33 Intrapartum Fetal Surveillance

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Factors Controlling Fetal Heart Rate

Fetal heart rate (FHR) analysis is the most common means of evaluating a fetus for adequate oxygenation. The rate and regulation of the fetal heart provide important information for the obstetrician. The average FHR is 155 beats/min at 20 weeks' gestation, 144 beats/min at 30 weeks, and 140 beats/min at term. This progression is thought to reflect maturation of vagal tone, with consequent slowing of the baseline FHR. Normal fetuses can have variations of 20 beats/min faster or slower than these baseline values.

The fetal heart is similar to the adult heart in that it has its own intrinsic pacemaker activity that results in rhythmic myocardial contractions. The sinoatrial node, found in one wall of the right atrium, has the fastest rate of depolarization and sets the rate in the normal heart. The next fastest rate is produced by the secondary pacemaker, the atrioventricular node within the atrial septum. The His-Purkinje system carries electrical signals throughout the ventricles at a slower rate than the sinoatrial or atrioventricular node. Complete or partial heart block in the fetus produces variations in rate that are markedly slower than normal. The rate in a fetus with a complete heart block is 60 to 80 beats/min.

FHR variability is important clinically, and its specific amplitude as part of the FHR pattern has prognostic value. The heart rate is the result of many physiologic factors that modulate the intrinsic rate of the fetal heart, the most common of which are signals from the autonomic nervous system.

PARASYMPATHETIC NERVOUS SYSTEM

The parasympathetic nervous system consists primarily of the vagus nerve (cranial nerve X), which originates in the medulla oblongata. The vagus nerve innervates the sinoatrial and atrioventricular nodes. Stimulation of the vagus nerve results in a decrease in FHR in the normal fetus because vagal influence on the sinoatrial node decreases its rate of firing. In a similar fashion, blockade of this nerve in a normal fetus causes an increase in the FHR of approximately 20 beats/min at term.[1] This finding demonstrates a normally constant vagal influence on the FHR, which tends to decrease it from its intrinsic rate.

The vagus nerve is also responsible for transmission of impulses causing beat-to-beat variability of the FHR; blockade of the vagus nerve eliminates this variability. The vagus nerve therefore has two possible effects on the heart: a tonic influence tending to decrease FHR and an oscillatory influence that results in FHR variability.[2] Vagal tone is not necessarily constant. Its influence increases with gestational age.[3] In fetal sheep, vagal activity increases as much as fourfold during acute hypoxia[4] or experimentally produced fetal growth restriction.[5]

SYMPATHETIC NERVOUS SYSTEM

Sympathetic nerves are widely distributed in the muscle of the heart at term. Stimulation of the sympathetic nerves releases norepinephrine and increases the rate and strength of fetal cardiac contractions, resulting in higher cardiac output. The sympathetic nerves are a reserve mechanism to improve the pumping activity of the heart during intermittent stressful situations. There is normally a tonic sympathetic influence on the heart. Blocking the action of these sympathetic nerves causes a decrease in FHR of approximately 10 beats/min. As with vagal tone, tonic sympathetic influence increases as much as twofold during fetal hypoxia.

CHEMORECEPTORS

Chemoreceptors are found in the peripheral and central nervous systems. They have their most dramatic effects on the regulation of respiration but are also important in control of the circulation. Peripheral chemoreceptors reside in the aortic and carotid bodies, which are located in the arch of the aorta and the area of the carotid sinus, respectively. The central chemoreceptors in the medulla oblongata respond to changes in oxygen and carbon dioxide tension in the blood or in the cerebrospinal fluid perfusing this area.

In the adult, a reflex tachycardia occurs when oxygen is decreased or the carbon dioxide content is increased in the arterial blood perfusing the central chemoreceptors. A substantial increase in arterial blood pressure occurs, particularly when the carbon dioxide concentration is increased. Both effects are thought to be protective, representing an attempt to circulate more blood through the affected areas and thereby decrease the carbon dioxide tension (Pco_2) or increase the oxygen tension (Po_2). In adults, selective hypoxia or hypercapnia of the peripheral chemoreceptors alone produces bradycardia, in contrast to the tachycardia and hypertension that result from central hypoxia or hypercapnia.

The interaction of central and peripheral chemoreceptors in the fetus is poorly understood. It is known that the net result of hypoxia or hypercapnia in the unanesthetized fetus is bradycardia with hypertension. During basal conditions, the chemoreceptors contribute to stabilization of the FHR and blood pressure.[6]

BARORECEPTORS

In the arch of the aorta and in the carotid sinus at the junction of the internal and external carotid arteries are small stretch receptors in the vessel walls that are sensitive to increases in blood pressure. When pressure rises, impulses are sent from

these receptors through the vagus or glossopharyngeal nerve to the midbrain. This results in further vagus stimulation, which tends to slow the heart. This is an extremely rapid response, occurring almost with the first systolic rise of blood pressure. It is a protective, stabilizing attempt by the body to lower blood pressure by decreasing the heart rate and cardiac output when blood pressure is increasing.

CENTRAL NERVOUS SYSTEM

In the adult, the higher centers of the brain influence the heart rate, which is increased by emotional stimuli such as fear and sexual arousal. In fetal lambs and monkeys, the electroencephalogram or electro-oculogram shows increased activity that sometimes is associated with increased variability of the FHR and body movements. When the fetus is sleeping, body movement slows, and FHR variability decreases, suggesting an association between these two factors and central nervous system activity.[7]

The medulla oblongata contains the vasomotor centers. These are integrative centers in which the net result of all the central and peripheral inputs is processed to generate irregular oscillatory vagal impulses, producing acceleration or deceleration of the heart (i.e., FHR variability).

HORMONAL REGULATION

Adrenal Medulla

The fetal adrenal medulla produces epinephrine and norepinephrine in response to stress. Both substances act on the heart and cardiovascular system in a way similar to sympathetic stimulation to produce a faster FHR (i.e., chronotropic effect), greater force of contraction of the heart (i.e., inotropic effect), and higher arterial blood pressure.

Renin-Angiotensin System

Angiotensin II may play a role in fetal circulatory regulation at rest. Its main activity is observed during hemorrhagic stress on a fetus.

Prostaglandins

Various prostaglandins and arachidonic acid metabolites are found in the fetal circulation and in many fetal tissues. Their main roles with respect to cardiovascular function are in regulating umbilical blood flow and maintaining the patency of the ductus arteriosus during fetal life.

Other Hormones

Fetal hormones such as nitric oxide, α-melanocyte-stimulating hormone, atrial natriuretic hormone, neuropeptide Y, thyrotropin-releasing hormone, and cortisol and metabolites such as adenosine participate in the regulation of circulatory function.

BLOOD VOLUME CONTROL

Capillary Fluid Shift

In the adult, when the blood pressure is elevated by excessive blood volume, fluid moves out of the capillaries into interstitial spaces, decreasing the blood volume toward normal. If the adult loses blood through hemorrhage, some fluid shifts out of the interstitial spaces into the circulation, increasing the blood volume toward normal. There is normally a delicate balance between the pressures inside and outside the capillaries. This mechanism of regulating blood pressure is slower than the almost instantaneous regulation observed with the reflex mechanisms discussed previously. Its role in the fetus is imperfectly understood, although imbalances may be responsible for the hydrops seen in some cases of red cell alloimmunization and the high-output failure sometimes seen with supraventricular tachycardia.

Intraplacental Pressures

Fluid moves along hydrostatic pressure gradients and in response to osmotic pressure gradients. The specific role of these factors within the human placenta, where fetal and maternal blood closely approximate, is unclear, but it seems likely that some delicate balancing mechanisms within the placenta prevent rapid fluid shifts between mother and fetus. The mean arterial blood pressure of the mother (≈100 mm Hg) is much higher than that of the fetus (≈55 mm Hg), but the spiral artery reduces this pressure to less than 70 mm Hg systolic, the pressure in the intervillous space is about 10 mm Hg, and the osmotic pressures are not substantially different.

Frank-Starling Mechanism

The amount of blood pumped by the adult heart is determined in part by the amount of blood returning to the heart. The heart normally pumps the blood that flows into it without excessive damming of blood in the venous circulation. When the cardiac muscle is stretched during diastole by increased venous return of blood, it contracts with greater force and is able to pump out more blood. This mechanism of response to preload is apparently not the same in the fetal heart as in the adult heart. In the fetus, increases in preload produce minor or no changes in combined ventricular output, suggesting that the fetal heart normally operates near the peak of its function curve.

The output of the fetal heart is related to the FHR. Some researchers have shown that spontaneous variations of the FHR are directly related to cardiac output (i.e., as the rate increases, output increases). However, different responses have been observed during right or left atrial pacing studies.[8] Additional factors are required to explain these differences. In addition to the FHR and preload, cardiac output depends on afterload and intrinsic contractility.[8,9]

The fetal heart is highly sensitive to changes in afterload, represented by the fetal arterial blood pressure. Increases in afterload dramatically reduce the stroke volume or cardiac output. The fetal heart is incompletely developed, and many ultrastructural differences between the adult and fetal heart account for its lower intrinsic capacity to alter its contraction efficiency. The determinants of cardiac output do not work separately; each interacts dynamically to modulate fetal cardiac output during changing physiologic conditions. In clinical practice, it is reasonable to assume that modest variations of the FHR from the normal range produce relatively small effects on cardiac output. However, at the extremes (e.g., tachycardia of >240 beats/min, bradycardia of <60 beats/min), cardiac output and umbilical blood flow are likely to be substantially decreased.

Umbilical Blood Flow

Umbilical blood flow is approximately 40% of the combined fetal ventricular output, and not all of this blood flow to the placenta exchanges with maternal blood. Umbilical blood flow

is unaffected by acute moderate hypoxia, but it is decreased by severe hypoxia affecting myocardial function. The umbilical cord lacks innervation, and there are no means of increasing umbilical flow. However, variable decelerations in the FHR commonly occur with transient umbilical cord compression, and flow is diminished or stopped for a time, depending on the degree and duration of cord compression or occlusion.

Monitoring the Fetal Heart Rate

The electronic FHR monitor is a device with two components. One establishes the FHR, and the other measures uterine contractions.[10] To recognize the FHR, the device uses the R wave of the fetal electrocardiogram (ECG) complex (i.e., fetal scalp electrode) or modulation of an ultrasound signal generated by movement of a cardiovascular structure (i.e., Doppler ultrasound transducer or cardiotachometer). Uterine contractions are detected directly by a pressure transducer attached to a catheter within the amniotic cavity (i.e., intrauterine pressure catheter) or by a beltlike external device (i.e., tocodynamometer) that recognizes tightening of the uterus during a contraction. Monitoring with devices attached directly to the fetus or placed within the uterine cavity is called *internal*, and monitoring with devices that are on the maternal abdomen is called *external*.

FETAL HEART RATE DETECTION

Fetal Electrode

The fetal electrode consists of a small, spiral, stainless steel wire that is typically attached to the fetal scalp. A second contact is bathed by the vaginal fluids. The wires traverse the vaginal canal and are connected to a maternal leg plate, which is attached to the fetal monitor. The internal mode gives the most accurate FHR tracing, because this technique directly measures the fetal cardiac electrical signal and true beat-to-beat variability.

Doppler Ultrasound Transducer

The FHR monitoring device most commonly employed is the cardiotachometer or Doppler ultrasound transducer. This device emits a high-frequency ultrasound signal (approximately 2.5 MHz) that is reflected from any moving structure (e.g., ventricle wall, valvular leaflets), with the reflected signal altered in frequency. The change in frequency with each systole is recognized as a cardiac contraction and is processed by the transducer. The interval between cardiac events is measured (in seconds) and then divided into 60 to yield a rate for each interval between beats. These calculated rates are transcribed onto a paper strip that is moving at a specific speed (usually 3 cm/min). The resulting tracing appears as a wavy line and is a very close representation of true FHR variability. If the intervals between heartbeats are persistently identical, the resultant FHR line is straight, suggesting minimal or absent variability.

Although this device is simple to apply, it is often inconsistent in obtaining a signal because of interference caused by maternal and fetal movements. Improvements in the logic and technology of the monitors have made the external devices more accurate and easier to use. The technique of *autocorrelation* is used to define the timing of the cardiac contraction more accurately. Analysis of a very large number of points on the curve depicting the Doppler frequency shift produces a signal that much more accurately represents the FHR variability. The signal must be confirmed as fetal rather than maternal in origin.

UTERINE ACTIVITY DETECTION

Intra-amniotic Catheter

The internal means of detecting uterine activity typically uses a soft, plastic, transducer-tipped catheter placed transcervically into the amniotic cavity. The pressure of the baseline uterine tone and that of any uterine contraction is translated into an electrical signal, which is calibrated and displayed directly (as millimeters of mercury [mm Hg]).

Tocodynamometer

The tocodynamometer is an external device that is placed on the maternal abdominal wall over the uterine fundus. Tightening of the fundus with each contraction is detected by pressure on a small button in the center of the transducer, and uterine activity is displayed on the recorder. It acts like a hand placed on the uterine fundus through the abdominal wall to detect uterine activity. This device detects the frequency and duration of uterine contractions but not true contraction intensity. One disadvantage of the tocodynamometer is that it works best with the mother in the supine position. This limitation may not always be compatible with maternal comfort, fetal well-being, or progression of labor. With repositioning of the patient, it is important to reestablish accurate monitoring of the fetal heart and uterine activity.

Fetal Responses to Hypoxia or Acidemia

Studies of chronically prepared animals have shown that a number of responses occur during acute hypoxia or acidemia in the previously normally oxygenated fetus. Little or no change in combined cardiac output and umbilical (placental) blood flow occurs, but there is a redistribution of blood flow favoring certain vital organs—heart, brain, and adrenal glands—and a decrease in blood flow to the gut, spleen, kidneys, and carcass.[11] This initial response is presumed to be advantageous to a fetus in the same way as the diving reflex is advantageous to an adult seal. Blood containing the available oxygen and other nutrients is supplied preferentially to vital organs. These responses are temporary compensatory mechanisms that enable a fetus to survive for moderately long periods (e.g., up to 30 minutes) of limited oxygen supply without decompensation of vital organs, particularly the brain and heart.

Close matching of blood flow to oxygen availability to achieve a constancy of oxygen consumption has been demonstrated in the fetal cerebral circulation[12] and in the fetal myocardium.[13] In studies of hypoxic lamb fetuses, cerebral oxygen consumption was constant over a wide range of arterial oxygen concentrations, because the decrease in arteriovenous oxygen content accompanying hypoxia was offset by an increase in cerebral blood flow. However, during more severe acidemia or sustained hypoxemia, these responses were no longer maintained, and decreases in cardiac output, arterial blood pressure, and blood flow to the brain and heart resulted.[14] These changes may be considered as a stage of decompensation after which tissue damage and even fetal death may follow.[15]

Fetal Acid-Base Balance

PHYSIOLOGY

Normal metabolism in the fetus results in the production of carbonic and organic acids. These acids are buffered by various mechanisms that regulate the fetal pH within a very narrow range. Although the concentration of hydrogen ions is extremely low, changes in fetal pH as small as 0.1 unit can have profound effects on metabolic activity and on the cardiovascular and central nervous systems. Extreme changes in pH can be fatal.

The maternal acid-base status can adversely affect the fetal acid-base status. In normal pregnancies, the difference between maternal and fetal pH is usually 0.05 to 0.10 units.[16]

Carbonic Acid

Carbonic acid (H_2CO_3) is a volatile acid that is produced from the metabolism of glucose and fatty acids. During fetal oxidative metabolism (i.e., aerobic glycolysis or cellular respiration), the oxidation of glucose uses oxygen (O_2) and produces carbon dioxide (CO_2).

From a practical standpoint, carbonic acid formation is equivalent to carbon dioxide generation, and most of the free hydrogen ion formed is buffered intracellularly. As blood passes through the placenta (or through the lung in the adult), bicarbonate ion (HCO_3^-) reenters erythrocytes and combines with hydrogen ions to form carbonic acid, which then dissociates to carbon dioxide and water. The carbon dioxide formed in the fetus diffuses across the placenta and is excreted by the maternal lung. Carbon dioxide diffuses rapidly across the human placenta, and even large quantities produced by the fetus can be eliminated rapidly if maternal respiration, uteroplacental blood flow, and umbilical blood flow are normal.

The rate of fetal carbon dioxide production is roughly equivalent to the fetal oxygen consumption rate.[17] For carbon dioxide to diffuse from fetus to mother, a gradient must be maintained between the P_{CO_2} in fetal umbilical blood and that in maternal uteroplacental blood. Adequate perfusion of both sides of the placenta also must be preserved. Because of progesterone-stimulated maternal hyperventilation, the arterial P_{CO_2} is reduced from a mean of 39 mm Hg in nonpregnant women to a mean of 31 mm Hg during pregnancy. Renal compensation results in increased bicarbonate excretion and plasma levels of 18 to 22 mEq/L during pregnancy.[18]

Nonvolatile Acids

Anaerobic metabolism in the fetus results in the production of nonvolatile or fixed organic acids by two mechanisms: use of non–sulfur-containing amino acids, which generates pyruvic and acetoacetic acids, and incomplete combustion of carbohydrates and fatty acids, which produces lactic acid and ketoacids (e.g., β-hydroxybutyric acid).

Because of relatively immature renal function, the fetus is unable to effectively excrete these acids; instead, they are transported to the placenta, where they diffuse slowly (unlike carbon dioxide) into the maternal circulation. The maternal kidney excretes fixed organic acids produced by maternal and fetal metabolism and helps to regenerate bicarbonate. Because the maternal glomerular filtration rate increases significantly during normal pregnancy, the maternal kidney filters and reabsorbs large quantities of bicarbonate daily.

The fetus does have the ability to metabolize accumulated lactate in the presence of sufficient oxygen. However, this is a slow process, and it is not thought to account for a large proportion of lactic acid elimination from the fetal compartment.

Buffers

Dramatic changes in pH are minimized by the action of buffers. The two major buffers are plasma bicarbonate and hemoglobin. Quantitatively less important buffers include erythrocyte bicarbonate and inorganic phosphates.[19]

Terms that are used for the expression of buffering capacity include the following:

- *Delta base:* measure of the change (Δ) in the buffering capacity of bicarbonate
- *Base deficit:* bicarbonate values lower than normal
- *Base excess:* bicarbonate values higher than normal

Although the fetus has a limited ability to buffer an increase in acid production with bicarbonate and hemoglobin, the placental bicarbonate pool also may play a role in buffering the fetus against changes in maternal pH or blood gas status. Aarnoudse and colleagues[20] studied bicarbonate permeability in the perfused human placental cotyledon model and found that acidification of the maternal circulation to pH 7.06 for 30 minutes did not significantly alter fetal pH. Instead, there was an efflux of total carbon dioxide from the placenta into the maternal circulation in the form of bicarbonate, which was not matched by an influx of total carbon dioxide from the fetal circulation. By this mechanism, bicarbonate transfer could take place between the placental tissue pool and the maternal circulation, whereas the transmission of maternal pH and blood gas changes to the fetal circulation would be minimized.

Ph Determination

The pH of a liquid is the negative logarithm of the hydrogen ion concentration in that liquid. It can be used to describe the acid-base status of any body fluid. It is directly related to the concentration of bicarbonate (base) and inversely related to the concentration of carbonic acid (acid). The H_2CO_3 equals $0.03 \times P_{CO_2}$, and the pK equals 6.11 for normal plasma at 37° C. This relationship is best illustrated by the Henderson-Hasselbalch equation for determining the pH of a buffered system, in which pK is the negative logarithm of the acid dissociation constant:

$$pH = pK + \log \frac{[base]}{[acid]}$$

In the case of fetal acid-base balance determinations,

$$pH = pK + \log \frac{[HCO_3^-]}{[H_2CO_3]}$$

$$pH = pK + \log \frac{[HCO_3^-](mEq/L)}{0.03[P_{CO_2}](mmHg)}$$

In simplest terms, the HCO_3 represents the metabolic component, and the H_2CO_3 (or P_{CO_2}) represents the respiratory component.[21]

TABLE 33-1 Terminology

Term	Definition
Acidemia	Increased concentration of hydrogen ions in blood
Acidosis	Increased concentration of hydrogen ions in tissue
Asphyxia	Hypoxia with metabolic acidosis
Base deficit	HCO_3^- concentration lower than normal
Base excess	HCO_3^- concentration higher than normal
Delta base	Measure of change (Δ) in buffering capacity of bicarbonate
Hypoxemia	Decreased oxygen content in blood
Hypoxia	Decreased level of oxygen in tissue
pH	Negative logarithm of hydrogen ion concentration

Adapted from American College of Obstetricians and Gynecologists (ACOG): Umbilical artery blood acid-base analysis, *technical bulletin no. 216, Washington, DC, 1995, ACOG.*

TABLE 33-2 Types of Acidemia

Acidemia*	Definition
Metabolic	Normal P_{CO_2} and decreased HCO_3^-
Respiratory	Increased P_{CO_2} and normal HCO_3^- (after correction of P_{CO_2})
Mixed	Increased P_{CO_2} and decreased HCO_3^-

*Umbilical artery pH < 7.10.

TERMINOLOGY

Acidemia refers to an increase in hydrogen ions in the blood; *acidosis* refers to an increase in hydrogen ions in tissue. Similarly, *hypoxemia* is a decrease in oxygen content in blood, whereas *hypoxia* is a decrease in oxygen content in tissue (Table 33-1).

Acidemia in the newborn can be classified as three types: metabolic, respiratory, and mixed. The type is based primarily on the levels of HCO_3^- and P_{CO_2} (Table 33-2). With marked elevations of the P_{CO_2}, there is a compensatory increase in HCO_3^- of 1 mEq/L for each 10 mm Hg increase in P_{CO_2}.[22]

FACTORS AFFECTING ACID-BASE BALANCE

For the acid-base balance in the fetus, the placenta acts as lungs and kidneys by supplying oxygen and removing carbon dioxide and various metabolites. The pH in the fetus is controlled within a very tight range. Umbilical blood oxygen content and saturation and fetal arterial delta base values depend primarily on uterine blood flow. Oxygen supply depends on the following:

- Adequate maternal oxygenation
- Blood flow to the placenta
- Transfer across the placenta
- Fetal oxygenation
- Delivery to fetal tissues

Removal of carbon dioxide depends on fetal blood flow to the placenta and transport across the placenta. Fixed-acid equilibrium depends on a continued state of balance between production and removal.

Respiratory Factors

Respiratory acidosis results from increased P_{CO_2} and subsequently from decreased pH. In the fetus, this picture is usually associated with decreased PO_2. The most common cause of acute respiratory acidosis in the fetus is a sudden decrease in placental or umbilical perfusion. Umbilical cord compression, uterine hyperstimulation, and abruptio placentae are examples, and transient cord compression is the most common factor.

Conditions associated with maternal hypoventilation or acute maternal hypoxemia can result in fetal hypoxemia and hypercarbia, potentially leading to fetal acidosis, which is a mixed respiratory and metabolic acidosis. Conditions associated with maternal hypoventilation or hypoxia can also result in respiratory acidosis in the fetus and, if severe enough, in metabolic acidosis. Coleman and Rund[23] reviewed the association between maternal hypoxia and non-obstetric conditions (e.g., asthma, epilepsy) during pregnancy. They found that the normal physiologic changes that occur during pregnancy might make early recognition of maternal hypoxia difficult. For example, in a mother with asthma, a pH of less than 7.35 and a P_{CO_2} higher than 38 mm Hg may indicate respiratory compromise.[24] To minimize the risk of concurrent hypoxemia in the fetus, early intubation in mothers who have borderline or poor blood gas values or evidence of respiratory compromise is recommended.

Other conditions can result in acute or chronic maternal hypoventilation during pregnancy. Induction of general anesthesia or narcotic overdose can depress the medullary respiratory center. Hypokalemia, neuromuscular disorders (e.g., myasthenia gravis), and toxic doses of drugs that impair neuromuscular transmission (e.g., magnesium sulfate) can result in hypoventilation or paralysis of the respiratory muscles. Airway obstruction by foreign bodies can cause maternal respiratory acidosis. Restoration of the normal fetal acid-base balance depends on the reversibility of maternal etiologic factors.

Maternal respiratory alkalosis may occur when hyperventilation reduces the P_{CO_2} and increases pH. Severe anxiety, acute salicylate toxicity, fever, sepsis, pneumonia, pulmonary emboli, and acclimation to high altitudes are etiologic factors. Severe respiratory alkalosis and hypocapnia can cause uterine artery vasospasm, reducing placental perfusion and causing fetal hypoxia and metabolic acidosis. As in respiratory acidosis, restoration of the maternal acid-base balance by appropriate treatment of causative factors results in normalization of fetal blood gases.

Metabolic Factors

Fetal metabolic acidosis is characterized by loss of bicarbonate, high base deficit, and a subsequent fall in pH. This type of acidosis results from protracted periods of oxygen deficiency to a degree that results in anaerobic metabolism. The cause can be fetal or maternal, and it usually implies the existence of a chronic metabolic derangement. Conditions such as growth restriction resulting from chronic uteroplacental hypoperfusion can be associated with fetal metabolic acidosis due to decreased oxygen delivery.

Maternal metabolic acidosis can cause fetal metabolic acidosis and is classified according to the status of the anion gap. In addition to bicarbonate and chloride, the remaining anions required to balance the plasma sodium concentration are referred to as *unmeasured anions* or the *anion gap*. Reduced excretion of inorganic acids (e.g., renal failure) or accumulation of organic acids (e.g., alcoholic, diabetic, or starvation ketoacidosis; lactic acidosis) results in metabolic acidosis characterized by an increased anion gap. Bicarbonate loss (e.g., renal tubular

TABLE 33-3 Fetal Scalp Blood Values in Labor

Measurement	Early First Stage*	Late First Stage*	Second Stage*
pH	7.33 ± 0.03	7.32 ± 0.02	7.29 ± 0.04
P_{CO_2} (mm Hg)	44.00 ± 4.05	42.00 ± 5.1	46.30 ± 4.2
P_{O_2} (mm Hg)	21.8 ± 2.6	21.3 ± 2.1	16.5 ± 1.4
Bicarbonate (mmol/L)	20.1 ± 1.2	19.1 ± 2.1	17 ± 2
Base excess (mmol/L)	3.9 ± 1.9	4.1 ± 2.5	6.4 ± 1.8

*All values are given as the mean ± standard deviation.
From Huch R, Huch A: Maternal-fetal acid-base balance and blood gas measurement. In Beard RW, Nathanielsz PW, editors: Fetal physiology and medicine, *New York, 1984, Marcel Dekker.*

acidosis, hyperparathyroidism, diarrheal states) or failure of bicarbonate regeneration results in metabolic acidosis characterized by a normal anion gap. Fetal responses to these maternal conditions are manifested by a pure metabolic acidosis with normal respiratory gas exchange as long as placental perfusion remains normal.

Prolonged fetal respiratory acidosis (e.g., cord compression, abruptio placentae) can result in accumulation of fixed organic acids produced by anaerobic metabolism. This condition is characterized by blood gas measurements that reflect a mixed respiratory and metabolic acidosis.

Effects of Labor

Each uterine contraction transiently diminishes uterine blood flow, reduces placental perfusion, and impairs transplacental gaseous exchange. A sample of blood may be obtained from the fetal presenting part to help evaluate fetal status during labor. Typical fetal scalp blood values during labor are shown in Table 33-3. This information is of limited value because fetal scalp blood sampling is rarely performed in the United States.

UMBILICAL CORD BLOOD ACID-BASE ANALYSIS

Acid-base analysis of umbilical cord blood provides an objective method of evaluating a depressed newborn's condition, especially with regard to hypoxia and acidemia.[16] Assessing umbilical cord blood pH has become an important adjunct in defining the degree of perinatal hypoxia when there is a question about whether it may be severe enough to result in acute neurologic injury.[25] Moreover, the technique is simple and relatively inexpensive.

Technique

For the depressed neonate of any gestational age, the umbilical cord should be immediately clamped and cut to allow delivery of the newborn to pediatric attendants for appropriate resuscitation. A segment of 10 to 20 cm of umbilical cord may then be clamped and cut separately. If other clinical issues require attention, aspiration of blood from this clamped, undisturbed, room-temperature cord segment may be delayed for up to 30 minutes without any effect on the accuracy of the initial blood gas values at the time of clamping. Specimens should be obtained ideally from the umbilical artery and the umbilical vein, but the umbilical artery sample provides a more direct assessment of fetal condition, whereas the umbilical vein reflects placental acid-base status. In cases such as cord prolapse, the umbilical artery pH may be extremely low despite a normal umbilical vein pH.[26] Nevertheless, some clinicians still prefer to use the umbilical vein, which is easier to access for drawing blood, especially in the very premature infant. In one study of 453 term infants, D'Souza and colleagues[27] determined that umbilical venous and arterial blood pH values were significantly related to each other and that umbilical venous pH measurements did provide useful information regarding acidemia at birth.

Samples should be drawn in plastic or glass syringes that have been flushed with heparin (1000 U/mL). Commercial syringes (1 to 2 mL) containing lyophilized heparin are also available for obtaining specimens. Kirshon and Moise[28] reported that the addition of 0.2 mL of 10,000 U/mL of heparin to 0.2 mL of blood significantly decreased the pH, P_{CO_2}, and bicarbonate values. Any residual heparin and air should be ejected, and the needle should be capped.

A few practical points merit mention. It is not necessary to draw the sample from the umbilical artery immediately if the cord is doubly clamped. Adequate specimens have been obtained from a clamped segment of cord as long as 60 minutes after delivery without significant changes in pH or P_{CO_2}.[29] After the specimens have been drawn into the syringe, they are relatively stable at room temperature for up to 60 minutes[30] and do not need to be transported to the laboratory on ice.[31] The same may not be true for specimens obtained from placental vessels.[32]

Chauhan and colleagues[33] prepared a mathematical model for calculating the umbilical artery pH for up to 60 hours after delivery. This model permits estimation of fetal pH at birth.

Normal Values

There is no consensus about the most appropriate umbilical artery pH cutoff for defining acidemia, but the mean pH values from four studies are shown in Table 33-4. The mean value for umbilical artery pH appears to be about 7.28. For example, in their study of cord blood respiratory gases and acid-base values, Riley and Johnson[26] determined a mean pH of 7.27 ± 0.07 for 3520 unselected women undergoing vaginal delivery.

The mean pH for umbilical venous blood has been reported to be 7.32 to 7.35 in various studies (see Table 33-4). In a study of umbilical venous blood, D'Souza and associates[27] reported a mean venous pH of 7.34 ± 0.07. Huisjes and Aarnoudse[34] reported good correlation between umbilical venous and arterial pH values.

Although the Apgar scores of premature infants may be low because of immaturity, mean arterial and venous pH and blood gas values are similar to those of the term infant. Mean values for almost 2000 premature infants are summarized in Table 33-5.

TABLE 33-4 Normal Umbilical Cord Blood pH and Blood Gas Values in Term Newborns

Measurement	Yeomans et al, 1985 (*N* = 146)*	Ramin et al, 1989 (*N* = 1292)*	Riley and Johnson, 1993 (*N* = 3520)*	Thorp et al, 1988 (*N* = 1924)*
ARTERIAL BLOOD (*N* = 1694)				
pH	7.28 ± 0.05	7.28 ± 0.07	7.27 ± 0.07	7.24 ± 0.07
P_{CO_2} (mm Hg)	49.20 ± 8.4	49.90 ± 14.2	50.30 ± 11.1	56.30 ± 8.6
HCO_3^- (mEq/L)	22.30 ± 2.5	23.10 ± 2.8	22.00 ± 3.6	24.10 ± 2.2
Base excess (mEq/L)	—	–3.60 ± 2.8	–2.70 ± 2.8	–3.60 ± 2.7
VENOUS BLOOD (*N* = 1820)				
pH	7.35 ± 0.05	—	7.34 ± 0.06	7.32 ± 0.06
P_{CO_2} (mm Hg)	38.20 ± 5.6	—	40.70 ± 7.9	43.80 ± 6.7
HCO_3^- (mEq/L)	20.40 ± 4.1	—	21.40 ± 2.5	22.60 ± 2.1
Base excess (mEq/L)	—	—	–2.40 ± 2.0	2.90 ± 2.4

*All values are given as the mean ± standard deviation.

Data from Yeomans ER, Hauth JC, Gilstrap LC, et al: Umbilical cord pH, PCO_2 and bicarbonate following uncomplicated term vaginal deliveries, Am J Obstet Gynecol *151:798, 1985; Ramin SM, Gilstrap LC, Leveno KJ, et al: Umbilical artery acid-base status in the preterm infant,* Obstet Gynecol *74:256, 1989; Riley RJ, Johnson JW: Collecting and analyzing cord blood gases,* Clin Obstet Gynecol *36:13, 1993; Thorp JA, Boylan PC, Parisi VM, et al: Effects of high-dose oxytocin augmentation on umbilical cord blood gas values in primigravid women,* Am J Obstet Gynecol *159:670, 1988.*

TABLE 33-5 Normal Arterial Blood Gas Values for Premature Infants

Measurement	Dickenson et al, 1992 (*N* = 949)*	Riley and Johnson, 1993 (*N* = 1015)*
pH	7.27 ± 0.07	7.28 ± 0.089
P_{CO_2} (mm Hg)	51.60 ± 9.4	50.20 ± 12.3
HCO_3^- (mEq/L)	23.90 ± 2.1	22.40 ± 3.5
Base excess (mEq/L)	–3.00 ± 2.5	–2.50 ± 3.0

*All values given as the mean ± standard deviation.

Data from Dickenson JE, Eriksen NL, Meyer BA, et al: The effect of preterm birth on umbilical cord blood gases, Obstet Gynecol *79:575, 1992; Riley RJ, Johnson JW: Collecting and analyzing cord blood gases,* Clin Obstet Gynecol *36:13, 1993.*

TABLE 33-6 Neonatal Morbidity and Mortality by pH Cutoff

pH	*N*	Neonatal Deaths	Seizures	Both
7.15-7.19	2236	3 (0.1%)	2 (0.1%)	1 (0.05%)
7.10-7.14	798	3 (0.4%)	1 (0.1%)	0 (0.00%)
7.05-7.09	290	0 (0.0%)	0 (0.0%)	1 (1.1%)
7.00-7.04	95	1 (1.1%)	1 (1.1%)	1 (1.1%)
<7.00	87	7 (8.0%)*	8 (9.2%)*	2 (2.3%)

*$P < .05$.

From Goldaber KG, Gilstrap LC, Leveno KJ, et al: Pathologic fetal academia, Obstet Gynecol *78:1103, 1991.*

Pathologic Fetal Acidemia

What level of umbilical artery pH should be considered abnormal, pathologic, or clinically significant? The former pH cutoff of 7.20 is no longer considered appropriate.[16,35] Most newborns with an umbilical artery pH lower than 7.20 are vigorous and have no systemic evidence of hypoxia. Evidence suggests that significant morbidity is more likely among neonates with umbilical artery pH values lower than 7.00, especially if associated with a low Apgar score (≤3). For example, in a study of 2738 term newborns, hypotonia, seizures, and required intubation were significantly correlated with an umbilical artery pH of less than 7.00 and an Apgar score of 3 or less at 1 minute.[36] The investigators concluded that a newborn must be severely depressed for birth hypoxia to be implicated as the cause of seizures.

Goldaber and coworkers,[22] in an attempt to better define the critical cutoff for pathologic fetal acidemia, studied the neonatal outcomes of 3506 term newborns. They determined the critical pH cutoff point to be 7.00 (Table 33-6). However, many had no complications and went to the newborn nursery. In a follow-up study from the same institution, King and associates[37] described 35 term newborns who appeared well at birth and were triaged to the newborn nursery but were found to have umbilical artery pH values less than or equal to 7.00 on routine screening. The study authors concluded that newborns born after 35 or more weeks' gestation who had this degree of acidemia at birth but had a stable appearance in the delivery room and no other complications did not have evidence of hypoxia or ischemia during the 48 hours after birth. Fewer than one half of neonates with an umbilical artery pH lower than 7.00 had neonatal complications.[38]

Human fetuses frequently tolerate much lower cord pH values without obvious injury than previously thought. Andres and colleagues[39] presented data from a retrospective cohort study of 93 neonates with an umbilical artery pH less than 7.00 (gestational age range, 23.5 to 42.9 weeks) and with a median pH of 6.92 (range, 6.62 to 6.99). The median pH was 6.75 for neonates with seizures (25th to 75th percentile, 6.72 to 6.88), compared with 6.93 for those without seizures ($P = .02$). The median pH for newborns with hypoxic-ischemic encephalopathy was significantly lower (6.69; 25th to 75th percentile, 6.62 to 6.75) than for those without this diagnosis (6.93; 25th to 75th percentile, 6.85 to 6.97; $P = .03$). The median pH was also less than 6.90 for newborns who required intubation (6.83) or cardiopulmonary resuscitation (6.83) and was significantly lower ($P < .05$) than for newborns without these complications. The median P_{CO_2} and base deficit values also were significantly higher for neonates with these morbidities.[39]

Acute Neurologic Injury

The 1-minute and the 5-minute Apgar scores are poor predictors of adverse neurologic outcomes for newborns. The correlation does improve if the scores remain between 0 and 3 at 10,

15, and 20 minutes; however, many of these newborns will be normal, if they survive. Similarly, a low umbilical artery pH in and of itself has poor correlation with adverse outcome.

The American College of Obstetricians and Gynecologists (ACOG)[40] has established the following essential criteria (all four must be met) to indicate hypoxia proximate to delivery severe enough to be associated with acute neurologic injury:

1. Evidence of a metabolic acidosis in fetal umbilical cord arterial blood obtained at delivery (pH <7 and base deficit ≥12 mmol/L). (Rates of neonatal encephalopathy, respiratory complications, and composite morbidity increase to approximately 10% for newborns with a base deficit of 12 to 16 mmol/L. The rate increases to 40% for newborns with umbilical artery base deficit values greater than 16 mmol/L.)
2. Early onset of severe or moderate neonatal encephalopathy in infants born at 34 or more weeks' gestation
3. Cerebral palsy of the spastic quadriplegic or dyskinetic type
4. Exclusion of other identifiable causes, such as trauma, coagulation disorders, infections, or genetic disorders

In two publications, Low and associates[41,42] described the association between severe or significant metabolic acidosis (determined by the umbilical artery blood gas profile) and newborn complications. Low[42] proposed a classification of intrapartum fetal asphyxia, the severity of which was based on newborn encephalopathy and other organ system dysfunction.

Other Clinical Events and Umbilical Blood Acid-Base Status

Beyond its use in assessing risk for neurologic injury with no obvious antecedent, umbilical blood gas analysis has been reported in a variety of clinical situations that entail more apparent risk, such as acute chorioamnionitis, nuchal cords, meconium-stained amniotic fluid, prolonged pregnancy, FHR anomalies, operative vaginal delivery, breech delivery, and use of oxytocin.[35] Analysis may also prove useful in assessing the interval from delivery of the head to complete delivery in cases of shoulder dystocia.

Acute Chorioamnionitis. In one study of 123 women with acute chorioamnionitis, compared with more than 6000 noninfected women, Maberry and coauthors[43] found no significant association between infection and fetal acidemia (Table 33-7). Hankins and colleagues[44] found no association between acute chorioamnionitis and newborn acidemia. Meyer and colleagues,[45] however, reported an association between neonatal blood cultures within the first 24 hours of life as a proxy for fetal sepsis and a decrease in umbilical artery pH compared with controls (7.21 versus 7.26).

TABLE 33-7 Umbilical Artery pH in Patients with or without Acute Chorioamnionitis

Umbilical Artery pH	Patients with Chorioamnionitis ($n = 123$)	Controls ($n = 6769$)
<7.20	18 (15.0%)	701 (10.0%)
<7.15	4 (3.0%)	242 (4.0%)
<7.00	0	6 (0.1%)
Metabolic acidemia	1 (0.8%)	9 (0.1%)

From Maberry MC, Ramin SM, Gilstrap LC, et al: Intrapartum asphyxia in pregnancies complicated by intraamniotic infection, Obstet Gynecol *76:351, 1990, with permission from the American College of Obstetricians and Gynecologists.*

Nuchal Cords. In a study of 110 newborns with nuchal cords, Hankins and colleagues[46] reported that significantly more newborns with nuchal cords were acidemic (umbilical artery pH <7.20), compared with controls (20% versus 12%; $P < .05$); however, there were no significant differences in mean pH (7.25 versus 7.27), Pco_2 (49 versus 48 mm Hg), or HCO_3^- (20.5 versus 21.0 mEq/L).

Meconium-Stained Amniotic Fluid. In a study of 53 term pregnancies with moderate to thick meconium, Mitchell and colleagues[47] reported that approximately one half of the newborns were acidemic and that significantly more acidemic newborns had meconium below the cords compared with controls (32% versus 0%; $P < .05$). However, these investigators used an umbilical artery pH cutoff of 7.25 to define acidemia.

In another report of 323 newborns with meconium by Yeomans and associates,[48] the frequency of meconium below the cords in acidemic fetuses was significantly increased compared with that for nonacidemic fetuses (31% versus 18%; $P < .05$). Meconium aspiration syndrome, however, was an uncommon event, occurring in only 3% of newborns. Ramin and colleagues,[49] reported that 55% of meconium aspiration syndrome cases were newborns with an umbilical artery pH greater than 7.20.

In a review of 4985 term neonates born to mothers with meconium-stained amniotic fluid, Blackwell and colleagues[50] identified 48 cases of severe meconium aspiration syndrome in which umbilical artery pH measurements were obtained. The pH was 7.20 or higher in 29 of these patients and less than 7.20 in 19. There was no difference in frequency of seizures between the two pH groups. The investigators concluded that severe meconium aspiration syndrome occurred in the setting of normal acid-base status at delivery in many of the cases, suggesting that a "preexisting injury or a nonhypoxic mechanism is often involved."[50]

Prolonged Pregnancy. In a study of 108 women with a prolonged pregnancy (≥41 weeks' gestation), Silver and colleagues[51] reported a mean umbilical artery pH of 7.25. Moreover, significantly more newborns who were delivered for FHR indications had acidemia than newborns who were not (45% versus 13%; $P < .05$).

Fetal Heart Rate Abnormalities. Gilstrap and colleagues,[52] in a study of 403 term newborns with FHR abnormalities in the second stage of labor compared with 430 control newborns, reported an association between abnormalities and acidemia (Table 33-8). This was confirmed in a follow-up study.[53] Honjo and Yamaguchi[54] also reported a correlation between second-stage baseline FHR abnormalities and fetal acidemia at birth. Although there may be an association between FHR abnormalities and acidemia, association with adverse long-term neurologic outcomes is uncommon. Nelson and colleagues[55] reported a population-based study of more than 115,000 children with birth weights of 2500 g or more, 78 of whom developed cerebral palsy and had electronic fetal monitoring during labor. Multiple late decelerations and decreased beat-to-beat variability were associated with an increased risk of cerebral palsy. However, of

TABLE 33-8 Umbilical Artery Acidemia and Second-Stage Fetal Heart Rate Abnormalities

Fetal Heart Rate Pattern	Number of Newborns	Umbilical Artery pH <7.20
Tachycardia*	117	15%
Mild bradycardia*	165	18%
Moderate or marked bradycardia*	121	27%
Normal	430	4%

*$P < .0001$ compared with normal patterns.
Modified from Gilstrap LC, Hauth JC, Toussaint S: Second stage fetal heart rate abnormalities and neonatal acidosis, Obstet Gynecol *63:209, 1984, with permission from the American College of Obstetricians and Gynecologists.*

TABLE 33-9 Method of Delivery and Fetal Acidemia

Method of Delivery	Number of Newborns	% with Acidemia*
Spontaneous	303	7
Elective outlet/low forceps	177	9
Indicated outlet/low forceps	293	18
Indicated mid-forceps	234	21
Cesarean delivery	111	18

*Umbilical artery pH <7.20.
From Gilstrap LC, Hauth JC, Shiano S, et al: Neonatal acidosis and method of delivery, Obstet Gynecol *63:681, 1984, with permission from the American College of Obstetricians and Gynecologists.*

all the children with these abnormal FHR findings, only 0.19% developed cerebral palsy.

Method of Delivery. Gilstrap and coworkers[56] found no significant difference in the frequency of newborn acidemia according to the method of delivery (Table 33-9). This was true even when the indication for delivery was concern about the FHR monitoring information.

Although the mean umbilical artery pH was lower for infants delivered vaginally in breech presentations compared with cephalic presentations in two studies,[57,58] in a total of 121 breech vaginal deliveries, pH levels were not significantly low from a clinical standpoint (7.23 and 7.16, respectively). The investigators in both studies concluded that uneventful vaginal breech labor and delivery at term was not associated with an increased risk of asphyxia.

Shoulder Dystocia. Most adverse outcomes associated with shoulder dystocia result from physical injury to the brachial plexus,[59] not acidemia or asphyxia (unless an extremely protracted period is needed to extract the fetus). In a review of 134 infants born after shoulder dystocia, Stallings and colleagues[60] reported that this complication was associated with a "statistically significant but clinically insignificant" reduction in mean umbilical artery pH levels compared with their obstetric population (7.23 versus 7.27).

Effect of Oxytocin. In a study of 556 women who received oxytocin compared with 704 who did not, Thorp and colleagues[61] found no significant difference in mean umbilical artery pH measurements (7.23 and 7.24, respectively).

MEASURING ACID-BASE STATUS

The fetus maintains its pH within a very limited range and depends on the placenta and the maternal circulation to maintain acid-base balance. Several methods for assessing fetal or newborn acid-base status have been described. Umbilical blood gas analysis is probably the most useful, the easiest, and the least expensive to perform.

Few data are available to justify a policy of umbilical blood gas analysis for all newborns. In a survey of 133 universities in the United States, Johnson and Riley[62] reported that approximately 27% of centers used cord blood for assessing newborn acid-base status in all deliveries. Two thirds of the programs used umbilical blood for tracing abnormal FHRs or for low Apgar scores. The Royal College of Obstetricians and Gynecologists and the Royal College of Midwives[63] recommend routine cord blood measurements for all cesarean deliveries and instrumental deliveries for fetal distress. The ACOG[40] recommends umbilical cord blood acid-base analysis in the following situations:

- Cesarean delivery for fetal compromise
- Low 5-minute Apgar score
- Severe growth restriction
- Abnormal FHR tracing
- Maternal thyroid disease
- Intrapartum fever
- Multifetal gestations

Characteristics of Fetal Heart Rate Patterns

BASIC PATTERNS

Characteristics of the FHR pattern are classified as baseline features and as periodic or episodic changes.[64,65] The baseline features are those recorded between uterine contractions. Periodic changes are associated with uterine contractions, and episodic changes are those not obviously associated with uterine contractions.

Baseline Features

The baseline features of the FHR are predominant characteristics that can be recognized between uterine contractions. They are the baseline rate and variability of the FHR.

Baseline Rate. The *baseline rate* is the FHR recorded between contractions. More accurately described, it is the approximate mean FHR rounded to 5 beats/min during a 10-minute segment, excluding the periodic or episodic changes, periods of marked FHR variability, and segments of the baseline that differ by at least 25 beats/min. In any 10-minute window, the minimum baseline duration must be at least 2 minutes; otherwise, the baseline for that period is indeterminate.

The normal baseline FHR is between 110 and 160 beats/min. Rates slower than 110 beats/min are called *bradycardia,* and rates faster than 160 beats/min are called *tachycardia.* Baseline bradycardia and tachycardia are quantified by the actual rate observed in keeping with the definition of baseline rate.

Fetal Heart Rate Variability. Electronic FHR monitoring (EFM) in most cases produces a tracing with an irregular line that demonstrates FHR variability. The irregularities represent

the slight differences in the time interval and calculated FHR that occur from beat to beat. If all intervals between heartbeats were identical, the line would be straight. Fluctuations in the baseline FHR are irregular in amplitude and frequency. A sinusoidal pattern (discussed later) is different from variability in that it has a smooth sine wave of regular frequency and amplitude and is therefore excluded from the definition of FHR variability.

Baseline variability is defined as fluctuations in the FHR of two or more cycles per minute and is quantitated as the peak-to-trough amplitude of the FHR in beats per minute. Variability is *absent* when the amplitude range is undetectable. It is *minimal* when there is an amplitude range that is less than 5 beats/min. Variability is *normal* or *moderate* when the amplitude range is between 6 and 25 beats/min. Variability is *marked* when the amplitude is greater than 25 beats/min[66] (see Classification and Significance of Baseline Variability, later).

Periodic Heart Rate Patterns

Periodic patterns are the alterations in FHR that are associated with uterine contractions or changes in blood flow within the umbilical cord vessels. These patterns include late decelerations, early decelerations, variable decelerations, and accelerations. In each case, the decrease or increase in the FHR is calculated from the most recently determined portion of the baseline.

Late Decelerations. In late deceleration of the FHR, there is a visually apparent decrease and subsequent return to the baseline FHR that is associated with a uterine contraction. The decrease is gradual; the time from onset of deceleration to nadir is at least 30 seconds. Its timing is delayed, with the nadir of the deceleration occurring late in relation to the peak of the uterine contraction. In most cases, the onset, nadir, and recovery are all late in relation to the beginning, peak, and ending of the contraction, respectively.

Early Decelerations. Early deceleration of the FHR is similar to late deceleration, except that the decrease is coincident in timing, with the nadir of the deceleration occurring at the same time as the peak of the uterine contraction. In most cases, the onset, nadir, and recovery are all coincident with the beginning, peak, and ending of the contraction, respectively.

Variable Decelerations. Variable deceleration is a visually apparent, abrupt decrease (<30 seconds from onset of deceleration to beginning of nadir) in FHR from the baseline. The decrease in FHR is at least 15 beats/min, and its duration from baseline to baseline is at least 15 seconds but not more than 2 minutes. When variable decelerations are associated with uterine contractions, their onset, depth, and duration commonly vary with successive contractions.

Prolonged deceleration is a visually apparent, abrupt decrease in FHR below the baseline of at least 15 beats/min that has a duration between 2 and 10 minutes from onset to return to baseline. If a deceleration lasts 10 minutes or longer, it is a baseline change.

Accelerations. Acceleration is a visually apparent, abrupt increase (<30 seconds from onset of acceleration to peak) in FHR above the baseline. The acme is at least 15 beats/min above the baseline, and the acceleration lasts between 15 seconds and 2 minutes from onset to return to baseline. A prolonged acceleration is one that lasts at least 2 minutes but less than 10 minutes. If an acceleration lasts for 10 minutes or longer, it is a baseline change.

Accelerations are closely associated with normal FHR variability. Sometimes, it may be difficult to decide whether a recorded pattern represents an acceleration or a normal, long-term variability complex. The final decision is not important, because both have the same reassuring prognostic significance, indicating normal fetal oxygenation.

Quantification. Deceleration is quantified by the depth of the nadir in beats per minute below the baseline. Duration is quantified in minutes and seconds from the beginning to the end of the deceleration. Acceleration is quantified similarly. Decelerations are defined as recurrent or persistent if they occur with more than 50% of uterine contractions in any 20-minute period. Bradycardia and tachycardia are quantified by the FHR in beats per minute.

NORMAL AND ABNORMAL HEART RATE PATTERNS

In the fetus, the normal heart rate pattern (Fig. 33-1) has a baseline FHR of between 110 and 160 beats/min, an FHR variability amplitude between 6 and 25 beats/min, and no decelerative periodic changes, although there may be periodic or episodic accelerations. It is widely accepted in clinical practice that a newborn is normally oxygenated if this normal FHR pattern is traced at the time of delivery.[4,67,68]

In contrast to the high predictability of fetal normoxia and vigor in the setting of the normal pattern, variant patterns are not as accurate for predicting fetal compromise. However, when these patterns are placed in the context of the clinical case (e.g., progressive change in the patterns, duration of the variant patterns), reasonable judgments can be made about the likelihood of fetal decompensation. With this screening approach, impending intolerable fetal acidosis can be presumed or, in certain cases, ruled out by the use of ancillary techniques (e.g., fetal scalp stimulation, vibroacoustic stimulation).

As a predictor of significant neurologic morbidity such as cerebral palsy, EFM has a very poor specificity and positive predictive value. The positive predictive value of a non-reassuring FHR is 0.14%. This means that for every 1000 fetuses born with a non-reassuring FHR tracing, 1 or 2 of them develop cerebral palsy.[55] The false-positive rate is greater than 99%. However, these results are from cases in which the clinicians were aware of the FHR patterns and managing the patients accordingly.

Baseline Rate

Bradycardia. Bradycardia is a baseline FHR slower than 110 beats/min. Some fetuses have a baseline FHR of less than 110 beats/min and are cardiovascularly normal. Their baseline FHR represents a variation outside the limits of normal. Others with an FHR slower than 110 beats/min may have congenital heart block and a well-compensated status despite a low FHR.

Bradycardia is related to baseline FHR and is distinguished from a deceleration. However, a prolonged deceleration resulting in a new baseline bradycardia may result from vagal activity in response to fetal hypoxia (Fig. 33-2). Decreases in FHR may be caused by the following:

- Sudden drop in oxygenation, such as occurs with placental abruption, maternal apnea, or amniotic fluid embolus

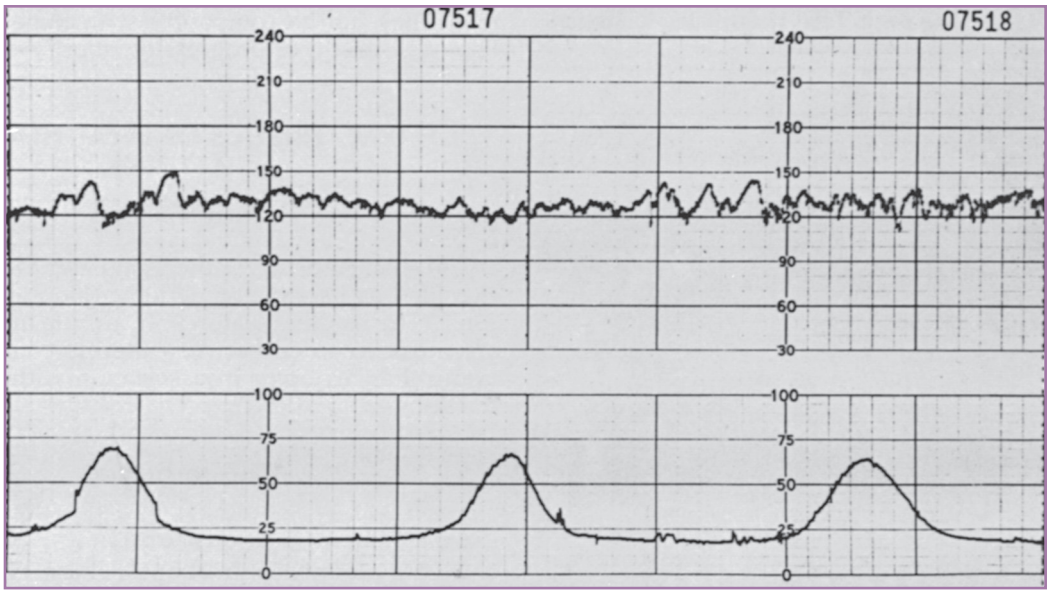

Figure 33-1 Normal fetal heart rate pattern. The tracing exhibits a normal rate (about 130 beats/min), normal variability (amplitude range about 15 beats/min), and absence of periodic changes. This pattern represents a nonacidemic fetus without evidence of hypoxic stress. Uterine contractions are 2 to 3 minutes apart and about 60 to 70 mm Hg in intensity.

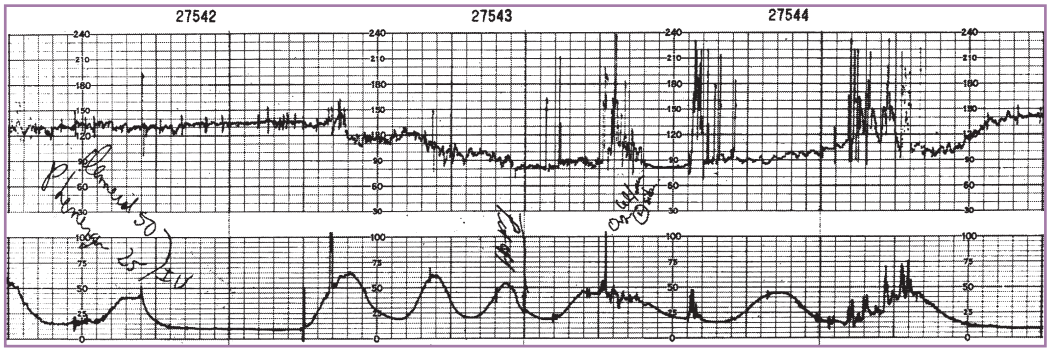

Figure 33-2 Prolonged fetal bradycardia. The prolonged fetal bradycardia resulted from excessive oxytocin-induced hyperstimulation of the uterus after intravenous infusion of meperidine (Demerol) and promethazine (Phenergan) into the same tubing. The heart rate is returning to normal at the end of the tracing after appropriate treatment (signified by the notes *Pit off, O_2 6 L/min,* and *side*). Notice that fetal heart rate variability was maintained throughout this asphyxial stress, signifying adequate central oxygenation.

- Decrease or cessation in umbilical blood flow, such as occurs with a prolapsed cord or uterine rupture
- Decrease in uterine blood flow, such as occurs with severe maternal hypotension

Tachycardia. Tachycardia is a baseline FHR faster than 160 beats/min. A duration of at least 10 minutes distinguishes it from an acceleration. With tachycardia, loss of FHR variability is common. Although fetal tachycardia is potentially associated with fetal hypoxia, particularly when it is accompanied by decelerations of the FHR, the more common association is with maternal fever or fetal infection (e.g., chorioamnionitis). In most instances, the fetus is not hypoxic but has an elevated baseline FHR.

It is not uncommon for the FHR baseline to rise in the second stage of labor. Certain drugs also cause tachycardia, such as β-mimetic agents used for attempted tocolysis or illicit drugs such as methamphetamine and cocaine.

Tachycardia should not be confused with the uncommon finding of a fetal cardiac tachyarrhythmia, in which the FHR is faster than 240 beats/min. These arrhythmias may be intermittent or persistent, and they are the result of abnormalities of the intrinsic determinants of cardiac rhythm. Findings of supraventricular tachyarrhythmias should be monitored closely and possibly treated with medical therapy or delivery, because they may be associated with deterioration of the fetal status.

Classification and Significance of Baseline Variability

Based on the amplitude range, FHR variability may be described as absent, minimal, moderate, or marked. The moderate (normal) amplitude range is between 6 and 25 beats/min. If the FHR variability is normal, regardless of what other FHR patterns may be present, the fetus is not experiencing cerebral tissue acidemia because the fetus can centralize the available oxygen and is physiologically compensated. However, if excessive hypoxic stress persists, this compensation may break down,

and the fetus may have progressive hypoxia in cerebral and myocardial tissues. In these cases, the FHR variability decreases and eventually is lost.

There are several possible nonhypoxic causes of decreased or absent FHR variability:

1. Absence of the cortex (i.e., anencephaly)
2. Narcotized or drugged higher centers (e.g., morphine, meperidine, diazepam) (Fig. 33-3)
3. Vagal blockade (e.g., atropine, scopolamine)
4. Defective cardiac conduction system (e.g., complete heart block) (Fig. 33-4)

Periodic Changes in Fetal Heart Rate

Late Decelerations. The two varieties of late decelerations are reflex and nonreflex (Fig. 33-5).[4,69-71] *Reflex late deceleration* sometimes occurs when an acute insult (e.g., reduced uterine blood flow resulting from maternal hypotension) is superimposed on a previously normally oxygenated fetus in the setting of contractions. These late decelerations are caused by a decrease in uterine blood flow (with the uterine contraction) beyond the capacity of the fetus to extract sufficient oxygen. The relatively deoxygenated fetal blood is carried from the placenta through

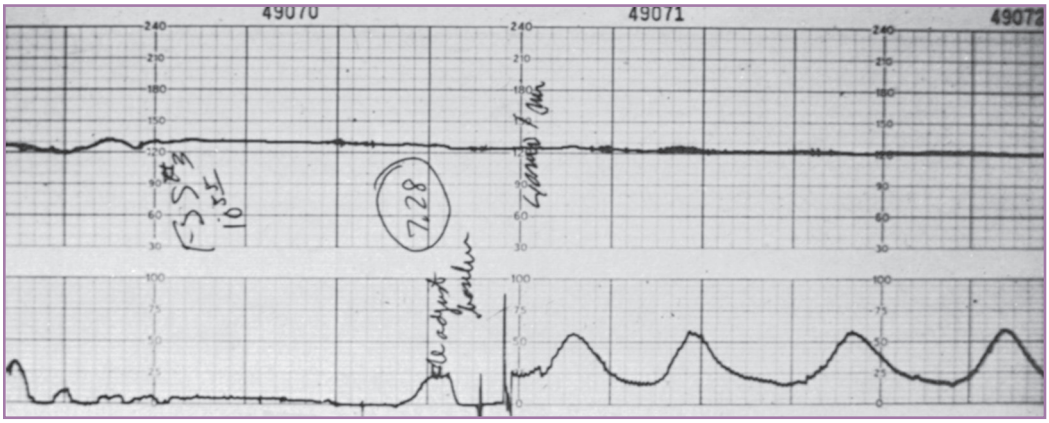

Figure 33-3 No variability of the fetal heart rate. The mother had severe preeclampsia and was receiving magnesium sulfate and narcotics. The normal scalp blood pH (7.28) ensures that the absence of variability is nonasphyxic in origin and that the fetus is not chronically asphyxiated and decompensating. The uterine activity channel has an inaccurate trace in the first half.

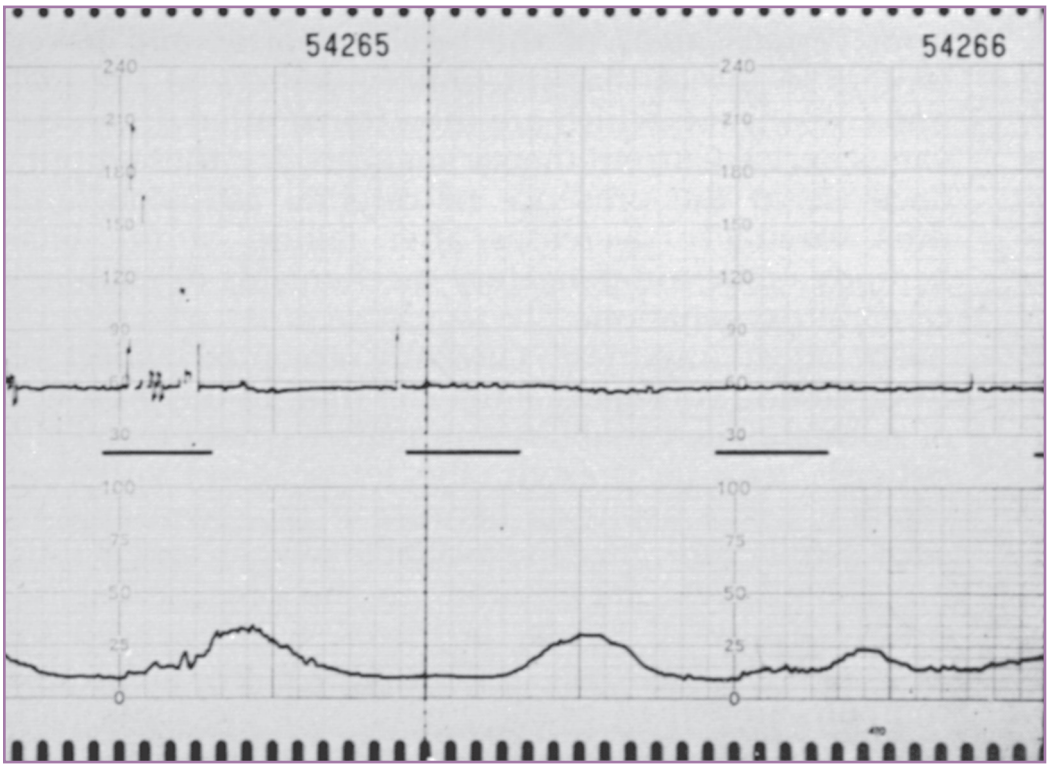

Figure 33-4 Unremitting fetal bradycardia. This tracing does not signify asphyxia, because this fetus had complete heart block, with a ventricular rate of about 55 beats/min. Notice the absence of fetal heart rate variability. There were serious cardiac structural defects, and the fetus died shortly after birth.

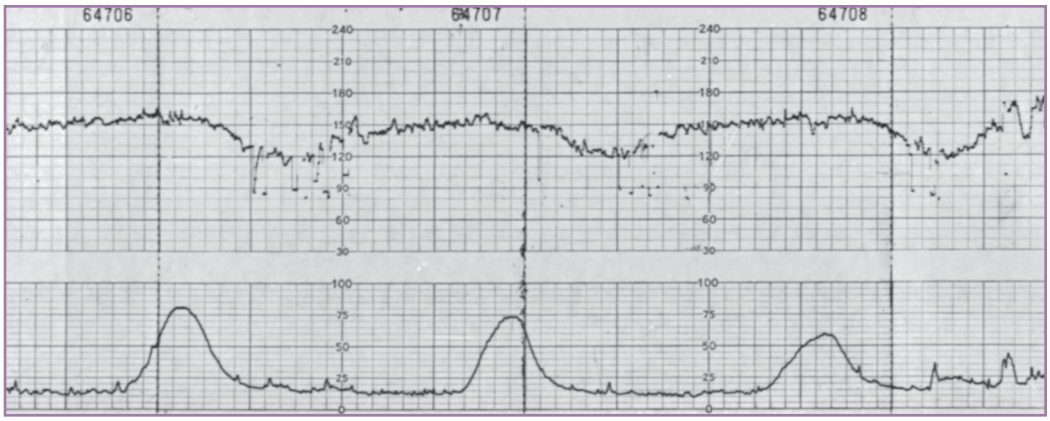

Figure 33-5 Late decelerations. The decelerations were recorded by Doppler ultrasound in the antepartum period in a severely growth-restricted (1700-g) term infant born to a 32-year-old preeclamptic primipara. Delivery was done by cesarean section because neither a direct fetal electrocardiogram nor a fetal blood sample could be obtained due to a firm, closed posterior cervix. The infant subsequently did well.

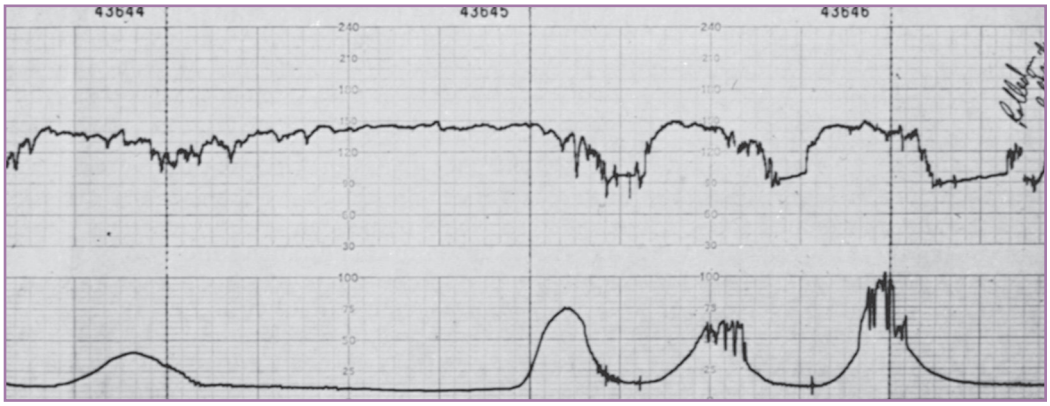

Figure 33-6 Reflex late decelerations. The fetal heart rate pattern was previously normal, but late decelerations appeared after severe maternal hypotension (70/30 mm Hg), which resulted from sympathetic blockade caused by a caudal anesthetic agent.

the umbilical vein to the heart and is distributed to the aorta, neck vessels, and head. The low Po_2 is sensed by chemoreceptors, and neuronal activity results in a vagal discharge that causes the transient deceleration. The deceleration is presumed to be late because of the circulation time from the fetal placental site to the chemoreceptors and because the progressively decreasing Po_2 must reach a certain threshold before vagal activity occurs. Baroreceptor activity also may cause the vagal discharge.[69] Because oxygen delivery is adequate and there is no additional vagal activity between contractions, the baseline FHR is normal. These late decelerations are accompanied by normal FHR variability and signify normal central nervous system integrity (i.e., vital organs are physiologically compensated) (Fig. 33-6).

The second type of late deceleration results from the same initial mechanism, except that the deoxygenated bolus of blood from the placenta is presumed to be insufficient to support myocardial action. For the period of the contraction, there is direct myocardial hypoxic depression (or failure) and vagal activity.[69,71] These *nonreflex late decelerations* occur without FHR variability (Fig. 33-7), signifying fetal decompensation (i.e., inadequate cerebral and myocardial oxygenation). They are seen most commonly in states of decreased placental reserve (e.g., preeclampsia, intrauterine growth restriction) or after prolonged hypoxic stress (e.g., long period of severe reflex late decelerations).

Further support for the two mechanisms of late decelerations comes from observations of chronically catheterized fetal monkeys in spontaneous labor during the course of intrauterine death.[72] The animals initially had normal blood gas values, normal FHR variability, FHR accelerations, and no persistent periodic changes. After various periods, they first demonstrated late decelerations and retained accelerations. This period was associated with a small decline in Po_2 in the ascending aorta (28 to 24 mm Hg) and a normal acid-base state. These late decelerations were probably vagal reflex types caused by chemoreceptor activity. At an average of more than 3 days after the onset of these reflex decelerations, accelerations were lost in the setting of worsening hypoxia (Po_2 = 19 mm Hg) and acidemia (pH = 7.22). Fetal death followed an average of 36 hours of persistent late decelerations without accelerations, which were presumed to be nonreflex decelerations associated with myocardial depression.

Late decelerations should prompt efforts to optimize placental blood flow and maternal oxygenation. The clinician should ensure that maternal blood pressure is normal.

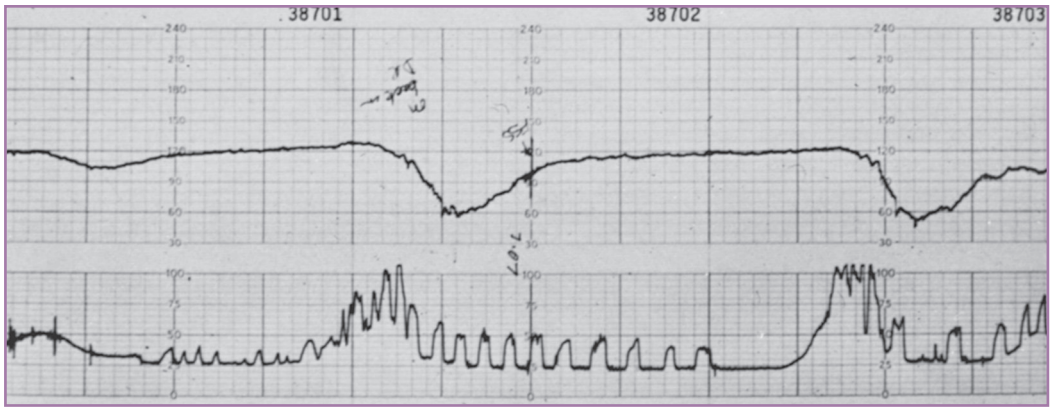

Figure 33-7 Nonreflex late decelerations with virtual absence of fetal heart rate (FHR) variability. The decelerations represent transient asphyxic myocardial failure and intermittent vagal decreases in heart rate. The lack of FHR variability also signifies decreased cerebral oxygenation. Notice the acidemia in fetal scalp blood (pH = 7.07). The infant, a 3340-g girl with Apgar scores of 3 (1 minute) and 4 (5 minutes), was delivered soon after this tracing. Cesarean section was considered to be contraindicated because of a severe preeclamptic coagulopathy.

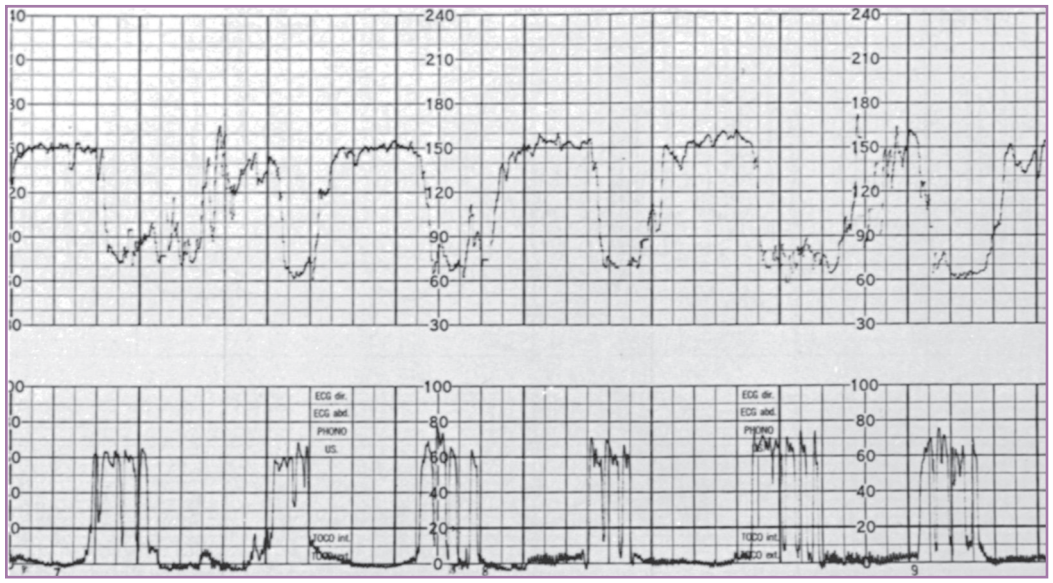

Figure 33-8 Variable decelerations. The intrapartum recording used a fetal scalp electrode and tocodynamometer. The spikes in the uterine activity channel represent maternal pushing efforts in the second stage of labor. The normal baseline variability between contractions signifies normal central oxygenation despite the intermittent hypoxic stress represented by the moderate variable decelerations.

Variable Decelerations. Variable decelerations (Fig. 33-8) have the following characteristics:

- They vary in duration, depth, and shape.
- Onset and cessation usually are abrupt.

Classification of Fetal Heart Rate Tracings. A three-tiered system for FHR pattern categorization is recommended by ACOG.[73] The three categories are described in Box 33-1.

Effect of in Utero Treatment. Fetal oxygenation can be improved, acidemia relieved, and variant FHR patterns abolished by certain modes of treatment. The events that result in fetal stress (recognized by FHR patterns) are provided in Table 33-10 with the recommended treatment maneuvers and presumed mechanisms for improving fetal oxygenation. They should be the primary maneuvers carried out. If the hypoxic event is acute and the fetus was previously normoxic, there is an excellent chance that the undesired FHR pattern will be abolished.

If the FHR pattern cannot be improved (i.e., nonreassuring patterns suggesting peripheral or central tissue hypoxia persist for a significant period), further diagnostic steps or delivery may be indicated. Certain severe patterns warrant immediate delivery if they cannot rapidly be relieved (Figs. 33-9 and 33-10).

Other Heart Rate Patterns

Sinusoidal Pattern. The sinusoidal pattern has a regular, smooth, sine wave–like baseline with a frequency of approximately 3 to 6 cycles per minute and an amplitude range of up to 30 beats/min that persists for 20 minutes or longer. Another distinguishing feature is the absence of beat-to-beat or short-term variability (Fig. 33-11).

BOX 33-1 THREE-TIERED FETAL HEART RATE INTERPRETATION SYSTEM

CATEGORY I

Category I FHR tracings include all of the following:

- Baseline rate: 110-160 beats per minute
- 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 with no 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 more than 2 minutes but less than 10 minutes
  - Recurrent late decelerations with moderate baseline variability
  - Variable decelerations with other characteristics, such as slow return to baseline, overshoots, or shoulders

CATEGORY III

Category III FHR tracings include either one of the following:

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

FHR, fetal heart rate.

From Macones GA, Hankins GD, Spong CY, et al: The 2008 National Institute of Child Health and Human Development workshop report on electronic fetal monitoring: update on definitions, interpretation, and research guidelines, Obstet Gynecol *112:661, 2008.*

TABLE 33-10 Intrauterine Treatment for Variant Fetal Heart Rate Patterns

Causes	Possible Resulting FHR Patterns	Corrective Maneuver	Mechanism
Hypotension (e.g., supine hypotension, conduction anesthesia)	Bradycardia, late decelerations	Intravenous fluids, position change, ephedrine	Return of uterine blood flow to normal
Excessive uterine activity	Bradycardia, late decelerations	Decrease in oxytocin, lateral position	Return of uterine blood flow to normal
Transient umbilical cord compression	Variable decelerations	Change in maternal position (e.g., left or right lateral, Trendelenburg)	Presumably removes fetal part from cord
		Amnioinfusion	Relieves compression of cord
Head compression	Early or variable decelerations	Push only with alternate contractions	Allows fetal recovery
Decreased uterine blood flow associated with uterine contraction	Late decelerations	Change in maternal position (e.g., left lateral, Trendelenburg)	Enhanced uterine blood flow toward optimum
		Tocolytic agents (e.g., terbutaline)	Decreased contractions or tone
Prolonged asphyxia	Decreasing FHR variability*	Change in maternal position (e.g., left lateral, Trendelenburg), establishment of maternal hyperoxia	Enhanced uterine blood flow toward optimum, increase in maternal-fetal oxygen gradient

*During labor, this usually is preceded by a heart rate pattern signifying asphyxial stress (e.g., late decelerations, usually severe), severe variable decelerations, or a prolonged bradycardia. This is not necessarily so in the antepartum period before the onset of uterine contractions.
FHR, Fetal heart rate.

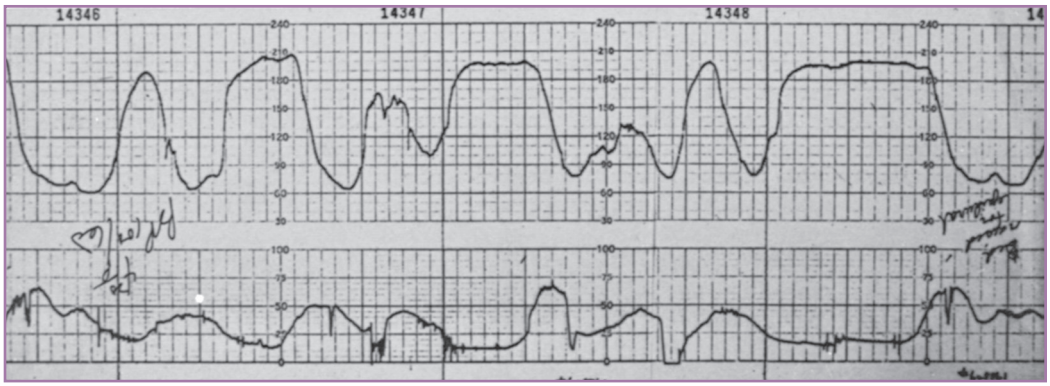

Figure 33-9 Sinister heart rate pattern in a 28-week fetus (gestational age determined after delivery) with baseline tachycardia, absence of heart rate variability, and severe periodic changes. The scalp blood pH was 7.0, and the fetus died shortly after this tracing was made. Cesarean section was not performed because the fetus was thought to be previable, although it weighed 1100 g. There is much artifact in the uterine activity channel.

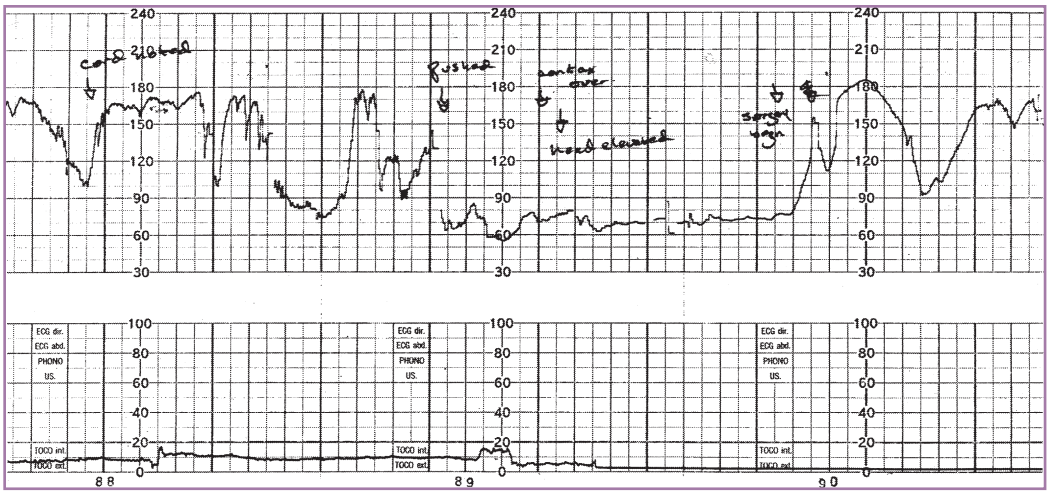

Figure 33-10 Bradycardia resulting from cord prolapse. The infant was delivered by cesarean section and did well.

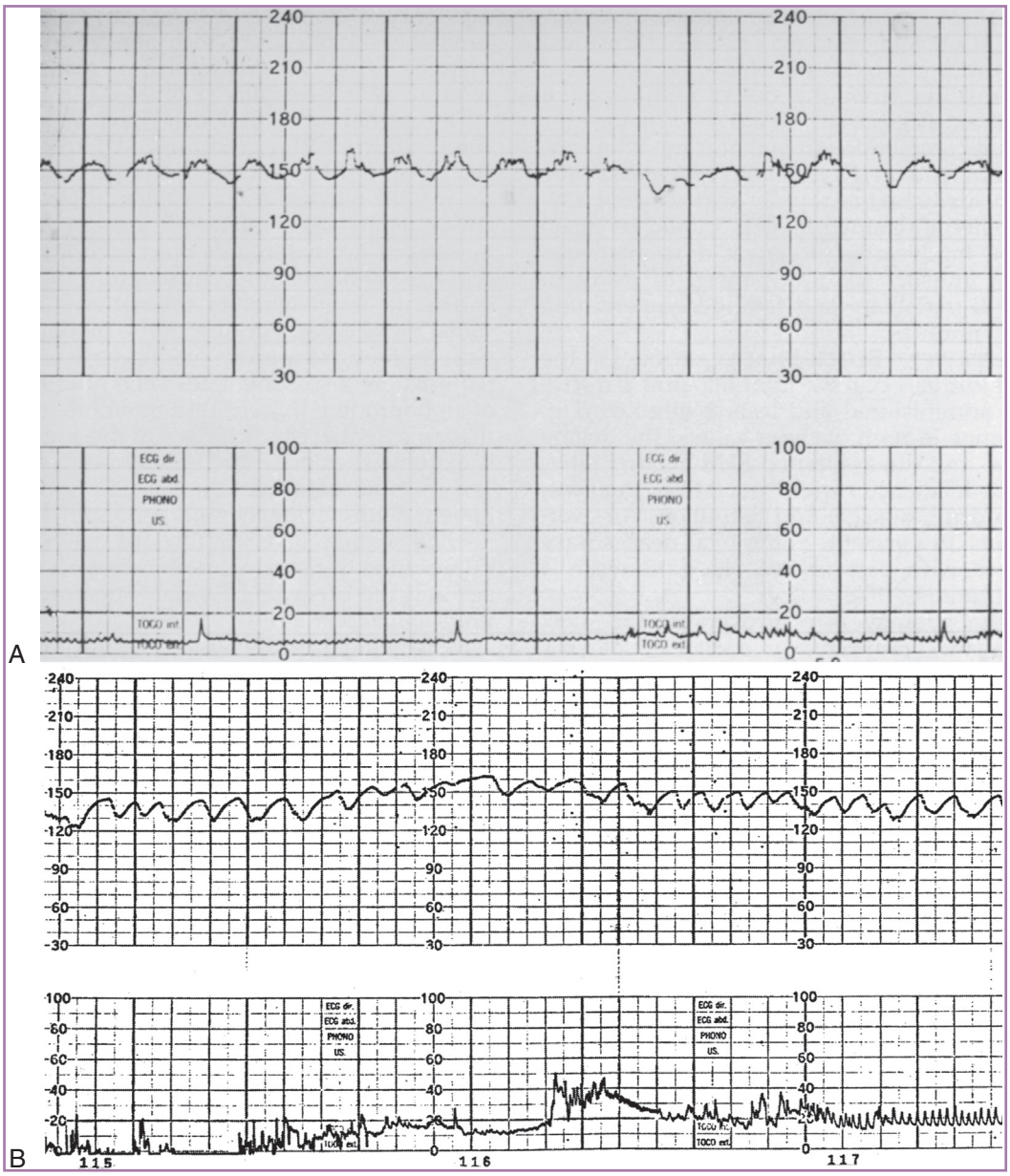

Figure 33-11 Sinusoidal pattern. A and B, Sinusoidal pattern in a term fetus with severe hemolysis caused by Rh disease. The cord hematocrit was 20%, and the infant, delivered by cesarean section, was subsequently normal. Recording was done by direct fetal electrode.

The sinusoidal pattern was first described in a group of severely affected Rh-alloimmunized fetuses but was subsequently identified in fetuses that were severely anemic for other reasons and in severely depressed infants. An essential characteristic of the sinusoidal pattern is extreme regularity and smoothness. Murata and colleagues[74] implicated elevated levels of arginine vasopressin in producing the sinusoidal pattern. A sinusoidal pattern or variant in an Rh-sensitized patient usually suggests anemia with a fetal hematocrit value of less than 20%.[75] Hydrops in the fetus suggests a fetal hematocrit of 15% or lower. Many severely anemic, Rh-affected fetuses do not have a sinusoidal pattern but do have a rounded, blunted pattern, and accelerations are usually absent.

Rapid intervention is needed when a sinusoidal pattern is seen in an Rh-sensitized patient and severe hemolysis is confirmed by peak systolic velocity measurement of flow in the middle cerebral artery of the fetus, by cordocentesis, or by the deviation in the amniotic fluid optical density at 450 nm determined by spectrophotometry. Intervention may take the form of delivery or intrauterine transfusion, depending on gestational age and the fetal status (see Chapter 36).

Management of a sinusoidal pattern in the absence of alloimmunization is somewhat more difficult to recommend. If the pattern is persistent, monotonously regular, and unaccompanied by variability and cannot be abolished by the maneuvers described, further workup and evaluation of the adequacy of fetal oxygenation are indicated using the contraction stress test, fetal stimulation test, biophysical profile, or fetal blood sampling. Nonalloimmune sinusoidal patterns have been associated with severe fetal acidemia and with fetal anemia resulting from fetal-maternal bleeding. The latter diagnosis is supported by identification of fetal red blood cells in maternal blood, often subsequently detected by the Kleihauer-Betke test.

Saltatory Pattern. The saltatory pattern consists of rapid variations in FHR with a frequency of 3 to 6 cycles per minute and an amplitude range greater than 25 beats/min (Fig. 33-12). It is qualitatively described as *marked variability*, and the variations have a strikingly bizarre appearance. The saltatory pattern is seen during labor rather than in the antepartum period. The cause is uncertain, but it may be similar to that of the increased FHR variability seen in animal experiments with brief and acute hypoxia in a previously normoxic fetus. Efforts should be made to optimize placental blood flow and fetal oxygenation if this pattern appears during labor.

CONGENITAL ANOMALIES

Except as described for dysrhythmias, most fetuses with congenital anomalies have essentially normal FHR patterns and respond to hypoxia in a manner similar to the normal fetus. There are several exceptions, including complete heart block and anencephaly. Aneuploid fetuses and fetuses with hypoplastic lungs, meningomyelocele, or hydrocephalus may give no FHR warning of such underlying defects, because they are not necessarily experiencing hypoxia or acidosis. Even though there was no pathognomonic pattern in these fetuses, the rate of cesarean section for fetal intolerance to labor was significantly increased, presumably because of abnormal FHR patterns during or preceding labor.[76]

Efficacy, Risks, and Recommendations for Monitoring

ELECTRONIC MONITORING VERSUS AUSCULTATION

Because there are no prospective, randomized clinical trials comparing EFM with no fetal heart monitoring during labor, most efforts to suggest its efficacy have relied on research reports comparing EFM with intermittent auscultation. The standard for efficacy usually is a decrease in complications, which for FHR monitoring may include fetal death in labor or severe neonatal and pediatric morbidity (e.g., neonatal seizures, cerebral palsy). Ideally, the improved outcomes are accompanied by appropriate interventions and restraint from inappropriate interventions.

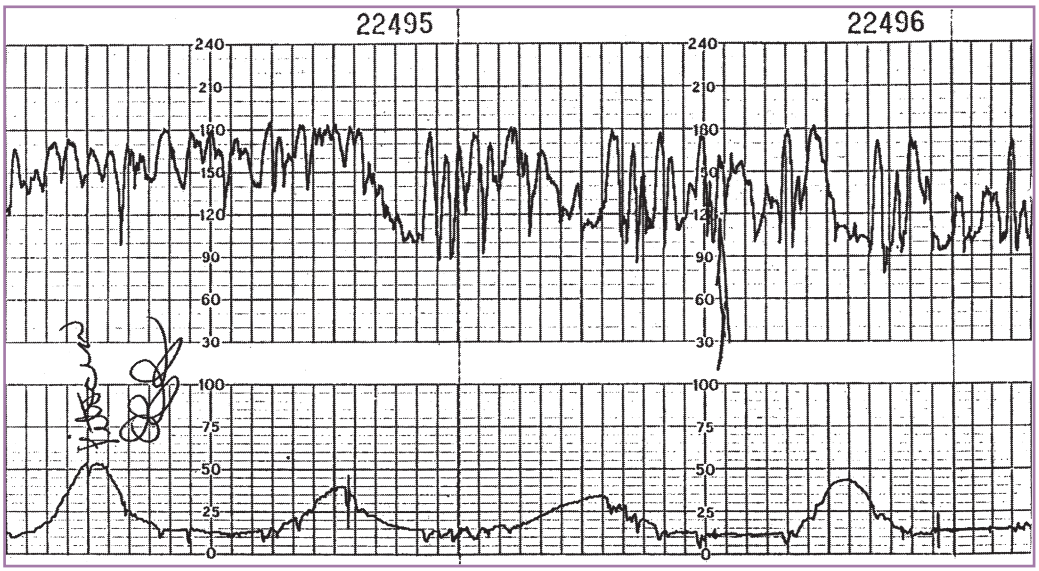

Figure 33-12 Saltatory pattern. The saltatory pattern, which shows excessive fetal heart rate variability of up to 60 beats/min in brief intervals, probably represents mild hypoxic stress.

In a meta-analysis of the nine published clinical studies comparing EFM with intermittent auscultation of the FHR, several conclusions were reached.[77] In several of these trials, patients with high-risk conditions were not randomized for study inclusion. The use of EFM was associated with significant increases in the rate of cesarean delivery for fetal intolerance to labor, in the overall cesarean delivery rate, and in the use of instrumentation (i.e., vacuum and forceps) for vaginal delivery. However, there was no reduction in overall perinatal mortality for these patients. Because of these findings, either option for monitoring the fetus during labor is acceptable for patients not considered to be at high risk.[66] The optimal frequency for intermittent auscultation in low-risk patients has not been established, but at a minimum, the FHR should be assessed at least every 30 minutes in the first stage of labor and every 15 minutes in the second stage.[66] Another method is to auscultate and record the FHR every 15 minutes in the active first stage of labor and every 5 minutes in the second stage, without limiting this approach to low-risk patients.[78]

ADJUNCTS TO ELECTRONIC FETAL HEART RATE MONITORING

When EFM was introduced into clinical practice more than 40 years ago, it was expected to identify fetuses at risk for impending stillbirth or damaging asphyxia in time to prevent problems and improve perinatal and neonatal outcomes. However, several well-designed, randomized clinical studies from different clinical centers involving almost 40,000 women in labor have demonstrated that intrapartum EFM, when compared with intermittent auscultation, does not result in any measurable improvement in outcomes.[79] This disappointing result suggests that the thinking about the risks associated with intrapartum events and adverse long-term neurologic outcomes should be reevaluated.

These outcome studies did reveal a strong correlation between certain EFM patterns and fetal acidosis as measured by the base deficit and the umbilical artery pH. In considering the specific outcome of neonatal seizures, several trials reported that there were significantly fewer such cases after intrapartum EFM than after labors monitored with intermittent auscultation. This end point, however, is not a good surrogate for long-term neurologic brain damage, because in many of these cases, damage was not evident with repeat examinations as the children grew older.

Because of the work intensity demanded of the nursing staff, the diminishing number of nurses available in hospital labor and delivery units, and the cost of this approach, it is not practical to offer most laboring patients the option of intermittent auscultation. This is particularly true for high-risk patients. As a result, EFM has become the default option for most modern obstetric units. Despite its limitations and disappointments, EFM will likely continue to be used in the management of labor for the foreseeable future.

Because of the significant lack of concordance among observers in the interpretation of monitor data showing anything but a category I pattern, efforts have been made to use EFM by providing appropriate adjuncts. Even so, it continues to have an unacceptably high false-positive rate and occasional false-negative results. Historically, fetal scalp blood sampling was used in an effort to accurately identify the fetus with an acidotic pH. Unfortunately, this cumbersome and technically challenging procedure was associated with complications, and because it ran into stiff regulatory headwinds, it is no longer an option in most hospitals providing obstetric care. Efforts to obtain a continuous measure of the fetal pH also have been unsuccessful for a number of reasons.

Efforts to directly assess fetal oxygenation (i.e., fetal pulse oximetry) or more closely interpret the fetal ECG (i.e., ST-waveform analysis) have been studied as complementary technologies to improve sensitivity and specificity for the prediction of fetal intrapartum hypoxia or acidosis. Adults experiencing metabolic acidosis, anaerobic metabolism, and hypoxia of the myocardium demonstrate changes in the ECG. There may be depression or elevation of the ST segment and T-wave changes. Increases in the ST segment and T wave of the fetal ECG in response to hypoxia have been demonstrated in fetal animal studies.[80] The T-wave height compared with QRS height (T/QRS ratio) can be used to express these changes.[81]

ST analysis (STAN, Neoventa Medical, Moelndal, Sweden) is a fetal monitoring technology that has been developed to assess the basic physiologic changes associated with hypoxia. STAN combines the routine visual assessment of the intrapartum EFM tracing with an automated analysis of the fetal ECG using a modified, gold-plated fetal scalp electrode. STAN performs a computer analysis of the fetal ECG specific to the detection of ST-segment changes that may predict fetal hypoxia during labor. In a randomized, controlled trial, Swedish investigators reported that fetal ECG analysis using STAN combined with standard EFM techniques lowered the rates of operative delivery for fetal intolerance to labor, severe fetal metabolic acidosis (i.e., pH <7.05 and base deficit >12 mmol/L), and neonatal encephalopathy compared with EFM alone for term laboring patients who were deemed to be candidates for continuous EFM.[82-84] The appropriate use of STAN technology was confirmed in a U.S. report.[85] In this prospective, uncontrolled, industry-sponsored feasibility study, recently trained clinicians demonstrated the ability to appropriately apply STAN in cases requiring delivery interventions or noninterventions compared with experienced STAN users. A negative predictive value of 95.2% was reported for nonintervention in cases with nonreassuring EFM patterns but normal STAN readings and normal neonatal outcomes with umbilical arterial pH values greater than 7.12. There was an 84% agreement for intervention and a 90% agreement for nonintervention between investigators and three STAN experts when the cases were retrospectively reviewed. These reports are limited to singleton pregnancies in labor at or beyond 36 weeks' gestation.

The STAN system requires a period of time, usually 20 minutes, to assess the fetal ECG and establish the normal and abnormal parameters before it is able to identify significant changes that suggest fetal hypoxia. Other concerns include a 2% to 3% frequency of missing ST data due to poor signal quality or continuous absence of data for unclear reasons. With correct scalp electrode placement and further improvements in processing the ECG signal, it is hoped that these limitations will diminish.

A meta-analysis of five randomized, controlled trials included 15,352 patients randomized to ST-waveform analysis in combination with cardiotocography or to conventional cardiotocography alone for intrapartum fetal monitoring of singleton gestations in cephalic presentation beyond 34 weeks' gestation.[86] The study authors concluded that the additional use of ST analysis significantly reduced the need for fetal blood

sampling and reduced the incidence of operative vaginal deliveries. However, there was no evidence of a reduced incidence of metabolic acidosis at birth. Because all studies in the meta-analysis were performed in countries where fetal blood sampling is part of the standard obstetric practice, their results may not be applicable to countries such as the United States, where fetal scalp blood sampling rarely occurs.

The National Institutes of Health is funding a randomized, controlled trial of intrapartum ST analysis of the fetal ECG combined with cardiotocography compared with conventional cardiotocography alone (NCT 01131260). Results regarding the efficacy of this combined modality will provide an interesting comparison with those of the European studies reported in the meta-analysis, all of which used fetal blood sampling in labor.

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The previous edition of this chapter included the contributions of Dr. Larry C. Gilstrap, III. This chapter is built upon his contributions and is still reflective of that work, for which the editors and author are greatly appreciative.

The complete reference list is available online at www.expertconsult.com.

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