CE 2.0 ANCC Contact Hours

CLINICAL IMPLICATIONS OF FETAL HEART RATE INTERPRETATION BASED ON UNDERLYING PHYSIOLOGY

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Fetal heart rate tracing interpretation is supported by physiologic evidence and expert consensus

Abstract

Understanding the physiology of fetal oxygenation and various influences on fetal heart rate control supports nurses, midwives, and physicians in interpreting and managing electronic fetal heart rate tracings during labor and birth. Maternal oxygenation, placental circulation and exchange, umbilical blood flow and fetal circulation affect fetal oxygenation, which is reflected in observed fetal heart rate patterns. Fetal heart control is further influenced by the central and autonomic nervous systems, baroreceptors, chemoreceptors, humoral factors, sleep–wake patterns, breathing movements, medications, painful stimuli, sound and vibrations, and temperature. Knowledge of the physiologic basis for fetal heart rate pattern characteristics guides interventions to improve fetal oxygenation when indicated. A review and update on clinical implications of fetal heart rate pattern interpretation based on underlying physiology is presented.

Key words Electronic fetal monitoring; Fetal; Heart rate; Intrauterine resuscitation; Physiology.

Electronic fetal monitoring is the most common method of fetal assessment (Sakala, Declercq, Turon, & Corry, 2018) for the nearly 3.8 million women who give birth each year in the United States (Hamilton, Martin, Osterman, & Rossen, 2019). The Association of Women's Health, Obstetric and Neonatal Nurses, American College of Nurse Midwives, and American College of Obstetricians and Gynecologists (ACOG) agree on common fetal heart rate (FHR) definitions as per the 2008 National Institute of Health Human Development workshop report on fetal monitoring (Macones, Hankins, Spong, Hauth, & Moore, 2008) and on evaluation and management of intrapartum FHR tracings (ACOG; 2010; O'Brien-Abel & Simpson, 2020). Understanding the physiology of fetal oxygenation and various influences on FHR control can help nurses, midwives, and physicians in interpreting and managing electronic FHR tracings during labor and birth. A review and update on clinical implications of FHR pattern interpretation based on underlying physiology is presented.

Physiologic Systems and Structures Supporting Fetal Wellbeing

Maternal Oxygenation

Well-oxygenated maternal blood is required for fetal oxygenation. Adequate maternal hemoglobin concentrations, maternal oxygen saturation (SaO_2), and maternal arterial oxygen tension (PaO_2) are components of oxygen transport physiology supporting fetal oxygen affinity and oxygen delivery to the fetus. Although most pregnant women are healthy and well-oxygenated, maternal conditions that may impair oxygenation of maternal blood include severe anemia, asthma, lung disease, congenital cardiac defects, or seizures.

Uterine Blood Flow

Adequate uterine blood flow is essential for transport of respiratory gases (oxygen and carbon dioxide) and substances across the placenta. In a singleton pregnancy, uterine blood flow progressively increases from approximately 50 mL per minute to approximately 500 to 800 mL per minute at term, or 10% to 15% of the maternal cardiac output (CO) (Carter, 2015; King, 2018; Parer, 1997). About 70% to 90% of uterine blood flow supplies the intervillous space within the placenta, whereas the remainder mostly supplies the myometrium and endometrium (King; Parer).

Any reduction in uterine blood flow can adversely affect uteroplacental exchange and fetal oxygenation. Table 1 lists clinical conditions that may decrease uteroplacental blood flow during labor. Normally during pregnancy, spiral arteries carrying oxygenated blood to the intervillous space are maximally dilated and have lost some ability to vasoconstrict or "autoregulate." If pressure in the spiral arteries drops, there is little physiologic function available to increase uterine blood flow (King, 2018; Parer, 1997). Clinicians can strive to maximize uteroplacental blood flow throughout labor and attempt to avoid clinical situations that are known to decrease uteroplacental blood flow. Bedside interventions to maximize uterine blood flow and maternal–fetal exchange may include promoting adequately spaced uterine contractions, use of side-lying positions or ambulation, and maintenance of normal maternal blood pressure during regional analgesia. Table 2 summarizes commonly used intrauterine resuscitation measures during labor and associated FHR patterns (Simpson, 2015a).

Placental Circulation and Exchange

Well-oxygenated maternal blood, propelled by maternal arterial blood pressure, enters the intervillous space of the placenta via the spiral arteries. Here, the oxygenated blood surrounds the fetal chorionic villi, allowing for bidirectional movement of gases, nutrients, drugs, waste products, and other substances between the maternal and fetal circulations. Chorionic villi are protrusions of fetal tissue exposed to circulating maternal blood within the intervillous space covered by the placental membrane. The newly oxygenated fetal blood flows into veins that converge into a single umbilical vein that carries the oxygenated blood and nutrients to the fetus. Eventually, deoxygenated blood and waste products return from the fetus via the two umbilical arteries which spiral around the umbilical vein to the placenta. At the placental membrane, exchange of substances between the fetal and maternal circulations occurs, and the deoxygenated blood and waste products are removed through the maternal endometrial veins.

Maternal–fetal exchange of gases, nutrients, drugs, waste products, and other substances is facilitated by the large surface area of the placental membrane separating

TABLE 1. Potential Clinical Causes of Decreased Uteroplacental Blood Flow and Maternal–Fetal Exchange

Maternal conditions
Chronic or gestational hypertension; preeclampsia
Cardiac disease
Maternal hypotension
Supine position (supine hypotensive syndrome)
Regional analgesia/anesthesia (sympathetic blockade)
Hemorrhage/hypovolemic shock
Placental changes
Degenerative (e.g., maternal hypertension, diabetes, nicotine use, prolonged pregnancy)
Infection (e.g., chorioamnionitis)
Edema (e.g., hydrops fetalis)
Decreased surface area (e.g., abruptio placenta, small placenta, infarcts)
Excessive uterine activity
Tachysystole
Hypertonus
Medications that cause contractions (e.g., oxytocin, prostaglandins)
Abruptio placenta
Cocaine
Vasoconstriction
Endogenous (e.g., catecholamines)
Exogenous (e.g., most sympathomimetics, except ephedrine; cocaine, amphetamines)

maternal and fetal blood. Table 3 summarizes transport mechanisms and lists examples of substances transported (Blackburn, 2018c; Moore, Persaud, & Torchia, 2020). Simple (passive) diffusion is the prime method of placental transport. Oxygen, carbon dioxide, water, electrolytes, and other substances move readily between maternal and fetal compartments, down the concentration gradient. Medications given to the mother (e.g., narcotics, antibiotics, barbiturates) and anesthetic gases (e.g., general anesthesia, nitrous oxide) readily diffuse into the fetal circulation often resulting in FHR changes. During labor, after administration of an intravenous narcotic, a temporary decrease in FHR variability may be observed due to diffusion of the narcotic across the placental membrane.

Umbilical Blood Flow

The umbilical cord contains three vessels, two arteries and one vein, surrounded by Wharton jelly, a gelatinous substance containing collagen, muscle, and mucopolysaccharide. Although the umbilical cord generally moves freely within the amniotic fluid, it can become wrapped around fetal body parts, particularly the fetal neck (nuchal cord), or compressed during uterine contractions. During partial umbilical cord compression, occlusion of the low-pressure vein results in decreased blood return to the fetal heart, decreased CO, hypotension, and a compensatory increase in FHR. With complete umbilical cord compression, the umbilical arteries become occluded, resulting in sudden fetal hypertension, stimulation of baroreceptors, and a sudden drop in FHR resulting in a variable deceleration or a prolonged deceleration.

With umbilical cord occlusion, carbon dioxide often accumulates, resulting in a relatively benign fetal respiratory acidemia. However, recurrent variable decelerations that progress to greater depth and longer duration or are accompanied with minimal or absent FHR variability are more indicative of metabolic acidemia. Metabolic acidemia is caused by an interruption in fetal oxygenation and the accumulation of lactic acid in excess of the fetus' ability to buffer. Variable decelerations in the presence of moderate variability or a spontaneous or induced acceleration suggest the fetus is not currently experiencing metabolic acidemia. Maternal repositioning is often the first bedside intervention to relieve variable decelerations. If variable decelerations persist during the first stage of labor, amnioinfusion may assist in their resolution (ACOG, 2010).

Fetal Oxygenation

The fetal umbilical vein, which carries relatively well-oxygenated blood from the placenta to the fetus, has approximately an equal amount of dissolved oxygen (partial pressure of oxygen or PO_2) as does the maternal uterine vein, which carries the deoxygenated blood from the placenta to the maternal heart and lungs (King, 2018; Meschia, 2019). Even with a comparably low PO_2, fetal blood transports large amounts of oxygen due to several physiologic adaptations. Compared with mother's blood, fetal blood has a higher hemoglobin concentration, providing greater oxygen-carrying capacity (King; King & Parer, 2000). Fetal hemoglobin has a higher affinity for oxygen than mother's hemoglobin, allowing for greater oxygen saturation (King; King & Parer). Finally, the fetus has a higher heart rate and CO, providing more rapid circulation of oxygenated blood (King; King & Parer; Martin & Gingerich, 1976). These physiologic adaptations allow the healthy fetus to tolerate transient decreases in uterine or umbilical blood flow. The fetus preserves aerobic metabolism until available oxygen in the intervillous space decreases to 50% of normal levels (Carter, 2015; King; King & Parer; Parer, 1997). Maternal oxygen administration at 10 L per nonrebreather facemask has been shown to improve fetal oxygenation during labor (Simpson & James, 2005) and may be considered after other intrauterine measures have been initiated without resolving the concerning FHR pattern (O'Brien-Abel & Simpson, 2020; Simpson, 2008, 2015b).

Response to Hypoxia

With acute hypoxia, the fetus redistributes its blood flow favoring vital organs. Blood flow to the heart, brain, and adrenal glands is increased two- to threefold, whereas blood flow to the gut, spleen, kidneys, and limbs is decreased (Giussani, 2016; King, 2018; King & Parer, 2000; Parer, 1997). As the FHR slows in response to hypoxia, the fetal myocardium decreases oxygen consumption, thereby using less oxygen (Giussani; King; King & Parer; Parer). These compensatory measures, referred to as the "brain sparing effect," are critical for survival if hypoxia is not too severe (Giussani; King). If these measures do not provide adequate oxygen, the fetus switches to anaerobic metabolism with production of lactate acid or hydrogen ions. Fetal bicarbonate, a base that buffers hydrogen ions, attempts to neutralize the lactic acid. Without restoration of fetal oxygenation, the responses are no longer maintained and metabolic acidosis will progress to asphyxia, myocardial depression, brain ischemia, and ultimately, fetal death (King; Parer).

Promoting adequate fetal oxygenation enhances fetal wellbeing.

Acidemia reflects an increased concentration of hydrogen ions (decrease in pH) in the blood. Acidosis reflects an increased concentration of hydrogen ions in the tissue. Although clinicians often interchange these terms, acidemia cannot reflect the duration nor predict the consequences of acidosis in tissue (King & Parer, 2000). The newborn's umbilical artery pH in combination with PCO_2 and base excess determine the type of acidemia (respiratory, metabolic, mixed) present at birth. Respiratory acidemia reflects a high PCO_2 with normal bicarbonate

TABLE 2. Intrauterine Resuscitation Measures

Intrauterine Resuscitation Measures		
Clinical Situation and/or FHR Characteristics	**Goal**	**Techniques/Measures**
Minimal or absent variability **Recurrent late decelerations** **Recurrent variable decelerations** **Prolonged decelerations** **Tachycardia** **Bradycardia** **Variable, late or prolonged decelerations occurring with maternal pushing efforts** **Tachysystole**	Promote fetal oxygenation	Lateral positioning (either left or right) IV fluid bolus of lactated Ringer's solution Oxygen administration at 10 L/min via non-rebreather facemask; may be considered if there is minimal to absent variability and/or recurrent late, variable, or prolonged decelerations (discontinue as soon as possible based on fetal status) Modification of pushing efforts; pushing with every other or every third contraction or discontinuation of pushing temporarily (during second-stage labor) Decrease in oxytocin rate Discontinuation of oxytocin/removal of Cervidil insert/withholding next dose of misoprostol If prolapsed umbilical cord is identified, elevate presenting fetal part while preparations are made for expedited operative birth
Tachysystole	Reduce uterine activity	IV fluid bolus of lactated Ringer's solution Lateral positioning (either left or right) Decrease in oxytocin rate Discontinuation of oxytocin/removal of Cervidil insert/withholding next dose of misoprostol If no response, terbutaline 0.25 mg subcutaneously may be considered
Recurrent variable decelerations	Alleviate umbilical cord compression	Repositioning Amnioinfusion (during first-stage labor) Modification of pushing efforts; pushing with every other or every third contraction or discontinuation of pushing temporarily (during second-stage labor)
Maternal hypotension	Correct maternal hypotension	Lateral positioning (either left or right) IV fluid bolus of lactated Ringer's solution If no response, ephedrine 5 mg to 10 mg IV push or phenylephrine infusion may be considered

Note. Adapted from Simpson (2015a).

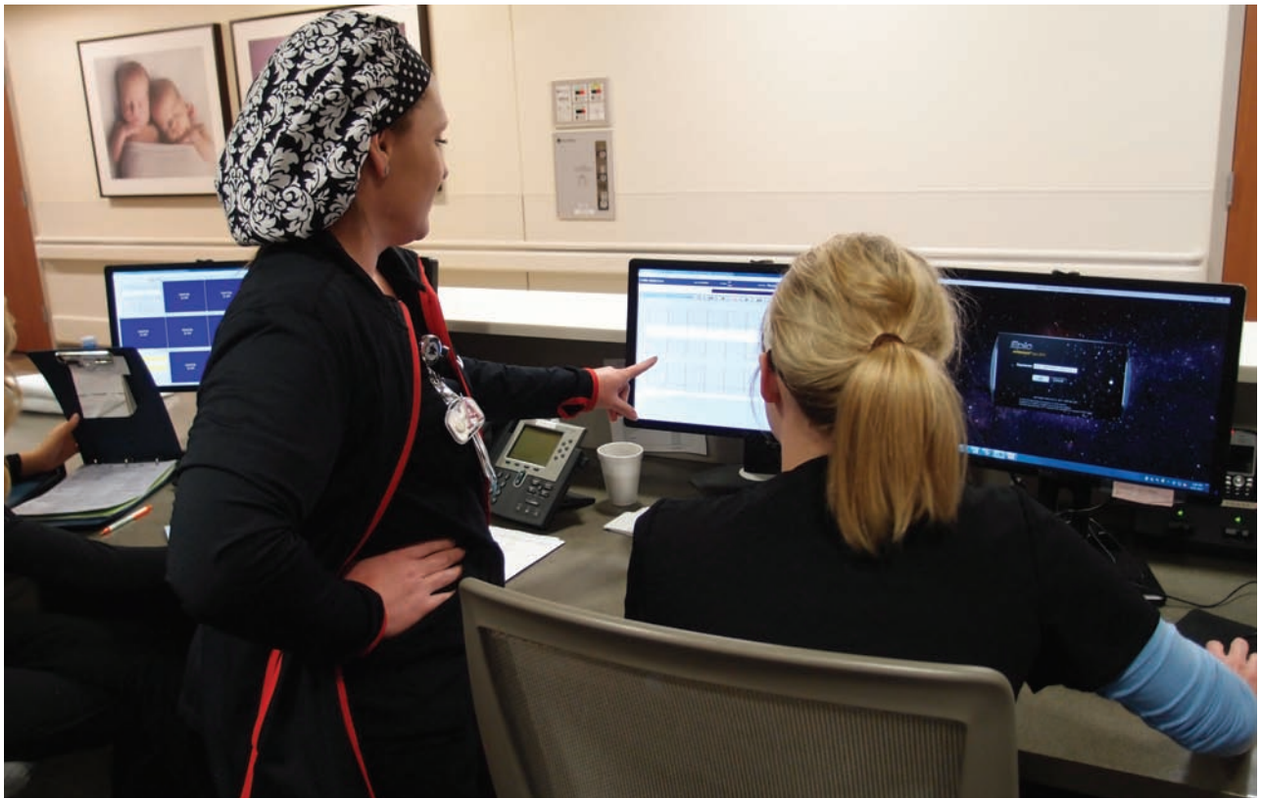

TABLE 3. Mechanisms of Exchange Between Maternal and Fetal Blood

Simple diffusion	Transfer down concentration gradient (higher to lower concentration); no energy required (e.g., water, electrolytes, oxygen, carbon dioxide, urea, simple amines, uric acid, creatinine, fatty acids, steroids, fat-soluble vitamins, narcotics, antibiotics, barbiturates, anesthetic gases)
Facilitated diffusion	Transfer down concentration gradient involving protein transporters or carrier molecules (e.g., glucose, possibly some oxygen, lactate)
Active transport	Transfer against concentration gradient; carrier molecules and energy required (e.g., amino acids, water-soluble vitamins, calcium, potassium, phosphate, iron, iodine)
Pinocytosis	Engulfing, and then suspending particles within small vesicles to transfer through cell (form of endocytosis) (e.g., immunoglobulin G [IgG] antibodies, phospholipids, lipoproteins, some viruses)
Bulk flow	Transfer resulting from changes in hydrostatic or osmotic gradient (e.g., water, electrolytes)
Accidental capillary breaks	Transfer of substances through microscopic breaks in placental membrane (e.g., intact red blood cells)
Independent movement	Transfer of cells under their own power (e.g., maternal leukocytes, microorganisms such as *Treponema pallidum (syphilis)*, some viruses)

Note. Content from Blackburn (2018c) and Moore et al. (2020).

levels (e.g., acute umbilical cord compression before anerobic metabolism has begun). Metabolic acidemia reflects a low bicarbonate (low base excess) due to an increase in hydrogen ions in the presence of normal PCO_2 levels (e.g., prolonged decrease in oxygen transfer secondary to uterine tachysystole, recurrent decelerations, and absent variability).

Fetal Circulation

Fetal and adult circulations differ significantly. Fetal circulation requires a series of anatomical "shunts" that normally close after birth. These fetal shunts direct well-oxygenated venous blood to the fetal systemic circulation and divert deoxygenated blood away from the immature fetal lungs, rather to the placenta for exchange. Ventricles of the fetal heart work in parallel, rather than in series, as in the adult heart.

Oxygen-rich blood returns from the placenta by way of the umbilical vein. As the vein enters the liver, some blood is shunted via the ductus venosus directly into the inferior vena cava and some traverses the hepatic parenchyma.

The blood shunted via the ductus venosus has greater oxygen content and kinetic energy, resulting in a distinct "stream" of blood as it enters the right atrium (Blackburn, 2018a; Edelstone & Rudolph, 1979; Fineman & Maltepe, 2019; Kiserud, 2005). As this oxygenated blood streams preferentially across the foramen ovale into the left atrium, it enters the left ventricle, and is ejected into the ascending aorta. The two fetal shunts, the ductus venosus and foramen ovale, allow the most highly oxygenated blood to supply the coronary circulation and the cerebral circuits that supply the head and neck, and upper extremities (Edelstone & Rudolph; Fineman & Maltepe; Kiserud; Parer, 1997).

Blood returning from the superior vena cava and coronary sinus mixes in the right atrium with hepatic and lower body desaturated blood, forming a second stream that flows through the tricuspid valve into the right ventricle. Most of this mixed blood is ejected through the pulmonary artery and the ductus arteriosus to reach the descending aorta, which supplies circulation to the gut, kidneys, lower body, and umbilical circulation. Only a relatively small amount of oxygen enters the fetal pulmonary circuit. This prefer-

ential streaming of desaturated blood is advantageous to the fetus because it directs the blood toward the placenta for reoxygenation (Fineman & Maltepe, 2019; Kiserud, 2005).

At birth, the neonate's circulation undergoes major physiologic and anatomic transitions. With removal of the low-resistance placental circulation, systemic vascular resistance increases, pulmonary vascular resistance decreases, and CO is redistributed (Blackburn, 2018a). The ventricles in the neonate's heart begin working in series, as in the adult heart. Fetal extracardiac and intracardiac shunts functionally close. Cessation of umbilical venous blood flow results in the closure of the ductus venosus. Hemodynamic changes within the heart chambers close the foramen ovale's flap valve, separating the two atria (Blackburn, 2018a). The increase in oxygen tension and decrease in prostaglandins constricts the ductus arteriosus, with obliteration of the lumen usually within 24 to 48 hours (Blackburn, 2018a; Kiserud, 2005; Schneider, 2012). Prematurity and certain genetic conditions may cause patency of the ductus arteriosus for much longer periods of time (Schneider).

Factors Influencing Fetal Heart Rate Control

The intrinsic rate of the fetal heart, like the adult heart, is determined by the sinoatrial node located in the right atrium. Average baseline FHR in a healthy fetus at term is 140 beats per minute (bpm) with a range of 110 to 160 bpm. Integration of FHR control is centralized in the medulla oblongata and influenced by parasympathetic and sympathetic nervous systems, baroreceptors, chemoreceptors, humoral factors, sleep–wake patterns, breathing movements, medications, painful stimuli, sound and vibrations, and temperature. Table 4 summarizes major physiologic factors influencing FHR control and their effect on FHR.

Parasympathetic Nervous System

The parasympathetic branch of the autonomic nervous system influences the fetal heart via the vagus nerve (10th cranial nerve) that innervates the sinoatrial and atrioventricular nodes within the heart. The gradual decrease in FHR observed with advancing gestational age is due to increased dominance of parasympathetic influence (vagal tone) on the fetal heart (Dalton, Dawes, & Patrick, 1983; Renou, Newman, & Wood, 1969). This vagal influence results in approximately 10 bpm difference in baseline FHR between 28 and 30 weeks' gestation and term (Freeman, Garite, Nageotte, & Miller, 2012).

The second important function of the vagus nerve is transmission of impulses that cause FHR variability. Although parasympathetic and sympathetic nervous systems work together to modulate baseline FHR, during second trimester vagal tone appears to have a greater influence on transmission of FHR variability (King, 2018; King & Parer, 2000; Nageotte, 2019; Parer, 1997). Presence of FHR variability represents integration of parasympathetic and sympathetic stimuli, cardiac conduction system, higher cortical centers, fetal states, and feedback from chemoreceptors and baroreceptors (King; King & Parer). Moderate FHR variability is highly predictive of normal fetal acid-base status at the time it is observed (ACOG, 2010; Freeman et al., 2012; Parer; Parer, King, Flanders, Fox, & Kilpatrick, 2006). Potential causes of minimal FHR variability include maternal medications (e.g., opioid, methadone, magnesium sulfate), gestational age, fetal sleep, hypoxia, or fetal acidemia (ACOG; King; King & Parer). Absent variability with recurrent late or variable decelerations, or bradycardia, is abnormal or category III, and signifies an increased risk for metabolic acidemia at time of observation (ACOG; Low, Victory, & Derrick, 1999; Macones et al., 2008; Paul, Suidan, Yeh, Schifrin, & Hon, 1975).

Sympathetic Nervous System

At term, sympathetic nervous system fibers are widely distributed throughout the fetal myocardium. Stimulation of sympathetic fibers releases catecholamines (e.g., norepinephrine, epinephrine) that increase FHR, resulting in an increased CO (King, 2018; Nageotte, 2019). During fetal stress (e.g., prolonged deceleration), sympathetic fibers are stimulated, resulting in a temporary, normal compensatory FHR acceleration or brief periods of fetal tachycardia to increase CO and oxygen flow to the tissues. Sustained fetal tachycardia is not normal in a term fetus. At term, fetal tachycardia is often associated with maternal fever or fetal infection (e.g., chorioamnionitis) and loss of FHR variability is common. In early pregnancy, sympathetic influence dominates, resulting in a slightly higher intrinsic FHR and minimal variability.

Cardiac Output Regulation

In adults, heart rate is related to CO and stroke volume in the following equation: Cardiac Output = Heart Rate × Stroke Volume. Because the fetal heart performs normally near the top of its cardiac function curve (Fineman & Maltepe, 2019), stroke volume does not fluctuate significantly, and fetal CO is essentially rate-dependent. Small FHR variations within normal FHR range (110–160 bpm) appear to have minimal effect on fetal CO. However, with fetal tachycardia greater than 240 bpm or fetal bradycardia less than 60 bpm, fetal CO and umbilical blood flow can be significantly decreased (Nageotte, 2019; Parer, 1997).

Clinical assessment at the onset of fetal bradycardia determines the need to proceed with emergent cesarean birth. Although the fetus has some ability to increase stroke volume in response to initial bradycardia, once the FHR decreases to 60 bpm, CO cannot be maintained, umbilical blood flow decreases, and eventual hypoxic decompensation ensues (Parer, 1997). In addition to depth and duration of fetal bradycardia, assessment of fetal reserve prior to the insult (category I FHR vs. category II or III) and type of insult (e.g., catastrophic uterine rupture vs. uterine tachysystole) are integral in determining clinical management and neonatal outcome.

Baroreceptors

Baroreceptors are small protective stretch receptors in the aortic arch and carotid sinus that quickly detect increases in fetal arterial blood pressure (Nageotte, 2019; Parer, 1997). When umbilical blood flow is occluded, fetal arterial blood pressure rises quickly, triggering the baroreceptors to send impulses via the vagus nerve to the midbrain, causing further vagal stimulation, which abruptly decreases the FHR and CO, thereby protecting the fetus.

Influence of baroreceptors may be seen in the mechanism of variable decelerations. As a contraction begins, partial umbilical cord compression causes occlusion of the low-pressure vein and decreased blood return to the fetal heart, resulting in decreased CO, hypotension,

TABLE 4. Influences on Fetal Heart Rate Control

Physiology	Effect on Fetal Heart Rate
Parasympathetic nervous system (branch of the autonomic nervous system): • Originates in medulla oblongata • Vagus nerve (10th cranial) innervates SA and AV nodes • Stimulation releases acetylcholine • Pathway for transmission of FHR variability • Variability represents an intact nervous pathway through cerebral cortex, midbrain, vagus nerve, and normal cardiac conduction system	• Decreases FHR • With increasing gestational age, slow, gradual decrease in FHR and increase in FHR variability • Moderate variability indicates absence of metabolic acidemia • Modulates baseline FHR with sympathetic branch
Sympathetic nervous system (branch of the autonomic nervous system): • Nerve fibers widely distributed throughout myocardium at term • Stimulation releases catecholamines (norepinephrine, epinephrine) • Reserve mechanism to initially improve the heart's pumping ability during intermittent hypoxemia/stress • Blocking with propranolol results in approximately 10 bpm decrease in FHR • Catecholamines may also cause fetal vasoconstriction and hypertension	• Increases FHR • With intermittent hypoxemia, initial normal fetal compensatory response is increase in FHR or brief tachycardia • At term, tachycardia is not normal • In early gestation, sympathetic dominance results in slightly higher FHR and decrease in variability • Modulates baseline FHR with parasympathetic branch
Cardiac output: • In the adult, CO increases or decreases in response to changes in HR or SV as in the following equation: CO = HR × SV • Because the fetal heart appears to operate near the top of its cardiac function curve, SV does not fluctuate significantly. Hence, fetal CO is dependent on HR	• Small FHR variations within normal FHR range (110–160 bpm) appear to have minimal effect on CO • With fetal tachycardia greater than 240 bpm or bradycardia less than 60 bpm, fetal CO and umbilical blood flow can be significantly decreased
Baroreceptors: • Protective, stretch receptors • Located in aortic arch and carotid sinuses at bifurcation of external and internal carotid arteries • With umbilical cord occlusion, arterial BP increases, baroreceptors quickly detect amount of stretch, sending impulses via vagus nerve to midbrain • Further vagal stimulation causes a sudden decrease in FHR, CO, and BP, thereby protecting fetus	• Abrupt decrease in FHR, CO, BP • Variable decelerations • Prolonged deceleration due to umbilical cord occlusion
Chemoreceptors: • Central—located in medulla oblongata • Peripheral—located in aortic arch and carotid sinuses • Interaction of central and peripheral chemoreceptors poorly understood; combined effect FHR slowing • When blood flow falls below threshold for normal respiratory gas exchange, increased PCO_2 stimulates chemoreceptors to slow FHR • Deceleration is late due to circulation time from fetal–placental site to chemoreceptors	• Late decelerations • Variable decelerations resulting from umbilical cord occlusion coupled with hypoxemia • Prolonged deceleration coupled with hypoxemia

TABLE 4. Influences on Fetal Heart Rate Control (*Continued*)

Physiology	Effect on Fetal Heart Rate
Hormonal influences: • Epinephrine and norepinephrine (adrenal medulla) ◦ Reserve mechanism to initially improve the heart's pumping ability during intermittent hypoxemia/stress ◦ In response to stress, fetal compensatory response shunts blood away from less vital organs and toward brain, heart, adrenal glands	• Increases FHR, strength of cardiac contractions, CO, arterial BP
• Renin–angiotensin system ◦ Regulates normal fetal circulation by tonic vasoconstriction on peripheral vascular bed ◦ Protects fetus during hemorrhagic stress	• Maintains systemic arterial BP and umbilical placental blood flow
• Prostaglandins ◦ Prostaglandins and arachidonic acid metabolites found in fetal circulation and in many tissues ◦ Maintains patency of fetal ductus arteriosus	• Regulation of umbilical blood flow
Sleep–wake patterns • Quiet sleep (state 1F) ◦ Quiescence (occasional brief body movements) ◦ Absent REM ◦ FHR stable with narrow oscillation bandwidth	• Normal baseline FHR, minimal variability, accelerations absent • Nonreactive NST • Responds to external stimuli (vibroacoustic stimulation)
• Active (REM) sleep (state 2F) ◦ Frequent gross body movements ◦ Rapid darting eye movements (REM) ◦ FHR with wider oscillation bandwidth and frequent accelerations with movements	• Moderate variability, accelerations present • Reactive NST • At term, duration of periods of active sleep are longer than quiet sleep

Note. AV = atrioventricular; BP = blood pressure; bpm = beats per minute; CNS = central nervous system; CO = cardiac output; FHR = fetal heart rate; HR = heart rate; NST = nonstress test; PCO2, = partial pressure of carbon dioxide; PO2 = partial pressure of oxygen; REM = rapid eye movements; SA = sinoatrial; SV = stroke volume. Content from Blackburn (2018a,b), Fineman & Maltepe (2019), Freeman et al. (2012), King (2018), King & Parer (2000), Nageotte (2019), Parer (1997), Richardson et al. (2019), and Rudolph (1985).

and a compensatory increase in FHR (Freeman et al., 2012; James et al., 1976; Lee, Di Loreto, & O'Lane, 1975). With complete umbilical cord occlusion, the two umbilical arteries also become occluded, resulting in sudden fetal hypertension, stimulation of the baroreceptors, resulting in an abrupt drop in FHR (Freeman et al.; Lee & Hon, 1963). As the contraction begins to dissipate, the umbilical arteries open first, followed by the vein, returning the FHR to baseline. For clinical implications, see the section on Umbilical Blood Flow.

Chemoreceptors

Chemoreceptors, located centrally in the medulla oblongata and peripherally in the aortic arch and carotid sinuses, have their greatest effect on regulation of respiration and control of circulation (Nageotte, 2019; Parer, 1997). When chemoreceptors detect a decrease in circulating oxygenation and an increase in carbon dioxide (PCO_2) (e.g., uteroplacental blood flow falls below a threshold needed for normal respiratory gas exchange), chemoreceptors stimulate a vagally mediated reflex fetal bradycardia and increase in blood pressure (King, 2018; Parer). Influence of chemoreceptors may be seen in the mechanism of late decelerations and in variable decelerations with minimal or absent variability due to hypoxemia.

Fetal Sleep–Wake Patterns

Fetal and infant sleep–wake patterns are commonly referred to as "states" because they are qualitatively and quantitatively different when compared with the adult (Blackburn, 2018b; Nijhuis, Prechtl, Martin, & Bots, 1982). Fetal state is determined by regularity of the FHR, presence or absence of fetal gross body movements, and presence or absence of rapid eye movements (REM). See Table 4 for information about the two most common fetal states during the third trimester, quiet sleep (fetal state 1F) and active REM sleep (fetal state 2F).

Although the healthy fetus at 24 to 26 weeks' gestation moves its extremities almost continuously, distinct periods of quiescence emerge with increasing maturation (Blackburn, 2018b). By approximately 36 weeks' gestation, FHR variations, fetal eye movements, and gross body movements demonstrate coordinated patterns of rest–activity cycles (quiet sleep and active REM sleep)

Suggested Clinical Implications

- Conditions that may impair oxygenation of maternal blood include severe anemia, asthma, lung disease, congenital cardiac defects, or seizures.
- Interventions to maximize uterine blood flow and maternal–fetal exchange may include promoting adequately spaced uterine contractions, side-lying positions or ambulation, and normal maternal blood pressure during regional analgesia.
- Medications given to the mother (e.g., narcotics, antibiotics, barbiturates) and anesthetic gases (e.g., general anesthesia, nitrous oxide) readily diffuse into the fetal circulation often resulting in FHR changes.
- Maternal repositioning is often the first intervention to relieve variable decelerations. If variable decelerations persist during the first stage of labor, amnioinfusion may be initiated.
- Absent variability with recurrent late or variable decelerations, or bradycardia, is abnormal or category III FHR, and signifies an increased risk for metabolic acidemia at the time of observation.
- In addition to depth and duration of fetal bradycardia, assessment of fetal reserve prior to the insult (category I FHR vs. category II or III FHR) and type of insult (e.g., catastrophic uterine rupture vs. uterine tachysystole) are integral in determining clinical management and neonatal outcome.
- Influence of chemoreceptors may be seen in the mechanism of late decelerations, and in variable decelerations with minimal or absent variability due to hypoxemia.
- A nonstress test (NST) during quiet sleep for a term fetus will most likely be interpreted as a nonreactive NST; vibroacoustic stimulation to elicit an acceleration may be used to rule out fetal metabolic acidemia.

(Nijhuis et al., 1982; Richardson, Harding, & Walker, 2019). However, not all fetuses, even at 36 to 38 weeks' gestation, demonstrate state organization with distinct cycles of sleep and wakefulness (Van den Bergh & Mulder, 2012). At term, a quiet sleep cycle generally lasts 20 minutes, but may persist for 60 minutes, with moderate variability returning once the cycle is complete (ACOG, 2010). A nonstress test (NST) during quiet sleep will most likely be interpreted as a nonreactive NST. In this situation, vibroacoustic stimulation to elicit an acceleration may be used to rule out fetal metabolic acidemia.

Clinical Implications

Knowledge of the underlying physiology of FHR patterns is useful for choosing appropriate clinical management strategies during labor when an indeterminate (category II) or abnormal (category III) tracing develops. Many factors influence FHR including maternal wellbeing, gestational age, maternal medications, maternal positioning, uterine activity, and placental status. Some of these factors are amenable to intrauterine resuscitation measures to increase fetal oxygenation; however, some may require urgent clinical intervention or expeditious birth. Periodic review of maternal and fetal physiology by clinicians enhances their ability to promote optimal maternity care. ✣

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