The Relationship Between Fetal Heart Rate Patterns and Fetal or Newborn Acidemia

In order to determine the relationship between specific fetal heart rate (FHR) patterns and fetal or newborn acidemia, one must first determine what biologic indices accurately identify acidemia in the fetus. Multiple studies have evaluated the relationships between umbilical cord gas results and subsequent neonatal morbidity.[1-4] As noted in Chapter 5, a pH of less than 7.0 with a base excess of less than −12 mmol/L has been identified as the threshold set of values that significantly increase the risk for newborn morbidity or mortality following metabolic acidosis.[1] This chapter reviews the evidence that links specific FHR patterns to fetal or newborn acidemia and adverse outcomes.

The relationship between FHR patterns and fetal or newborn acidemia would ideally be determined in a prospectively gathered, unselected series of cases that includes the full range of FHR patterns recorded up until the time of birth, measurements of umbilical cord arterial blood gases and acid–base state, and other measures of newborn outcome. This would enable the determination of validity with regard to the relationship. However, these studies are not available because obstetric intervention occurs before some FHR patterns develop or persist. Therefore, current research is limited to analyses of FHR patterns that are mostly benign, as it is not ethically possible to observe all FHR patterns, in particular, those that are believed to be associated with an increased risk of fetal acidemia. For this reason, modern accumulated series of FHR tracings rarely contain FHR tracings that are believed to be very abnormal for any length of time.[5-7] Thus, the compilation of older smaller observational studies that were conducted before the advent of wide-spread intervention is the best evidence available to determine the association between some FHR patterns and newborn acid–base outcome.

I. DEVELOPMENT OF ACIDEMIA WITH RECURRENT DECELERATIONS OVER TIME

There are four common assumptions about FHR patterns and the progression of these patterns that are in clinical usage for term fetuses during labor:

1. Moderate (normal) FHR variability is associated with the absence of acidemia and the birth of a nondepressed (vigorous) neonate.

2. Minimal or undetectable FHR variability in the presence of recurrent late decelerations or variable decelerations is associated with the presence of acidemia and/or a depressed neonate.

3. The depth of decelerations is positively related to the degree of fetal or newborn acidemia and/or neonatal depression.

4. In the presence of recurrent and progressive decelerations in a fetus without an a priori increased risk for developing acidemia, and the absence of catastrophic events, the development of clinically significant acidemia evolves over a period of time.

This chapter presents the findings of a research group that reviewed the literature published between 1967 and 2003 to determine if FHR patterns are related to fetal acidemia and/or Apgar scores, and to support or refute the above four assumptions.[8] Subsequently published research is also presented.

The four themes related to FHR monitoring are all supported by the evidence, though not always with high predictive value. Most of the studies identified that evaluated FHR patterns and newborn indices of acidemia were judged to be grade III evidence (observational and uncontrolled studies) using the U.S. Preventive Services task force criteria, although two were case-control studies (grade II).[9] However, within each hypothesis, the relevant studies came to similar conclusions, which tends to strengthen the quality of the evidence. A further strength is that much of the data were gathered before widespread intervention began.

These four themes have a number of limitations. First, the older studies that observed "abnormal" FHR patterns over a period of time generally used a pH of less than 7.15 or less than 7.2 for the definition of acidemia. In a number of cases, umbilical blood gasses were not available so an Apgar score of less than 7 at 5 minutes was used as a surrogate for metabolic acidemia. Nonetheless, subsequent work has largely supported these themes and they have been used to inform the development of interpretation and management classification systems.[10-14]

A. The Relationship Between Moderate Fetal Heart Rate Variability and the Nonacidemic Vigorous Neonate

Within the 50 studies that met inclusion criteria for the original analysis, five publications correlated moderate FHR variability, with or without decelerations or bradycardia to acid–base status determined via fetal scalp samples, newborn cord blood gases, or Apgar scores (where blood gases were not available).[15-19] The FHR tracings were examined in the last 30 minutes before delivery, or when the fetal blood samples were obtained.

A total of 1551 fetuses or newborns were included in these studies. Of these, 1518 were either nonacidemic, with pH greater than 7.15 or a 5-minute Apgar score higher than 7, depending on the endpoint used by the authors. Thus, 98% of the fetuses with moderate FHR variability, whether or not there were decelerations or a bradycardia present, were either nonacidemic or vigorous at birth.

Subsequently published studies have validated these findings. Samueloff et al. evaluated a cohort of 1968 consecutive intrapartum FHR tracings and found that the presence of moderate variability had greater than 99% negative predictive value for adverse outcome, defined as 5-minute Apgar score less than 7, and greater than 85% negative predictive value for umbilical arterial blood pH less than 7.2.[20] In a further publication from the same group, of 2200 consecutive deliveries, fetuses that had either a normal FHR tracing, mild variable decelerations, decreased FHR variability without concomitant decelerations, mild bradycardia, or accelerations present, in the last 30-minute segment before delivery, had an Apgar score of 7 or higher in 99.7% of cases, and umbilical arterial cord pH 7.15 or higher in 96.9% of cases.[21] Williams and Galerneau, in a study of 488 term FHR tracings that included 2 hours before delivery and at least part of the last 30 minutes, found that the fetuses with moderate variability, even if late or variable decelerations were present, had a cord pH of 7.0 or higher in more than 97% of cases.[22]

These correlations are impressive and have been consistently demonstrated in additional studies from different populations.[6,23,24] The presence of moderate FHR variability, even in the presence of decelerations, is highly associated with the absence of clinically significant fetal or newborn acidemia. The 98% association should be considered a minimal value, and the actual predictive value may be somewhat higher. This is because the FHR tracing was not always available in the last minutes before the birth and subsequent acute events after the monitor was removed might have resulted in newborn acidemia. Also in some cases, the acidemia reflected by a low umbilical artery pH may be respiratory rather than metabolic, and newborns with respiratory acidemia only generally do not have significant morbidity.[25,26] A further reason for lack of a higher correlation might be that there are degrees of metabolic acidemia in the fetus that reflect maternal acidemia, and not

intrinsic anaerobic metabolism within the fetus. It is not possible to determine the influence of this potential limitation without concomitant maternal acid–base values.

B. The Relationship Between Minimal or Undetectable Fetal Heart Rate Variability in the Presence of Recurrent Decelerations, and Fetal Acidemia and/or a Depressed Neonate

Minimal or absent variability without concomitant decelerations does not predict the presence or absence of newborn acidemia. Similarly, although multiple studies have shown a relationship between recurrent late or variable decelerations and lower umbilical cord pH values, decelerations alone are not predictive of outcome.[7,15,16,27]

Eight studies that included a total a 588 FHR tracings with minimal or absent variability and recurrent late or variable decelerations found the combination of decelerations and reduced variability to be related to fetal acidemia or adverse newborn outcomes.[15-18,23,28-30] It was not always possible to clearly distinguish between absent or minimal variability in the published data because these two categories of FHR variability were often grouped together in individual studies. In this combined series of studies, 137 patients had fetuses or newborns with a pH less than 7.2, or a base excess less than –12 mEq/L in fetal blood, or a 5-minute Apgar score of less than 7. Thus, approximately 23% of the newborns in this series with reduced FHR variability and recurrent decelerations had fetal acidemia or newborn depression.

In the subsequent series of tracings of 488 term fetuses with linked umbilical cord blood gas values analyzed by Williams and Galerneau, 31% had an umbilical artery pH 7.0 or less when the FHR variability was minimal or absent in the last hour before delivery.[22] In those with decreased FHR variability and late decelerations, 24% had pH of 7.0 or less; in those with decreased FHR variability and variable decelerations, 13% had a pH of 7.0 or less. Base excess in the same study group was less than –16 mEq/L in 32% with decreased FHR variability and late decelerations, 13% of those with decreased FHR variability and variable decelerations, and 38.5% in those with minimal or absent variability for an hour before delivery.[22] Sameshima and Ikenoue found similar results in an analysis of nonrecurrent and recurrent late decelerations in the last hour before birth ($n = 301$ out of a large population of women who were low risk for fetal acidemia on admission [$n = 5546$]). The positive predictive value for a pH less than 7.1 was 12% for late decelerations with moderate variability and 53% for late decelerations with variability that was less than 5 bpm variation.[6]

Further support for an association between minimal or absent FHR variability with decelerations and newborn complications has become evident in case-control and cohort studies of term newborns with umbilical artery pH less than 7.0 and/or base deficit less than –12 mEq/L or less than –16 mEq/L. The majority of newborns with metabolic acidemia at birth had an FHR tracing prior to birth that exhibited minimal or absent variability with recurrent decelerations.[3,5,23,31,32]

In summary, although it is generally accepted that there is an association between acidemia and minimal or undetectable FHR variability in the presence of decelerations, the overall correlation was only 23% in this analysis.[15-18,23,28-30] With absent, as opposed to minimal FHR variability and severe decelerations, the association is probably much higher.[16] The lower correlation with simply reduced FHR variability may be secondary to a number of factors. First the cases with minimal variability and recurrent decelerations may reflect cases in which the fetus is transitioning from physiologic compensation to decompensation due to asphyxia. Second, the studies that evaluated these tracings did so for varying periods of time. The association may also be diluted due to different timeframes before intervention occurred. Those studies that evaluated these FHR

patterns for varying periods of time found a stronger correlation with longer time frames.[5,33] The true relationship would ideally need confirmation from a large prospective unselected series. However, the ability of investigators to accumulate such cases will be limited, as most clinicians will intervene rather than tolerate such patterns.

Despite strong evidence that reduced FHR variability and recurrent decelerations in the last hour before birth indicate a significant risk for newborn acidemia, many nonacidemic newborns also have combinations of these FHR patterns prior to birth. Thus, the predictive power of these FHR patterns is not high.[6,31,34] Case-control studies that compared the incidence of reduced variability, late or variable decelerations, and prolonged decelerations in infants with and without metabolic acidosis at birth have found higher incidences of these FHR patterns in the infants with metabolic acidemia.[3,5,6,22,23,31,32,34] The incidence of these FHR patterns increases as the severity of metabolic acidemia increases. However, the range of sensitivity is 7.7% to 29%, specificity is 93% to 98.9%, the positive predictive power is 2.6% to 53%, and negative predictive is 88.6% to 99.5%.[6,31,34] Although the positive predictive value of these particular FHR patterns for fetal acidemia is low, the association is high enough, particularly if there is potential for brain morbidity that intervention is recommended.[12-14] Thus, intervention for these patterns becomes a decision involved with risk tolerance for individuals, rather than strict scientific certainty.

C. The Relationship of the Depth of Decelerations to the Severity of Acidemia and/or Neonatal Depression

It is commonly accepted clinically that the deeper the decelerations the greater the likelihood of acidemia and/or depression. Three older studies with a total of 703 patients contain information addressing this subject.[16,27,30] The FHR deceleration patterns had variable degrees of FHR variability, not always stated. Severe late decelerations (defined as the nadir $\geq$ 45 bpm) were associated with a pH less than 7.15, or an Apgar score less than 7 at 5 minutes in 35 of 55 (64%) cases, whereas only 3/199 (3%) of cases with mild late decelerations (defined as the nadir < 15 bpm below the baseline) were associated with only a pH of less than 7.15. In the cases where there was reduced FHR variability and severe late decelerations, all of 28 cases (100%) had a pH of less than 7.15. With reduced variability and mild late decelerations none of 33 were acidemic (**Table 8-1**).[8] Severe variable decelerations were associated with fetal acidemia or low 5-minute Apgar scores in 22/201 (11%) patients in this series whereas mild variable decelerations were associated with fetal acidemia or low 5-minute Apgar score in only 10/287 (3%) cases (Table 8-1). Subsequent studies have also demonstrated the positive relationship between severity of deceleration and lower pH values.[6]

Table 8-1 Association Between Depth of Decelerations and Acidemia (pH < 7.15) or Low 5-Minute Apgar Score (< 7)

	FHR Variability Not Stated		Reduced FHR Variability	
	Severe	**Mild**	**Severe**	**Mild**
Late decelerations	64% (35/55)	3% (3/119)	100% (28/28)	0% (0/33)
Variable decelerations	11% (22/201)	3% (10/287)		

Data from Paul RH, Suidan AK, Yeh S, Schifrin BS, Hon EH. Clinical fetal monitoring. VII. The evaluation and significance of intrapartum baseline. FHR variability. *Am J Obstet Gynecol.* 1975;123:206-210[16]; Krebs HB, Petres RE, Dunn LJ, Jordaan HV, Sergeti A. Intrapartum fetal heart rate monitoring. I. Classification and prognosis of fetal heart rate patterns. *Am J Obstet Gynecol.* 1979;133:762-772[17]; Kubli FW, Hon EH, Khazin AF, Takemura H. Observations on heart rate and pH in the human fetus during labor. *Am J Obstet Gynecol.* 1969;104:1190-1206.[27]

Reproduced with permission from Parer JT, King TL, Flanders S, Fox M, Kilpatrick SJ. Fetal acidemia and electronic fetal heart rate patterns: is there evidence of an association? *J Matern Fetal Neonatal Med.* 2006:19:289-294.[8] www.tandfonline.com.

More recently, studies have used current technologies to more precisely measure the depth and duration of recurrent decelerations and have calculated the duration of time that an FHR is below thresholds of bpm before birth. These studies have concluded that the total deceleration area has the best predictive power for identifying newborn acidemia.[7,35-37] This technique combines duration and severity into one measure. A small study by Tranquilli et al. correlated the total deceleration area to umbilical artery pH values and found an umbilical artery pH of less than 7.1 following 25 minutes of FHR of 80 bpm, 13 minutes for an FHR of 70 bpm, 8 minutes for an FHR of 60 bpm, 6 minutes for an FHR of 50 bpm, and only 5 minutes for an FHR of 40 bpm. The positive predictive value was 78.5% and the negative predictive value was 68.4%.[37]

D. Time Course of Development of Significant Acidemia in the Presence of Decelerations When There Is an Initially Normal Fetal Heart Rate Pattern and Absence of Catastrophic Events

Three studies were found that analyzed the time from onset of decelerations to fetal or newborn acidemia. In a study of term fetuses with an initially normal FHR tracing and normal scalp blood pH, but who subsequently developed an "abnormal" tracing based on a scoring system, it was found that the fetuses remained nonacidemic (scalp blood pH > 7.25), for at least 90 minutes of the "abnormal" pattern. After this initial 90 minutes, the average time for 50% of the fetuses to develop acidemia was 115 minutes with recurrent late decelerations and 145 minutes with recurrent variable decelerations.[33]

Low et al. showed that there was an approximately 1-hour window from the start of FHR patterns containing minimal baseline variability and late or prolonged decelerations, which preceded fetal asphyxial decompensation and newborn morbidity.[38] In a case-control study of fetuses with umbilical artery pH less than 7.05, Ingemarsson et al. concluded that the 4-year follow-up developmental screening test was worse when a "pathologic" tracing lasted for more than 60 minutes.[39]

A more recent case-control study of infants with hypoxic ischemic encephalopathy (*n* = 35) assessed the timing of the onset of abnormal FHR patterns to outcome.[40] These authors found that when the FHR pattern was normal on admission and declined gradually before delivery, the median duration between the development of the abnormal FHR pattern and delivery was 145 minutes in all the infants who exhibited hypoxic ischemic changes. This study documents that in the absence of a sentinel event, acidemia develops over time during the course of labor. However, because this study was a case-control study in which all the infants were delivered with acidemia, it does not shed light on when the developing acidemia crossed the threshold that causes brain injury.

Overall, these studies support the concept that in the absence of catastrophic events, in a fetus with an initial normal FHR pattern, the development of significant acidemia in the presence of recurrent variant FHR patterns evolves over a significant period of time, of the order of at least 1 hour.

II. FETAL HEART RATE PATTERN EVOLUTION

During labor, there are three ways the FHR pattern can develop abnormal characteristics: (1) most commonly, the FHR tracing is normal at the outset of labor and decelerations gradually develop over time; (2) when a sentinel event such as uterine prolapse or placental abruption occurs, the FHR may suddenly develop a terminal bradycardia; and (3) although not common, the fetus may experience an adverse event prior to labor and the FHR tracing on admission reveals abnormal characteristics. By far, the most common pattern is the one that develops over time.
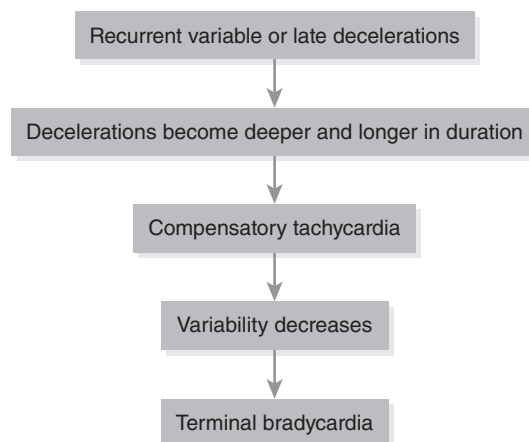
A. Fetal Heart Rate Pattern Evolution

To date, studies that have prospectively or retrospectively evaluated the relationship between FHR patterns and metabolic academia confirm the value of the four assumptions commonly used for interpreting FHR patterns in clinical practice. When this work is analyzed as a whole, the pattern of developing acidemia that is reflected in FHR changes becomes apparent. Acute acidemia starts interruptions in fetal oxygenation and the occurrence of decelerations, e.g., late or variable. When the decelerations are unabated, the first evolutional change is decelerations becoming deeper and longer in duration. Tachycardia usually develops due to increase of sympathetic stimulation such as increase of blood catecholamine level. The fetus cannot increase stroke volume to increase cardiac output. Therefore, a faster heart rate is the mechanism by which the fetus increases cardiac output to compensate for recurrent hypoxial events. FHR baseline variability begins to diminish indicating acidemic change when compensatory measures begin to fail. If the acidemia is not reversed, the heart rate decreases and ultimately a terminal bradycardia will occur (**Figure 8-1** and **Figure 8-2**).[41,42]
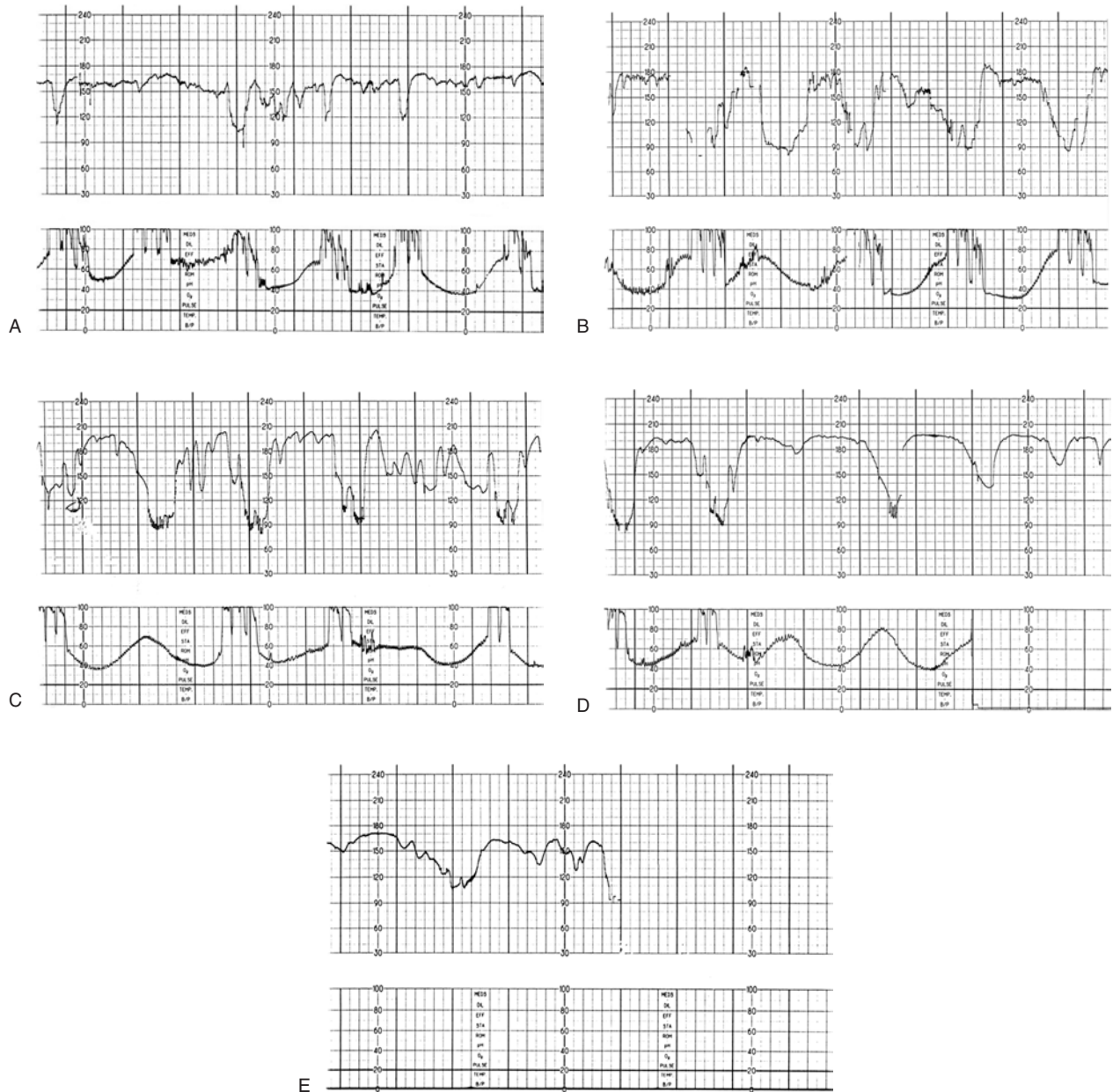
It is important to remember that this pattern undoubtedly has variations. For example, this pattern progresses through each phase faster in a fetus with a preexisting chronic hypoxia as can occur in preeclampsia, or a fetus at increased risk for asphyxial damage for other reasons such as infection or prematurity. Similarly, the fetus exposed to tachysystole may not have an intercontraction interval of long enough duration to increase the heart rate and in this instance, the expected tachycardia may not develop before the variability decreases and a terminal bradycardia ensues.[43] However, this pattern should be taught to healthcare providers who manage women in labor and viewed as a trigger for close observation. This pattern includes consideration of the effect of duration and severity (deceleration index or area under the curve). Action should be taken when the variability becomes persistently minimal in the presence of worsening decelerations. The dose-response relationship is sufficiently impressive to use at least tentatively as an indicator of the acceptability of this form of management.

B. Abrupt Change: Sentinel Events and Fetal Heart Rate Bradycardia

In contrast to the slower development of fetal acidemia in the presence of deepening decelerations, tachycardia, and decreasing variability, fetal metabolic acidemia can develop very quickly when

Recurrent variable or late decelerations

↓

Decelerations become deeper and longer in duration

↓

Compensatory tachycardia

↓

Variability decreases

↓

Terminal bradycardia

Figure 8-1 Evolution of the FHR pattern as the fetus becomes acidemic.

Figure 8-2 Case of pattern evolution: G1 P0 at 40 6/7 gestational weeks. She had an unremarkable prenatal course and has had a normal labor course with the FHR between 140 and 150 bpm, moderate variability, and occasional variable decelerations. The second stage of labor was complicated by recurrent variable decelerations that gradually became deeper and more frequent. Cesarean section as performed after a 3-hour second stage. Cord gas values for pH/ CO_2/O_2/base excess were: umbilical artery 6.82/114/14/−13 and umbilical vein 6.93/84/27/−8. The 1-, 5-, and 10-minute Apgar scores were 3, 7, and 8. The infant was transferred to the NICU for sepsis evaluation and head cooling.

the fetus sustains an acute bradycardia.[44] Sudden bradycardia can be secondary to uterine rupture, placental abruption, cord prolapse, shoulder dystocia, maternal hypotension, amniotic fluid embolism, vasa previa, and other causes. In general, if the FHR remains above 80 bpm, variability will be retained and both coronary and cerebral oxygenation will be preserved.[45] In contrast, when the FHR falls below 80 bpm, the variability will diminish rapidly as a metabolic acidemia accumulates. In this scenario, the fetus rapidly becomes unable to maintain circulation. **Figure 8-3** and **Figure 8-4** illustrate this concept.
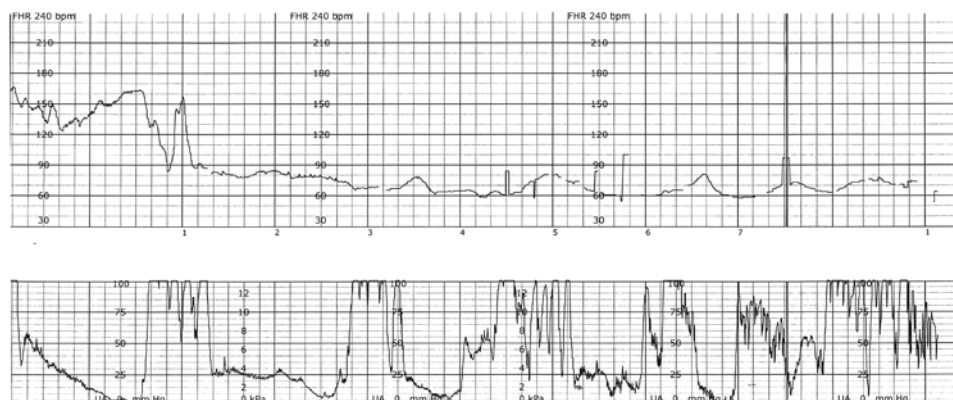
Figure 8-3 G1 P0 at term in spontaneous labor. Her medical history and prenatal course was unremarkable. The first stage of labor was normal with an FHR at 145 bpm, moderate variability, accelerations present, and occasional variable decelerations. Over the course of a 1.5 hour second stage, the FHR has continued to have a baseline of 140 bpm, moderate variability, and recurrent variable decelerations with a nadir of 100 bpm and duration of 40 seconds. The FHR dropped to between 60 and 70 bpm with absent variability for 7 minutes prior to the birth. The umbilical cord gas values for pH/ CO_2/base excess were: umbilical artery 6.88/112/−15 and the umbilical vein 6.91/99/−15. The 1- and 5-minute Apgar scores were 1 and 7 (with vigorous resuscitation). The infant was transferred to an NICU for head cooling.

Several authors have correlated the depth, duration, and presence or absence of variability in acute bradycardias in relation to newborn acidemia.[46-49] In a retrospective analysis of cases of uterine rupture, Leung et al. found none of the newborns in this series had significant metabolic acidemia when the bradycardia to birth time was less than 18 minutes if a normal FHR without decelerations preceded the bradycardia.[46] However, when the bradycardia was preceded by recurrent decelerations, metabolic acidemia occurred as early as 10 minutes after the bradycardia started. Other authors have noted that a pH less than 7.0 occurs with increasing frequency at approximately 10 minutes during a severe bradycardia associated with a sentinel event such as uterine rupture or placental abruption.[47] Kamoshita et al. evaluated the effect of bradycardia to delivery time on newborn acidemia ($n = 2267$) and outcomes at 2 years of age from a population that included women who gave birth at the index hospital and those who were transported to the hospital from outlying settings. The episodes range from acute bradycardia without known etiology, placental abruption, uterine rupture and prolapsed cord. The cases were divided into three groups: (1) unfavorable outcome including cerebral palsy or death; (2) uncomplicated pregnancy and favorable newborn outcome; and (3) complicated pregnancy (e.g., preeclampsia, intrauterine growth restriction) and favorable outcome. All term infants had a favorable outcome if the bradycardia to delivery time was less than 25 minutes, whereas a bradycardia to delivery time that was more than 25 minutes was more likely to be associated with an adverse neonatal outcome (1 out of 15 vs 3 out of 4, respectively, $P < 0.01$).[48] In summary, although the optimal bradycardia to delivery time has not been fully determined, these studies of acute bradycardia have consistently identified depth and loss of variability as the strongest associations with subsequent newborn metabolic acidosis.

C. The Initial Fetal Heart Rate Tracing with Abnormal Characteristics

Clearly evolutionary change from normal acid-base balance to fetal or newborn acidemia can be categorized into gradual/subacute change versus abrupt/acute change. In clinical practice, however, there are cases in which a sentinel event occurs prior to admission and the FHR tracing on admission is reflective of the problem. For example, cases of placenta abruption can
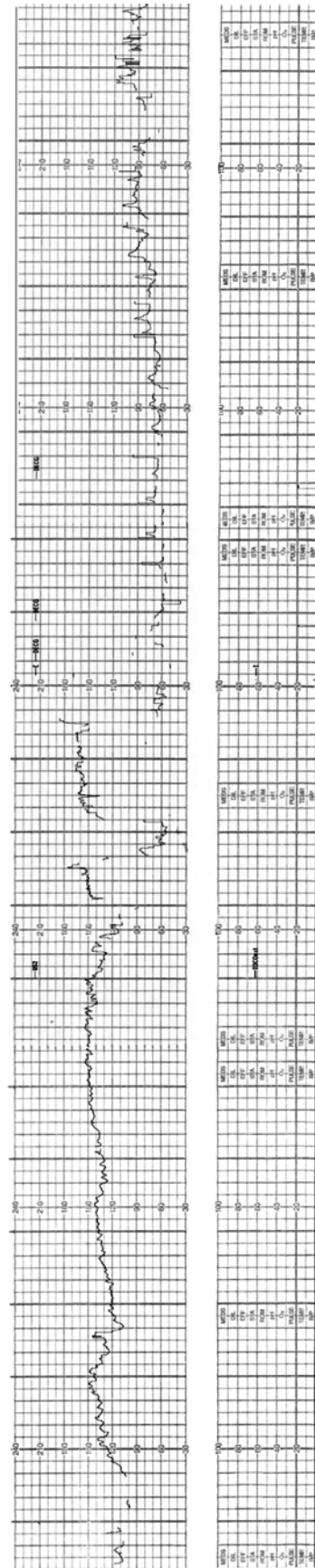
Figure 8–4 G2 P1 with twin gestation at 39 weeks. The fetuses are normally grown and vertex presentation. Twin A has just been born and uterine contractions have restarted. Approximately 10 minutes after contractions resume, a bradycardia is noted and occult umbilical prolapse is diagnosed during a vaginal examination. The FHR rose to above 90 bpm and variability was retained. The infant was born 10 minutes after the bradycardia started. The umbilical cord gas values for pH/ CO_2/O_2/base excess were: umbilical artery 7.05/84/12/−7 and the umbilical vein 7.15/68/20/−4. The 1- and 5-minute Apgar scores were 7 and 9.

manifest as severe bradycardia on admission. When the fetal acidemia has been so severe that fetal brain damage ensued and resulted in FHR changes such as decreased variability, one might observe the abnormal FHR pattern on admission. The clinical management of FHR patterns that are abnormal on admission is discussed further in Chapter 10.

III. VARIANT FETAL HEART RATE PATTERNS AND TIMING OF ACIDEMIA

Although the relationship between FHR variability, recurrent decelerations, bradycardia, and fetal or newborn acidemia has been identified, most fetuses demonstrate a variety of different FHR patterns over the course of labor. Thus, researchers conducting case-control studies of infants with abnormal neurologic findings at birth have identified FHR patterns associated with cerebral palsy, newborn seizures, and hypoxic ischemic encephalopathy. [3,5,6,22,23,31,32] The results of these studies are complementary and in whole, support the relationships defined in this chapter.

The categorization of Phelan and Ahn is useful in contemplating the timing of the asphyxial event that causes fetal brain injury. This work comes from the analysis of a registry of more than 300 brain-damaged infants in which the intrapartum courses and FHR patterns were known. [32] In analyzing these FHR patterns Phelan and Ahn identified six patterns as noted in **Table 8**-2.

Figure 8-5 shows the estimation of timing and pattern of asphyxia causing brain damage according to the five different patterns of FHR evolution. The case of bradycardia on admission

Table 8-2 Classification of Fetal Heart Rate Patterns Prior to Birth in Brain-Damaged Infants

Group	Description
Group 1 (Bradycardia on admission)	Terminal FHR pattern such as severe prolonged deceleration, bradycardia, and recurrent severe deceleration with decreased variability, usually emergent delivery followed when this group of FHR patterns was identified; this pattern was found in 3% ($n = 9$) of cases
Group 2 (Persistent nonreassuring)	Composed of nonreassuring admission tests that remained nonreassuring with or without FHR decelerations until delivery; this pattern was present in 45% ($n = 135$) of these cases, and was the most common FHR pattern identified in this cohort
Group 3 (Reassuring—abrupt change)	Reassuring fetal admission test and later FHR abruptly changes such as prolonged deceleration or bradycardia are identified; a sudden catastrophic event occurs just before delivery; 20% ($n = 61$) of cases exhibited this pattern
Group 4 (Reassuring—gradual change)	Phelan and Ahn called this the "Hon pattern," which described reassuring fetal admission tests that evolved with recurrent late and/or variable decelerations, increasing baseline, and decreased variability followed by decreasing baseline rate and terminal bradycardia; Hon called "stair-step to death"; this pattern was the second most frequent and was present in 22% ($n = 67$) of the cases
Group 5 (Persistent reassuring)	Reassuring admission test and the FHR remained within normal range during delivery; this pattern was present in 8% ($n = 24$) of the cases
Unclassified	1% ($n = 4$) of the cases had an FHR pattern that could not be classified

Based on Phelan JP, Ahn MO. Fetal heart rate observations in 300 term brain-damaged infants. *J Matern Fetal Investig.* 1998;8(1):1-5. [32]
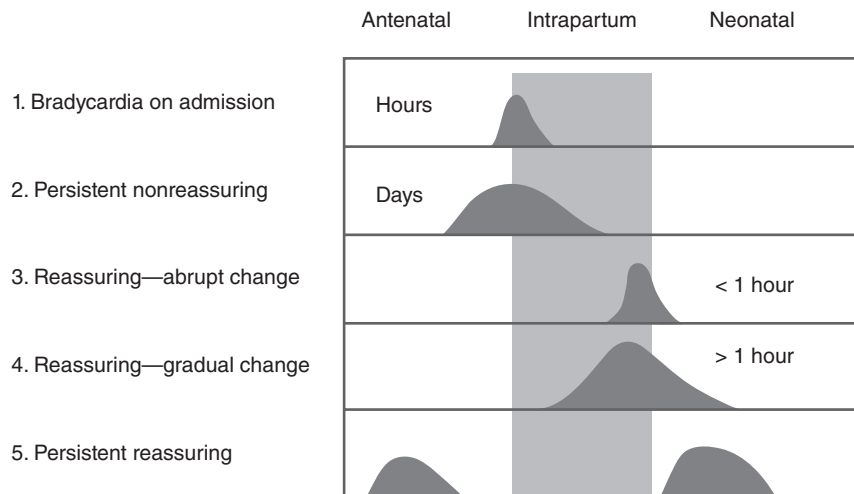
Figure 8-5 Estimated timing and pattern of asphyxia causing brain damage according to each 5 different FHR evolution patterns. Data from Phelan JP, Ahn MO. Fetal heart rate observations in 300 term brain-damaged infants. *J Matern Fetal Investig.* 1998;8(1):1-5.[32]

(Group 1) is thought to be an event that occurred just hours before the admission. Placental abruption is the most common disorder that would result in this bradycardia group. Persistent nonreassuring (Group 2) indicates causal events have occurred over a relatively long period such as days before admission. In the case of the reassuring then abrupt change group (Group 3), acute sentinel events such as cord prolapse and uterine rupture cause hypoxic-ischemic damage within 1 hour. The reassuring then gradual change (Group 4) suggests relatively subacute (usually more than 1 hour) stress affected the fetal brain causing hypoxic-ischemic encephalopathy over a period of time. In cases of persistent reassuring FHR patterns (Group 5), the causes of brain injury might have occurred antenatally, such as congenital brain anomaly, or in the neonatal period, as can occur following neonatal GBS infection. The very few cases that could not be placed into one of these five categories were placed into a final "Other" (Group 6) category.

IV. CLINICAL IMPLICATIONS

The fact that acidemia develops over a reasonably long period of time, approximately 1 hour following the onset of recurrent FHR decelerations and the common FHR pattern evolution, is support for the practice of observing the FHR tracing over time to identify acidemia. Unless a sentinel event such as uterine rupture occurs, one has time for obstetric decision-making, attempts to ameliorate variant FHR patterns, and to institute appropriate intervention, i.e., delivery, before serious acidemia sufficient to damage the fetus has occurred. However, this raises the importance of the decision-delivery time, which may be vastly different in various institutions. The logistical realities of each individual labor and delivery suite will determine at what stage of the evolution of the FHR pattern intervention should occur.

These findings support the use of standard algorithms for management of FHR patterns, with the aim of avoiding metabolic acidemia and minimizing unnecessary obstetric intervention. Algorithms will decrease variability in practice[50] while studies of the reliability and validity of FHR monitoring are ongoing, due to the almost universal use of monitoring in North America, and the diversity of clinical management schemas being used. We also believe that standardization of management is required even while awaiting agreement with regard to the results of trials of ancillary techniques, such as computerized decision support tools, because of the substantial lag time that often accompanies the clinical acceptance or rejection of new obstetric technologies, even after the publication of results of randomized control trials.[51]

Once algorithms based on reasonable evidence have been formulated, they should then be subjected to prospective examination by appropriately designed trials. This would then allow a much more rational usage of FHR monitoring in contemporary obstetric practice. One such algorithm forms the basis of the next chapter.

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