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Fetal Considerations in the Critically Ill Gravida

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Introduction

Unlike any other medical or surgical specialty, obstetrics deals with the simultaneous management of two – and sometimes more – individuals. Under all circumstances, the obstetrician must delicately balance the impact of each treatment decision on the pregnant woman and her fetus, seeking, when possible, to minimize the risks of harm to each person. Throughout this text, the primary focus has been on the critically ill obstetric patient and, secondarily, her fetus. Although the fetal effects of those illnesses were reviewed in part, the goal of this chapter is to highlight, especially for the non-obstetric clinician, the important clinical fetal considerations encountered when caring for these complicated pregnancies. To achieve that objective, this chapter reviews: (1) current techniques for assessing fetal well-being, (2) fetal assessment in the intensive care unit (ICU), (3) fetal considerations in several maternal medical and surgical conditions, (4) the contemporary management of the gravida who is brain-dead or in a persistent vegetative state, and (5) the role of perimortem cesarean delivery in contemporary obstetrics.

Detection of fetal distress in the critically ill obstetric patient

Almost a half century ago, Hon and Quilligan [1] demonstrated the relationship between certain fetal heart rate (FHR) patterns and fetal condition by using continuous electronic FHR monitoring in laboring patients. Since then, continuous electronic FHR monitoring has become a universally accepted method of assessing fetal well-being during labor [2,3] with the goal of permitting the clinician to identify those fetuses at a greater likelihood of intrapartum fetal death [4] and to intervene when certain FHR abnormalities are present.

In addition to the intrapartum assessment of fetal well-being, the fetal monitor has been used to assess fetal health before labor [5] and to attempt to identify those fetuses at risk for intrauterine death. Once that fetus is identified, the maternal-fetal unit is moved from outpatient to inpatient care. Once in labor and delivery, continuous fetal monitoring is used to determine whether continued expectant management or delivery by induction of labor or cesarean is the next form of intervention. It is this area of fetal monitoring, antepartum rather than intrapartum fetal assessment, that is used more frequently in the arena of the critically ill gravida. In keeping with those core principles, the focus of this chapter will be on applications of fetal monitoring to assess fetal status both in the ICU setting and intrapartum during labor.

Although the presence of a reassuring FHR tracing is virtually always associated with a well-perfused and oxygenated fetus [5,6], an "abnormal tracing" is not necessarily predictive of an adverse fetal outcome. While it was anticipated that the detection of abnormal FHR patterns during labor and expeditious delivery of such fetuses would impact the subsequent development of cerebral palsy, this expectation has not been realized because the number of fetuses injured during labor was highly overestimated and the number of fetuses injured before labor was highly underestimated [7]. However, with the ubiquitous use of electronic FHR monitoring during labor and a rise in the cesarean delivery rate for the past two decades from 5% to over 25%, a decline

Critical Care Obstetrics, Sixth Edition. Edited by Jeffrey P. Phelan, Luis D. Pacheco, Michael R. Foley, George R. Saade, Gary A. Dildy, and Michael A. Belfort.

in the rate of asphyxia-induced cerebral palsy among singleton term infants has been observed [8,9]. For example, Smith and associates [9] documented a 56% decline over two decades in the incidence of hypoxic ischemic encephalopathy (HIE) among singleton term infants. During this time, the incidence of HIE dropped from 1 per 8000 to 1 per 12,500 births.

While the specific entity of cerebral palsy is, in most cases, unrelated to the events associated with labor and delivery, it is more often related to prenatal developmental events, infection, or complications of prematurity. Nevertheless, the basic physiologic observations relating to specific FHR patterns remain, for the most part, valid. The critically ill mother will necessarily shunt blood from the splanchnic bed (including the uterus) in response to shock. Because of this and the fact that the fetus operates on the steep portion of the oxyhemoglobin dissociation curve, any degree of maternal hypoxia or hypoperfusion may first be manifested as an abnormality of the FHR. In this sense, the late second- and third-trimester fetus serves as a physiologic oximeter and cardiac output computer. Observation of FHR changes, thus, may assist or alert the clinician to subtle degrees of physiologic instability, which would be unimportant in a nonpregnant adult but may have potentially detrimental effects to the fetus [10].

The next few pages present an overview of FHR patterns pertinent to the critically ill gravida. Interpretations of FHR patterns, like all diagnostic tests, depend on the index population, and consequently, certain of these observations may not be applicable to the laboring but otherwise well mother. For a more detailed description of antepartum and intrapartum FHR tracings associated with fetal brain injury, the reader is referred to the classic descriptions by Phelan and Ahn [11], Phelan and Kim [12], Phelan [13], and Phelan and associates [14].

Of note, FHR interpretation was changed into a category system [15] in 2008 (Table 8.1). As demonstrated in Table 8.1, a Category 1 strip includes a normal baseline rate, moderate variability, the absence of late and variable decelerations, and early decelerations, and accelerations may be present or absent. A Category 1 strip is consistent with a healthy fetus and a lack of metabolic acidosis. In contrast, a Category 3 FHR strip is the polar opposite of Category 1, and is indicative of a fetus in significant potential fetal jeopardy. This means, according to the "consensus," that the fetus with a Category 3 tracing, if uncorrected, is at a higher probability of metabolic acidosis and the potential for *fetal neurologic injury*. A Category 2 strip is somewhere in between 1 and 3. In other words, a Category 2 FHR tracing includes everything that is not Category 1 or Category 3. However, it is important to remember the following:

Table 8.1 Category system for interpretation of intrapartum fetal heart rate tracings.

Category I
Category I fetal heart rate (FHR) tracings include all of the following:
- Baseline rate: 110–160 beats per minute (bpm)
- Baseline FHR variability: Moderate
- Late or variable decelerations: Absent
- Early decelerations: Present or absent
- Accelerations: Present or absent

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

Baseline rate
- Bradycardia not accompanied by absent baseline variability
- Tachycardia

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

Accelerations
- Absence of induced accelerations after fetal stimulation

Periodic or episodic decelerations
- Recurrent variable decelerations accompanied by minimal or moderate baseline variability
- Prolonged deceleration ≥2 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
Category III FHR tracings include either:
- Absent baseline FHR variability and any of the following:
  - Recurrent late decelerations
  - Recurrent variable decelerations
  - Bradycardia

Sinusoidal pattern

Source: Ref. [15].

1) Not all fetuses require systemic metabolic acidosis to become neurologically impaired.
2) No one pH can determine whether a fetus will become permanently neurologically impaired [16].
3) The key issue with a Category 2 FHR tracing is that the recruitment of two or more FHR abnormalities, in all likelihood, increases the risk of an adverse outcome.

With that as a backdrop, we will now break down the numerous FHR characteristics into their separate parts for purposes of understanding.

Baseline fetal heart rate

The baseline FHR is the intrinsic heart rate of the fetus. The baseline FHR is determined by approximating the mean FHR rounded to increments of 5 beats per minute (bpm) during a 10 min window, excluding accelerations and decelerations and periods of marked variability (>25 bpm). A normal baseline FHR is between 110 bpm and 160 bpm. A baseline FHR below 110 bpm is termed a bradycardia, and a baseline FHR in excess of 160 bpm is termed a FHR tachycardia [15].

A persistent slow FHR or an intrinsic bradycardia

Bradycardia is defined as the intrinsic heart rate of the fetus of less than 110 bpm, as opposed to a sudden, rapid, and sustained deterioration of the FHR from a previously normal or tachycardic rate that lasts until delivery, or, under today's parlance, a FHR bradycardia. As such, a persistent slow FHR may be associated with an underlying congenital fetal abnormality, such as a structural defect of the fetal heart. In addition, congenital bradyarrhythmias may involve fetal heart block secondary to a prior maternal infection, a structural defect of the fetal heart, or systemic lupus erythematosus with anti-Ro/SSA antibodies [17]. In these circumstances, the FHR bradycardia is not usually a threat to the fetus. But, alternative methods of fetal assessment, such as the fetal biophysical profile (BPP) [18], are necessary in this select group of patients to assure fetal well-being before and during labor. Given the inherent difficulties in providing continuous fetal monitoring and assuring fetal well-being in fetuses with a bradyarrhythmia, cesarean delivery may well represent the preferred route of delivery for these patients. Obviously, the decision to proceed directly to a cesarean will depend on the overall clinical circumstances and appropriate patient informed consent.

A sudden rapid and sustained deterioration of the fetal heart rate

Prolonged FHR deceleration is distinctly different from a bradycardia. In the former, the fetal monitor strip is typically reactive with a normal or tachycardic baseline rate; but, due to a sentinel hypoxic event, such as those depicted in Table 8.2, the FHR suddenly drops and remains at a lower level unresponsive to remedial measures and/or terbutaline therapy. In the critical care setting, a sudden, rapid, and sustained deterioration of the FHR or a prolonged FHR deceleration may arise from a partial or complete abruption in cases of markedly and persistently elevated maternal blood pressures or an aggressive lowering of maternal BP with antihypertensive agents [19]. This type of FHR pattern may also herald a sudden maternal hypoxic event, such as amniotic fluid embolus syndrome [20], acute respiratory insufficiency, or an eclamptic seizure [19,21]. Prolonged FHR decelerations have also been associated with maternal operative procedures such as cardiopulmonary bypass with inadequate maternal flow rates [22,23], and brain surgery during hypothermia [24].

Table 8.2 Examples of sentinel hypoxic events associated with a sudden, rapid, and sustained deterioration of the fetal heart rate that can be unresponsive to remedial measures and/or terbutaline lasting until delivery from a previously reactive fetal heart rate.

Umbilical cord prolapse
Uterine rupture
Placental abruption
Maternal arrest (e.g., AFE syndrome)
Fetal exsanguination

AFE, Amniotic fluid embolus.

In a patient with a prior normal baseline FHR, the abrupt occurrence and persistence of a fetal heart rate of less than 110 bpm for an extended period of time unresponsive to remedial measures and/or terbutaline therapy constitute an obstetric emergency. Under these circumstances, and assuming the pregnant woman is hemodynamically and clinically stable and the fetus is potentially viable, these patients should be managed as if the fetus has had a cardiac arrest and be delivered as rapidly as it is technically feasible in keeping with the level of the institution in question.

Tachycardia

Fetal tachycardia is defined as a baseline FHR of 160 bpm or greater lasting 10 min or longer. Most commonly, this type of baseline FHR abnormality can be associated with prematurity, maternal pyrexia, or chorioamnionitis. In addition, betamimetic administration, hyperthyroidism, or fetal cardiac arrhythmias may also be responsible. The clinical observation of a FHR tachycardia, in and of itself, is probably not an ominous finding but probably reflects a normal physiologic adjustment to an underlying maternal or fetal condition. Although operative intervention is infrequently required, a search for the underlying basis for the FHR tachycardia and a reanalysis of the admission FHR pattern may be helpful.

For example, a patient with a previously reactive FHR pattern with a normal baseline rate (Figure 8.1) who develops the Hon pattern of intrapartum asphyxia or ischemia [11] that is characterized by a substantial rise in the baseline rate – often to a level of tachycardia

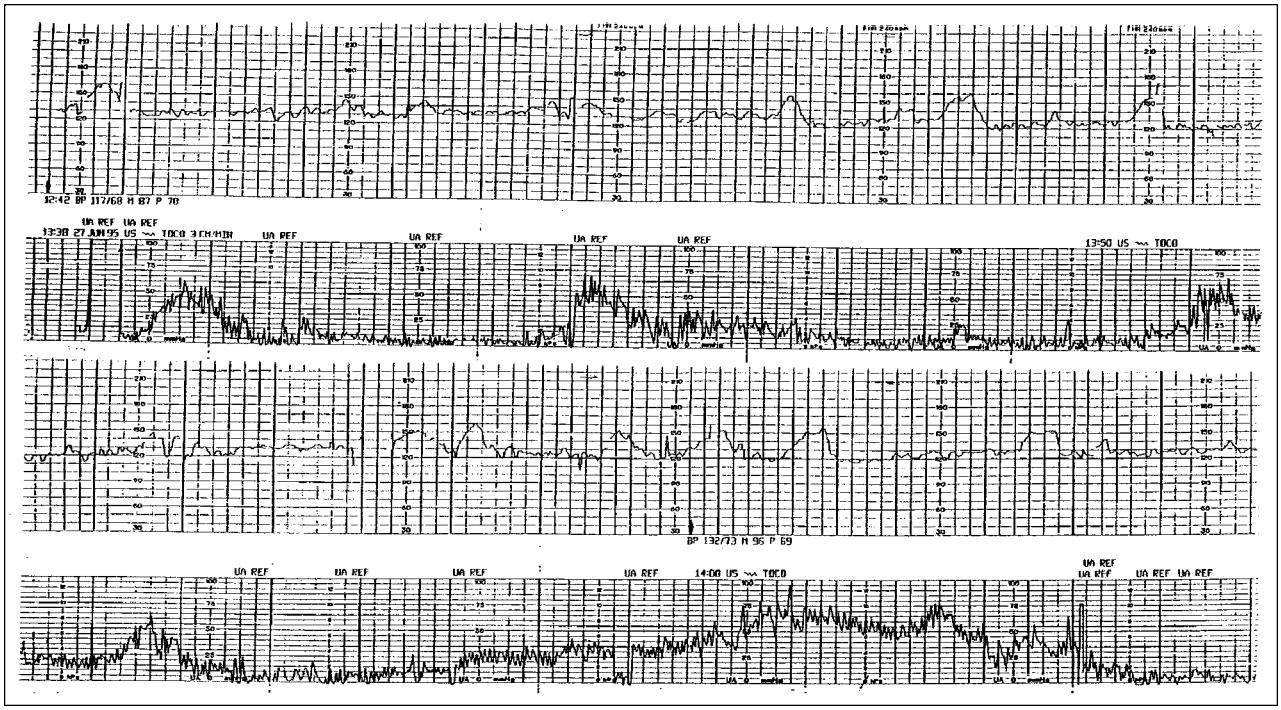


Figure 8.1 Admission FHR of this term pregnancy with spontaneously ruptured membranes exhibits a baseline rate of around 120 bpm and numerous FHR accelerations or a reactive FHR pattern. This FHR pattern would now be characterized as a Category 1 fetal monitor strip.

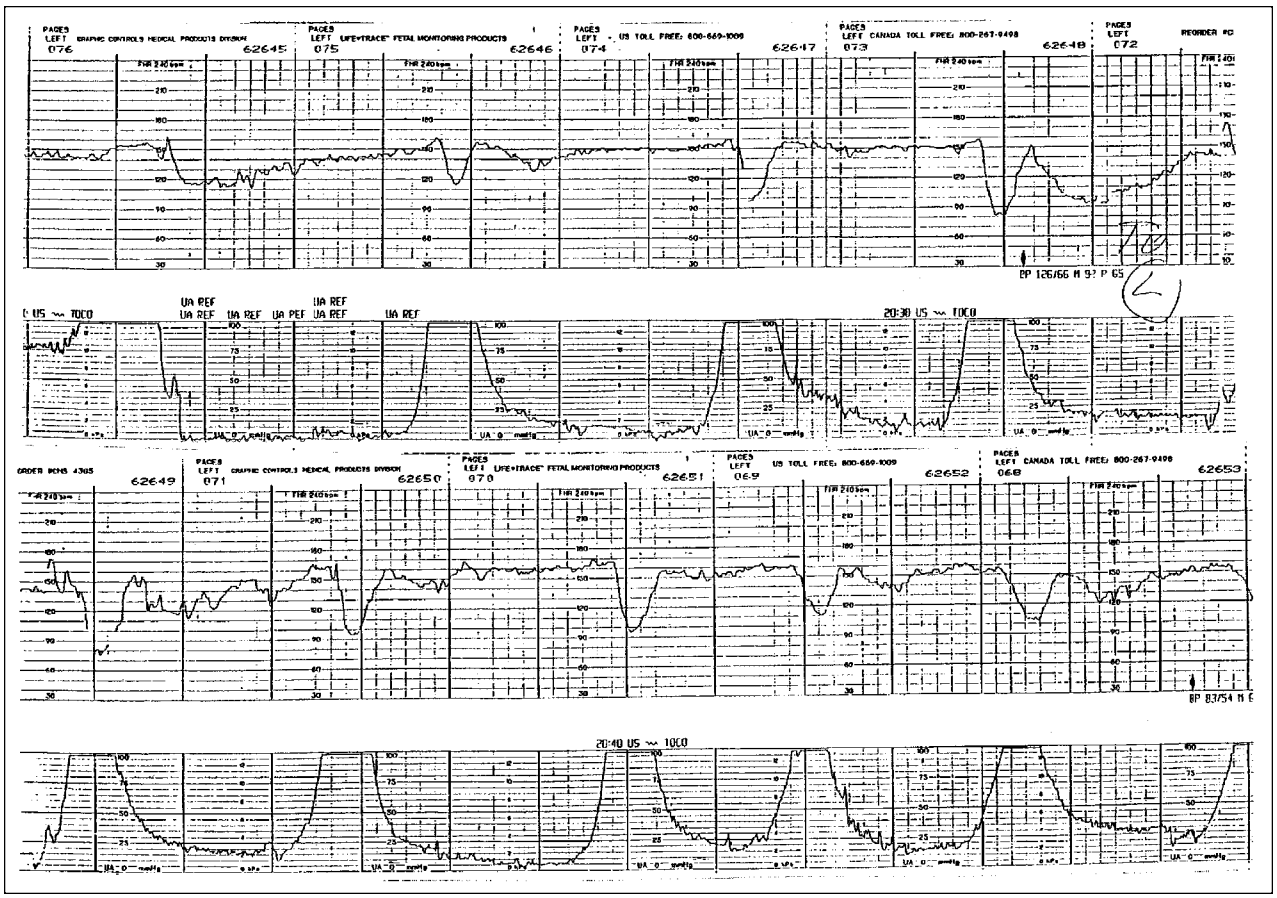


Figure 8.2 Some time later, the fetus exhibits an FHR tachycardia around 160 bpm, repetitive FHR decelerations, and nonreactivity. Under current characterizations, this FHR pattern would be considered a Category 2 fetal monitor strip.

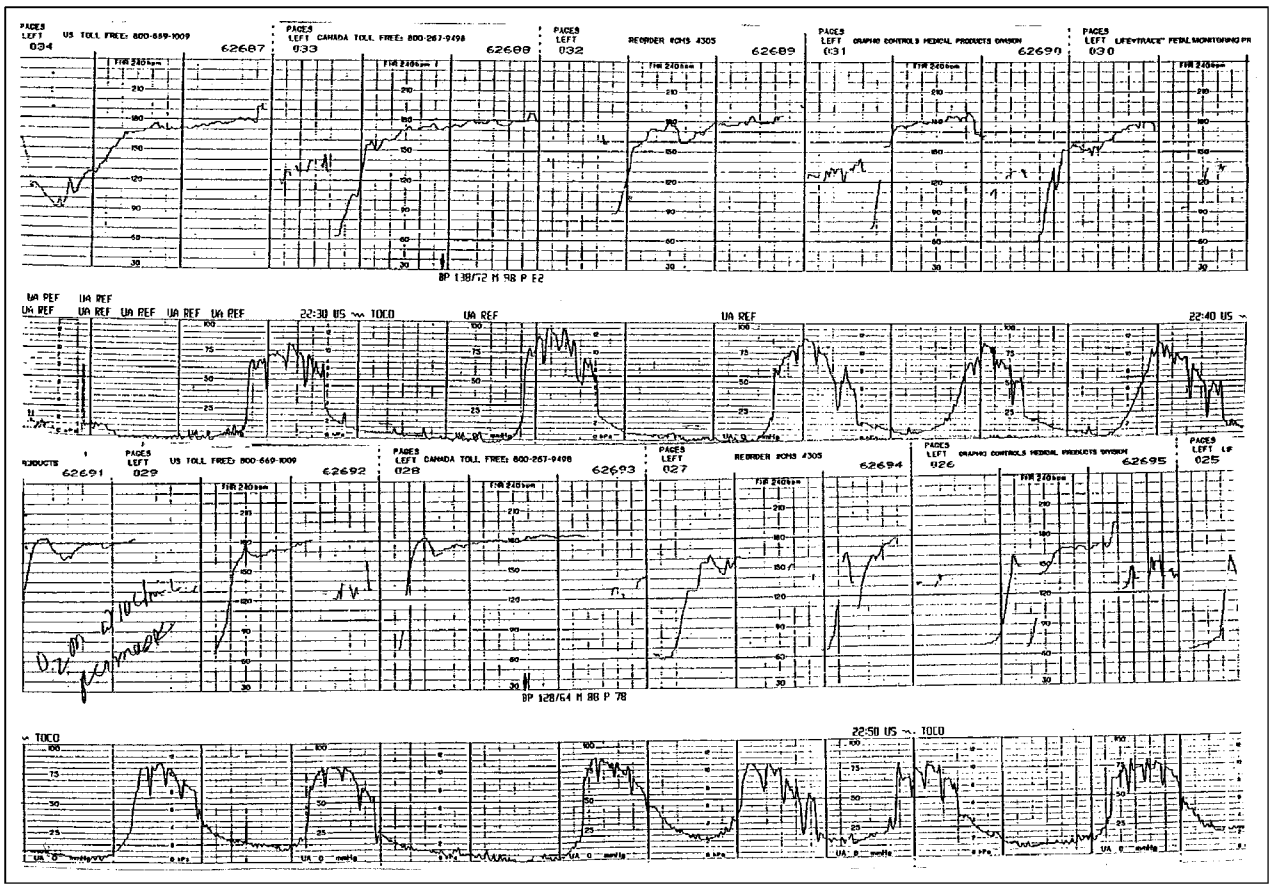


Figure 8.3 Later in labor, the baseline FHR reaches 180 bpm and continues to exhibit repetitive FHR decelerations, nonreactivity, and diminished variability. Despite the changes in the FHR pattern from the time of admission (Category 1 strip) and even though multiple Category 2 characteristics are now present, some would still characterize this FHR pattern as a Category 2 because the variability is not absent. Of note, this fetus was born with spastic quadriplegia due to hypoxic ischemic encephalopathy.

(Figures 8.2 and 8.3) in association with an inability to accelerate or nonreactivity, repetitive FHR decelerations, and usually a loss of FHR variability – flags fetal brain injury [25], and the fetus is at risk for hypoxic ischemic brain injury [11–13]. In this clinical setting, assessment of the usual causes of FHR tachycardia should be undertaken. If the mother does not have a fever to account for the change in fetal status, assessment of fetal acid–base status with scalp or acoustic stimulation [6,12] or delivery as soon as it is practical, in keeping with the capability of the hospital, should be considered. If the gravida has a fever, she should be cultured, and treated with antibiotics and antipyretics. If the FHR pattern does not return to normal (i.e., the same FHR pattern the fetus had on admission – normal baseline FHR and reactive) within approximately an hour of the initiation of medical therapy and regardless of whether the FHR variability is average [11–13,26], the patient should be delivered as expeditiously as possible.

Baseline fetal heart rate variability

Fetal heart rate variability (FHRV) is determined in a 10 min window, excluding accelerations and decelerations. Baseline FHRV is defined as fluctuations in the baseline FHR that are irregular in amplitude and frequency. The fluctuations are visually quantitated as the amplitude of the peak-to-trough in BPM. In other words, FHRV is the beat-to-beat variation in the FHR resulting from the continuous interaction of the parasympathetic and sympathetic nervous systems on the fetal heart [15].

Since the last edition of this textbook, the two approaches to define FHRV [11,14,15,27] have been consolidated under the National Institutes of Child Health and Human Development (NICHD) [15,27] umbrella to classify FHRV. The NICHD approach subclassifies FHRV into four categories:

1) Undetectable or absent FHRV
2) Minimal (more than absent but ≤5 bpm)

3) Moderate (6–25 bpm)
4) Marked (>25 bpm).

A loss of FHRV, in and of itself, may not necessarily be an ominous observation. In most cases, the loss of FHRV can represent normal fetal physiologic adjustments to a number of medications, illicit substances, or simply behavioral state changes such as 1 F to 4 F [28]. For example, narcotic administration [29] or magnesium sulfate infusion [30] can alter FHRV by inducing a change in the behavioral state of the fetus to one of a sleep state or behavioral state 1 F. Clinically, the change in FHRV to minimal or absent appears to be clinically significant in cases of the Hon pattern of intrapartum asphyxia [11–13]. As observed herein (Figures 8.1–8.3), the FHR pattern was first reactive and exhibited a normal baseline rate or a Category 1 FHR tracing. Subsequently, the FHR pattern changed. Then, minimal FHRV was associated with a loss of FHR reactivity (the ability of the fetus to accelerate), a substantial rise in the baseline FHR to the level of a FHR tachycardia, and repetitive (more than recurrent) FHR decelerations. Under these circumstances, the potential for fetal asphyxia is increased. Additionally, the presence of loss of FHRV [26] in the setting of the Hon pattern of intrapartum asphyxia has been associated with significantly higher rates of neonatal cerebral edema.

Sinusoidal fetal heart rate pattern

A sinusoidal FHR pattern is a specific FHR pattern that has a visually apparent, smooth, sine wave–like undulating pattern in the baseline FHR. It is further defined as a persistent regular sine wave variation of the baseline FHR that has a cycle frequency of 3–5 cycles per minute for 20 min or longer [15]. The degree of oscillation correlates with fetal outcome [31]. For instance, infants with oscillations of 25 bpm or more have a significantly greater perinatal mortality rate than do infants whose oscillations are less than 25 bpm (67% vs. 1%). A favorable fetal outcome also is associated with the presence of FHR accelerations and/or nonpersistent sinusoidal FHR pattern.

The key to the management of a persistent sinusoidal FHR pattern is recognition and prompt evaluation. Once a sinusoidal FHR pattern is recognized, a timely clinical evaluation of the patient and a search for the underlying cause should be considered. Nonpersistent or an intermittent sinusoidal FHR pattern is commonly related to maternal narcotic administration [32]. In the absence of maternal narcotic administration, the sudden appearance of a persistent sinusoidal FHR pattern and a lack of FHR accelerations do suggest the potential for fetal anemia and fetal-maternal hemorrhage.

Fetal anemia may be associated with a number of obstetric conditions such as placental abruption or previa, fetal-maternal hemorrhage, vasa previa, Rh sensitization, and non-immune hydrops [32]. If, for example, a persistent sinusoidal FHR pattern is observed in a patient who recently has been involved in a motor vehicle accident or a victim of domestic violence, placental abruption is one consideration. Evidence of an abruption or other forms of fetal hemorrhage may also be suggested by a positive Kleihauer–Betke (K-B) test for fetal red blood cells (RBCs) in the maternal circulation. Finally, as suggested by Katz and associates [31], a persistent sinusoidal FHR pattern in the absence of accelerations is a sign of potential fetal compromise. In this latter circumstance, a K-B test with either delivery or some form of fetal acid–base assessment with scalp or acoustic stimulation should be considered [33,34]. Often, patients with a persistent sinusoidal FHR pattern will have a history of reduced fetal activity, usually a stair-step reduction over several days [35], and, occasionally, an abnormal K-B test [34,36].

Periodic changes or FHR changes in response to uterine contractions

The focus of this section is on periodic FHR changes that occur in response to uterine contractions, such as FHR accelerations and variable and late decelerations. FHR decelerations, in and of themselves, are not associated with an increased risk of perinatal morbidity and mortality. To be associated with adverse fetal outcome (i.e., cerebral palsy due to hypoxic ischemic encephalopathy), FHR decelerations should be repetitive and in association with usually diminished FHR variability, a rising baseline rate to a level of FHR tachycardia, and a nonreactive FHR pattern [11,14]. To understand these periodic changes, the reader is encouraged to review the NICHD and CIPF approaches to the interpretation of periodic FHR decelerations. The CIPF approach is based on the criteria established in the 1960s and 1970s and published in Corometric's Teaching Program around 1974 [37] for FHR interpretation. Each of these periodic changes will be discussed separately here to assist the reader in their understanding of FHR patterns during labor.

Accelerations

An FHR acceleration is a visually apparent abrupt increase in the FHR above baseline. An abrupt increase is defined as an increase from the onset of the acceleration to the peak in less than 30 s. To be called an acceleration, the peak must

15 bpm or higher and the acceleration must last 15 s or longer from the onset to the return [15]. In preterm fetuses at <32 weeks gestation, accelerations are defined as having a peak of 10 bpm or higher and a duration of 10 s or longer. Of note, a FHR acceleration lasting longer than 10 min is considered a baseline change.

FHR accelerations can occur spontaneously or in relation to uterine activity, fetal body movement, or fetal breathing. Whenever spontaneous or induced FHR accelerations are present, a healthy and non-acidotic fetus is probably present. This is true, regardless of whether otherwise "worrisome" features of the FHR tracing are present [5,6,38]. The presence of FHR accelerations is the basis to assess fetal well-being both before and during labor [5,6].

The presence of FHR accelerations is a sign of fetal well-being with a low probability of fetal compromise [5], brain damage [39], or death within several days to a week of fetal surveillance testing [5]. This observation persists irrespective of whether the acceleration is spontaneous or induced [5]. In contrast, the findings of a persistent nonreactive FHR pattern lasting longer than 120 min from admission to the hospital or the physician's office is a sign of preexisting compromise due to a preadmission to the hospital or pre-NST fetal brain injury [14], structural [40] or chromosomal abnormality [41], fetal infection due to cytomegalovirus or toxoplasmosis [42], or maternal substance abuse.

Briefly, the clinical approach to assessing fetal health begins with monitoring the baseline FHR for a reasonable period to determine the presence of FHR accelerations or reactivity. In using an outpatient approach such as the NST, the goal is to identify the fetus at risk of death in utero. In this circumstance, a certain number of accelerations are required within a 10 or 20 min window to satisfy the criteria for a reactive NST. In contrast, in the patient in the hospital or ICU, the criteria for reactivity can be less because surgical intervention is readily available.

If the NST is considered nonreactive after a 40 min monitoring period, several options are available to the clinician. These include but are not limited to the following: to continue fetal monitoring, or to perform a contraction stress test [42], fetal biophysical profile (BPP) [43,44], or some form of fetal stimulation. If, after acoustic stimulation, the fetus has a persistent nonreactive pattern, a contraction stress test [42] or the BPP [18,44] can be used to evaluate fetal status.

In the critical care setting, the BPP (Table 8.3) is the easiest approach to use after fetal monitoring. Since the introduction of the BPP, this technique has been modified to include the amniotic fluid index to estimate the amniotic fluid volume [45,46]. Based on the work of Phelan and associates [5,45,46], an amniotic fluid index (AFI) of ≤5.0 cm is considered oligohydramnios. Consequently, if a patient has an AFI ≤5.0 cm, her BPP score for that component will be 0. Additional components of the BPP include fetal breathing movements, fetal limb movements, fetal tone, and reactivity on an NST. Based on the presence or absence of each component, the patient receives 0 or 2 points.

Table 8.3 Fetal biophysical profile (BPP) components required over a 30-min period.[a]

Components[b]	Normal result	Score
Non-stress test	Reactive	2
Fetal breathing	Duration ≥1 min	2
Fetal movement	≥3 movements	2
Fetal tone	Flexion and extension of limb	2
Amniotic fluid volume	Amniotic fluid index >5.0 cm	2
Maximum score		10

[a]This represents one approach to the BPP.
[b]Components of the BPP include the modification for determining the amniotic fluid volume using the amniotic fluid index.
Source: Refs. [44–46].

A BPP score of 8 or 10 is considered normal. In patients whose score is 6, the test is considered equivocal or suspicious. In such patients, a repeat BPP is recommended in 12–24 h. If the patient is considered to be at term, she should be evaluated for delivery [44]. The patient with a biophysical profile score of 0, 2, or 4 is considered for delivery; but this BPP score does not mandate a cesarean. A trial of labor is reasonable whenever the cervix is favorable for induction, the amniotic fluid volume is normal (AFI >5.0 cm), and the fetus is not growth impaired. In the preterm fetus with a BPP score of 4 or less, the subsequent clinical management does not mandate delivery but does require an evaluation and a balancing of the risks of prematurity with those of continued intrauterine existence. If delivery is determined to be the best course of action under the circumstances, and with proper informed consent, the options of induction of labor and cesarean are available.

Variable deceleration

Variable FHR decelerations have a variable or nonuniform shape and bear no consistent relationship to a uterine contraction. A variable deceleration is a visually apparent abrupt decrease in the FHR. An *abrupt decrease* is defined as a decrease from the onset of the deceleration to the beginning of the FHR nadir of 30 s or longer. The decrease in FHR is calculated from the onset of the deceleration to the nadir of the deceleration. This decrease in the FHR is 15 bpm or more, lasting less than 2 min in

duration [15]. Of note, NICHD suggests that variable decelerations be observed over successive contractions because the onset, depth, and duration of variable decelerations commonly vary with successive contractions.

Umbilical cord compression leading to an increased fetal BP and baroreceptor response is felt to be the most likely etiology. Umbilical cord compression is more likely to occur in circumstances of nuchal cords, knots, cord prolapse [47], or a diminished amniotic fluid volume [48,49].

To simplify intrapartum management, investigators such as Kubli *et al*. [50] and Krebs *et al*. [51] have attempted to classify variable decelerations. For example, Kubli and associates [50] used the depth of the deceleration to determine the fetal risk status and have correlated fetal outcome with mild, moderate, or severe variable decelerations. Kubli's criteria, however, are cumbersome and do not reliably lend themselves to easy clinical use. In contrast, Krebs *et al*.'s [51] criteria rely on the visual characteristics of the variable decelerations rather than on the degree or amplitude of the FHR deceleration. If the variable deceleration did not maintain the usual characteristics of a variable FHR deceleration, such as a variable deceleration with a late component or a slow return to the baseline, a rising baseline rate or tachycardia after the deceleration, or biphasic decelerations (Figures 8.2 and 8.3 illustrate a biphasic or "w" deceleration), Krebs termed these FHR decelerations as atypical variable decelerations. In addition, Krebs was able to demonstrate that when repetitive, atypical, variable decelerations are present over a prolonged period of time in a patient with a previously normal FHR tracing, the risk of low Apgars was increased. However, the presence of nonrepetitive atypical variables, in and of themselves, are clinically insignificant.

However, these atypical features in the circumstance of a Hon pattern of intrapartum asphyxia [11–13,52] can be associated with fetal brain injury. As demonstrated in Figures 8.1–8.3, when persistent, atypical variable FHR decelerations occur in association with a substantial rise in the baseline FHR to a level of tachycardia, in the absence of FHR accelerations or nonreactivity and with or without a loss of FHRV (Figures 8.1–8.3), expeditious delivery should be considered.

Late decelerations

A late deceleration is a visually apparent, usually symmetrical gradual decrease and return of the FHR associated with a uterine contraction. The gradual FHR decrease is defined as from the onset of the FHR nadir of 30 s or longer. The decrease in FHR is calculated from the onset to the nadir of the deceleration. Additionally, the deceleration is delayed in timing, with the nadir of the deceleration occurring after the peak of the contraction. This usually means that the onset, nadir, and recovery of the deceleration occur after the beginning, peak, and ending of the contraction. In general, classic late decelerations are a uniform deceleration pattern with onset at the peak of the uterine contraction, the nadir in heart rate at the offset of the uterine contraction, and a delayed return to baseline after the contraction has ended [37].

In the past, late decelerations were considered clinically significant when they were repetitive (i.e., occurring with each contraction of similar magnitude) and associated with a substantial rise in baseline FHR, a loss of reactivity, with or without a loss of FHRV [11–14]. At the same time, nonpersistent or intermittent late decelerations were probably variables and, consequently, appeared to have no bearing on fetal outcome [53]. Now, the definition has been broadened. Rather than repetitive late decelerations, late decelerations need only be recurrent if they occur in 50% or more of the contractions in a 20 min segment [15]. Decelerations less frequent than 50% of the contractions in a 20 min window are termed intermittent [15]. In fact, Nelson and associates [53] found that 99.7% of late decelerations observed on a fetal monitor strip were associated with a favorable fetal outcome.

Whenever a patient with a reactive admission FHR pattern develops recurrent late decelerations in association with a fetal tachycardia and a loss of reactivity, traditional maneuvers of intrauterine resuscitation such as maternal repositioning, oxygen administration, and increased intravenous fluids are warranted. If this pattern persists, assessment of the fetal ability to accelerate its heart rate [5,6] or delivery should be considered.

In the critical care setting, late decelerations are frequently reversed in a number of clinical circumstances, such as diabetic ketoacidosis [54,55], sickle cell crisis [56], acute hypovolemia, or anaphylaxis [57,58]. With correction of the underlying maternal metabolic and hemodynamic abnormality, the FHR abnormality usually will resolve, and operative intervention is often unnecessary. Persistence of the FHR pattern after maternal metabolic recovery, however, may suggest an underlying fetal diabetic cardiomyopathy [59] or preexisting fetal compromise [11–13,52] and should, when accompanied by the aforementioned additional signs of fetal compromise, lead to assessment for fetal reactivity or delivery.

Overview of periodic changes

In summary, the NICHD changes [15] are as follows:

1) The NICHD criteria broadened the definition of a late deceleration to include a deceleration with its onset at any time during the contraction as opposed to at the

peak of the contraction. Additionally, the nadir or the lowest point of a late deceleration can occur after the peak of the contraction rather than at the offset of the contraction [27].

2) To determine whether a variable deceleration is present, the NICHD approach requires the practitioner to review successive contractions but does not appear to impose a similar requirement for late or early decelerations [15].
3) Recurrent FHR decelerations are persistent decelerations with more than 50% of contractions in any 20 min segment [27]. This definition is broader than the previous requirement of "repetitive" FHR decelerations, or decelerations that occur with each and every contraction.
4) The characterization of variable decelerations is patterned after those of Kubli [50], which is based on the depth and duration of the deceleration ("the big, the bad and the ugly"). This contrasts with the approach described by Krebs and associates [51]. With the latter approach, an atypical deceleration is defined as one that has lost its normal characteristics such as the loss of the primary and secondary accelerations associated with a typical or normal variable.
5) The key component to management of FHR patterns is to recognize the presence of changes in the FHR from admission to the hospital or the doctor's office until the time the FHR is currently under evaluation. As exemplified by the Childbirth Injury Prevention Foundation [CIPF] approach [13,14], the questions to be asked are: Has the status of the fetus changed over time, and has the fetal risk of an adverse outcome increased since admission to the hospital? This risk of asphyxia appears to increase with the presence of prolonged FHR decelerations and/or the development of a Hon pattern of intrapartum asphyxia [13,14].

Two important "FHR" patterns

The prolonged FHR deceleration

As previously noted in this chapter, a prolonged FHR deceleration is considered present when there is a visually apparent decrease in the FHR from baseline that is 15 bpm or more, lasting 2 min or longer but less than 10 min [15]. According to the NICHD [15], cases with a "single prolonged FHR deceleration" are placed in Category 2. When the FHR pattern is placed in Category 2, a clinical evaluation is required and a plan [60] is established. Unanswered in the category approach to FHR interpretation is the circumstance of "two or more prolonged FHR decelerations."

Under the circumstance of two or more prolonged FHR decelerations in a term infant, the focus is on the fetal risk for "acute" asphyxia [13]. With this approach, the issue is whether the asphyxia and resultant fetal brain injury were reasonably foreseeable. Foreseeability begs the question as to whether the nurse or physician were on notice of, in this case, a crash in the FHR or a sudden, rapid, and sustained deterioration of the FHR that could potentially last until delivery. In this circumstance, the focus is on the presence, if any, of prolonged FHR decelerations and the risk of a FHR bradycardia. As noted by NICHD [15], a "single prolonged deceleration" places the tracing into Category 2. As such, a Category 2 tracing requires clinical evaluation. If there are two or more prolonged FHR decelerations and there is subsequent crash in the FHR, has the subsequent crash become reasonably foreseeable and hence preventable? In other words, has the FHR with the presence of two or more prolonged FHR decelerations become a Category 3 FHR tracing?

Recording the maternal heart rate: A confounding variable

Since the development of the external fetal monitor, the maternal heart rate (MHR) rather than the FHR has been infrequently picked up by the fetal monitor. When this happens, the features of the MHR intrapartum may prevent proper interpretation of the FHR [61–63]. At the same time, the "true" status of the fetus may go unrecognized for an extended period of time until it is too late to intervene and to potentially prevent fetal brain injury or death. Many clinicians and nurses understand that the electronic fetal monitor can sometimes misrepresent the MHR as the FHR. As such, one has to be mindful of the circumstances in which this MHR/FHR pattern can emerge and recognize it in a timely manner.

Typically, the MHR/FHR pattern occurs in the second stage of labor, with an external fetal monitor, and in the presence of maternal tachycardia [61–63]. During this stage of labor, maternal efforts to deliver the fetus dramatically increase. In response, the MHR escalates to a level of tachycardia (>100 bpm) due to her increased cardiac work brought on by maternal pushing. Under these circumstances, the external fetal monitor picks up the faster MHR and loses or fails to detect the FHR. This tends to happen when the FHR slows for whatever reason and the fetal monitor shifts to the faster MHR. The picking up of the MHR as opposed to the FHR appears to occur more frequently in the second stage of labor and usually during maternal pushing. It is during this time that the woman develops a tachycardia from the work of labor. Almost simultaneously, the MHR accelerates with every push during a contraction. Over time, the baseline

MHR continues to rise in response to the increase in cardiac work. This rise in MHR also provides an indirect indicator of the presence of maternal exhaustion and a justification for the use of operative vaginal delivery to deliver the fetus.

Figure 8.4A illustrates the recording of the apparent "FHR" immediately prior to the delivery of a stillborn infant. As noted in Figure 8.4A, the baseline rate is at or around 160 bpm. After delivery of the stillbirth, the external fetal monitor was left on for a period of time. Figure 8.4B illustrates that the heart rate is at or around 160–170 bpm, in keeping with the pulse rate of the mother postpartum. While the entire tracing is not shown, this case illustrates two things:

1) As illustrated by the MHR at or around 160 bpm, the pregnant woman exerts considerable cardiac work during the second stage of labor.
2) When the MHR becomes tachycardic during the second stage of labor in a woman on an external monitor, the external monitor may pick up the MHR, as it did in this case, rather than the FHR.

When the MHR is picked up by an external fetal monitor during the second stage of labor, the FHR pattern can appear normal or demonstrate a Category 1 FHR tracing. The accelerations observed during this period of time are related to accelerations of the MHR in response to maternal pushing during a contraction. As noted by Sherman and associates [61], "the absence of decelerations in the second stage of labor and marked accelerations coinciding with uterine contractions [does] suggest the MHR rather than the FHR is being recorded [61]. Along these same lines, VanVeen and associates [63] concluded that second-stage tracings that show repetitive accelerations with contractions should be considered MHR until proven otherwise. An example of this FHR pattern is illustrated in Figure 8.5A and 8.5B. Figure 8.5A picks up the labor as the mother is in the second stage of labor. The fetal monitor strip demonstrates evidence of maternal pushing with each contraction and the presence of "accelerations" during those contractions. Figure 8.5B is a continuation of Figure 8.5A. Here, there are accelerations with each contraction and maternal pushing. Throughout this window of time, the fetal monitor strip appears to be a Category 1. In the lower portion of Figure 8.5B, one can see that the fetus was crowning and that the strip ends around 07:45. Two minutes later, the fetus was born at 07:47 with an umbilical artery pH of 6.82 and a base deficit of 17. The child was later diagnosed with spastic

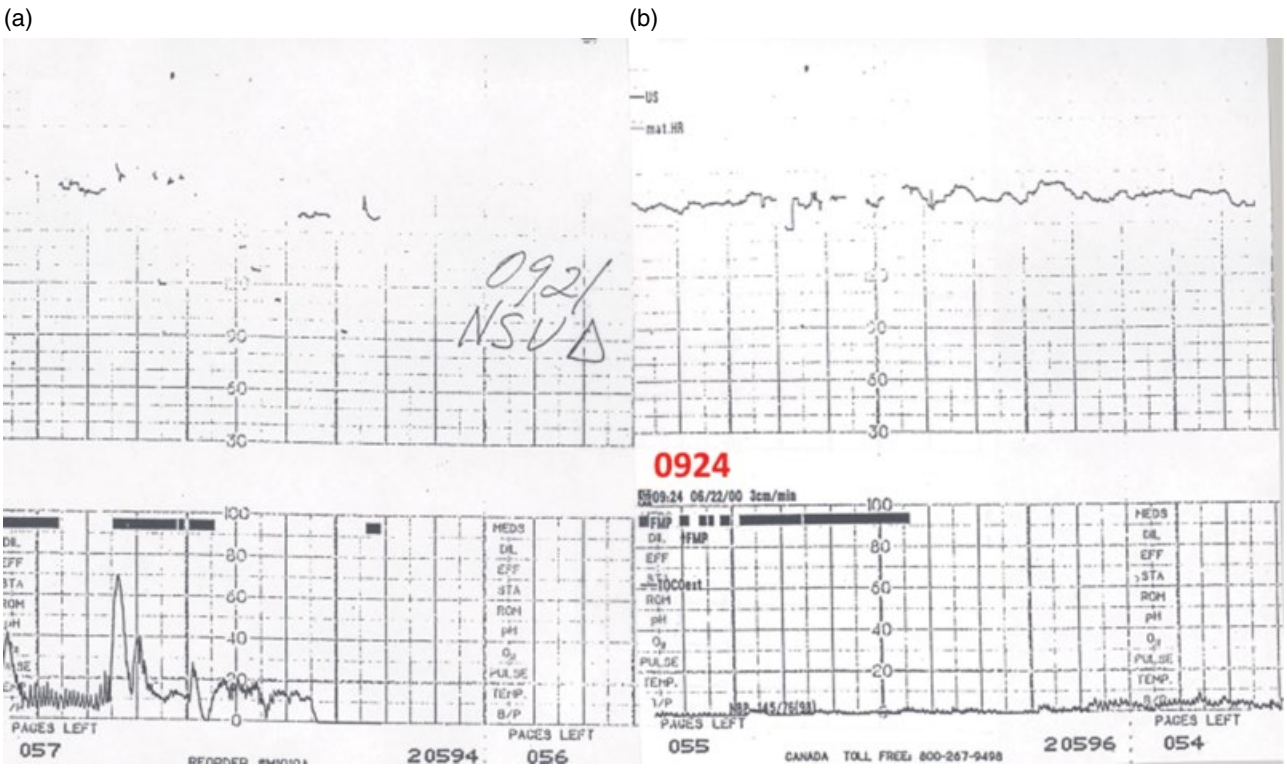
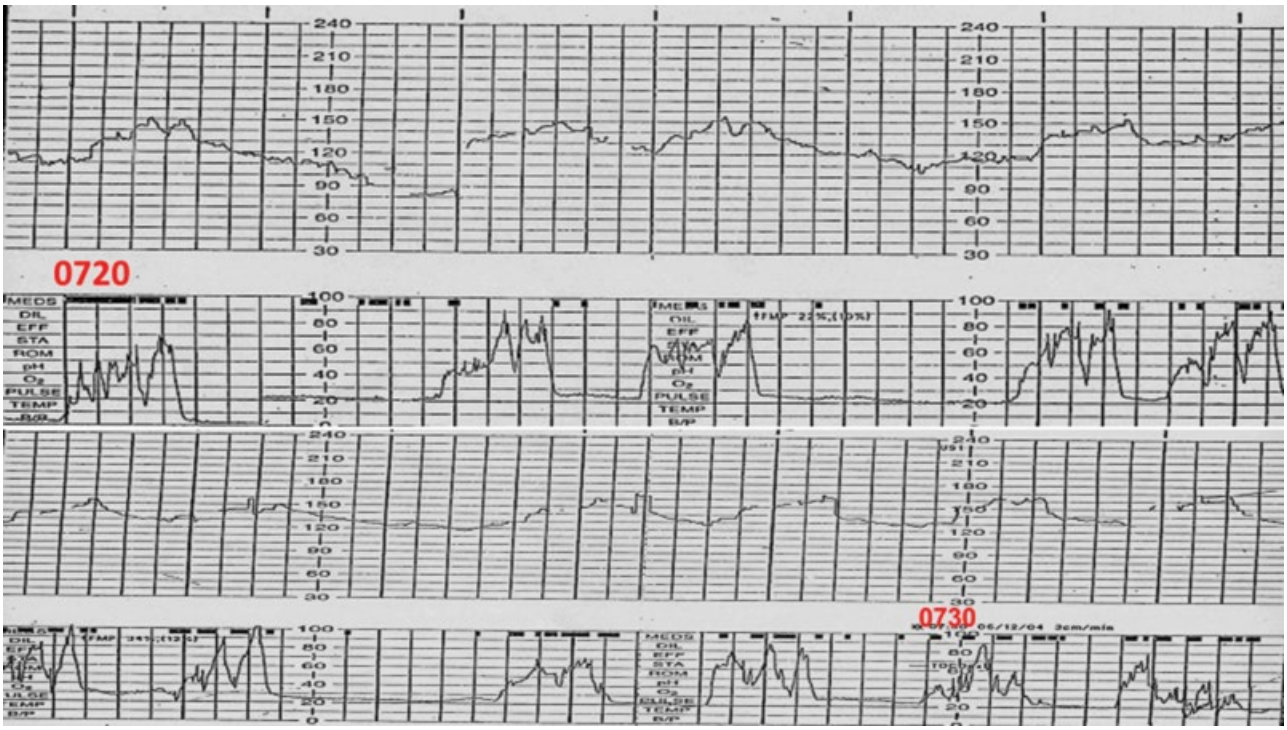


Figure 8.4 (A) A recording of the apparent "FHR" immediately prior to the delivery of a stillborn infant. (B) The heart rate is around 160–170 bpm, in keeping with the pulse rate of the mother postpartum.

(a)



(b)

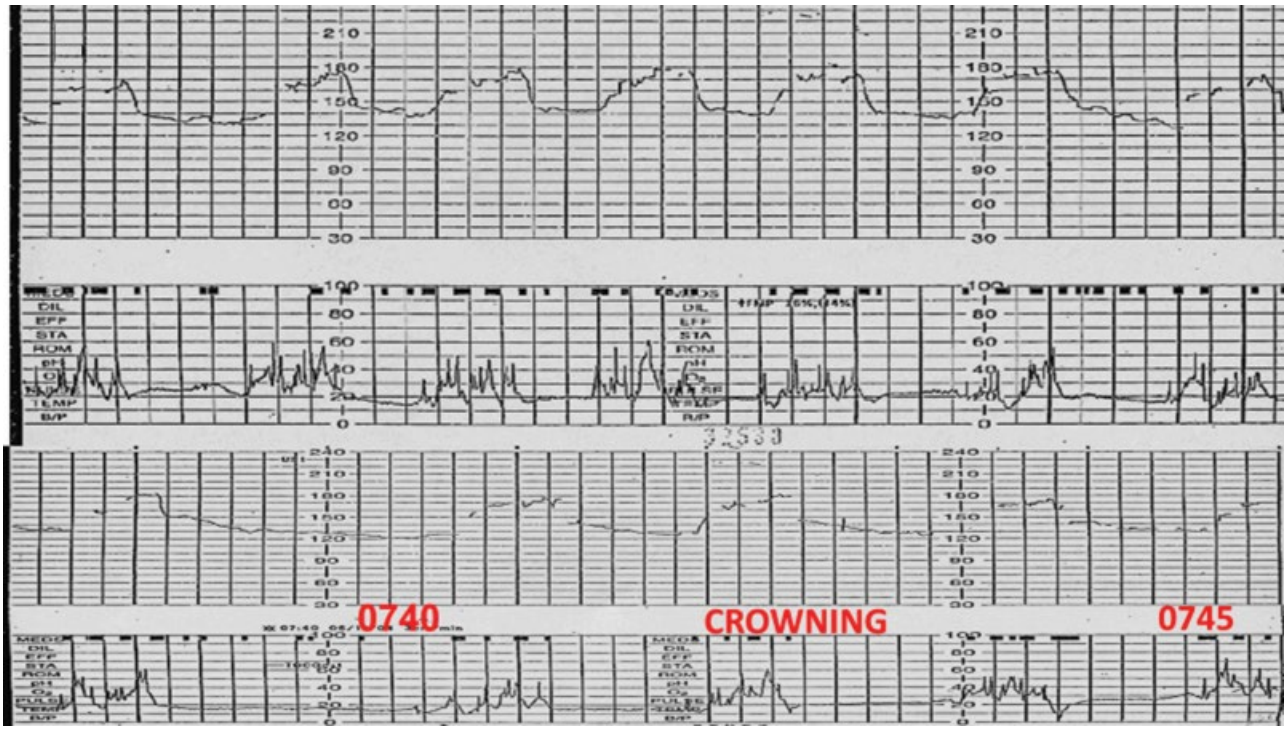


Figure 8.5 (A,B) Second stage of labor.

quadriplegic cerebral palsy. This case illustrates the following:

1) During the second stage of labor, the MHR may be recorded rather than the FHR may be recorded.
2) When the MHE is being recorded, accelerations that coincide with each contraction are usually seen.
3) To potentially avoid MHR/FHR confusion, simultaneous recording of the MHR should be considered. In the absence of the simultaneous recording of the MHR, the MHR should be correlated with the FHR.

Fetal acid–base assessment

Even though many clinicians continue to focus on fetal acid–base status as an indicator of fetal neurologic outcome, fetal acid–base assessment continues to have minimal to no role in the contemporary practice of obstetrics. In the past, fetal acid–base status was thought to be a valuable adjunct for the assessment of fetal health during labor. This practice stemmed from the work of Saling [64]. In that work, Saling found that infants with a pH of less than 7.2 were more likely to be delivered physiologically depressed. Conversely, a normal fetal outcome was more likely to be associated with a non-acidotic fetus (pH ≥7.20) [65]. Even at the peak of its popularity, fetal scalp blood sampling was used in a limited number of pregnancies (~3%) [66]. Notwithstanding, Goodwin and associates [67] concluded in 1994 that fetal scalp blood sampling "has been virtually eliminated without an increase in the cesarean rate for fetal distress or an increase in indicators of perinatal asphyxia. [Its continued role] in clinical practice is questioned."

A profound metabolic acidemia or mixed acidemia at birth, as reflected by an umbilical artery pH of less than 7.00 and a base deficit of 12 or greater, although often a direct result of a sentinel hypoxic event, usually reflects the impact of a slow heart rate (<100 bpm) at the time of birth [68] and seems to be a poor predictor of long-term neurologic impairment [69]. For example, Myers [70] demonstrated that animals whose blood pH was maintained at 7.1 showed no hypoxic brain injury, and that fetuses who had a pH of less than 7.00 could survive several hours before they died. Thus, the initial abnormal pH that surrounds a given birth may not be, in and of itself, indicative of an intrapartum injury [14].

If the clinical circumstances suggest the need for fetal acid–base assessment and the clinician is concerned about fetal status, the clinician should look alternatively for the presence of FHR accelerations. In key studies, Phelan [5], Skupski and colleagues [6], and NICHD [15] have demonstrated (with labor stimulation tests such as scalp or acoustic stimulation) that FHR accelerations were associated with a significantly greater likelihood of normal fetal acid–base status and a favorable fetal outcome. If the fetus fails to respond to sound or scalp stimulation, delivery should be considered.

As with fetal scalp blood sampling, umbilical cord blood gas data do not appear to be useful in predicting long-term neurologic impairment. It is interesting to note that of 314 infants with severe umbilical artery acidosis identified in the world literature, 27 (8.6%) children were subsequently found to have permanent brain damage [69]. In the Fee study [71], for example, minor developmental delays or mild tone abnormalities were noted at the time of hospital discharge in 9 of 110 (8%) singleton term infants. When 108 of these infants were seen on long-term follow-up, all were considered neurologically normal, and none of these infants, which included a neonate with an umbilical artery pH of 6.57 at birth, demonstrated major motor or cognitive abnormality. In contrast, the neonatal outcomes for 113 infants in the Goodwin study [67] were known. Of these, 98 (87%) had normal outcomes. In the remaining 15 infants with known outcomes, five neonates died and 10 infants were brain damaged. Of interest, Dennis and colleagues [72] commented in their series of patients that "the very acidotic children did not perform worse than [the non-acidotic children]. Thus, the finding of severe fetal acidosis on an umbilical artery cord gas does not appear to be linked to subsequent neurologic deficits."

In contrast, the absence of severe acidosis does not ensure a favorable neurologic outcome. For example, Korst and associates [73,74] had previously shown that neonates with sufficient intrapartum asphyxia to produce persistent brain injury did not have to sustain severe acidosis (umbilical arterial pH ≤7.00). When her two studies are combined, 42 (60%) fetuses did not have severe acidosis, and all were neurologically impaired. Of 94 infants with reported permanent brain damage, Dennis and associates [72] also noted that children without acidosis appeared to fare worse than acidotic children. Thus, it appears that factors other than the presence of severe acidosis are probably responsible for fetal brain injury.

It is interesting to note that severe acidosis may not be a proper endpoint to study intrapartum asphyxia [75] or to define whether a fetus has sustained intrapartum brain damage [76–78]. These findings suggest that the pathophysiologic mechanisms responsible for fetal brain damage appear to operate independently of central fetal acid–base status and to be more likely related to the adequacy of cerebral perfusion and the presence of neurocellular acidemia [14].

Severe acidosis, rather than fetal brain damage, continues to be used as an endpoint in the study of intrapartum asphyxia [78] and to define whether a fetus has sustained

intrapartum brain damage [76–78]. This alleged clinical relationship remains a puzzlement when you consider that "there is no pH value that separates cleanly those babies who have experienced intrapartum injury from those who have not – no prognosis can be made or refuted on the basis of a single laboratory study" [16]. The lack of a consistent relationship between the presence or absence of fetal acidosis suggests that the pathophysiologic mechanisms that are responsible for fetal brain damage seem more likely to be related to the adequacy of cerebral perfusion [14] in that fetus rather than the mere presence of metabolic acidosis. Thus, as has happened with fetal scalp blood sampling, the use of umbilical cord blood gases to define or time fetal brain damage or the quality of care may not have a role in the contemporary or future practice of obstetrics.

FHR patterns in the brain-damaged infant

Term infants found to be brain damaged do not manifest a uniform FHR pattern [11–14,52]. However, these fetuses do manifest distinct FHR patterns intrapartum that can be easily categorized and identified based on the admission FHR pattern and subsequent changes in the baseline rate.

Reactive admission test and subsequent fetal brain damage

When a pregnant woman is admitted to hospital, the overwhelming number of obstetric patients will have a reactive or Category 1 FHR pattern. Of these, more than 98% will go through labor uneventfully, and most will deliver vaginally. In the few patients (typically 1–2%) that develop intrapartum "fetal distress" [79,80], the characteristic "fetal distress" is usually, but not always, acute, usually precipitated by a sentinel hypoxic event and manifested by a sudden, rapid, and sustained deterioration of the FHR that is unresponsive to remedial measures and/or terbutaline and lasts until delivery. Of these, an even smaller number of fetuses will ultimately experience a central nervous system (CNS) injury. So, while unusual, fetal brain injury in the fetus with a reactive fetal admission test may arise, in the absence of trauma, as a result of a sudden, rapid, and sustained deterioration of the FHR or a Hon pattern of intrapartum asphyxia.

Acute fetal brain injury

In this group (Table 8.1), the FHR pattern is reactive or Category 1 on admission. This FHR pattern may be followed by a sudden, rapid, and sustained deterioration of the FHR or bradycardia that lasts until the time of delivery. In the circumstances of a bradycardia, there is typically a sentinel hypoxic event associated with it. If the sentinel hypoxic event is associated with an abruption and/or a uterine rupture, the resultant bradycardia is usually unresponsive to remedial measures and/or subcutaneous or intravenous terbutaline. In contrast, the presence of a bradycardia unrelated to an abruption or uterine rupture will usually respond to terbutaline therapy. As with rapid delivery of the fetus in this situation, the goal of terbutaline is to resuscitate the fetus to avoid any brain injury.

As many of you know, the window to fetal brain injury is relative short in this situation, and the window to brain injury is no different than if one of us coded. For example, a fetus who has a sudden, rapid, and sustained deterioration of the FHR or bradycardia that is unresponsive to remedial measures and/or terbutaline and lasts for a prolonged period of time typically has an injury to the basal ganglia or the deep gray matter. Injury to this area of the brain, the deep gray matter, such as the basal ganglia, gives rise to athetoid or dyskinetic cerebral palsy [14,81]. In this circumstance, the fetal brain injury is the result of a sudden reduction of fetal cardiac output and blood pressure or "cerebral hypotension due to an ineffective or non-functional cardiac pump." That is not to say that the fetus cannot have injury to both the deep gray matter and the cerebral hemisphere, or white matter with this specific FHR pattern. Whether both areas of the fetal brain are affected often depends on the five factors illustrated in Table 8.4. Fetal brain injuries that arise from this FHR pattern are associated with an array of hypoxic sentinel events (Table 8.2), such as uterine rupture, placental abruption, and cord prolapse. Given the acute nature of this FHR pattern, limited time is available to preserve normal brain function.

Timing of fetal neurologic injury in this specific FHR group is a function of multiple factors (Table 8.4). Each variable plays a role in determining the length of time required to sustain fetal brain damage. For example, the admission FHR pattern provides an indicator of fetal status before the catastrophic event. If, for example, the

Table 8.4 Five factors useful in determining the susceptibility of a fetus to fetal brain injury under the circumstances of a sudden, rapid, and sustained deterioration of the fetal heart rate (FHR) from a previously reactive FHR.

Prior FHR pattern
Fetal growth pattern
Degree of intrafetal shunting
Duration of the FHR deceleration
Intactness of the placenta

Source: Refs. [13].

FHR pattern is reactive with a normal baseline rate or a Category 1 FHR tracing and a sudden prolonged FHR deceleration occurs, the window to fetal brain injury will be longer than in the patient with a tachycardic baseline [82]. As with the baseline rate, the other variables also play a role. However, it is not within the scope of this chapter to detail this information. The reader is referred to the work of Phelan and associates [14]. In general, our experience [11–14] would suggest an even shorter time to neurologic injury of less than 16 min whenever the placenta has completely separated. If the placenta remains intact, a longer period of time appears to be available before the onset of CNS injury. Thus, the intactness of the placenta plays an important role in determining long-term fetal outcome.

Hon pattern of asphyxia

The Hon pattern of intrapartum asphyxia (Figures 8.1–8.3) is uniquely different because the asphyxia evolves over a longer period of time [11–14,52]. This FHR pattern begins with a reactive FHR pattern or a Category 1 FHR tracing on admission to the hospital. Subsequently during labor, the fetus develops a nonreactive FHR pattern or loses its ability to accelerate its heart rate [11–14,37]. As the labor continues, a rise in the baseline FHR to a level of tachycardia develops in association with a reduction in FHR variability. If uncorrected, a substantial rise in baseline heart rate from admission (135 ± 10 bpm) to a mean maximum (186 ± 15 bpm) baseline heart rate is seen [11]. The maximum FHR ranged from 155 bpm to 220 bpm. This constituted a 39 ± 13% mean percentage rise in baseline heart rate from admission and ranged from 17 to 82% [11]. This rise in baseline FHR is usually not accompanied by maternal pyrexia. When a substantial rise in baseline FHR is encountered, the FHR pattern is also associated with repetitive FHR decelerations but not necessarily late decelerations and usually a change to minimal FHR variability [11–14,52]. If the condition remains uncorrected, the variability becomes absent. "If labor continues to progress and the fetus nears death, the slopes become progressively less steep until the FHR does not return to its baseline rate and ultimately terminates in a profound bradycardia" [83] or a "stairsteps-to-death" (or heaven) FHR pattern [11,12].

Once a FHR tachycardia begins in association with the fetal inability to accelerate its heart rate at least 15 bpm for 15 s from the time the FHR leaves baseline until it returns, repetitive FHR decelerations, and usually a loss of FHR variability, the subsequent FHR pattern [11] does one of the following: (1) the FHR pattern remains tachycardic and/or continues to rise until the fetus is delivered; (2) the fetus develops a sudden, rapid, and sustained deterioration of the FHR that lasts until delivery; or (3) the fetus initiates a stairsteps-to-death pattern or a progressive bradycardia. Of particular clinical relevance is that all patients manifested a substantial rise in their baseline heart rates, lost their ability to generate FHR accelerations, became nonreactive, and exhibited repetitive FHR decelerations. Of note, the repetitive FHR decelerations were not necessarily late decelerations and were frequently variable decelerations [11–13,78].

In the Hon FHR group, FHR variability appeared to be a predictor of neonatal cerebral edema [11]. For example, many brain-damaged fetuses exhibited average FHR variability at the time of their deliveries [11]. In the neonatal period, brain-damaged fetuses that had the Hon pattern of intrapartum asphyxia with average FHR variability had significantly less cerebral edema [26]. Kim's cerebral edema [26] findings suggest that the use of "diminished," which included those fetuses with minimal and absent FHR variability, as an endpoint for the Hon pattern of intrapartum asphyxia to decide the timing of operative intervention is probably unreasonable. This means that the fetal brain may well be injured before the loss of FHR variability.

The Hon pattern characteristically results in damage to both cerebral hemispheres and gives rise to spastic quadriplegia [14,81]. Here, the mechanism for injury is not an ineffective pump, because these fetuses usually demonstrate a tachycardic baseline heart rates and the pump is working, albeit at a higher rate. The brain damage in this situation relates more to cerebral ischemia (Figure 8.6). The triggering mechanism may be meconium [84,85] or infection [86,87] that may be bacterial, anaerobic or aerobic, or viral [88,89], but is not related to uterine contractions [14]. The resultant fetal vasoconstriction or intrafetal shunting probably reflects the fetal efforts to maintain blood pressure and/or enhance fetal cerebral blood flow. Nevertheless, once the fetus develops ischemia or is unable to perfuse its brain cells, neurocellular hypoxia or injury occurs. Thus, the hypoxia encountered in the fetus is at the cellular level and not yet at the central or systemic level. By the time the fetus develops systemic or central hypoxia, the fetus, in our

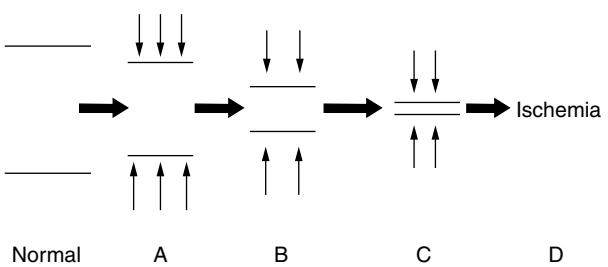


Figure 8.6 Persistent fetal vasoconstriction over time or intrafetal shunting leads to progressive narrowing of the fetal vascular tree, leading ultimately to ischemia.

opinion, has already been brain injured and is probably near death [12,14]. Thus, cerebral perfusion deficits due to intrafetal and intracerebral shunting rather than fetal systemic hypoxia are most likely responsible for the fetal brain injury [90].

This means, for example, that a fetus that develops the Hon pattern of intrapartum asphyxia would appear to move to ischemia or from point C to point D (Figure 8.6). During this transition, a progressive and substantial rise in FHR is observed in an effort to preserve cerebral perfusion and neurocellular oxygenation. During this period, fetal systemic oxygenation and oxygen saturation are maintained. In our opinion [11], only after progressive and prolonged ischemia and brain injury do central fetal oxygen saturations begin to fall. These observations would be in keeping with American College of Obstetricians and Gynecologists (ACOG) recommendations on the intrapartum management of a Category 2 FHR tracing with the presence of FHR tachycardia, minimal variability, and no accelerations. Under these circumstances, one cannot reliably exclude fetal academia [60].

Additionally, it is important to emphasize that the pattern of fetal brain injury may change depending on the circumstances that gave rise to the delivery of the fetus. For example, and as previously discussed, this FHR pattern characteristically results in cerebral palsy of the spastic quadriplegic type due to cerebral hemispheric injury. If, however, the FHR pattern moves from a Hon pattern followed by a sudden, rapid, and sustained deterioration of the FHR that lasts until delivery, the pattern of brain damage becomes more global and involves not only the cerebral hemispheres but also the deep gray matter. As such, the fetuses with this latter FHR pattern have a more severe injury and shorter life expectancies.

The persistent nonreactive FHR pattern

The persistent nonreactive FHR pattern from admission to the hospital or a non-stress test accounted for 45% of the FHR patterns observed in a population of 300 brain-damaged babies [11] and 33% of an updated population of 423 singleton term brain-damaged children [13,14]. This population is typically, but not always, characterized by the presence of reduced fetal activity before admission to the hospital, male fetuses, old meconium, meconium sequelae such as meconium aspiration syndrome and persistent pulmonary hypertension, and oligohydramnios [90]. Along with these observations, these fetuses usually but not always have elevated nucleated red blood cell counts [91,92], prolonged NRBC clearance times [91], low initial platelet counts [93], significant multi-organ system dysfunction [73,74,91], delayed onset of seizures from birth [94,95], and cortical or hemispheric brain injuries [13,14]. The typical FHR pattern is nonreactive with a fixed baseline rate that normally does not change from admission until delivery [13,14] in association with diminished or average variability.

When looking at the admission FHR pattern, the persistent nonreactive FHR pattern group can be divided into three phases. These three phases, in our opinion, represent a post-CNS insult compensatory response in the fetus. Moreover, this FHR pattern, in our opinion, does not represent ongoing asphyxia or worsening of the CNS injury [11–14]. For a fetus to have ongoing fetal asphyxia, a FHR pattern similar to the Hon pattern of intrapartum asphyxia would have to be seen. There, a progressive and substantial rise in baseline heart rate in association with repetitive FHR decelerations is observed in response to ongoing fetal asphyxia (Figures 8.1–8.3). In contrast, the FHR baseline in the nonreactive group usually but not always remains fixed. Infrequently, a FHR tachycardia is seen; however, the rise in baseline rate is usually insubstantial. Thus, the phase of recovery appears to equate with the length of time from the fetal CNS insult. Thus, phase I would appear to be closer to the time of the insult, and phase III would appear to be more distant in time from the injury-producing event [12].

The persistent nonreactive FHR pattern is not, in our opinion, a sign of ongoing fetal asphyxia but rather represents a static encephalopathy [11–14]. This means that earlier intervention in the form of a cesarean on admission to the hospital would not, in our opinion, substantially alter fetal outcome.

Fetal monitoring made simple during labor

In light of the lessons learned from the children damaged in utero before and during labor, current fetal monitoring interpretation will need to change to reflect and include the significance of the initial fetal monitoring period. When a patient presents to labor and delivery, the initial fetal assessment should include an initial fetal monitoring period to assess reactivity (the presence of FHR accelerations) and to ascertain from the patient the quality and quantity of fetal movement. In the patient with a reactive FHR pattern and normal fetal movement, the key to clinical management before and during labor is to follow the baseline fetal heart rate.

This means that the physician and nurse will need to watch for persistent elevations of the baseline rate to a level of tachycardia or higher or look for the potential for the baseline rate to fall suddenly. To assist with the identification of the Hon pattern, medical and nursing personnel should try to compare the current tracing with

the one obtained on admission. If the characteristics of the Hon pattern of intrapartum asphyxia develop, subsequent clinical management will depend on whether the gravida is febrile and as outlined earlier in this chapter. In the nonreactive group, clinical management is to first evaluate the maternal and fetal status with respect to the etiology of the FHR pattern. These causes include, but are not limited to, the following: maternal substance abuse, fetal-maternal hemorrhage, fetal anomaly, and the potential for a fetal chromosomal abnormality. During this period of maternal and fetal evaluation, continuous fetal monitoring is used, if technically feasible, to assess fetal status. In addition, fetal stimulation tests, a contraction stress test, or a biophysical profile may be used to further determine fetal status. Once fetal status is clarified in the nonreactive group, the subsequent management with respect to the route of delivery in the term or near-term pregnancy will depend on the discussion with the family and the clinical findings.

Maternal and surgical conditions

Anaphylaxis

Anaphylaxis is an acute allergic reaction to food ingestion or drugs. The reported incidence of anaphylaxis during pregnancy has been reported to be 3–5 cases per 10,000 births [58]. It is generally associated with rapid onset of pruritus and urticaria and may result in respiratory distress, edema, vascular collapse, and shock. Anaphylaxis during pregnancy has been linked with the use of misoprostol [96]; after laminaria insertion; the administration of antibiotics, such as ampicillin, cefazolin, penicillin, iron, ranitidine, and snake antivenom; insect stings, primarily bees and wasps; local anesthetics; and general anesthesia [58]. When anaphylaxis occurs during pregnancy, the typical fetal heart rate response is repetitive late deceleration [57] or a bradycardia [58].

When an anaphylactic reaction occurs during pregnancy, the accompanying maternal physiologic changes may result in the manifestation of fetal distress, as noted earlier in this chapter. For example, in a case described by Klein and associates [57], a woman at 29 weeks' gestation presented with an acute allergic reaction after eating shellfish. On admission, she had evidence of regular uterine contractions and repetitive, severe late decelerations. The "fetal distress" was believed to be the result of maternal hypotension and relative hypovolemia, which accompanied the allergic reaction. Prompt treatment of the patient with intravenous fluids and ephedrine corrected the FHR abnormality. Subsequently, the patient delivered a healthy male infant at term with normal Apgar scores.

As suggested by these investigators, acute maternal allergic reactions can pose a threat to the fetus, and treatment directed at the underlying cause often remedies the accompanying fetal distress. As such, treatment is directed toward maternal cardiorespiratory support with the goal of maternal stabilization. Maternal stability should be re-evaluated in the presence of a persistent fetal heart rate tachycardia or bradycardia, or other abnormal FHR patterns. The persistence of these FHR patterns suggests the need for additional maternal hemodynamic support or oxygenation. This means that intervention is delayed until the mother is sufficiently stabilized to be able to withstand a cesarean. Although Schoen [96] provides a clinical algorithm for the acute management of anaphylaxis during pregnancy, Schoen suggests that "if there is not rapid improvement in the maternal clinical condition, the [clinician should] move to immediate delivery" [96]. According to Gei [58], "poor neonatal outcomes have been experienced after an emergency cesarean prior to maternal stabilization." The issue comes down to Schoen's clinical meaning of "not rapid improvement." As discussed later in this chapter, the cornerstone of the clinical management of these complicated clinical conditions is that maternal health trumps fetal health.

Once maternal stabilization has been achieved, the clinical focus should be shifted toward the status of the fetus. Generally, to afford the fetus a wider margin of safety, efforts should be directed at maintaining maternal systolic BP above 90 mmHg. In addition, oxygen should be administered to correct maternal hypoxia; in the absence of maternal hypovolemia, a maternal P_aO_2 in excess of 60–70 mmHg will assure adequate fetal oxygenation [57,58].

In summary, the obstetrician should be prepared for anaphylaxis in the office as well as the hospital setting [97] and incorporate this type of emergency into their obstetrical drills [98]

Eclampsia

Maternal seizures are a well-known but infrequent sequel of preeclampsia [19]. Although the maternal hemodynamic findings in patients with eclampsia are similar to those with severe preeclampsia [99], maternal convulsions require prompt attention to potentially prevent harm to both mother and fetus [19]. During a seizure, the fetal response usually is manifested as an abrupt, prolonged FHR deceleration [21,100]. During the seizure, which generally lasts less than 1–2 min [21], transient maternal hypoxia and uterine artery vasospasm occur and combine to produce a decline in uterine blood flow. In addition, uterine activity increases secondary to the release of norepinephrine, resulting in

additional reduction in uteroplacental perfusion. Ultimately, the reduction of uteroplacental perfusion causes the FHR deceleration. Such a deceleration may last up to 10 min after the termination of the convulsions and the correction of maternal hypoxemia [19,21]. Following the seizure and recovery from the FHR deceleration, a loss of FHRV and a compensatory rise in baseline FHR are characteristically seen. Transient late decelerations are not uncommon but usually resolve once maternal metabolic recovery is complete. During this recovery period, it is reasonably believed to be beneficial for the fetus to permit recovery in utero from convulsion induced hypoxia and hypercarbia [19]. During this time, the patient should not be rushed to an emergency cesarean based on the FHR changes associated with an eclamptic seizure [19]. This is especially true if the patient is unstable.

The cornerstone of patient management during an eclamptic seizure is to maintain adequate maternal oxygenation and to administer appropriate anticonvulsants. After a convulsion occurs, an adequate airway should be maintained and oxygen administered. To optimize uteroplacental perfusion, the mother is repositioned onto her side. Anticonvulsant therapy with intravenous magnesium sulfate [19,101–103] to prevent seizure recurrence is recommended. In spite of adequate magnesium sulfate therapy, adjunctive anticonvulsant therapy occasionally may be necessary in about 10% of patients [19,21,101].

In the event of persistent FHR decelerations, intrauterine resuscitation with a betamimetic [104] or additional magnesium sulfate [105] may be helpful in relieving eclampsia-induced uterine hypertonus. Continuous electronic fetal monitoring should be used to follow the fetal condition. After the mother has been stabilized, and if the fetus continues to show signs of a FHR bradycardia and/or repetitive late decelerations after a reasonable period of recovery, delivery should be considered.

Disseminated intravascular coagulopathy

Disseminated intravascular coagulopathy (DIC) occurs in a variety of obstetric conditions, such as abruptio placentae, amniotic fluid embolus syndrome, severe preeclampsia and eclampsia, and the dead fetus syndrome. The pathophysiology of this condition is discussed in greater detail in Chapter 31.

Infrequently, DIC may be advanced to a point of overt bleeding [106]. Under these circumstances, laboratory abnormalities accompany the clinical evidence of consumptive coagulopathy. In the rare circumstance of overt "fetal distress" and a clinically apparent maternal coagulopathy, obstetric management requires prompt replacement of deficient coagulation components before attempting to deliver the distressed fetus. This frequently requires balancing the interests of the pregnant woman with those of her unborn child.

For example, a 34-year-old woman presented to the hospital at 33 weeks' gestation with the FHR tracing illustrated in Figure 8.7. Real-time sonography demonstrated asymmetric intrauterine growth restriction. Oxygen was administered, and the patient was repositioned on her left side. Appropriate laboratory studies were drawn, and informed consent for a cesarean was obtained. When a Foley catheter was inserted, grossly bloody urine was observed. The previously drawn blood did not clot, and she was observed to be bleeding from the site of her intravenous line. The abnormal FHR pattern persisted.

In this circumstance, the interests of the mother and fetus are at odds with one another, and a difficult clinical decision must now be made. Whose interest does the obstetrician protect in this instance? Immediate surgical intervention without blood products would have lessened the mother's chances of survival. On the other hand, if the clinician waits for fresh frozen plasma and platelet infusion before undertaking surgery, the fetus will be at significant risk of death or permanent neurologic impairment. Ideally, the mother and/or her family should participate in such decisions. In reality, because of the unpredictable nature of these dilemmas and the need for rapid decision making, family involvement is not always possible. Under such circumstances, it is axiomatic that maternal interests take precedence over those of the fetus.

Because blood products were not readily available, the decision was made to stabilize the mother and to move the patient to the operating room. Once in the operating room, the clinical management would include, but is not limited to, the following: to continue to oxygenate the mother; to maintain her in the left lateral recumbent position; to have an anesthesiologist, operating room personnel, and surgeons present; and to be prepared to operate. As soon as the blood products are available, and the fetus is alive, transfuse with appropriate blood products. Then, the clinician should begin the cesarean under general anesthesia. In this case, maternal and fetal outcomes were ultimately favorable.

In summary, the cornerstone of management of the patient with full-blown DIC and clinically apparent fetal distress is to stabilize the mother by correcting the maternal clotting abnormality before initiating surgery. While waiting for the blood products to be infused, the patient should be prepared and ready for immediate cesarean delivery. If the fetus dies in the interim, the cesarean should not be performed, and the patient should be afforded the opportunity to deliver vaginally, to reduce maternal hemorrhagic risks.

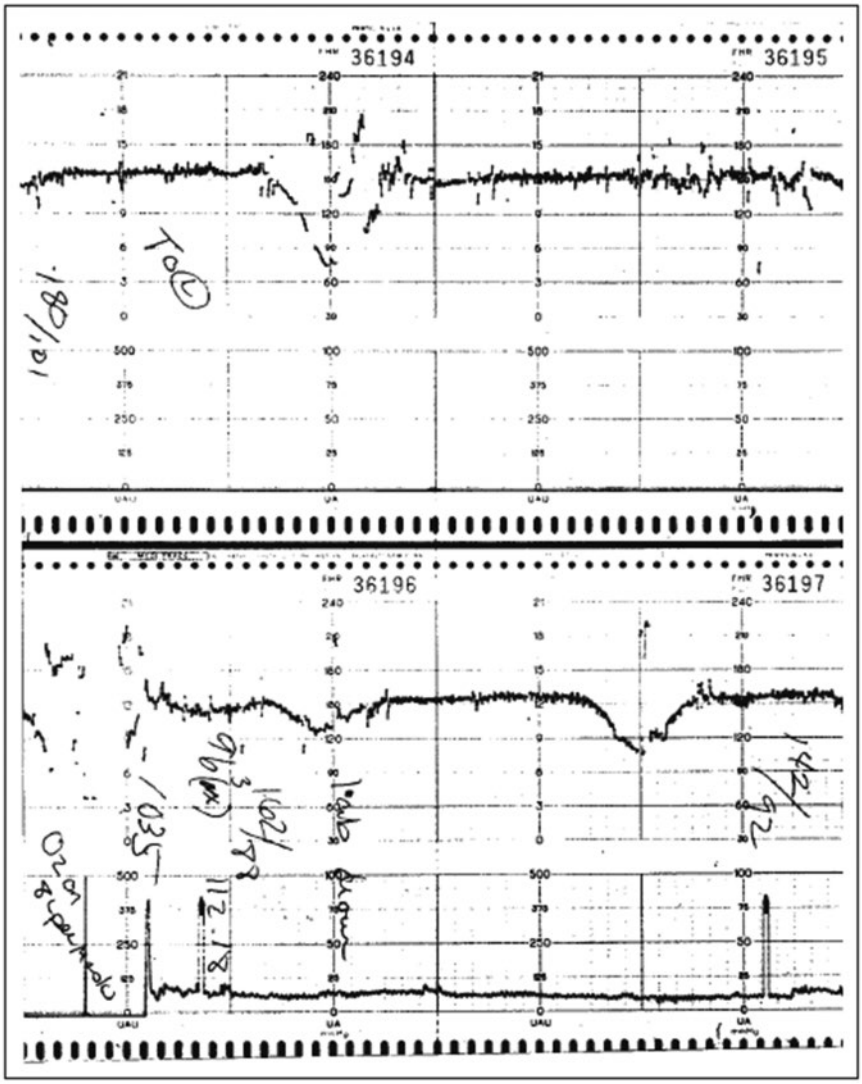


Figure 8.7 The FHR pattern from a 33-week fetus with asymmetric intrauterine growth impairment whose mother presented with clinical disseminated intravascular coagulation.

The burn victim

Although burn victims are uncommonly encountered in high-risk obstetric units, the pregnant burn patient is sufficiently complex to require a team approach to enhance maternal and perinatal survival [107,108]. In most cases, this will require maternal–fetal transfer to a facility skilled to handle burn patients. Transfer will depend primarily on the severity of the burn and the stability of the pregnant woman and her fetus. For greater detail and discussion on the clinical management of various types of thermal injuries, the reader is referred to Chapter 53.

As an overview, the first step in the management of the pregnant burn patient is to determine the depth and size of the burn. The depth of a burn may be partial or full thickness. A full-thickness burn, formerly called a third-degree burn, is the most severe and involves total destruction of the skin. As a result, regeneration of the epithelial surface is not technically feasible.

The second element of burn management is to determine the percentage of body surface area involved (Table 8.5). The percentage of maternal total body surface area covered by the burn is linked to maternal and perinatal outcome. The more severe the maternal burn, the higher the maternal and perinatal mortality is [107,108]. The risk of mortality becomes significant whenever 40% or more of the maternal total body surface area is burned [107].

The subsequent clinical management of the pregnant burn patient will depend on the patient's burn phase (e.g., acute, convalescent, or remote) or burn period [109] (e.g., resuscitation, postresuscitation, inflammation/infection,

Table 8.5 Classification of burn patients based on the percentage of body surface area involved.

Classification	Body surface area (%)
Minor	<10
Major	
Moderate	10–19
Severe	20–39
Critical	≥40

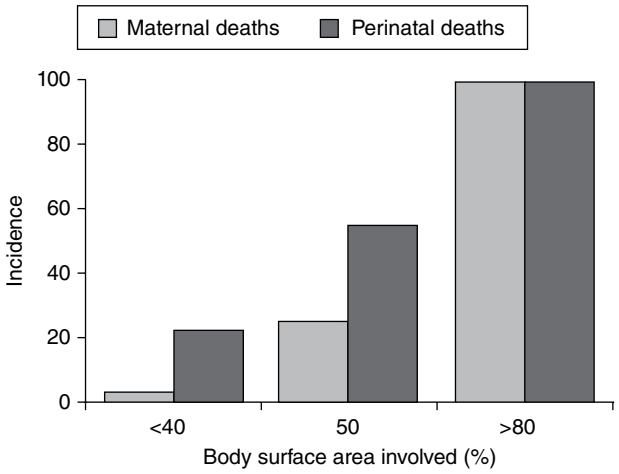


Figure 8.8 Estimated maternal and perinatal mortality rates following maternal burn injuries according to the amount of body surface area involved.

or rehabilitation). Each phase has unique problems. For example, the acute phase is characterized by premature labor, electrolyte and fluid disturbances, maternal cardiopulmonary instability, and the potential for fetal compromise. In contrast, the convalescent and remote periods are unique for their problems of sepsis and abdominal scarring, respectively. Because the potential for fetal compromise is greatest during the window of time immediately following the burn, the focus in this chapter is on acute-phase burn patients.

In the acute phase of a severe burn, the primary maternal focus centers on stabilization [108]. Here, electrolyte disturbances due to transudation of fluid and altered renal function mandate close attention to the maternal intravascular volume and prompt and aggressive fluid resuscitation. To estimate fluid resuscitation requirements, multiple formulas are available and include but are not limited to the following formulas: Evans, Third Military Medical University, and Ruijins. The latter two formulas are used primarily in China and other Asian countries. At the same time, these patients are also potentially compromised from airway injury and/or smoke inhalation, and ventilator support may be necessary to maintain cardiopulmonary stability. Additionally, a high index of suspicion for venous thrombosis and sepsis with early and aggressive treatment should be considered. Given the complexities of these patients, invasive hemodynamic monitoring may be necessary. Because most of these patients will be in an ICU, appropriate medical consultation and intensive nursing care for the mother and fetus are essential.

Assessing fetal well-being in the burn patient may be difficult. The ability to determine fetal status with ultrasound or fetal monitoring will depend on the size and location of the burn. If, for example, the burn involves the maternal abdominal wall, alternative methods of fetal assessment, such as fetal kick counts (alone or in response to acoustic stimulation) [28] or a modified BPP [18,43,44] using vaginal ultrasound, may be necessary. Whenever abdominal burns are present, a sterile transducer cover for the ultrasound device, fetal monitor, or doptone should be used to reduce the risk of infection. In the absence of a maternal abdominal burn, continuous electronic fetal monitoring can generally be used. Because of such monitoring difficulties and the direct relationship between the size of the maternal burn and perinatal outcome (see Figure 8.8), Matthews [110] and Shi [107] have recommended immediate cesarean delivery (assuming maternal stability) in any pregnant burn patient with a potentially viable fetus and a burn that involves 50% or more of the maternal body surface area. In contrast, Guo [108] recommends early delivery if the pregnancy is in the third trimester. As a reminder, burn patients with electrolyte disturbances may exhibit alterations in fetal status similar to those of a patient in sickle cell crisis [56] or diabetic ketoacidosis [54,55]. Once the maternal electrolyte disturbance is corrected, fetal status may return to normal and intervention often can be avoided.

Fetal considerations specific to cardiac bypass procedures and electrical shock are discussed in Chapters 17 and 53.

Maternal brain death or persistent vegetative state

With the advent of artificial life-support systems, prolonged viability of the brain-dead pregnant woman [111–122] or one in a persistent vegetative state (PVS) [123–137] is no longer unusual in a perinatal unit. As a consequence, an increasing number of obstetric patients on artificial life support will be encountered in the medical community. In addition to maternal somatic survival to prolong fetal life, the recent clinical focus has also been on the role of the brain-dead gravida as a potential organ donor [138–140]. "Where one life ends and another begins" has been captured in the articles by Ecker [139] and Esmaeilzadeh [140]. As one can readily see, maternal brain death and persistent vegetative state

pose an array of medical, legal, and ethical dilemmas for the obstetric healthcare provider [118,136,141–145].

In each case of maternal brain death or PVS, multiple questions need to be addressed depending on the role, if any, of continued somatic survival. When first confronted by the clinical circumstances of confirmed maternal brain death or PVS, the focus shifts to that of the fetus. If the fetus is alive, the question arises as to whether extraordinary care for the brain-dead patient should be initiated to preserve the life of her unborn child, and if so, at what gestational age? If artificial life support is elected to permit further maturation of the fetus, how should the pregnancy be managed, and when and under what circumstances should the fetus be delivered? When should maternal life support be terminated? Is consent required to maintain the pregnancy? If so, from whom should consent be obtained? Such questions barely touch the surface of the complexities associated with these cases. But it is clearly not within the scope of this chapter to deal with the ethical, moral, and legal issues related to the obstetric care of the brain-dead gravida or the gravida with PVS. Rather, the emphasis is on the clinical management of these patients when a decision has been made to maintain somatic support for the benefit of the unborn child.

The key distinction between brain death and PVS is that in PVS, the brainstem is usually but not always functioning normally. In the initial phases, it is arguably difficult to separate the two entities. With time, the distinction becomes clearer. For example, a PVS patient could appear to be awake, be capable of swallowing, and have normal respiratory control, but have no purposeful interactions. PVS patients are "truly unconscious because, although they are wakeful, they lack awareness" [136]. Nevertheless, the clinical management of the brain-dead or PVS gravida is similar initially.

To date, more than 13 cases of maternal brain death [111–122] and more than 17 cases of PVS [123–137] during pregnancy have been reported (Tables 8.6 and 8.7). In this update, recent cases have not been added to the tables or references unless the case was clinically necessary because the care of these complicated pregnancies has changed little since the fifth edition. In general, PVS patients require less somatic support than do brain-dead pregnant women but can require a similar degree of medical management. The review by Bush and associates [136] illustrates the key differences between these two groups. When compared with the brain-dead group, the PVS population is more likely to demonstrate the following [136]:

Table 8.6 Perinatal outcome in 13 reported cases of maternal brain death during pregnancy.

Gestation age (weeks)							
Reference	**Year**	**Brain death**	**Delivery**	**Indication for delivery**	**Mode of delivery**	**Apgar score at 5 min**	**Birth Weight (grams)**
Dillon 1 [116]	1982	25	26	Fetal distress	Cesarean	8	850
Dillon 2	1982	18	19	Life support Terminated	SVD	NA	NA
Heikkinen [117]	1985	21	31	Maternal hypotension	Cesarean	7	1600
Field [118]	1988	22	31	Growth impaired Maternal sepsis	Cesarean	8	1440
Bernstein [119]	1989	15	32	Fetal distress	Cesarean	9	1555
Wuermeling [120]	1994	14	NA	NA	SVD	NA	NA
Iriye [121]	1995	30	30	Maternal hypotension FHR decelerations	Cesarean	8	1610
Vives [122]	1996	27	27	Fetal distress Maternal Hypotension	Cesarean	10	1150
Catanzarite [123]	1997	25	29	Chorioamnionitis	Cesarean	7	1315
Lewis [124]	1997	25	31	Fetal Lung Maturity	Cesarean	NA	NA
Spike [125]	1999	16	31	Maternal Hypotension	Cesarean	8	1440
Souza [126]	2006	25	28	Oligohydramnios Growth Impaired	Cesarean	10	815
Hussein [12]	2006	26	28	Oligohydramnios	Cesarean	NA	1285

FHR, Fetal heart rate; NA, not available; SVD, spontaneous vaginal delivery.

Table 8.7 Perinatal outcome in 17 reported cases of persistent vegetative state during pregnancy.

Gestation age (weeks)							
Reference	**Year**	**PVS**	**Delivery**	**Indication for delivery**	**Mode of delivery**	**Apgar score at 5 min**	**Birth Weight (grams)**
Lucas [123]	1976	6 mo	8 mo	None	SVD-breech	NA	1760
Sampson [124]	1979	6	34	Premature labor	Forceps	5	1640
BenAderet [125]	1984	17	35	Premature rupture of membranes	Cesarean	9	2450
Hill [126]	1985	14	34	Fetal lung maturity	Cesarean	9	1600
Diamond [127]	1986	22	34	Contraction stress test	Cesarean	5	2835
Landye [128]	1987	5 mo	37		Vacuum	9	2530
Koh [129]	1993	13	37	Failed VBAC	Cesarean	9	3680
Webb [130]	1996	14	31	Abruption	Cesarean	7	2240
Wong [131]	1997	22	33	Chorioamnionitis	Cesarean	9	2150
Finerty-1 [132]	1999	12	NA	NA	Cesarean	NA	NA
Finerty-2 [132]	1999	17	33	NA	SVD	NA	NA
Ayorinde [133]	2000	12	35	Premature labor	SVD	10	2200
Feldman [134]	2000	15	31	Seizures/hypertension	Cesarean	9	1506
Sim [135]	2001	4	33	Premature rupture of membranes	Cesarean	6	1680
Bush [136]	2003	15	24	FHR bradycardia	Cesarean	1	740
Chiossi-1 [137]	2006	10	34	Hypotension Fetal lung maturity	Cesarean	9	2680
Chiossi-2 [137]	2006	19	31	Abnormal FHR pattern Biophysical profile 6/10	Cesarean	7	1701

FHR, Fetal heart rate; mo, months; NA, not available; PVS, persistent vegetative state; SVD, spontaneous vaginal delivery.

1) Longer time interval between maternal brain injury and delivery
2) Heavier birth weights at delivery
3) Delivery at a more advanced gestational age.

It is important to note that these differences may be more a reflection of the severity of the maternal condition in the brain-dead gravida [136]. Moreover, prolonged "maternal survival" is related to the ability to maintain euthermia, to have spontaneous respirations, and to have a functioning cardiovascular system [136].

Therefore, it is easy to see that for optimal care of such patients and fetuses, a cooperative effort among various healthcare providers is essential. The goal is to maintain maternal somatic survival until the fetus is viable and reasonably mature. To achieve this goal, a number of maternal and fetal considerations must be addressed to enhance fetal outcome (Table 8.8) [113].

Table 8.8 Medical and obstetric considerations in providing artificial life support to the brain-dead gravida.

Maternal considerations
Mechanical ventilation
Cardiovascular support
Temperature lability
Hyperalimentation
Panhypopituitarism
Infection surveillance
Prophylactic anticoagulation
Fetal considerations
Fetal surveillance
Ultrasonography
Steroids
Timing of delivery

As demonstrated in Table 8.8, Field and associates [113] have tried to capture the complexities associated with the medical management of these patients. Maternal medical management involves the regulation of most, if not all, maternal bodily functions. For example, the loss of the pneumotaxic center in the pons, which is responsible for cyclic respirations, and the medullary center, which is responsible for spontaneous respirations, make mechanical ventilation mandatory. Ventilation, under

these circumstances, is similar to that for the non-pregnant patient. In contrast to the non-pregnant patient, the desirable gas concentrations are stricter due to the presence of the fetus. As such, the maternal P_aCO_2 should be kept between 30 and 35 mmHg [141] and the maternal P_aO_2 greater than 60–70 mmHg to avoid deleterious effects on uteroplacental perfusion.

Maternal hypotension occurs frequently in these patients and may be due to a combination of factors, including hypothermia, hypoxia, and panhypopituitarism. Maintenance of maternal BP can often be achieved with the infusion of low-dose dopamine, which elevates BP without affecting renal or splanchnic blood flow. Along with vasopressors to support the maternal blood pressure and organ perfusion, the patient should be kept, when possible, in the lateral recumbent position to maintain uteroplacental blood flow. At the same time, care should be exercised to avoid decubitus ulcers.

With maternal brain death, the thermoregulatory center located in the ventromedian nucleus of the hypothalamus does not function, and maternal body temperature cannot be maintained normally. As a result, maternal hypothermia is the rule. Maintenance of maternal euthermia is important and usually can be accomplished through the use of warming blankets and the administration of warm, inspired, humidified air.

Maternal pyrexia suggests an infectious process and the need for a thorough septic workup. Thus, infection surveillance for, and the treatment of, infectious complications is helpful to prolong maternal somatic survival [141]. If the maternal temperature remains elevated for a protracted period, cooling blankets may be necessary to avoid potentially deleterious effects on the fetus [145].

Nutritional support, usually in the form of enteral or parenteral hyperalimentation, is required for maternal maintenance and fetal growth and development (see Chapter 15). Because of poor maternal gastric motility, parenteral rather than enteral hyperalimentation is often preferred [113] to maintain a positive nitrogen balance. The use of hyperalimentation during pregnancy does not appear to have deleterious effects on the fetus [146]. As a rule, the amount of hyperalimentation should be in keeping with the caloric requirements for that gestational age of the pregnancy and be sufficient to avoid maternal hyperglycemia.

In such patients, panhypopituitarism frequently occurs. As a result, a variety of hypoendocrinopathies, such as diabetes insipidus, secondary adrenal insufficiency, and hypothyroidism, may develop, each mandating therapy to maintain the pregnancy. Treatment of these conditions requires the use of vasopressin, corticosteroids, and thyroid replacement, respectively.

Because of the hypercoagulable state of pregnancy and the immobility of the brain-dead gravida, these patients also are at an increased risk for thromboembolism. Therefore, to minimize the potential for deep venous thrombosis or pulmonary embolus, heparin prophylaxis (5000–7500 units twice or three times a day) and/or intermittent pneumatic calf compression are recommended [147].

By artificially supporting the maternal physiologic system, the intrauterine environment can be theoretically maintained to allow for adequate fetal growth and development (Table 8.8). Obstetric management should focus on monitoring fetal growth with frequent ultrasound evaluations, antepartum FHR assessment, and the administration of corticosteroids between 24 and 34 weeks of gestation to enhance fetal lung maturation [113,148]. For stimulation of fetal lung maturity, betamethasone or dexamethasone is recommended. Repeated steroid injections in subsequent weeks are not recommended due to the concern over the effect of repeated steroid injections on fetal brain growth [148]. However, "a single repeat course of antenatal corticosteroids should be considered in women who are less than 34 0/7 weeks of gestation who have an imminent risk of preterm delivery within the next 7 days, and whose prior course of antenatal corticosteroids was administered more than 14 days previously" [148].

Another obstetric concern is the development of premature labor. Here, tocolytic therapy has been used successfully [118,149]. Catanzarite *et al*. [123] described the use of a magnesium sulfate infusion and indomethacin to control uterine contractions, allowing prolongation of the pregnancy for 25 days. Other agents available for tocolysis include betamimetics, calcium-channel blockers, and oxytocin antagonists. The hemodynamic effects of betamimetics and calcium-channel-blocking agents may make these drugs less than ideal choices in these settings, in which maternal hemodynamic instability is common [118].

The timing of delivery is based on the deterioration of maternal or fetal status or the presence of fetal lung maturity. Classical cesarean is the procedure of choice [113,141] and is the least traumatic procedure for the fetus. To assure immediate cesarean capability, a cesarean pack and neonatal resuscitation equipment should be immediately available in the ICU.

Perimortem cesarean delivery

For centuries, perimortem cesarean delivery has been described as an attempt to preserve the life of the unborn child when the pregnant woman dies [151]. The first description of a perimortem cesarean was by Pliny the Elder in 237 AD. This delivery related to that of Scipio Africanus. Over a thousand years later, in 1280, the

Catholic Church at the Council of Cologne decreed that a perimortem cesarean delivery must be performed to permit the unborn child to be baptized and to undergo a proper burial. Failure to perform the delivery constituted a punishable offense. Moreover, perimortem cesarean was mandated specifically in those women whose pregnancies were advanced beyond 6 months. To date, there have been 307 cases of perimortem cesarean delivery reported in the English literature [151,152]. Of these cesareans, there have been 222 surviving infants [151,152].

Since Weber's monumental review of the subject in 1971, the causes of maternal death leading to a perimortem cesarean delivery have not changed substantially [151,152] but are more reflective of contemporary obstetric care [130,131]. These include traumatic events, pulmonary embolism from amniotic fluid, clot or air, acute respiratory or cardiac failure, and sepsis. In the case of a sudden, unanticipated maternal arrest, the timing of cesarean delivery becomes the quintessential element [151,152].

If a pregnant woman does sustain a cardiopulmonary arrest, cardiopulmonary resuscitation (CPR) should be initiated immediately (Chapter 11). Optimal performance of CPR in the non-pregnant patient results in a cardiac output less than one-third of normal [152]. In the pregnant woman at term, CPR, under optimal circumstances, produces a cardiac output around 10% of normal. To optimize maternal cardiac output, the patient should be placed in the supine position. Dextrorotation of the uterus and compression of the major vessels of the uterus may impede venous return and may further compromise this effort. Lateral uterine displacement may help to remedy this problem, but CPR in this position is extremely awkward. Ultimately, a cesarean may be necessary to alleviate this impedance to CPR.

If maternal and fetal outcomes are to be optimized, the timing of the cesarean delivery is critical. According to Katz and associates [151] in 1986 and reaffirmed in 2005 [152], the theory behind a perimortem cesarean is that if CPR fails to produce a pulse within 4 min, a cesarean delivery should be begun and the baby delivered within 5 min of maternal cardiac arrest. Once the baby is delivered, maternal CPR should continue because many women will have "sudden and profound improvement" [152] after evacuation of the uterus. Hence, the "4-minute rule" came into effect and had been adopted by the American Heart Association when maternal CPR has been ineffective [153–155]. Thus, the standard ABCs of cardiopulmonary resuscitation (airway, breathing, and circulation) have been expanded to include D (delivery).

As demonstrated in Table 8.9, fetal survival is linked consistently with the interval between maternal arrest and delivery. It is clear from the available data [154,156] that the longer the time interval from maternal death to the delivery of the fetus, the greater is the likelihood of permanent neurologic impairment of the fetus. Ideally, the fetus should be delivered within 5 min of maternal arrest. Within that 5 min window rests the greatest likelihood of delivering a child who will be neurologically normal (Table 8.9). However, the potential exists for a favorable fetal outcome beyond 15 min of maternal cardiac arrest, and therefore, delivery should not be withheld even if beyond 5 min, if the fetus is still alive [151,152]. Recently, Benson and associates [157] have challenged the "Katz rule" [151,152]. In their review of 53 perimortem cesareans, Benson found that the fetal/neonatal injury-free interval was longer than 5 min. These investigators found the injury-free window to be around 9–10 min from arrest to delivery interval. Analysis of misses and near misses will have to be evaluated in the future to determine the optimal interval.

Table 8.9 Perimortem cesarean delivery with the outcome of surviving infants from the time of maternal death until delivery.

Time interval (min)	Surviving infants (no.)	Intact neurologic status of survivors (%)
0–5	9	8 (89%)
6–15	5	2 (60%)
>15	7	4 (57%)

Source: Refs. [150–152].

While the timing of cesarean delivery is a major determinant of subsequent fetal outcome, the gestational age of the fetus also is an important consideration. The probability of survival is related directly to the neonatal birth weight and gestational age [154–156,158]. At what gestational age should a perimortem cesarean delivery be considered? Is there a lower limit? It becomes obvious immediately that there are no clear answers to these questions. As a general rule, intervention appears prudent whenever the fetus is potentially viable or is "capable of a meaningful existence outside the mother's womb" [159]. According to ACOG [148], the gray zone rests between 23 and 24 weeks' gestation. But, this threshold is continually pushed to earlier gestational ages in keeping with the advances in obstetrical and neonatal care. Ideally, criteria for intervention in such circumstances should be formulated with the aid of an institution's current neonatal survival statistics and guidance from its bioethics committee. In light of the continual technologic advances in neonatology, care must be taken to periodically review these criteria because the gestational age and weight criteria may be lowered in the future [154–156,158,159].

When maternal death is an anticipated event, is informed consent necessary? For instance, patients hospitalized with terminal cancer, class IV cardiac disease, pulmonary hypertension, or previous myocardial infarction are at an increased risk of death during pregnancy. Although these cases are infrequent, it seems reasonable to prepare for such an eventuality. Decisions regarding intervention should be made in advance with the patient and family. When intervention has been agreed to, one consideration is to have a cesarean delivery pack and neonatal resuscitation equipment immediately available in the ICU.

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