression from occiput posterior or transverse positions, particularly during second-stage labor (Young, 1976). Such mild bradycardias were observed in 2 percent of monitored pregnancies and averaged approximately 50 minutes in duration. Freeman and associates (2003) have concluded that bradycardia within the range of 80 to 120 bpm and with good variability is reassuring. Interpretation of rates less than 80 bpm is problematic, and such rates generally are considered nonreassuring.

Some causes of fetal bradycardia include congenital heart block and serious fetal compromise (Jaeggi, 2008; Larma, 2007). Figure 24-5 shows bradycardia in a fetus dying from placental abruption. Maternal hypothermia under general anesthesia for repair of a cerebral aneurysm or during maternal cardiopulmonary bypass for open-heart surgery can also cause fetal bradycardia. Sustained fetal bradycardia in the setting of severe pyelonephritis and maternal hypothermia also has been reported (Hankins, 1997). Involved fetuses apparently are not harmed by several hours of such bradycardia.

FIGURE 24-5

Fetal bradycardia measured with a scalp electrode (*upper panel*) in a pregnancy complicated by placental abruption and subsequent fetal death. Concurrent uterine contractions are shown in the lower panel.



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Tachycardia

Fetal tachycardia is defined as a baseline heart rate greater than 160 bpm. The most common explanation for fetal tachycardia is maternal fever from chorioamnionitis, although fever from any source can produce this. In some cases, fetal tachycardia may precede overt maternal fever (Gilstrap, 1987). Fetal tachycardia caused by maternal infection typically is not associated with fetal compromise unless there are associated periodic heart rate changes or fetal sepsis.

Other causes of fetal tachycardia include fetal compromise, cardiac arrhythmias, and maternal administration of parasympathetic inhibiting (atropine) or sympathomimetic (terbutaline) drugs. Prompt relief of the compromising event, such as correction of maternal hypotension caused by epidural analgesia, can result in fetal recovery. The key feature to distinguish fetal compromise in association with tachycardia seems to be concomitant heart rate decelerations.

Wandering Baseline

This baseline rate is unsteady and “wanders” between 120 and 160 bpm (Freeman, 2003). This rare finding is suggestive of a neurologically abnormal fetus and may occur as a preterminal event. In contrast, changes of the normal baseline are common in labor and do not predict morbidity (Yang, 2017).

Beat-to-Beat Variability

Baseline variability is an important index of cardiovascular function and appears to be regulated largely by the autonomic nervous system (Kozuma, 1997). That is, a sympathetic and parasympathetic “push and pull” mediated via the sinoatrial node produces moment-to-moment or beat-to-beat oscillation of the baseline heart rate. Such heart rate change is defined as baseline variability. Variability can be further analyzed over the short term and long term, although these terms have fallen out of use. *Short-term variability* reflects the instantaneous change in fetal heart rate from one beat—or R wave—to the next. This variability is a measure of the time interval between cardiac systoles (Fig. 24-6). Short-term variability can most reliably be determined to be normally present only when electrocardiac cycles are measured directly with a scalp electrode. *Long-term variability* is used to describe the oscillatory changes during 1 minute and result in the waviness of the baseline (Fig. 24-7). The normal frequency of such waves is three to

five cycles per minute (Freeman, 2003).

FIGURE 24-6

Schematic representation of short-term beat-to-beat variability measured by a fetal scalp electrode. t = time interval between successive fetal R waves. (Adapted with permission from Klavan M, Laver AT, Boscola MA: Clinical concepts of fetal heart rate monitoring. Waltham, Hewlett-Packard, 1977.)

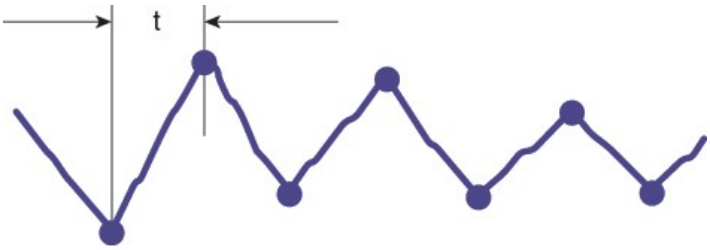
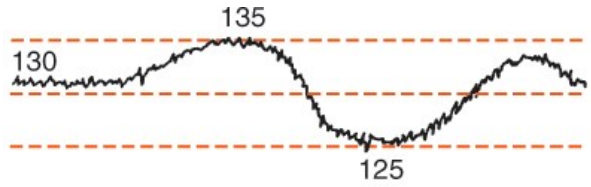


FIGURE 24-7

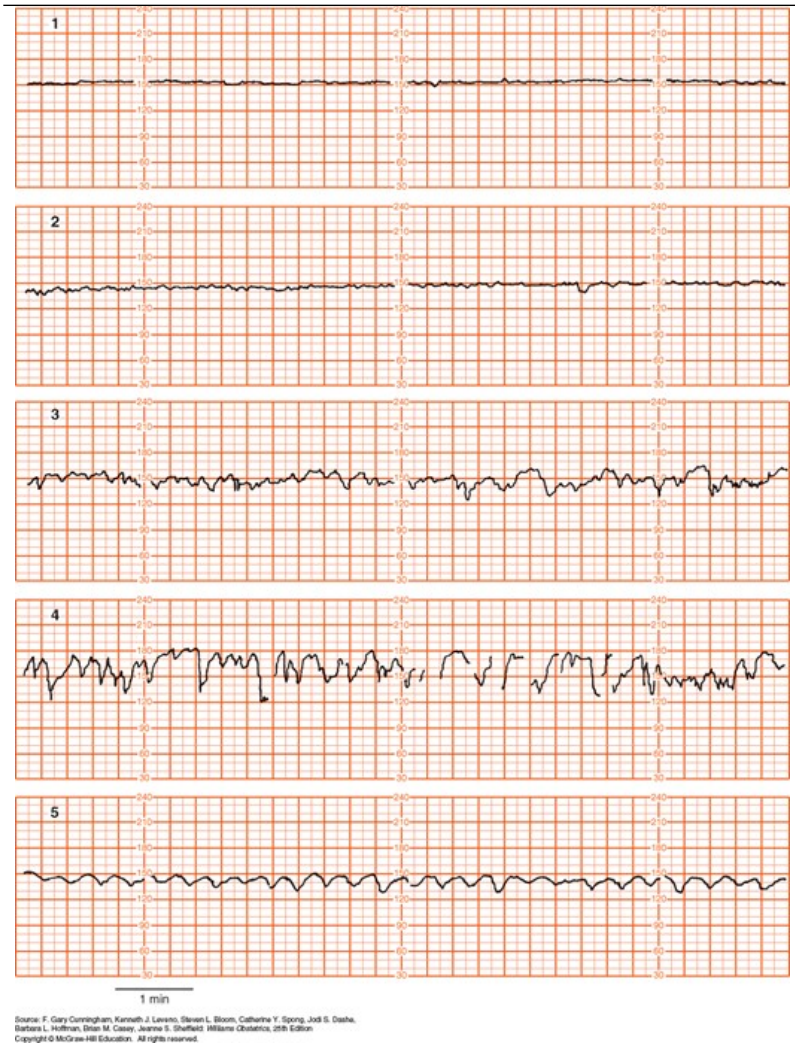
Schematic representation of long-term beat-to-beat variability of the fetal heart rate ranging between 125 and 135 bpm. (Adapted with permission from Klavan M, Laver AT, Boscola MA: Clinical concepts of fetal heart rate monitoring. Waltham, Hewlett-Packard, 1977.)



It should be recognized that precise quantitative analysis of both short- and long-term variability presents several frustrating problems due to technical and scaling factors (Parer, 1985). Thus, most clinical interpretation is based on visual analysis with subjective judgment of the smoothness or flatness of the baseline. According to Freeman and associates (2003), no evidence suggests that the distinction between short- and long-term variability has clinical relevance. Similarly, the NICHD Workshop (1997) did not recommend differentiating short- and long-term variability because in actual practice they are visually determined as a unit. The workshop panel defined baseline variability as those baseline fluctuations of two cycles per minute or greater. They recommended the criteria shown in Figure 24-8 for quantification of variability. Normal beat-to-beat variability was accepted to be 6 to 25 bpm.

FIGURE 24-8

Grades of baseline fetal heart rate variability shown in the following five panels. **1.** Undetectable, absent variability. **2.** Minimal variability, ≤5 bpm. **3.** Moderate (normal) variability, 6 to 25 bpm. **4.** Marked variability, >25 bpm. **5.** Sinusoidal pattern. This differs from variability in that it has a smooth, sinelike pattern of regular fluctuation and is excluded in the definition of fetal heart rate variability. (Adapted with permission from National Institute of Child Health and Human Development Research Planning Workshop, 1997.)



Increased Variability

Several physiological and pathological processes can affect beat-to-beat variability. Greater variability accompanies fetal breathing and body movements (Dawes, 1981; Van Geijn, 1980). Pillai and James (1990) reported increased baseline variability with advancing gestation. Up to 30 weeks, baseline characteristics were similar during both fetal rest and activity. After 30 weeks, fetal inactivity was associated with diminished baseline variability, but fetal activity enhanced it. Last, the baseline fetal heart rate becomes more physiologically fixed (less variable) as the rate rises. This phenomenon presumably reflects less cardiovascular physiological wandering as beat-to-beat intervals shorten with a higher heart rate.

Decreased Variability

A common cause of diminished beat-to-beat variability is administration of analgesic drugs during labor (Chap. 25, Analgesia and Sedation During Labor). Various central nervous system depressant drugs can cause transient diminished beat-to-beat variability. Included are narcotics, barbiturates, phenothiazines, tranquilizers, and general anesthetics. Corticosteroids also dampen variability (Knaven, 2017). As one specific example, variability regularly diminishes within 5 to 10 minutes following intravenous meperidine administration, and the effects may last up to 60 minutes or longer (Hill, 2003; Petrie, 1993). Butorphanol given intravenously has similar effects (Schucker, 1996). And, chronically administered buprenorphine suppresses fetal heart rate and movement (Jansson, 2017).

Magnesium sulfate, widely used in the United States for tocolysis or management of hypertensive gravidas, is associated with diminished beat-to-beat variability. In a study of nearly 250 term gestations, magnesium sulfate administration led to decreased variability but without evidence of adverse neonatal effects (Duffy, 2012). Others have echoed these findings (Hallak, 1999; Lin, 1988). With magnesium sulfate tocolysis of preterm labor,

variability was also diminished in most reviewed studies (Nensi, 2014; Verdurmen, 2017).

Of greatest concern, diminished beat-to-beat variability can be an ominous sign indicating a seriously compromised fetus. Paul and coworkers (1975) reported that loss of variability in combination with decelerations was associated with *fetal acidemia*. Decreased variability was defined as an excursion of the baseline of ≤5 bpm (see Fig. 24-8). Severe *maternal acidemia* can also lower fetal beat-to-beat variability, for example, in a mother with diabetic ketoacidosis.

According to Dawes (1985), metabolic acidemia that causes depression of the fetal brainstem or the heart itself creates the loss of variability. Thus, diminished beat-to-beat variability, when it reflects fetal compromise, likely reflects acidemia rather than hypoxia. Indeed, mild degrees of fetal hypoxemia have been reported actually to *enhance* variability, at least initially (Murotsuki, 1997).

Reduced baseline heart rate variability is the single most reliable sign of fetal compromise. Smith and coworkers (1988) performed a computerized analysis of beat-to-beat variability in growth-restricted fetuses before labor. Diminished variability (≤4.2 bpm) maintained for 1 hour was diagnostic of developing acidemia and imminent fetal death. In contrast, Samueloff and associates (1994) evaluated variability in 2200 consecutive deliveries and concluded that variability by itself could not be used as the only indicator of fetal well-being. They also warned that good variability should not be interpreted as necessarily reassuring. Blackwell and associates (2011) found that even experts often disagreed as to whether variability was absent or minimal (≤5 bpm).

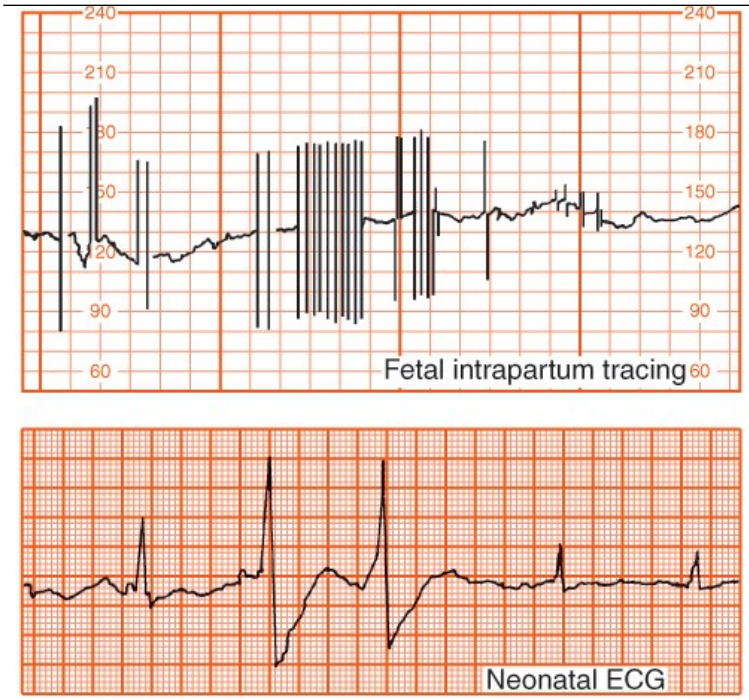
In sum, beat-to-beat variability is affected by fetal physiology, and its meaning differs depending on the clinical setting. Decreased variability in the absence of decelerations is unlikely to reflect fetal hypoxia (Davidson, 1992). A persistently flat fetal heart rate baseline—absent variability—within the normal baseline rate range and without decelerations may reflect a previous fetal insult that has resulted in neurological damage (Freeman, 2003).

Cardiac Arrhythmia

When fetal cardiac arrhythmias are first suspected using electronic monitoring, findings can include baseline bradycardia, tachycardia, or most commonly in our experience, *abrupt baseline spiking* (Fig. 24-9). An arrhythmia can only be documented, practically speaking, when scalp electrodes are used. Some fetal monitors can be adapted to output the scalp electrode signals into an ECG recorder. Because only a single lead is obtained, analysis and interpretation of rhythm and rate disturbances are severely limited.

FIGURE 24-9

Internal fetal monitoring at term demonstrated occasional abrupt beat-to-beat fetal heart rate spiking due to erratic extrasystoles shown in the corresponding fetal electrocardiogram. The normal newborn was delivered spontaneously and had normal cardiac rhythm in the nursery.



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Southall and associates (1980) studied fetal cardiac rate and rhythm disturbances in 934 normal pregnancies between 30 and 40 weeks. Arrhythmias, episodes of bradycardia <100 bpm, or tachycardia >180 bpm were encountered in 3 percent. Most supraventricular arrhythmias are of little significance during labor unless there is coexistent fetal heart failure as evidenced by hydrops. Many supraventricular arrhythmias disappear in the immediate neonatal period, although some are associated with structural cardiac defects (Api, 2008). Intermittent baseline bradycardia is frequently due to congenital heart block. Conduction defects, most often complete atrioventricular (AV) block, usually are found in association with maternal connective tissue diseases (Chap. 59, Perinatal Mortality and Morbidity). Antepartum evaluation of the fetus with an identified arrhythmia and potential treatment options are discussed in Chapter 16 (Tachyarrhythmias).

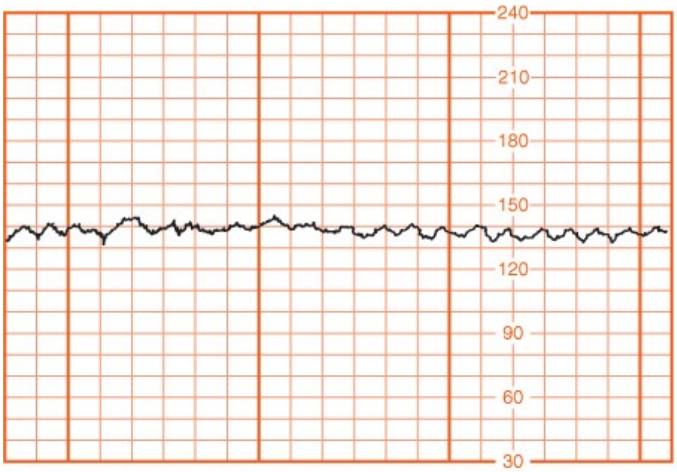
Most fetal arrhythmias without comorbid fetal hydrops are inconsequential during labor, but they may hinder interpretation of fetal heart rate tracings. Sonographic evaluation of fetal anatomy and echocardiography can be useful. Generally, in the absence of fetal hydrops, neonatal outcome is not measurably improved by pregnancy intervention. At Parkland Hospital, intrapartum fetal cardiac arrhythmias, especially those associated with clear amnionic fluid, are typically managed conservatively.

Sinusoidal Heart Rate

A true sinusoidal pattern such as that shown in panel 5 of Figure 24-8 can be observed with fetal intracranial hemorrhage, with severe fetal asphyxia, and with severe fetal anemia. The last may stem from anti-D alloimmunization, fetomaternal hemorrhage, twin-twin transfusion syndrome, fetal parvoviral infection, or vasa previa with bleeding. Insignificant sinusoidal patterns have been reported following administration of meperidine, morphine, alphaprodine, and butorphanol (Angel, 1984; Egley, 1991; Epstein, 1982). Shown in Figure 24-10 is a sinusoidal pattern seen with maternal meperidine administration. An important characteristic of this pattern when due to narcotics is the sine frequency of 6 cycles per minute. A sinusoidal pattern also has been described with chorioamnionitis, fetal distress, and umbilical cord occlusion (Murphy, 1991). Young (1980a) and Johnson (1981) with their coworkers concluded that intrapartum sinusoidal fetal heart patterns were not generally associated with fetal compromise. Thus, management is usually dictated by the clinical setting. Modanlou and Freeman (1982), based on their extensive review, proposed adoption of a strict definition:

FIGURE 24-10

Sinusoidal fetal heart rate pattern associated with maternal intravenous meperidine administration. Sine waves are occurring at a rate of 6 cycles per minute.



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1. Stable baseline heart rate of 120 to 160 bpm with regular oscillations
2. Amplitude of 5 to 15 bpm (rarely greater)
3. Long-term variability frequency of 2 to 5 cycles per minute
4. Fixed or flat short-term variability
5. Oscillation of the sinusoidal waveform above or below a baseline
6. Absent accelerations.

Although these criteria were selected to define a sinusoidal pattern that is most likely ominous, they observed that the pattern associated with alphaprodine is indistinguishable. Other investigators have proposed a classification of sinusoidal heart rate patterns into mild—amplitude 5 to 15 bpm, intermediate—16 to 24 bpm, and major—≥25 bpm to quantify fetal risk (Murphy, 1991; Neesham, 1993).

Some have defined intrapartum sine wavelike baseline variation with periods of acceleration as *pseudosinusoidal.* Murphy and colleagues (1991) reported that pseudosinusoidal patterns were seen in 15 percent of monitored labors. Mild pseudosinusoidal patterns were associated with use of meperidine and epidural analgesia. Intermediate pseudosinusoidal patterns were linked to fetal sucking or transient episodes of fetal hypoxia caused by umbilical cord compression. Egley and associates (1991) reported that 4 percent of fetuses demonstrated sinusoidal patterns transiently during normal labor. These authors observed patterns persisting for up to 90 minutes in some cases.

The pathophysiology of sinusoidal patterns is unclear, in part due to various definitions. There seems to be general agreement that *antepartum* sine wave baseline undulations portend severe fetal anemia. Still, few anti-D alloimmunized fetuses develop this pattern (Nicolaides, 1989). The sinusoidal pattern has been reported to develop or disappear after fetal transfusion (Del Valle, 1992; Lowe, 1984). Ikeda and associates (1999) proposed that the pattern is related to waves of arterial blood pressure, reflecting oscillations in the baroreceptor-chemoreceptor feedback mechanism.

Periodic Fetal Heart Rate Changes

These refer to deviations from baseline that are temporally related to uterine contractions. *Acceleration* refers to a rise in fetal heart rate above baseline, and *deceleration* is a drop below the baseline rate. The nomenclature most commonly used in the United States is based on the *timing* of the deceleration in relation to contractions—thus, *early, late,* or *variable*. The waveform of these decelerations is also significant for pattern recognition. In early and late decelerations, the slope of fetal heart rate change is gradual, resulting in a curvilinear and uniform or symmetrical waveform. With variable decelerations, the slope of fetal heart rate change is abrupt and erratic, giving the waveform a jagged appearance. The NICHD Workshop (1997) proposed that decelerations be defined as *recurrent* if they accompanied ≥50 percent of contractions in any 20-minute period.

Another system now used less often to describe decelerations is based on the pathophysiological events considered most likely to underlie the

pattern. In this system, early decelerations are termed *head compression,* late decelerations are termed *uteroplacental insufficiency,* and variable decelerations are *cord compression patterns.*

Accelerations

These are abrupt heart rate increases above the fetal heart rate baseline and defined by an onset-to-peak rise within 30 seconds (American College of Obstetricians and Gynecologists, 2017a). At 32 weeks' gestation and beyond, an acceleration has a peak ≥15 bpm above baseline. Its duration is ≥15 sec but <2 minutes from onset to baseline return (see Table 24-1). Before 32 weeks, a peak ≥10 bpm for 10 seconds to 2 minutes is considered normal. Prolonged acceleration is defined as ≥2 minutes but <10 minutes.

According to Freeman and coworkers (2003), accelerations most often occur antepartum, in early labor, and in association with variable decelerations. Proposed mechanisms for intrapartum accelerations include fetal movement, stimulation by uterine contractions, umbilical cord occlusion, fetal stimulation during pelvic examination, scalp blood sampling, and acoustic stimulation. Accelerations are common during labor. These are virtually always reassuring and almost always confirm that the fetus is not acidemic at that time.

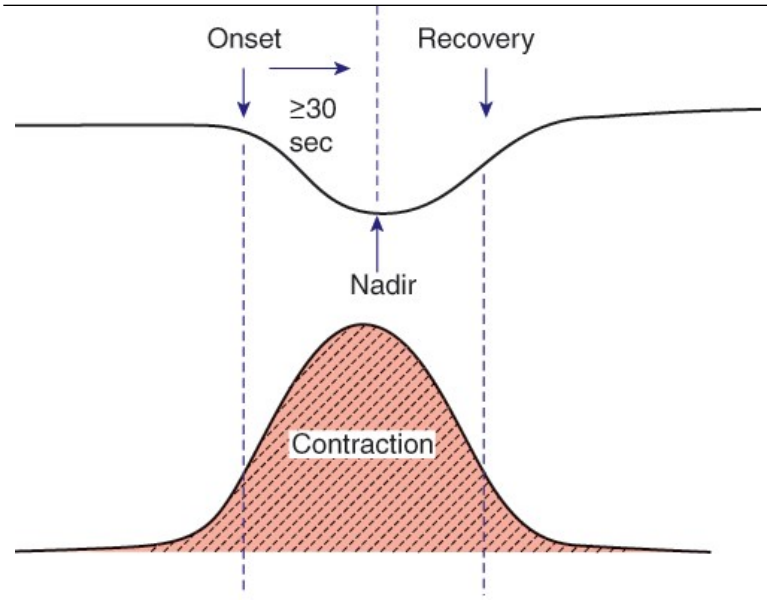
As with beat-to-beat variability, accelerations represent intact neurohormonal cardiovascular control mechanisms linked to fetal behavioral states. Krebs and colleagues (1982) analyzed electronic heart rate tracings in nearly 2000 fetuses and found sporadic accelerations during labor in 99.8 percent. Fetal heart rate accelerations during the first or last 30 minutes during labor, or both, were a favorable sign for fetal well-being. The absence of such accelerations during labor, however, is not necessarily an unfavorable sign unless coincidental with other nonreassuring changes. The chance of acidemia in the fetus that fails to respond to stimulation in the presence of an otherwise nonreassuring pattern approximates 50 percent (Clark, 1984; Smith, 1986).

Early Deceleration

This physiological response shows a gradual fetal heart rate decline and then return to baseline associated with a contraction (Fig. 24-11). Freeman and associates (2003) defined early decelerations as those generally seen in active labor between 4 and 7 cm cervical dilation. In their definition, the degree of deceleration is generally proportional to the contraction strength and rarely falls below 100 to 110 bpm or 20 to 30 bpm below baseline. Such decelerations are common during active labor and not associated with tachycardia, loss of variability, or other fetal heart rate changes. Importantly, early decelerations are not associated with fetal hypoxia, acidemia, or low Apgar scores.

FIGURE 24-11

Features of early fetal heart rate deceleration. Characteristics include a gradual decline in the heart rate with both onset and recovery coincident with the onset and recovery of the contraction. The nadir of the deceleration is 30 seconds or more after the deceleration onset.



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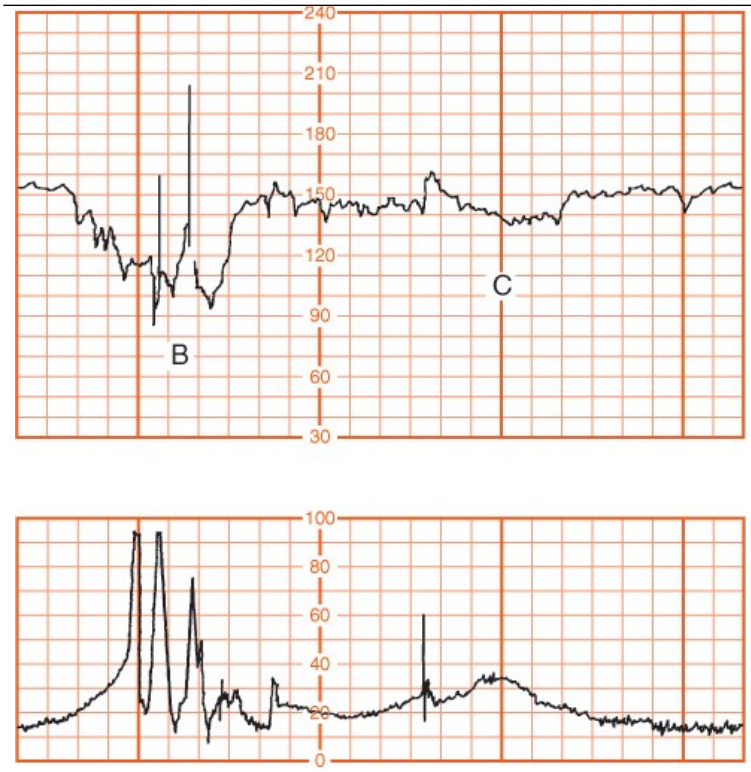
Head compression probably causes vagal nerve activation as a result of dural stimulation, and this mediates the heart rate deceleration (Paul, 1964). Ball and Parer (1992) concluded that fetal head compression is a likely cause not only of the deceleration shown in Figure 24-11 but also of those shown in Figure 24-12, which typically occur during second-stage labor. Indeed, they observed that head compression is the likely cause of many variable decelerations classically attributed to cord compression.

FIGURE 24-12

Two different fetal heart rate patterns during second-stage labor that are likely both due to head compression (*upper panel*). Maternal pushing efforts (*lower panel*) correspond to the spikes with uterine contractions. Fetal heart rate deceleration (*C*) is consistent with the pattern of head compression shown in Figure 24-11. Deceleration (*B*), however, is "variable" in appearance because of its jagged configuration and may alternatively represent cord occlusion.

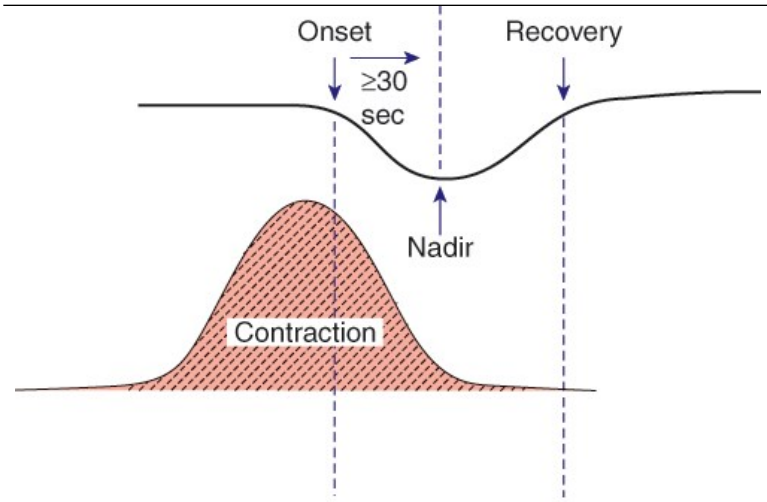


Late Deceleration

The fetal heart rate response to uterine contractions can reflect uterine perfusion or placental function. A late deceleration is a smooth, gradual, symmetrical decline in fetal heart rate beginning at or after the contraction peak and returning to baseline only after the contraction has ended. This deceleration reaches its nadir within 30 seconds of its onset. In most cases, the onset, nadir, and recovery of the deceleration occur after the beginning, peak, and ending of the contraction, respectively (Fig. 24-13). The magnitude of late decelerations is seldom more than 30 to 40 bpm below baseline and typically not more than 10 to 20 bpm. Late decelerations usually are not accompanied by accelerations. Myers and associates (1973) studied monkeys in which they compromised uteroplacental perfusion by lowering maternal aortic blood pressure. The interval or lag from the contraction onset until the late deceleration onset was directly related to basal fetal oxygenation. They demonstrated that the length of the lag was predictive of the fetal Po_2 but not fetal pH. The lower the fetal Po_2 before contractions, the shorter the lag to the onset of late decelerations. This lag reflected the time necessary for the fetal Po_2 to fall below a critical level necessary to stimulate arterial chemoreceptors, which mediated the decelerations.

FIGURE 24-13

Features of late fetal heart rate deceleration. Characteristics include gradual decline in the heart rate with the contraction nadir, and recovery occurring after the end of the contraction. The nadir of the deceleration occurs 30 seconds or more after the onset of the deceleration.



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Murata and coworkers (1982) also showed that a late deceleration was the first fetal heart rate consequence of uteroplacental-induced hypoxia. During the course of progressive hypoxia that led to death over 2 to 13 days, monkey fetuses invariably exhibited late decelerations before development of acidemia. Variability of the baseline heart rate disappeared as acidemia developed.

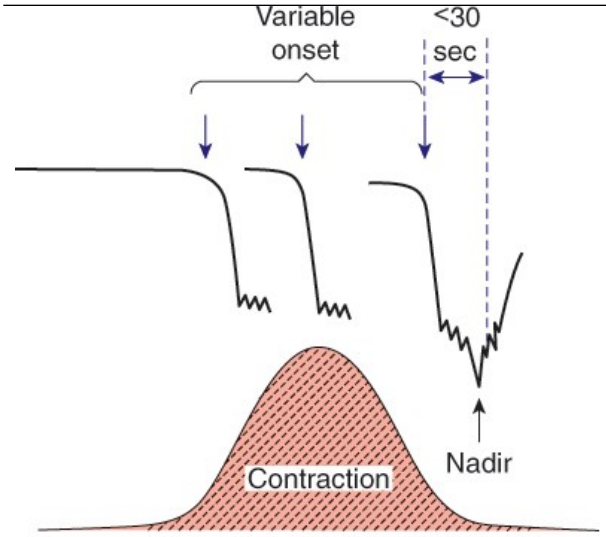
Generally, any process that produces maternal hypotension, excessive uterine activity, or placental dysfunction can induce late decelerations. The two most common sources are hypotension from epidural analgesia and uterine hyperactivity from oxytocin stimulation. Maternal diseases such as hypertension, diabetes, and collagen vascular disorders can cause chronic placental dysfunction. Placental abruption can produce acute late decelerations.

Variable Deceleration

The most frequent deceleration patterns encountered during labor are variable decelerations attributed to umbilical cord occlusion. In a study of more than 7000 monitor tracings, variable decelerations were identified in 40 percent when labor had progressed to 5 cm dilation and in 83 percent by the end of first-stage labor (Melchior, 1985). A variable deceleration is defined as an abrupt drop in the fetal heart rate beginning with the onset of the contraction and reaching a nadir in less than 30 seconds. The decrease must last between 15 seconds and 2 minutes and must be ≥15 bpm in amplitude. The onset of deceleration typically varies with successive contractions (Fig. 24-14).

FIGURE 24-14

Features of variable fetal heart rate decelerations. Characteristics include an abrupt decline in the heart rate, and onset that commonly varies with successive contractions. The deceleration measures ≥15 bpm for ≥15 seconds and has an onset-to-nadir phase of <30 seconds. Total duration is <2 minutes.



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Hon (1959) tested the effects of umbilical cord compression on fetal heart rate (Fig. 24-15). In experimental animals, complete occlusion of the umbilical cord produces abrupt, jagged-appearing deceleration of the fetal heart rate (Fig. 24-16). Concomitantly, fetal aortic pressure rises. Itskovitz and colleagues (1983) observed that variable decelerations in fetal lambs occurred only after umbilical blood flow was reduced by at least 50 percent.

FIGURE 24-15

A. The effects of 25-second cord compression compared with those of 40 seconds in panel **(B)**. (Redrawn with permission from Hon EH: The fetal heart rate patterns preceding death in utero, Am J Obstet Gynecol. 1959 Jul;78(1):47–56.)

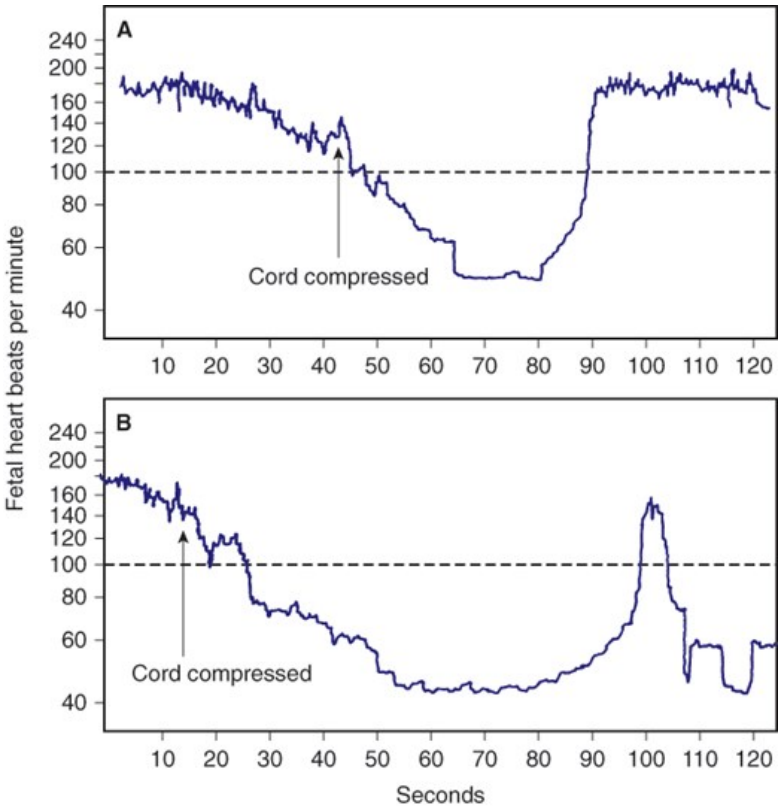

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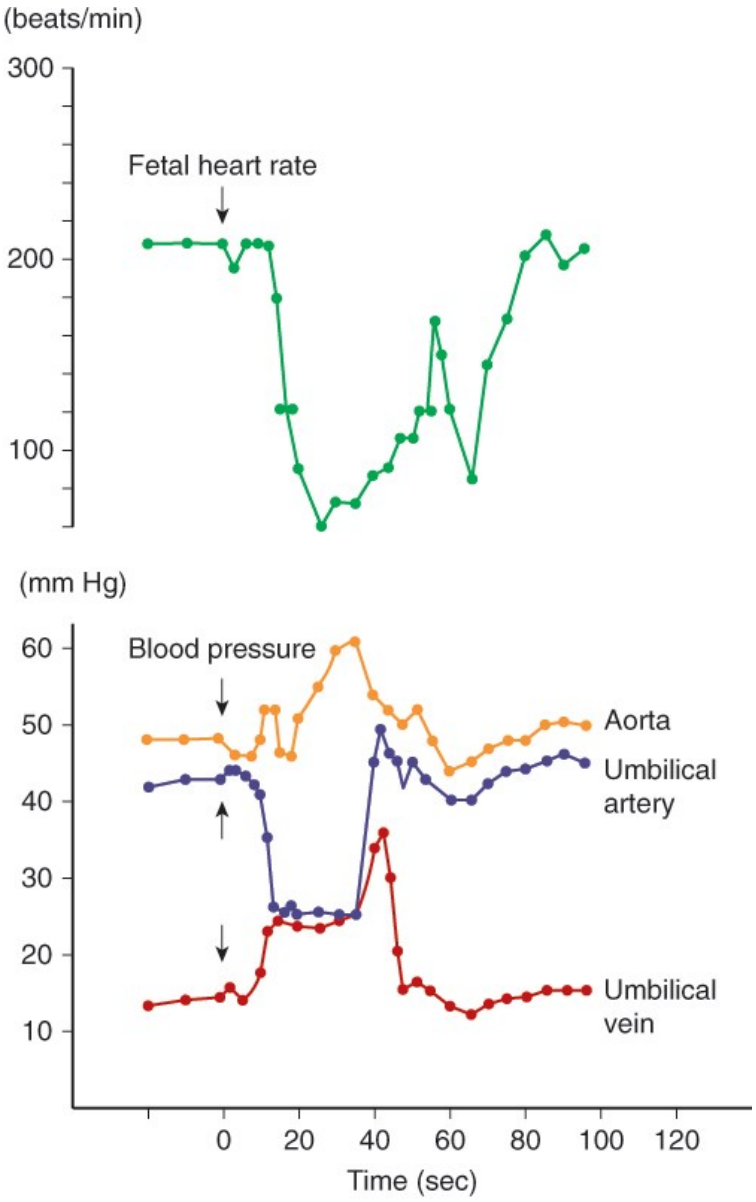
FIGURE 24-16

Total umbilical cord occlusion (*arrow*) in the sheep fetus is accompanied by an increase in fetal aortic blood pressure. Blood pressure changes in the umbilical vessels are also shown. (Redrawn with permission from Künzel W: Fetal heart rate alterations in partial and total cord occlusion. In Künzel W (ed): Fetal Heart Rate Monitoring: Clinical Practice and Pathophysiology. Berlin, Springer, 1985.)



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Two types of variable decelerations are shown in Figure 24-17. The deceleration denoted by "A" is very much like that seen with complete umbilical cord occlusion in experimental animals (see Fig. 24-16). Deceleration "B," however, has a different configuration because of the "shoulders" of acceleration before and after the deceleration component. Lee and coworkers (1975) proposed that this form of variable deceleration was caused by differing degrees of partial cord occlusion. In this physiological scheme, occlusion of only the vein reduces fetal blood return, thereby triggering a baroreceptor-mediated acceleration. With increasing intrauterine pressure and subsequent complete cord occlusion, fetal systemic hypertension develops due to obstruction of umbilical artery flow. This stimulates a baroreceptor-mediated deceleration. Presumably, the aftercoming shoulder of the acceleration represents the same events occurring in reverse (Fig. 24-18).

FIGURE 24-17

Varying (variable) fetal heart rate decelerations. Deceleration (*B*) exhibits "shoulders" of acceleration compared with deceleration (*A*).

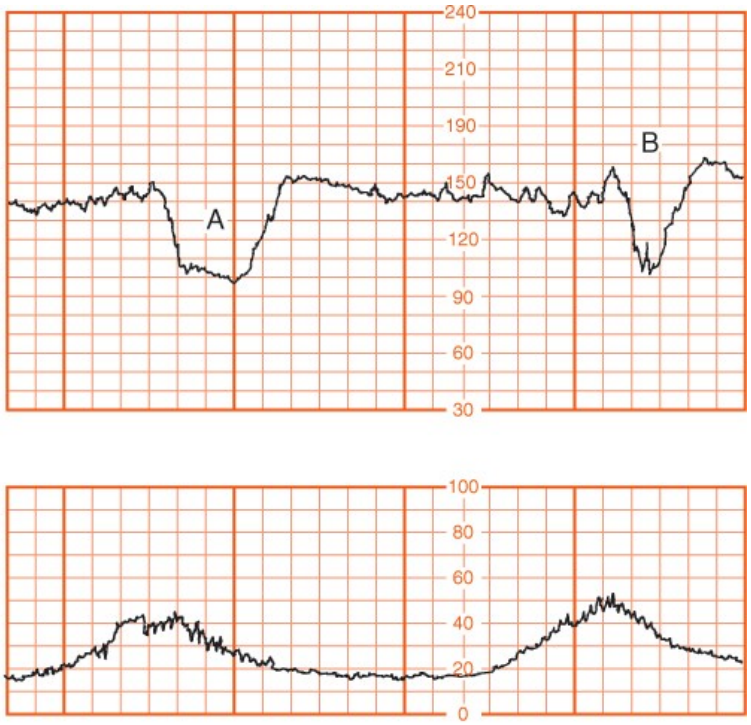
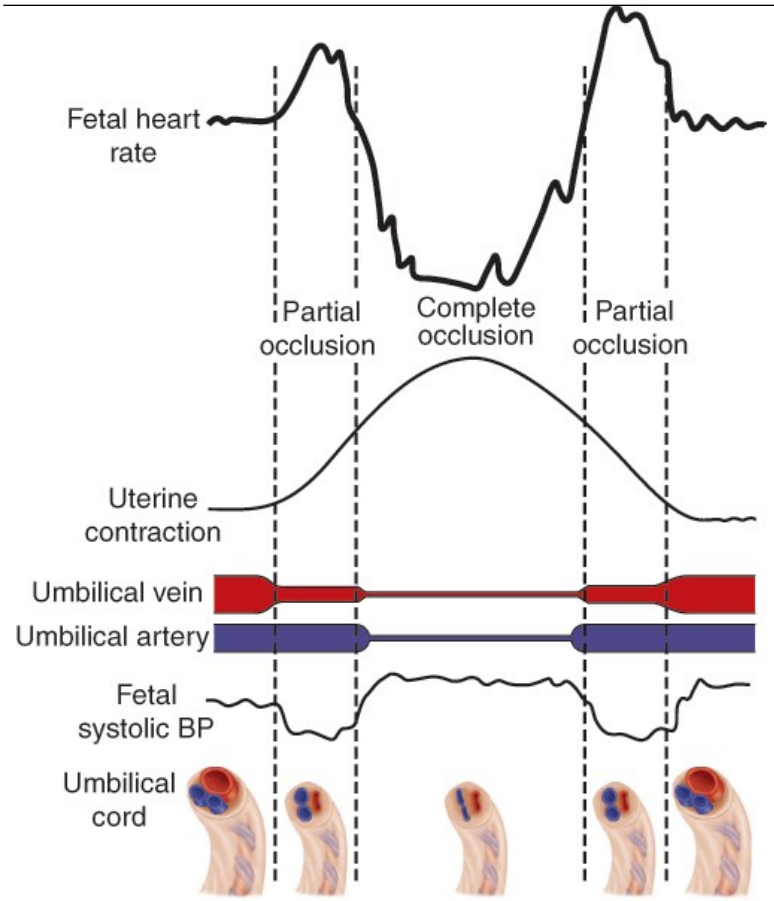


FIGURE 24-18

Schematic representation of the fetal heart rate effects with partial and complete umbilical cord occlusion. Uterine pressures generated early in a contraction cause cord compression predominantly of the thin-walled umbilical vein. The resulting decrease in fetal cardiac output leads to an initial compensatory rise in fetal heart rate. As cord compression intensifies, umbilical arteries are then also compressed. The resulting rise in fetal systolic blood pressure leads to a vagal-mediated fetal heart rate deceleration. As the contraction abates and compression is relieved first on the umbilical arteries, elevated fetal systolic blood pressures drop and the deceleration resolves. A final increase in fetal heart rate is seen as a result of persistent umbilical vein occlusion. With completion of the uterine contraction and cord compression, the fetal heart rate returns to baseline. BP = blood pressure. (Adapted with permission from Lee CV, DiLaretto PC, Lane JM: A study of fetal heart rate acceleration patterns, Obstet Gynecol. 1975 Feb;45(2):142–146.)



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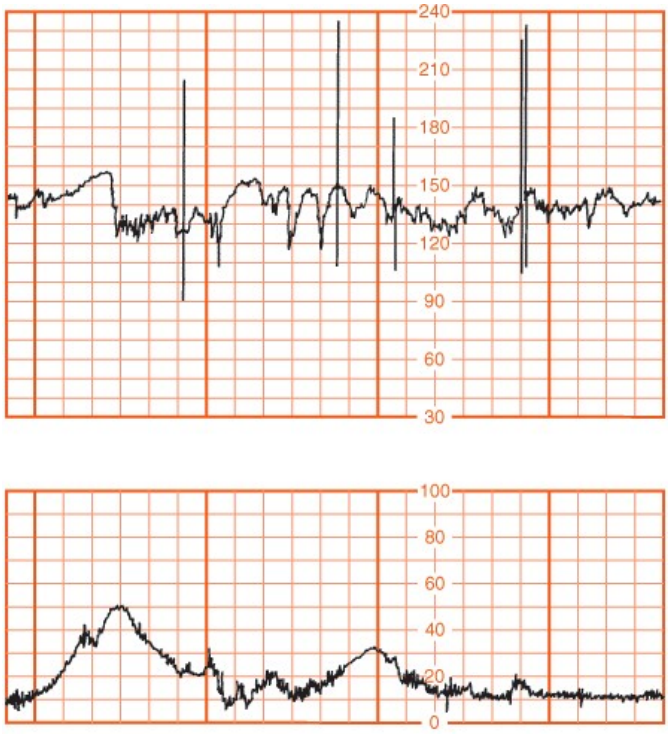
Ball and Parer (1992) concluded that variable decelerations are mediated vagally and that the vagal response may be due to chemoreceptor or baroreceptor activity or both. Partial or complete cord occlusion produces an increase in afterload (baroreceptor) and a drop in fetal arterial oxygen content (chemoreceptor). These both result in vagal activity leading to deceleration. In fetal monkeys, the baroreceptor reflexes appear to operate during the first 15 to 20 seconds of umbilical cord occlusion followed by decline in Po_2 at approximately 30 seconds, which then serves as a chemoreceptor stimulus (Mueller-Heubach, 1982).

Thus, variable decelerations represent fetal heart rate reflexes that reflect either blood pressure changes due to interruption of umbilical flow or changes in oxygenation. It is likely that most fetuses have experienced brief but recurrent periods of hypoxia due to umbilical cord compression during gestation. The frequency and inevitability of cord occlusions undoubtedly have provided the fetus with these physiological mechanisms as a means of coping. The great dilemma for the obstetrician in managing variable fetal heart rate decelerations is determining when variable decelerations are pathological. According to the American College of Obstetricians and Gynecologists (2017a), recurrent variable decelerations with minimal-to-moderate beat-to-beat variability are *indeterminate,* whereas those with absent variability are *abnormal.*

Other fetal heart rate patterns have been associated with umbilical cord compression. *Saltatory* baseline heart rate (Fig. 24-19) was first linked to umbilical cord complications during labor (Hammacher, 1968). The pattern consists of rapidly recurring couplets of acceleration and deceleration causing relatively large oscillations of the baseline fetal heart rate. We also observed a relationship between cord occlusion and the saltatory pattern in postterm pregnancies (Leveno, 1984). In the absence of other fetal heart rate findings, these do not signal fetal compromise. *Lambda* is a pattern involving an acceleration followed by a variable deceleration with no acceleration at the end of the deceleration. This pattern typically is seen in early labor and is not ominous (Freeman, 2003). This lambda pattern may result from mild cord compression or stretch. *Overshoot* is a variable deceleration followed by acceleration. The clinical significance of this pattern is controversial (Westgate, 2001).

FIGURE 24-19

Saltatory baseline fetal heart rate showing rapidly recurring couplets of acceleration combined with deceleration.



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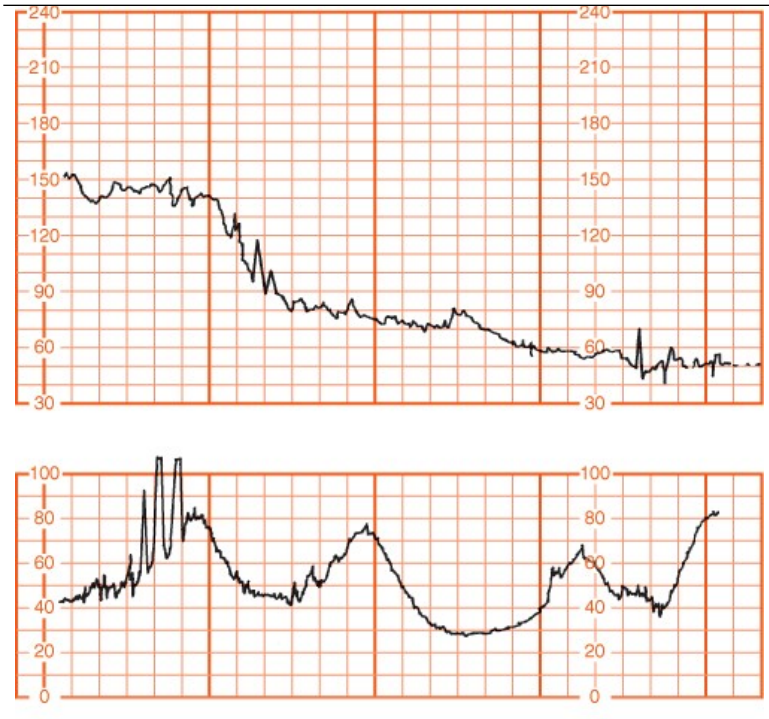
Prolonged Deceleration

This pattern, which is shown in Figure 24-20, is defined as an isolated deceleration ≥15 bpm that lasts ≥2 minutes but <10 minutes from onset to return to baseline. Prolonged decelerations are difficult to interpret because they are seen in many different clinical situations. Some of the more frequent causes are cervical examination, uterine hyperactivity, cord entanglement, and maternal supine hypotension.

FIGURE 24-20

Prolonged fetal heart rate deceleration due to uterine hyperactivity. Approximately 3 minutes of the tracing are shown, but the fetal heart rate returned to normal after uterine hypertonus resolved. Vaginal delivery later ensued.



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Epidural, spinal, or paracervical analgesia may induce a prolonged deceleration (Eberle, 1998). Hill and associates (2003) observed prolonged deceleration in 1 percent of women given epidural analgesia during labor at Parkland Hospital. Other causes of prolonged deceleration include maternal hypoperfusion or hypoxia from any cause, placental abruption, umbilical cord knots or prolapse, maternal seizures including eclampsia and epilepsy, application of a fetal scalp electrode, impending birth, or maternal Valsalva maneuver. In one example, Ambia and colleagues (2017) described prolonged decelerations lasting 2 to 10 minutes following an eclamptic seizure.

The placenta is effective in resuscitating the fetus if the original insult does not recur immediately. Occasionally, such self-limited prolonged decelerations are followed by loss of beat-to-beat variability, baseline tachycardia, and even a period of late decelerations, all of which resolve as the fetus recovers. Freeman and colleagues (2003) emphasize that the fetus may die during prolonged decelerations. Thus, management of prolonged decelerations can be extremely tenuous. Management of isolated prolonged decelerations is based on bedside clinical judgment, which inevitably will sometimes be imperfect given the unpredictability of these decelerations.

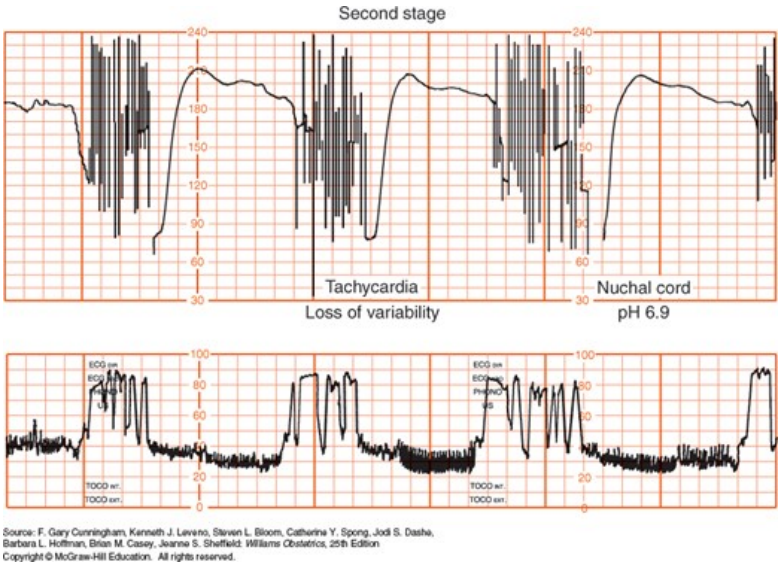
Fetal Heart Rate Patterns During Second-Stage Labor

Decelerations are virtually ubiquitous during the second stage of labor. In one study, only 1.4 percent of more than 7000 deliveries lacked decelerations during second-stage labor (Melchior, 1985). Both cord and fetal head compressions have been implicated as causes of decelerations and baseline bradycardia in this stage. Profound, prolonged fetal heart rate deceleration in the 10 minutes preceding vaginal delivery has been described (Boehm, 1975). And, similar prolonged second-stage decelerations were associated with a stillbirth and neonatal death (Herbert, 1981). These experiences attest to the unpredictability of the fetal heart rate during second-stage labor.

Spong and associates (1998) analyzed the characteristics of second-stage variable fetal heart rate decelerations in 250 deliveries. They found that as the total number of decelerations <70 bpm increased, the 5-minute Apgar score decreased. Of other patterns in second-stage labor, Picquard and coworkers (1988) reported that loss of beat-to-beat variability and baseline fetal heart rate <90 bpm predicted fetal acidemia. Krebs and associates (1981) also found that persistent or progressive baseline bradycardia or baseline tachycardia was associated with lower Apgar scores. Gull and colleagues (1996) observed that abrupt fetal heart rate deceleration to <100 bpm associated with loss of beat-to-beat variability for 4 minutes or longer was predictive of fetal acidemia. Thus, abnormal baseline heart rate—either bradycardia or tachycardia, absent beat-to-beat variability, or both—in the presence of deep second-stage decelerations is associated with a greater risk for fetal compromise (Fig. 24-21).

FIGURE 24-21

Cord-compression fetal heart rate decelerations in second-stage labor associated with tachycardia and loss of variability. The umbilical cord arterial pH was 6.9.



Admission Fetal Monitoring in Low-Risk Pregnancies

With this approach, women with low-risk pregnancies are monitored for a short time on admission for labor. In one study, 3752 low-risk women in spontaneous labor at admission were randomly assigned either to auscultation of the fetal heart or to 20 minutes of electronic fetal monitoring (Mires, 2001). Use of admission electronic fetal monitoring did not improve neonatal outcome. Moreover, its use resulted in a greater number of interventions, including operative delivery. A similar study echoed these neonatal outcomes (Impey, 2003). More than half of the women enrolled in these studies eventually required continuous monitoring. A review by Devane and associates (2017) found that admission fetal monitoring programs for low-risk pregnancy are associated with a higher risk for cesarean delivery. Somewhat related, with the increasing rate of scheduled cesarean deliveries in the United States, clinicians and hospitals must decide whether fetal monitoring is required before the procedure in low-risk women.

Computerized Interpretation

Fetal heart rate pattern interpretations are subjective. Thus, the potential for computer assistance to enhance the precision of identifying abnormal patterns appeared promising. The INFANT Collaborative Group (2017) studied whether the addition of computer-based decision-support software for interpretation of fetal heart rate patterns lowered the number of poor neonatal outcomes. In this trial, 23,515 women were randomized to computer-assisted interpretation compared with 23,055 women in a conventional clinical interpretation arm. Perinatal outcomes such as intrapartum stillbirth, early neonatal death, and neonatal encephalopathy were not improved by computer assistance. Cesarean delivery rates were similar in both groups. Moreover, a 2-year follow-up of a subset of the surviving children showed no differences in their neurological development.

OTHER INTRAPARTUM ASSESSMENT TECHNIQUES

Fetal Scalp Blood Sampling

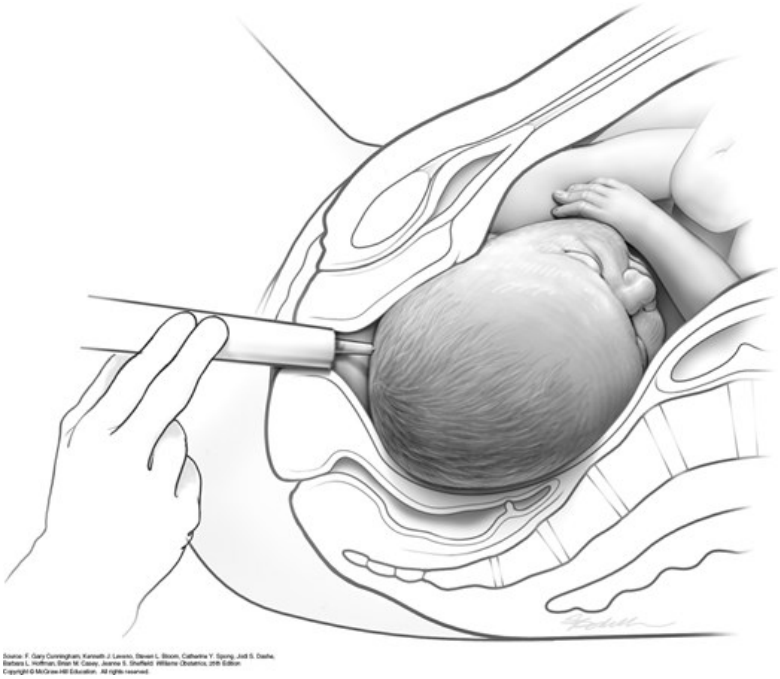
According to the American College of Obstetricians and Gynecologists (2017a), measurements of the pH in capillary scalp blood may help identify the fetus in serious distress. However, this group also emphasizes that neither normal nor abnormal scalp pH results are predictive of neonatal outcome. Notably, the procedure is now used uncommonly and is not available at most hospitals in the United States.

With sampling, an illuminated endoscope is inserted through the dilated cervix after membrane rupture and is pressed firmly against the fetal scalp (Fig. 24-22). The skin is wiped clean with a cotton swab and coated with a silicone gel, which allows fetal blood to accumulate as discrete globules. An incision is made through the fetal scalp to a depth of 2 mm with a special blade on a long handle. As a drop of blood forms on the surface, it is

immediately collected into a heparinized glass capillary tube. The pH of the blood is measured promptly.

FIGURE 24-22

The technique of fetal scalp sampling using an amnioscope. The end of the endoscope is displaced from the fetal vertex approximately 2 cm to show the disposable blade against the fetal scalp before incision.



The pH of fetal capillary scalp blood is usually lower than that of umbilical venous blood and approaches that of umbilical arterial blood. In one algorithm, if the pH is ≥7.25, labor is observed, and if between 7.20 and 7.25, the pH measurement is repeated within 30 minutes (Zalar, 1979). If the pH is <7.20, another scalp blood sample is collected immediately, and the mother is taken to an operating room and prepared for surgery. Delivery is performed promptly if the low pH is confirmed. Otherwise, labor is allowed to continue, and scalp blood samples are repeated periodically.

The only benefits reported for scalp blood pH testing are fewer cesarean deliveries for fetal distress (Young, 1980b). However, Goodwin and coworkers (1994) showed a decrease in the scalp pH sampling rate from approximately 1.8 percent in the mid-1980s to 0.03 percent by 1992. This drop in sampling rate was not associated with a higher cesarean delivery rate for fetal distress. They concluded that scalp blood pH sampling was unnecessary.

Kruger and colleagues (1999) have advocated the use of fetal scalp blood lactate concentration as an adjunct to pH. Wiberg-Itzel and associates (2008) randomly assigned 1496 fetuses to scalp blood pH analysis and 1496 to scalp blood lactate analysis. They found either to be equivalent in predicting fetal acidemia. The advantage of lactate measurement was that a smaller amount of blood was needed, which led to a lower procedural failure rate compared with scalp blood sampling for pH.

Scalp Stimulation

Clark and coworkers (1984) have suggested that fetal scalp stimulation is an alternative to scalp blood sampling. This proposal was based on the observation that heart rate acceleration in response to pinching the fetal scalp with an Allis clamp just before obtaining blood was invariably associated with a normal pH. Conversely, failure to provoke acceleration was not uniformly predictive of fetal acidemia. Later, Elimian and associates (1997) reported that of 58 cases in which the fetal heart rate accelerated >10 bpm after 15 seconds of gentle digital stroking of the scalp, 100 percent had a scalp blood pH of >7.20. Without an acceleration, however, only 30 percent had a scalp blood pH >7.20. Following a prospective cohort study, Tahir Mahmood and coworkers (2017) concluded that fetal scalp stimulation was a reliable alternative to scalp blood pH determination.

Vibroacoustic Stimulation

Fetal heart rate acceleration in response to vibroacoustic stimulation has been recommended as a substitute for fetal scalp blood sampling

(Edersheim, 1987). The technique uses an electronic artificial larynx placed approximately 1 cm from or directly onto the maternal abdomen (Chap. 17, Acoustic Stimulation Tests). Response to vibroacoustic stimulation is considered normal if a fetal heart rate acceleration of at least 15 bpm for at least 15 seconds occurs within 15 seconds after the stimulation and with prolonged fetal movements (Sherer, 1994).

Lin and colleagues (2001) prospectively studied vibroacoustic stimulation in 113 women in labor with either moderate-to-severe variable or late fetal heart rate decelerations. They concluded that this technique is an effective predictor of fetal acidosis in the setting of variable decelerations. The predictability for fetal acidosis, however, is limited in the setting of late decelerations. Other investigators have reported that vibroacoustic stimulation in second-stage labor did not predict neonatal outcome or enhance labor management (Anyaegbunam, 1994).

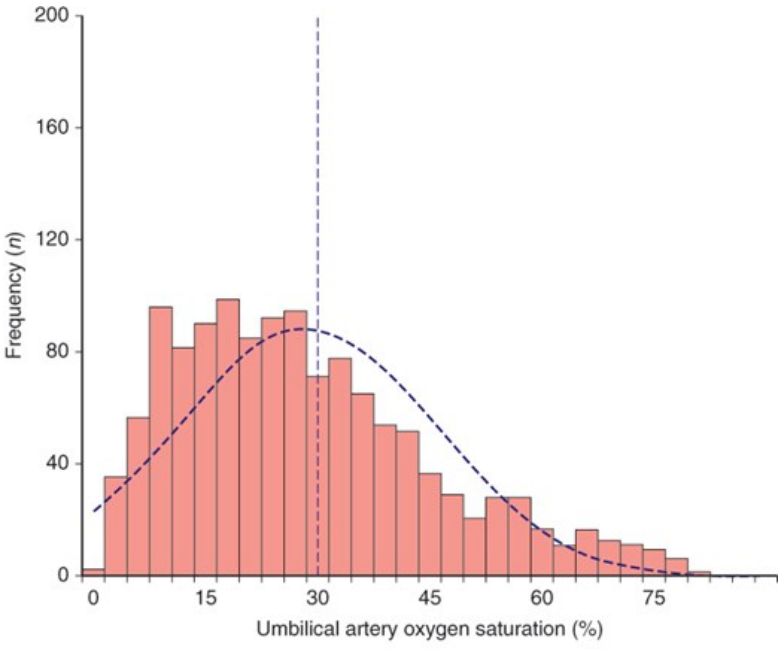
Skupski and coworkers (2002) performed a metaanalysis of reports on intrapartum fetal stimulation tests published between 1966 and 2000. Four types of fetal stimulation were analyzed and included fetal scalp puncture for blood pH testing, Allis clamp pinching of the fetal scalp, vibroacoustic stimulation, and digital stroking of the fetal scalp. Results were similar for all four methods. These investigators concluded that intrapartum stimulation tests were useful to exclude fetal acidemia. They cautioned, however, that these tests are “less than perfect.”

Fetal Pulse Oximetry

Using technology similar to that of adult pulse oximetry, this instrumentation allows assessment of fetal oxyhemoglobin saturation once membranes are ruptured. A unique padlike sensor is inserted through the cervix and positioned against the fetal face. The transcervical device reliably registers fetal oxygen saturation in 70 to 95 percent of women throughout 50 to 88 percent of their labors (Yam, 2000). Using fetal pulse oximetry, the lower limit for normal fetal oxygen saturation is generally considered to be 30 percent (Gorenberg, 2003; Stiller, 2002). However, when measured in umbilical arterial blood, fetal oxygen saturation normally varies greatly, as shown in Figure 24-23. Bloom and associates (1999) reported that brief, transient fetal oxygen saturations <30 percent were common during labor because such values were observed in 53 percent of fetuses with normal outcomes. When persistent for 2 minutes or longer, however, saturation values <30 percent were associated with a greater risk of potential fetal compromise.

FIGURE 24-23

Frequency distribution of umbilical artery oxygen saturation values in 1281 vigorous newborn infants. Dotted line indicates normal distribution. (Redrawn with permission from Arikan GM, Scholz HS, Petru E, et al: Cord blood oxygen saturation in vigorous infants at birth: what is normal? BJOG. 2000 Aug;107(8):987–994.)



Source: F. Gary Cunningham, Kenneth J. Leveno, Steven L. Bloom, Catherine Y. Spong, Jodi S. Dashe, Barbara L. Hoffman, Brian M. Casey, Jeanne S. Sheffield: *Williams Obstetrics*, 25th Edition

Garite and colleagues (2000) randomly assigned 1010 women with term pregnancies and in whom predefined abnormal fetal heart rate patterns

developed. Patients received either conventional fetal monitoring alone or fetal monitoring plus continuous fetal pulse oximetry. The use of fetal pulse oximetry significantly reduced the cesarean delivery rate for nonreassuring fetal status from 10.2 to 4.5 percent. Alternatively, the cesarean delivery rate for dystocia rose significantly from 9 to 19 percent when pulse oximetry was used. No neonatal benefits or adverse effects were associated with fetal pulse oximetry. Based on these observations, the Food and Drug Administration approved marketing of the Nellcor N-400 Fetal Oxygen Monitoring System.

Since then, three other randomized trials have compared fetal pulse oximetry with standard care. In all three trials, neonatal outcomes were similar between the two study arms. East and coworkers (2006) reported that the addition of oximetry significantly reduced cesarean delivery rates for a nonreassuring fetal heart rate pattern. However, Bloom (2006) and Klauser (2005), each with their colleagues, found no difference in cesarean delivery rates between the two study groups. Because of these findings, in 2005, the manufacturer discontinued sale of the fetal oximeter system in the United States.

Fetal Electrocardiography

As fetal hypoxia worsens, the fetal ECG changes. Namely, the mature fetus exposed to hypoxemia develops an elevated ST segment and a progressive rise in the T-wave height that can be expressed as a T:QRS ratio (Fig. 24-24). Increasing T:QRS ratios are thought to reflect the fetal cardiac ability to adapt to hypoxia and appear before neurological damage. Further worsening of hypoxia then leads to progressively negative ST-segment deflection that takes on a biphasic form (Fig. 24-25). It is reasonable to consider that ST-segment abnormalities might occur late in the course of fetal compromise. Indeed, it has been hypothesized that ST-segment changes reflect myocardial tissue hypoxia.

FIGURE 24-24

A. ST segment changes in normal and hypoxic conditions. **B.** Generation of T:QRS ratios. (Redrawn with permission from Devoe L: ECG analysis: the next generation in electronic fetal monitoring? Contemporary Ob/Gyn, September 15, 2006.)

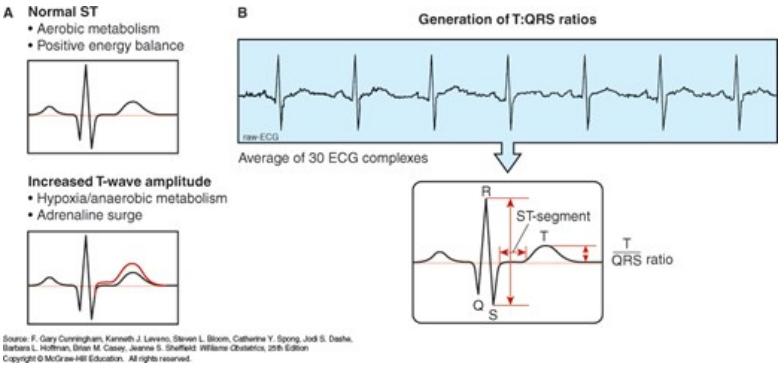
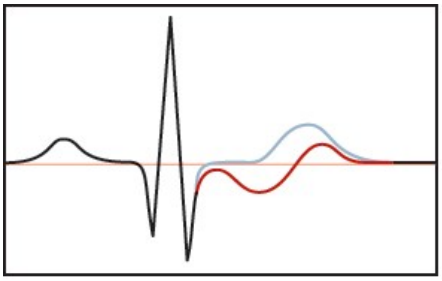


FIGURE 24-25

Biphasic ST-segment waveform with progressive fetal hypoxia. (Adapted with permission from Devoe L: ECG analysis: the next generation in electronic fetal monitoring? Contemporary Ob/Gyn, September 15, 2006.)



Because of these findings, several investigators have assessed the value of analyzing these parameters as an adjunct to conventional fetal monitoring.

The technique requires internal fetal heart monitoring and special equipment to process the fetal ECG. In 2005, the manufacturer—Neoventa Medical—received Food and Drug Administration approval for their ST analysis program named the STAN system.

Several studies have evaluated ST-segment changes with fetal monitoring. In one randomized trial of 2400 pregnancies, neonatal outcomes were not improved compared with those in which conventional fetal monitoring alone was used (Westgate, 1993). However, the cesarean delivery rate for fetal distress declined in those with ST-segment analysis. Amer-Wåhlin and colleagues (2001, 2007) found that the addition of ST-segment analysis to conventional fetal monitoring significantly lowered cesarean delivery rates for fetal distress and reduced metabolic acidemia in umbilical artery blood.

Subsequently, Doria and associates (2007) introduced STAN as a clinical practice and reported no changes in the incidence of operative delivery or neonatal encephalopathy. And, one metaanalysis of five randomized trials comprising 15,352 patients found that ST-segment analysis did not lower rates of cesarean delivery or fetal metabolic acidemia at birth (Becker, 2012).

Last, in a trial by the NICHD, 5532 women were randomly assigned to an ST-segment analysis arm (the open group) and 5576 to standard intrapartum management (the masked group). The primary outcome was a composite of one or more of seven events associated with fetal compromise (Belfort, 2015). In the open group, clinical practice was directed to some degree by predetermined ST-segment analysis guidelines. These stipulated that intervention should be withheld, that is, expectant management adopted, for at least 60 minutes despite the presence of minimal variability; variable decelerations lasting ≥60 seconds or dropping to ≥60 bpm; recurrent late decelerations; or prolonged decelerations lasting >2 minutes, so long as no ST-event was present. These guidelines did not pertain to the standard usual management group. Notably, in the open group, 55 women were delivered when STAN guidelines indicated that labor should continue. This composed 20 percent of the total 287 cesarean deliveries performed for fetal distress in this group. Clearly, the attending physicians abandoned the open group protocol that stipulated nonintervention. They likely perceived the fetal heart rate patterns to reflect those formerly accepted in their usual practice as nonreassuring.

The results of this trial showed that STAN had no effect on neonatal outcome or cesarean delivery rates (Belfort, 2015). In their review, Neilson and colleagues (2015) reached similar conclusions. These results have essentially eliminated use of ST-segment analysis in the United States, but this technology is still used in Europe.

Intrapartum Doppler Velocimetry

Doppler interrogation of the umbilical artery has been studied as another potential adjunct to conventional fetal monitoring. Further described in Chapter 10 (Doppler), abnormal Doppler waveforms may signify pathological umbilical-placental vessel resistance. From their review, Farrell and associates (1999) concluded that this technique, used intrapartum, was a poor predictor of adverse perinatal outcomes.

NONREASSURING FETAL STATUS

The term *fetal distress* is too broad and vague to be applied with any precision to clinical situations (American College of Obstetricians and Gynecologists, 2014). Uncertainty regarding the diagnosis based on interpretation of fetal heart rate patterns has given rise to descriptions such as *reassuring* or *nonreassuring.* The term “reassuring” suggests a restoration of confidence in the health of the fetus by a particular pattern. In contrast, a “nonreassuring” designation suggests inability to remove doubt. These patterns during labor are dynamic, and they can rapidly change from reassuring to nonreassuring and vice versa. *These assessments are subjective clinical judgments that are inevitably subject to imperfection and must be recognized as such.*

The difficulty in assigning a nonreassuring label to fetal heart rate patterns stems in part from the fact that these patterns are more a reflection of fetal physiology than of pathology. Physiological control of heart rate includes various interconnected mechanisms that depend on blood flow and oxygenation. Moreover, the activity of these control mechanisms is influenced by the preexisting state of fetal oxygenation, for example, as seen with chronic placental insufficiency. Importantly, the fetus is tethered by an umbilical cord, whereby blood flow is constantly in jeopardy. Moreover, normal labor is a process of increasing acidemia (Rogers, 1998). Thus, normal labor is a process of repeated fetal hypoxic events that can infrequently lead to significant acidemia.

Diagnosis

Identification of “fetal distress” based on fetal heart rate patterns is imprecise and controversial. Experts in interpretation of these patterns often disagree with each other. Ayres-de-Campos and colleagues (1999) investigated interobserver agreement of fetal heart rate pattern interpretation and

found that agreement—or conversely, disagreement—was related to whether the pattern was normal, suspicious, or pathological. Specifically, experts agreed on 62 percent of normal patterns, 42 percent of suspicious patterns, and only 25 percent of pathological patterns. Keith and coworkers (1995) asked each of 17 experts to review 50 tracings on two occasions, at least 1 month apart. Approximately 20 percent changed their own interpretations, and approximately 25 percent did not agree with the interpretations of their colleagues.

To develop standardized and unambiguous definitions of fetal heart rate (FHR) tracings, the NICHD (1997) held a succession of workshops in 1995 and 1996 and published recommendations for interpreting these patterns. As previously shown in Table 24-1, a second workshop was convened to reevaluate these recommendations and clarify terminology (Macones, 2008). A major result was the recommendation of a three-tier system for classification of FHR patterns (Table 24-2). The American College of Obstetricians and Gynecologists (2017b) has recommended use of this tiered system.

TABLE 24-2

Three-Tier Fetal Heart Rate Interpretation System

Category I—Normal

Include all of the following:

- Baseline rate: 110–160 bpm
- Baseline FHR variability: moderate
- Late or variable decelerations: absent
- Early decelerations: present or absent
- Accelerations: present or absent

Category II—Indeterminate

Include all FHR tracings not categorized as Category I or III.

Category II tracings may represent an appreciable fraction of those encountered in clinical care. Examples 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 min but <10 min
- Recurrent late decelerations with moderate baseline variability
- Variable decelerations with other characteristics, such as slow return to baseline, “overshoots,” or “shoulders”

Category III—Abnormal

Include either:

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

bpm = beats per minute; FHR = fetal heart rate.

Reproduced with permission 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: update on definitions, interpretation, and research guidelines, Obstet Gynecol. 2008 Sep;112(3):661–666.

A few studies have assessed this three-tiered system. Jackson and coworkers (2011) studied 48,444 women in labor and found that category I (normal FHR) patterns were observed during labor in 99.5 percent of tracings. Category II (indeterminate FHR) patterns were found in 84.1 percent of tracings, and category III (abnormal FHR) patterns were seen in 0.1 percent (54 women). Most—84 percent of women—had a mix of categories during labor. Cahill and colleagues (2012) retrospectively correlated the incidence of umbilical cord acidemia (pH ≤7.10) with fetal heart rate characteristics during the 30 minutes preceding delivery. None of the three categories demonstrated a significant association with cord blood acidemia. The American

College of Obstetricians and Gynecologists and the American Academy of Pediatrics (2014) concluded that a category I or II tracing with a 5-minute Apgar score >7 or with normal arterial blood acid–base values was not consistent with an acute hypoxic-ischemic event.

Sholapurkar (2012) challenged the validity of the three-tier system because most abnormal fetal heart rate patterns fall into the indeterminate category II. It was further suggested that this resulted from most fetal heart rate decelerations being inappropriately classified as *variable* decelerations due to cord compression. A group of 19 experts led by Clark (2013) observed that more than 80 percent of fetuses have FHR patterns in tier II. They proposed a management algorithm for these fetuses, however, their hypothetical algorithm was not clinically tested.

Parer and King (2010) compared this situation in the United States with that of other countries in which a consensus on classification and management has been reached by several professional societies. Some of these include the Royal College of Obstetricians and Gynaecologists, the Society of Obstetricians and Gynaecologists of Canada, the Royal Australian and New Zealand College of Obstetricians and Gynaecologists, and the Japan Society of Obstetrics and Gynecology. These authors further comment that the NICHD three-tier system is inadequate because category II—an indeterminate FHR pattern—contains a vast heterogenous mixture of patterns that prevents development of a management strategy.

Parer and Ikeda (2007) had previously proposed a color-coded five-tier system for both FHR interpretation *and* management. Two subsequent reports have compared the five- and three-tier systems. Bannerman and associates (2011) found that the two systems were similar in fetal heart rate interpretations for tracings that were either very normal or very abnormal. Coletta and coworkers (2012) found that the five-tier system had better sensitivity than the three-tier system. Elliott and colleagues (2010) used computerization to measure the performance of a five-tier classification system but failed to successfully analyze and categorize 2472 fetal heart recordings.

It is apparent that, after 50 years of continuous electronic fetal heart rate monitoring use, there is not a consensus on interpretation and management recommendations for FHR patterns (Parer, 2011).

Meconium in the Amnionic Fluid

Obstetricians have long realized that meconium during labor is problematic in the prediction of fetal distress or asphyxia. Indeed, although 12 to 22 percent of labors are complicated by meconium, only a few are linked to neonatal mortality. In an investigation from Parkland Hospital, meconium was found to be a “low-risk” obstetrical hazard because the perinatal mortality rate attributable to meconium was only 1 death per 1000 live births (Nathan, 1994).

Three theories regarding fetal passage of meconium may explain, in part, the tenuous connection between its detection and neonatal mortality. First, fetuses may pass meconium in response to hypoxia, and meconium therefore signals fetal compromise (Walker, 1953). Second, in utero passage of meconium may represent normal gastrointestinal tract maturation under neural control (Mathews, 1979). A final theory posits that meconium passage follows vagal stimulation from common but transient umbilical cord entrapment with resultant increased bowel peristalsis (Hon, 1961).

Ramin and associates (1996) studied almost 8000 pregnancies with meconium-stained amnionic fluid delivered at Parkland Hospital. Meconium aspiration syndrome was significantly associated with fetal acidemia at birth. Other significant correlates of aspiration included cesarean delivery, forceps to expedite delivery, intrapartum heart rate abnormalities, depressed Apgar scores, and need for assisted ventilation at delivery. Analysis of the type of fetal acidemia based on umbilical blood gases suggested that the fetal compromise associated with meconium aspiration syndrome was an acute event. This is because most acidemic fetuses had abnormally increased P_{CO_2} values rather than a pure metabolic acidemia.

Dawes and coworkers (1972) observed that such hypercarbia in fetal lambs induces gasping and resultant increased amnionic fluid inhalation. Jovanovic and Nguyen (1989) observed that meconium gasped into the fetal lungs caused aspiration syndrome only in asphyxiated animals.

Ramin and colleagues (1996) hypothesized that the pathophysiology of meconium aspiration syndrome includes, but is not limited to, fetal hypercarbia, which stimulates fetal respiration leading to aspiration of meconium into the alveoli. Lung parenchymal injury is secondary to acidemia-induced alveolar cell damage. In this pathophysiological scenario, meconium in amnionic fluid is a fetal environmental hazard rather than a marker of preexistent compromise. This proposed pathophysiological sequence is not all-inclusive, because it does not account for approximately half of the cases of meconium aspiration syndrome in which the fetus is not acidemic at birth.

Thus, it was concluded that the high incidence of meconium observed in the amnionic fluid during labor often represents fetal passage of gastrointestinal contents in conjunction with normal physiological processes. Although normal, such meconium becomes an environmental hazard

when fetal acidemia supervenes. Importantly, such acidemia occurs acutely, and therefore meconium aspiration is unpredictable and likely unpreventable. Moreover, Greenwood and colleagues (2003) showed that clear amnionic fluid was also a poor predictor. In a prospective study of 8394 women with clear amnionic fluid, they found that clear fluid was an unreliable sign of fetal well-being.

Growing evidence indicates that many newborns with meconium aspiration syndrome have suffered chronic hypoxia before birth (Ghidini, 2001). Blackwell and associates (2001) found that 60 percent of neonates diagnosed with meconium aspiration syndrome had umbilical artery blood pH ≥7.20, implying that the syndrome was unrelated to the neonatal condition at delivery. Similarly, markers of chronic hypoxia, such as elevated fetal erythropoietin levels and increased nucleated red blood cell counts in newborns, suggest that chronic hypoxia is involved in many meconium aspiration syndrome cases (Dollberg, 2001; Jazayeri, 2000).

In the recent past, routine obstetrical management of a newborn with meconium-stained amnionic fluid included intrapartum suctioning of the oropharynx and nasopharynx. In 2005, management guidelines were significantly modified. Now, the American College of Obstetricians and Gynecologists (2017c) recommends that newborns with meconium-stained amnionic fluid, regardless of their vigor, should no longer routinely receive intrapartum suctioning. Suctioning is reserved for those with airway obstruction. They also recommend that an appropriately credentialed team with full resuscitation skills be available (Chap. 32, Care in the Delivery Room).

Management Options

Principal management options for variant fetal heart rate patterns consist of correcting any fetal insult, if possible. Suggestions are listed in Table 24-3. The woman is moved to a lateral position, and supplemental oxygen is provided by mask. Correcting maternal hypotension caused by regional analgesia and discontinuing oxytocin both serve to improve uteroplacental perfusion. Vaginal examination excludes a prolapsed cord or impending delivery. Simpson and James (2005) assessed the benefits of three maneuvers in 52 women with fetal oxygen saturation sensors already in place. They used intravenous hydration—500 to 1000 mL of lactated Ringer solution given over 20 minutes; lateral versus supine positioning; and administration of supplemental oxygen at 10 L/min using a nonrebreathing mask. Each of these maneuvers significantly raised fetal oxygen saturation levels.

TABLE 24-3

Some Resuscitative Measures for Category II or Category III Tracings

Fetal Heart Rate Abnormality[a]	Interventions[b]
Recurrent late decelerations Prolonged decelerations or bradycardia Minimal or absent FHR variability	Lateral decubitus positioning; administer maternal oxygen; intravenous fluid bolus; reduce uterine contraction frequency
Tachysystole with category II or III tracing	Discontinue oxytocin or prostaglandins; Give tocolytics: terbutaline, magnesium sulfate
Recurrent variable decelerations Prolonged decelerations or bradycardia	Reposition mother; amnioinfusion; with cord prolapse, manually elevate the presenting part while preparing for immediate delivery

[a]Simultaneous evaluation of the suspected cause(s) is also an important step in management of abnormal FHR tracings.

[b]The combination of multiple interventions simultaneously may be appropriate and potentially more effective than doing them individually or serially.

FHR = fetal heart rate.

Tocolysis

Terbutaline sulfate given to relax the uterus can be a temporizing maneuver in the management of nonreassuring fetal heart rate patterns during labor. A single 250-µg intravenous or subcutaneous injection is used to inhibit uterine contractions and thereby improve fetal oxygenation. Cook and Spinnato (1994) described their 10-year experiences with terbutaline tocolysis in 368 pregnancies. Such resuscitation improved fetal scalp blood pH values, although all fetuses underwent cesarean delivery. These investigators concluded that although the studies were small and rarely randomized, most reported favorable results with terbutaline tocolysis for nonreassuring patterns. Small intravenous doses of nitroglycerin—60 to 180 µg—also have been reported to be beneficial (Mercier, 1997). Bullens and associates (2015) concluded in their review that tocolysis was beneficial. Still, the American College of Obstetricians and Gynecologists (2017b) cites that evidence is insufficient to recommend tocolysis for nonreassuring fetal heart rate patterns.

Amnioinfusion

Miyazaki and Taylor (1983) infused saline through an intrauterine pressure catheter in laboring women who had either variable or prolonged decelerations attributed to cord entrapment. Such therapy improved the heart rate pattern in half of the women studied. Later, Miyazaki and Nevarez (1985) randomly assigned 96 nulliparas in labor with cord compression patterns and found that those who were treated with amnioinfusion required cesarean delivery for fetal distress less often. Based on many of these early reports, transvaginal amnioinfusion has been extended into three clinical areas (Dad, 2016). These include: (1) treatment of variable or prolonged decelerations; (2) prophylaxis for women with oligohydramnios, as with prolonged ruptured membranes; and (3) attempts to dilute or wash out thick meconium (Chap. 33, Neonatal Encephalopathy and Cerebral Palsy).

Many different amnioinfusion protocols have been reported, but most provide a 500- to 800-mL bolus of warmed normal saline followed by a continuous infusion of approximately 3 mL/min (Owen, 1990; Pressman, 1996). In another study, Rinehart and colleagues (2000) gave either a 500-mL bolus of normal saline at room temperature alone or a similar bolus plus a continuous infusion at 3 mL/min. Their study included 65 women with variable decelerations, and the investigators found neither method to be superior. Wenstrom and associates (1995) surveyed use of amnioinfusion in teaching hospitals in the United States. The procedure was used in 96 percent of the 186 centers surveyed, and it was estimated that 3 to 4 percent of all women delivered at these centers received such infusion. Potential complications of amnioinfusion are summarized in Table 24-4.

TABLE 24-4

Complications Associated with Amnioinfusion from a Survey of 186 Obstetrical Units

Complication	No. of Centers (%)
Uterine hypertonus	27 (14)
Abnormal fetal heart rate tracing	17 (9)
Chorioamnionitis	7 (4)
Cord prolapse	5 (2)
Uterine rupture	4 (2)
Maternal cardiac or respiratory compromise	3 (2)
Placental abruption	2 (1)
Maternal death	2 (1)

Data from Wenstrom, 1995.

For variable decelerations, Hofmeyr and Lawrie (2012) reviewed the effects of amnioinfusion in the management of fetal heart rate patterns associated with umbilical cord compression. They concluded that amnioinfusion appeared to be useful in reducing the occurrence of variable decelerations,

improving neonatal outcome, and lowering cesarean delivery rates. The American College of Obstetricians and Gynecologists (2016) has concluded that amnioinfusion is a reasonable approach in the treatment of repetitive variable decelerations regardless of meconium status.

For oligohydramnios, amnioinfusion has been used prophylactically to avoid intrapartum fetal heart rate patterns from cord occlusion. Nageotte and coworkers (1991) found that this resulted in significantly fewer and less severe variable decelerations in labor. However, the cesarean delivery rate or condition of term newborn was not improved. In a randomized investigation, Macri and colleagues (1992) studied prophylactic amnioinfusion in 170 term and postterm pregnancies complicated by both thick meconium and oligohydramnios. Amnioinfusion significantly reduced meconium aspiration syndrome rates and cesarean delivery rates for fetal distress. In contrast, Ogundipe and associates (1994) randomly assigned 116 term pregnancies with an amnionic fluid index <5 cm to receive prophylactic amnioinfusion or standard obstetrical care. Overall cesarean delivery rates, delivery rates for fetal distress, or umbilical cord acid–base studies did not differ significantly between groups.

For meconium-stained amnionic fluid, Pierce and associates (2000) reviewed 13 prospective trials of intrapartum amnioinfusion for 1924 women with meconium-stained fluid. In the amnioinfusion group, newborns were significantly less likely to have meconium below the vocal cords, and meconium aspiration syndrome rates were lower. The cesarean delivery rate was also reduced in the amnioinfusion group. Similar results were reported by Rathore and coworkers (2002).

In contrast, several investigators were not supportive of amnioinfusion for meconium staining. For example, Usta and associates (1995) reported that amnioinfusion was not feasible in half of women with moderate or thick meconium who were randomized to this treatment. These investigators were unable to demonstrate improved neonatal outcomes with this treatment. Spong and coworkers (1994) also concluded that although prophylactic amnioinfusion did dilute meconium, it did not improve perinatal outcome. Last, Fraser and colleagues (2005) randomized amnioinfusion in 1998 women with thick meconium-stained amnionic fluid in labor and found no benefits. Hofmeyr and associates (2014) reported mixed results from their review. Because of these findings, the American College of Obstetricians and Gynecologists (2016) does not recommend amnioinfusion to dilute meconium-stained amnionic fluid.

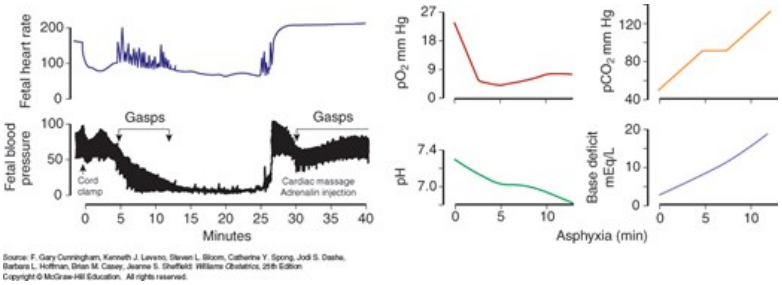
Fetal Heart Rate Patterns and Brain Injury

Studies that have attempted to correlate fetal heart rate patterns with brain injury primarily have examined infants identified in medicolegal actions. Phelan and Ahn (1994) reported that among 48 fetuses later found to be neurologically impaired, a persistent nonreactive fetal heart rate tracing was already present at the time of admission in 70 percent. They concluded that fetal neurological injury occurred predominately before arrival to the hospital. When they looked retrospectively at heart rate patterns in 209 brain-injured newborns, they concluded that there was not a single unique pattern associated with fetal neurological injury (Ahn, 1996). Graham and associates (2006) reviewed the world literature published between 1966 and 2006 on the effect of fetal heart rate monitoring to prevent perinatal brain injury and found no benefit.

Fetal heart rate patterns necessary for perinatal brain damage have been studied in experimental animals. Myers (1972) described the effects of complete and partial asphyxia in rhesus monkeys. Complete asphyxia was produced by total occlusion of umbilical blood flow that led to prolonged deceleration (Fig. 24-26). Fetal arterial pH did not drop to 7.0 until approximately 8 minutes after complete cessation of oxygenation and umbilical flow. At least 10 minutes of such prolonged deceleration was required before there was evidence of brain damage in surviving fetuses.

FIGURE 24-26

Prolonged deceleration in a rhesus monkey shown with blood pressure and biochemical changes during total occlusion of umbilical cord blood flow. (Data from Myers, 1972.)



Myers (1972) also produced partial asphyxia in rhesus monkeys by impeding maternal aortic blood flow. This resulted in late decelerations due to

uterine and placental hypoperfusion. He observed that several hours of these late decelerations did not damage the fetal brain unless the pH fell below 7.0. Indeed, Adamsons and Myers (1977) reported subsequently that late decelerations were a marker of partial asphyxia long before brain damage occurred.

The most common fetal heart rate pattern during labor—due to umbilical cord occlusion—requires considerable time to significantly affect the fetus in experimental animals. Clapp and colleagues (1988) partially occluded the umbilical cord for 1 minute every 3 minutes in fetal sheep. Rocha and associates (2004) totally occluded the umbilical cord for 90 seconds every 30 minutes for 3 to 5 hours a day for 4 days without producing necrotic brain cell injury. Results from such studies suggest that the effects of umbilical cord entrapment depend on the degree of occlusion—partial versus total, the duration of individual occlusions, and the frequency of such occlusions.

The contribution of intrapartum events to subsequent neurological handicaps has been greatly overestimated, as discussed in further detail in Chapter 33 (Neonatal Encephalopathy). It is clear that for brain damage to occur, the fetus must be exposed to much more than a brief period of hypoxia. Moreover, the hypoxia must cause profound, just barely sublethal metabolic acidemia. Because of this, the American College of Obstetricians and Gynecologists (2014) has recommended umbilical cord blood gases be obtained whenever cesarean delivery is performed for fetal compromise, a low 5-minute Apgar score, severe fetal-growth restriction, an abnormal fetal heart rate tracing, maternal thyroid disease, or multifetal gestation (Chap. 32, Umbilical Cord Blood Acid–Base Studies).

Until recently, the prognosis for moderately affected newborns with hypoxic-ischemic encephalopathy (HIE) was poor. This has stimulated research aimed at mitigating these consequences. Animal experiments beginning in the late 1990s suggested that reducing brain temperature after an inciting event could reduce the incidence of cerebral damage (Gunn, 1997, 2000; Nedelcu, 2000; Tooley, 2003; Wagner, 2002). These findings led to several studies worldwide that showed brain cooling administered to newborns suffering neonatal HIE could ameliorate the development of subsequent cerebral palsy. These studies are further described in Chapter 33 (Cerebral Palsy).

Benefits of Electronic Fetal Heart Rate Monitoring

Several false assumptions underlie the expectation of improved perinatal outcome with electronic monitoring. One is that fetal distress is a slowly developing phenomenon and that electronic monitoring permits early detection of the compromised fetus. Another presumption is that all fetal injury develops in the hospital. Within the past 20 years, attention has focused on the reality that most neurologically damaged fetuses suffered insults before arrival at labor units. The very term *fetal monitor* implies that this inanimate technology in some fashion "monitors." The assumption is made that if a dead or damaged fetus is delivered, the tracing strip must provide some clue, because this device was monitoring fetal condition. All of these assumptions led to great expectations and fostered the belief that all neonatal deaths or injuries were preventable.

By the end of the 1970s, questions regarding the efficacy, safety, and costs of electronic monitoring were being voiced from the Office of Technology Assessment, the United States Congress, and the Centers for Disease Control and Prevention. Banta and Thacker (2002) reviewed 25 years of the controversy on the benefits, or lack thereof, of electronic fetal monitoring. More recently, Alfirevic and colleagues (2017) reviewed 13 randomized trials involving more than 37,000 women. They concluded that electronic fetal monitoring was associated with fewer neonatal seizures but a higher rate of cesarean and operative vaginal deliveries. Importantly, rates of perinatal mortality or cerebral palsy did not decline. Grimes and Peipert (2010) wrote a *Current Commentary* on electronic fetal monitoring in *Obstetrics & Gynecology.* They summarized that such monitoring, although it has been used in 85 percent of the almost 4 million annual births in the United States, has failed as a public health screening program. They noted that the positive-predictive value of electronic fetal monitoring for fetal death in labor or cerebral palsy is near zero—meaning that "almost every positive test result is wrong."

There have been at least two attempts to study the epidemiological effects of electronic fetal monitoring in the United States. Chen and coworkers (2011) used 2004 data on more than 1.7 million singleton live births, 89 percent of which underwent electronic fetal monitoring. They reported that monitoring raised operative delivery rates but lowered early neonatal mortality rates. This benefit was gestational-age dependent, however, and the highest effect was seen in preterm fetuses. Later, Ananth and colleagues (2013) reported a similar but larger epidemiological study in the United States. They studied nearly 58 million nonanomalous singleton liveborn neonates delivered between 1990 and 2004. The temporal increase in fetal monitoring use was associated with a decline in neonatal mortality rates, especially in preterm gestations. In an accompanying editorial, Resnik (2013) cautioned that an epidemiological association between fetal monitoring and reduced neonatal death does not establish causation. He suggested that the limitations of the study by Ananth should make the reader skeptical of the findings.

At Parkland Hospital in July 1982, an investigation began to ascertain whether all women in labor should undergo electronic monitoring (Leveno,

1986). In alternating months, universal electronic monitoring was rotated with selective heart rate monitoring, which was the prevailing practice. During the 3-year investigation, more than 17,000 labors were managed using universal electronic monitoring, and these outcomes were compared with a similar-sized cohort of women selectively monitored electronically. No significant differences were found in any perinatal outcome. With universal monitoring, a small but significant increase in the cesarean delivery rate for fetal distress was noted. Thus, greater application of electronic monitoring at Parkland Hospital did not improve perinatal results but did slightly raise the frequency of cesarean delivery for fetal distress. More recently, a Cochrane Database review found that intermittent auscultation had a higher cesarean delivery rate compared with continuous monitoring (Martis, 2017).

Current Recommendations

Methods most commonly used for intrapartum fetal heart rate monitoring include auscultation with a fetal stethoscope or a Doppler ultrasound device, or continuous electronic monitoring of the fetal heart rate and uterine contractions. No scientific evidence has identified the most effective method, including the frequency or duration of fetal surveillance that ensures optimum results. Summarized in Table 24-5 are the recommendations of the American Academy of Pediatrics and the American College of Obstetricians and Gynecologists (2017). Intermittent auscultation or continuous electronic monitoring is considered an acceptable method of intrapartum surveillance in both low- and high-risk pregnancies. The recommended interval between checking the heart rate, however, is longer in the uncomplicated pregnancy. When auscultation is used, it is recommended that it be performed after a contraction and for 60 seconds. It also is recommended that a 1-to-1 nurse–patient ratio be used if auscultation is employed. The position taken by the American College of Obstetricians and Gynecologists (2017b) acknowledges that available data do not show a clear benefit to the use of electronic monitoring over intermittent auscultation. At Parkland Hospital, all high-risk labors are continuously monitored electronically. In low-risk pregnancies, both intermittent auscultation and continuous electronic monitoring are used depending on clinical circumstances, including the woman's desire to ambulate.

TABLE 24-5

Guidelines for Methods of Intrapartum Fetal Heart Rate Monitoring

Surveillance	Low-Risk Pregnancies	High-Risk Pregnancies
Acceptable methods		
Intermittent auscultation	Yes	Yes[a]
Continuous electronic monitoring (internal or external)	Yes	Yes[b]
Evaluation intervals		
First-stage labor (active)	30 min	15 min[a,b]
Second-stage labor	15 min	5 min[a,c]

[a]Preferably before, during, and after a uterine contraction.

[b]Includes tracing evaluation and charting at least every 15 min.

[c]Tracing should be evaluated at least every 5 min.

Data from the American Academy of Pediatrics and the American College of Obstetricians and Gynecologists, 2017.

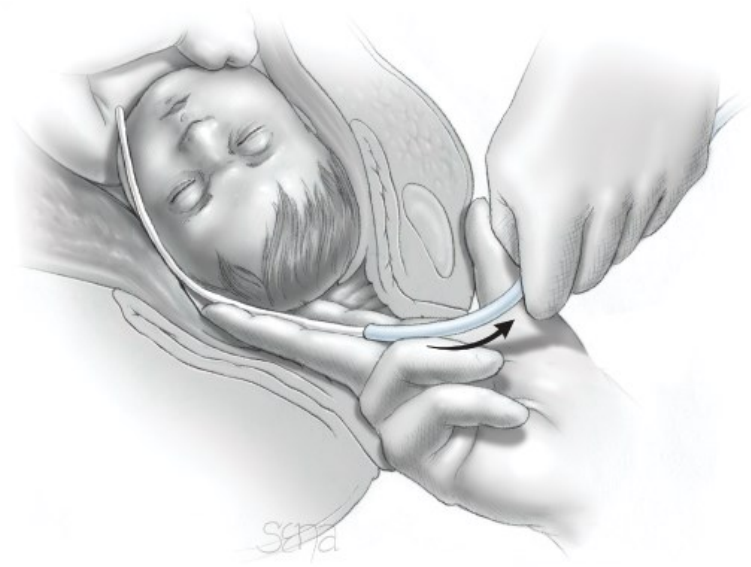
INTRAPARTUM SURVEILLANCE OF UTERINE ACTIVITY

Analysis of electronically measured uterine activity permits some generalities concerning the relationship of certain contraction patterns and labor outcome. However, uterine muscle efficiency to bring about delivery varies greatly. Thus, caution should be exercised before diagnosing true labor or its absence solely from a monitor tracing.

With *internal monitoring* of contractions, amnionic fluid pressure is measured between and during contractions. In the past, a fluid-filled plastic catheter with its distal tip located above the presenting part was used (Fig. 24-27). The catheter was connected to a strain-gauge pressure sensor adjusted to the same level as the catheter tip in the uterus. The amplified electrical signal produced in the strain gauge by variation in pressure within the fluid system was recorded on a calibrated moving paper strip simultaneously with the fetal heart rate recording. Today, intrauterine pressure catheters are used that have the pressure sensor in the catheter tip, which obviates the need for the fluid column.

FIGURE 24-27

Placement of an intrauterine pressure catheter to monitor contractions and their pressures. The catheter, contained within the introducer, is inserted into the birth canal and placed along one side of the fetal head. The catheter is then gently advanced into the uterus, and the introducer is withdrawn.



With *external monitoring*, uterine contractions can be measured by a displacement transducer in which the transducer button, or "plunger," is held against the maternal abdominal wall. As the uterus contracts, the button moves in proportion to the strength of the contraction. This movement is converted into a measurable electrical signal that indicates the *relative* intensity of the contraction. It has generally been accepted that internal monitoring provided a more accurate measure of intensity. That said, Bakker and associates (2010) performed a randomized trial comparing internal versus external monitoring of uterine contractions in 1456 women. The two methods were equivalent in terms of operative delivery rates and neonatal outcomes.

Patterns of Uterine Activity

Caldeyro-Barcia and Poseiro (1960), from Montevideo, Uruguay, were pioneers in elucidating the patterns of spontaneous uterine activity throughout pregnancy. Contractile waves of uterine activity were usually measured using intraamnionic pressure catheters. But early in their studies, as many as four simultaneous intramyometrial microballoons were also used to record uterine pressure. Contraction intensity was defined as the rise in this pressure above a resting pressure baseline. These investigators also introduced the concept of *Montevideo units* to define uterine activity (Chap. 23, Active-Phase Protraction). With this definition, uterine performance is the product of contraction intensity in mm Hg multiplied by the number of contractions in a 10-minute span. For example, three contractions in 10 minutes, each of 50 mm Hg intensity, would equal 150 Montevideo units.

During the first 30 weeks of pregnancy, uterine activity is comparatively quiescent. Contractions are seldom greater than 20 mm Hg, and these have been equated with those first described by John Braxton Hicks. Uterine activity increases gradually after 30 weeks, and it is noteworthy that these

Braxton Hicks contractions also increase in intensity and frequency. Uterine activity is further enhanced during the last weeks of pregnancy. During this phase, the cervix ripens (Chap. 21, Cervical Ripening).

According to Caldeyro-Barcia and Poseiro (1960), clinical labor usually commences when uterine activity reaches values between 80 and 120 Montevideo units. This translates into approximately three contractions of 40 mm Hg every 10 minutes. Importantly, no clear-cut division marks labor onset, which is a gradual and progressive transition.

In first-stage labor, uterine contractions progressively grow in intensity from approximately 25 mm Hg at labor commencement to 50 mm Hg at its end. At the same time, the frequency advances from three to five contractions per 10 minutes, and uterine baseline tone rises from 8 to 12 mm Hg. Uterine activity is further enhanced during second-stage labor, aided by maternal pushing. Indeed, contraction intensity of 80 to 100 mm Hg is typical, and the uterus contracts as frequently as five to six times each 10 minutes. Hauth and coworkers (1986) quantified uterine contraction pressures in 109 women at term who received oxytocin for labor induction or augmentation. Most of these women achieved 200 to 225 Montevideo units, and 40 percent had up to 300 units to effect delivery. The authors suggested that these levels of uterine activity should be sought before consideration of cesarean delivery for presumed dystocia (Chap. 23, Active-Phase Protraction).

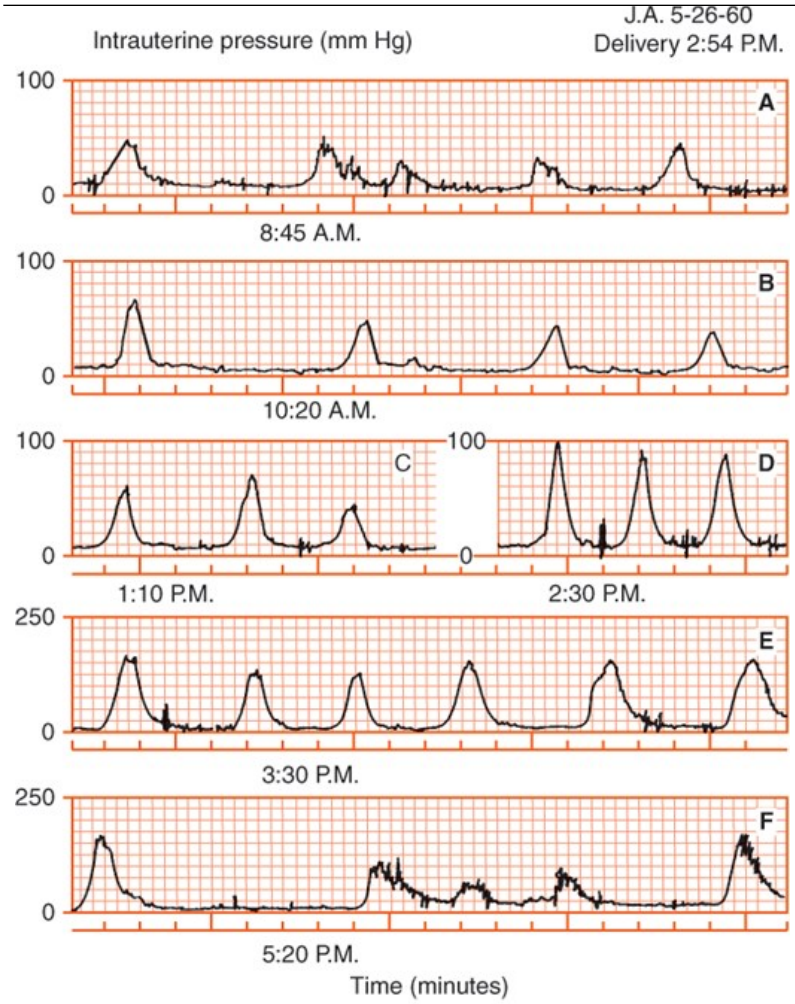
Interestingly, the duration of uterine contractions—60 to 80 seconds—does not lengthen appreciably from early active labor through the second stage (Bakker, 2007; Pontonnier, 1975). Presumably, this duration constancy serves fetal respiratory gas exchange. During a uterine contraction, as the intrauterine pressure exceeds that of the intervillous space, respiratory gas exchange is halted. This leads to functional fetal "breath holding," which has a 60- to 80-second limit that remains relatively constant.

Caldeyro-Barcia and Poseiro (1960) also observed empirically that uterine contractions are clinically palpable only after their intensity exceeds 10 mm Hg. Moreover, until the intensity of contractions reaches 40 mm Hg, the uterine wall can readily be depressed by the finger. At greater intensities, the uterine wall then becomes so hard that it resists easy depression. Uterine contractions usually are not associated with pain until their strength exceeds 15 mm Hg. Presumably, this is the minimum pressure required to distend the lower uterine segment and cervix. It follows that Braxton Hicks contractions exceeding 15 mm Hg may be perceived as uncomfortable because distention of the uterus, cervix, and birth canal is generally thought to produce discomfort.

Hendricks (1968) observed that "the clinician makes great demands upon the uterus." The uterus is expected to remain well relaxed during pregnancy, to contract effectively but intermittently during labor, and then to remain in a state of almost constant contraction for several hours postpartum. Figure 24-28 demonstrates an example of normal uterine activity during labor. Uterine activity progressively and gradually rises from early through late labor. Interestingly, uterine contractions after birth are identical to those resulting in delivery of the newborn. Logically, the uterus that performs poorly before delivery is also prone to atony and puerperal hemorrhage.

FIGURE 24-28

Intrauterine pressure recorded through a single catheter. **A.** Prelabor. **B.** Early labor. **C.** Active labor. **D.** Late labor. **E.** Spontaneous activity ½ hour postpartum. **F.** Spontaneous activity 2½ hours postpartum. (Redrawn from Hendricks CH: Uterine contractility changes in the early puerperium, Clin Obstet Gynecol. 1968 Mar;11(1):125–144.)



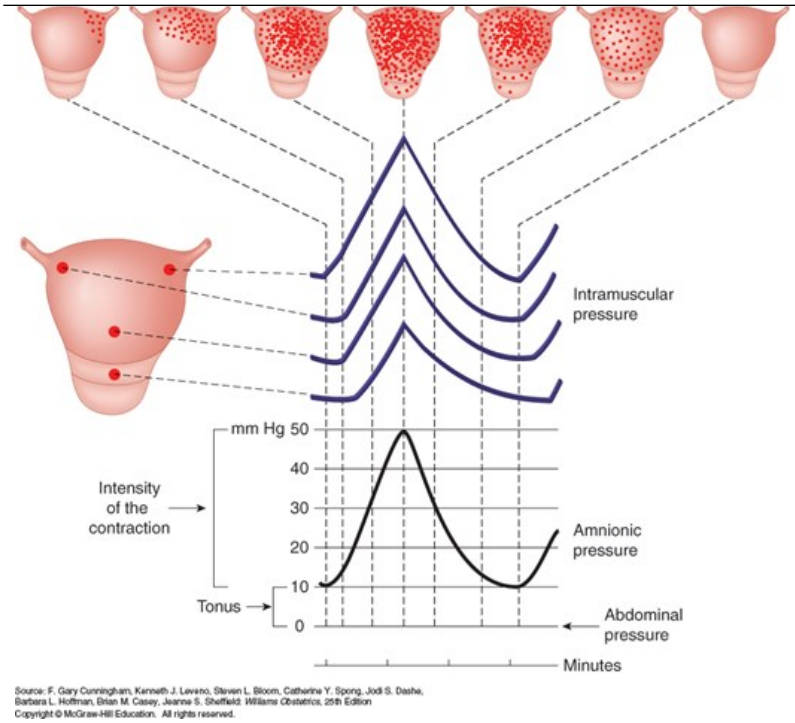
Source: F. Gary Cunningham, Kenneth J. Leveno, Steven L. Bloom, Catherine Y. Spong, Jodi S. Dashe, Barbara L. Hoffman, Brian M. Casey, Jeanne S. Sheffield: *Williams Obstetrics*, 25th Edition

Origin and Propagation of Contractions

The normal contractile wave of labor originates near the uterine end of one of the fallopian tubes. Thus, these areas act as "pacemakers" (Fig. 24-29). The right pacemaker usually predominates over the left and starts most contractile waves. Contractions spread from the pacemaker area throughout the uterus at 2 cm/sec, and the whole organ is depolarized within 15 seconds. This depolarization wave propagates downward toward the cervix. Intensity is greatest in the fundus, and it diminishes in the lower uterus. This phenomenon is thought to reflect the reduced myometrial thickness from the fundus to the cervix. Presumably, this descending pressure gradient serves to direct fetal descent toward the cervix and to efface the cervix. Importantly, all parts of the uterus are synchronized and reach their peak pressure almost simultaneously, giving rise to the curvilinear waveform shown in Figure 24-29. Young and Zhang (2004) have shown that the initiation of each contraction is triggered by a tissue-level bioelectrical event.

FIGURE 24-29

Schematic representation of the normal contractile wave of labor. Large uterus on the left shows the four points at which intramyometrial pressure was recorded with microballoons. Four corresponding pressure tracings are shown in relation to each other by shading on the small uteri at top. (Adapted with permission from Caldeyro-Barcia R, Poseiro JJ: Physiology of the uterine contraction. Clin Obstet Gynecol 1960 3:386.)



Source: F. Gary Cunningham, Kenneth J. Leveno, Steven L. Bloom, Catherine Y. Spong, Jodi S. Dashe, Barbara L. Hoffman, Brian M. Casey, Jeanne S. Sheffield: *Williams Obstetrics*, 25th Edition

The pacemaker theory also serves to explain the varying intensity of adjacent coupled contractions shown in panels A and B of Figure 24-28. Such coupling was termed *incoordination* by Caldeyro-Barcia and Poseiro (1960). A contractile wave begins in one cornual-region pacemaker but does not synchronously depolarize the entire uterus. As a result, another contraction begins in the contralateral pacemaker and produces the second contractile wave of the couplet. These small contractions alternating with larger ones appear to be typical of early labor. Indeed, labor may progress with such uterine activity, albeit at a slower pace. These authors also observed that labor would progress slowly if regular contractions were hypotonic—that is, contractions with intensity <25 mm Hg or frequency <2 per 10 minutes.

Uterine Contraction Terminology

Terms for the description and quantification of uterine contractions have been recommended by the American College of Obstetricians and Gynecologists (2017b). *Normal uterine activity* is defined as five or fewer contractions in 10 minutes, averaged during a 30-minute span. *Tachysystole* is more than five contractions in 10 minutes, averaged over 30 minutes. Tachysystole can be applied to spontaneous or induced labor. The term *hyperstimulation* was abandoned.

Stewart and associates (2012) prospectively studied uterine tachysystole in 584 women undergoing labor induction with misoprostol at Parkland Hospital. A higher rate of adverse neonatal outcomes was not associated with an increasing number of contractions per 10 minutes or per 30 minutes. Counts of six or more contractions in 10 minutes, however, were significantly associated with fetal heart rate decelerations.

Electronic Fetal Monitoring Complications

Electrodes for fetal heart rate evaluation and catheters for uterine contraction measurement are both associated with infrequent but potentially serious complications. Rarely, an intrauterine pressure catheter during placement may lacerate a fetal vessel in the placenta. Also with insertion, placental and possibly uterine perforation can cause hemorrhage, abruption, serious morbidity, and spurious recordings that have resulted in inappropriate management. Severe cord compression has been described from entanglement with the pressure catheter. Injury to the fetal scalp or breech by a heart rate electrode is rarely severe. However, application at some other site—such as the eye in face presentations—can be serious.

Both the fetus and the mother may be at greater risk of infection from internal monitoring (Faro, 1990). Scalp wounds from the electrode may become infected, and subsequent cranial osteomyelitis has been reported (Brook, 2005; Eggink, 2004; McGregor, 1989). The American Academy of Pediatrics and the American College of Obstetricians and Gynecologists (2017) have recommended that certain maternal infections, including human immunodeficiency virus (HIV), herpes simplex virus, and hepatitis B and C virus, are relative contraindications to internal fetal monitoring.

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