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Physiology of Fetal Heart Rate Monitoring

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Abstract: Fetal heart tracings (FHTs) are useful as a window into the oxygenation status of the fetal brain. Patterns in the FHT reflect the oxygen status of the fetal brain. Fetal adaptive response to progressive hypoxemia and acidosis are detectable and produce recognizable patterns in the fetal heart rate. The basic physiology and adaptive responses that regulate the fetal heart rate and physiological fetal adaptations to stress as reflected in the FHTs are described. Mechanisms of oxygen delivery to the fetus including ways in which those mechanisms can be disrupted are reviewed.
Key words: hypoxia, labor, fetal heart rate

An understanding of the physiology underlying fetal heart tracings (FHTs) is the key to appropriate interpretation and intrapartum management. FHTs are useful as a window into the oxygenation status of the fetal brain. The basic premise underlying FHT as a tool is that patterns in the FHT reflect the oxygen status of the fetal brain. Interpretation is further predicated on the notion that fetal adaptive response to progressive hypoxemia and acidosis are detectable and produce recognizable patterns in the fetal heart rate (FHR).

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The author declares that there is nothing to disclose.

The basic physiology and adaptive responses that regulate the FHR are reviewed, physiological fetal adaptations to stress as reflected in the FHT are described, and mechanisms of oxygen delivery to the fetus including ways in which those mechanisms can be disrupted are reviewed.

Direct evidence of human FHT physiology is obviously difficult to obtain. While some laboratory studies exist based on animal experimentation with interruption of oxygen delivery to the fetus, such studies are obviously unethical in humans. Thus, most information is either observational in nature or extrapolation of animal studies.

General Principles

ACID-BASE PHYSIOLOGY AND PATHWAY TO FETAL INJURY

Homeostasis, derived from the Greek words for "same" and "steady," is the concept that living organisms actively maintain stable physiological conditions necessary for survival. The changes and patterns seen in the FHR in response to changes in oxygenation and acid/base status should be considered as the fetal organism attempting to maintain homeostasis.

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Several unique characteristics of fetal and placental biology result in the FHR playing a critical role in fetal homeostasis. First, fetal stroke volume is relatively fixed. Thus, the heart rate is the primary mechanism for changing cardiac output. Second, in contrast to nonfetal physiology in which multiple organ systems (ie, respiratory, renal, cardiovascular, neurological) respond to physiological changes, the placenta has limited ability to increase fetal oxygen intake or carbon dioxide removal. Finally, several fetal organ systems (notably the cardiovascular and neurological) are particularly sensitive to very small changes in pH.

There are also a number of general human physiological principles that do not differ between fetal and nonfetal physiology and which have a direct bearing on FHR interpretation. A brief review of cellular physiology reminds us that at the most basic level; energy generation is central for any biological organism and occurs at the subcellular level via aerobic and anaerobic metabolism. Aerobic metabolism is the slower of the 2 processes, but the most effective, thus the primary pathway for energy generation in higher-order organisms. This process occurs in the mitochondria via the Krebs cycle and the electron transport chain, resulting in 34 molecules of ATP for every molecule of glucose metabolized. Other byproducts are water and carbon dioxide, which diffuses across the placenta and is ultimately eliminated via maternal respiration. Carbon dioxide transfer from the fetus is of course enhanced by the reduction in the partial pressure of carbon dioxide in the maternal circulation during pregnancy, which is a result of the increased maternal respiratory rate. In situations of compromised gas transfer across the placenta, carbon dioxide will accumulate and result in respiratory acidosis.

When the oxygen is not available for aerobic metabolism, cells will use anaerobic metabolism in an attempt to continue energy production. Anaerobic metabolism occurs in the cytoplasm and is much less efficient, producing only 2 molecules of ATP for each molecule of glucose. The other byproduct of anaerobic metabolism is lactic acid, which is removed at a slower rate and requires oxygen for removal (via oxidation to carbon dioxide). When oxygen debt exists in the cell, lactic acid accumulates intracellularly and extracullarly via transport across the cell membrane. The other noncarbonic (organic acids) produced by fetal metabolism include uric acid (from the metabolism of amino acids) and keto acids (from the metabolism of fatty acids). Fetal organic acids diffuse slowly across the placenta and are ultimately eliminated by the maternal kidneys. Thus hypoxia and placental dysfunction can lead to metabolic acidosis.

Bicarbonate buffering systems (below) in the fetal blood can initially compensate for acidosis. However, prolonged or severe acidosis will overwhelm the system, leading to loss of homeostasis, cell death, and potential long-term neurological consequences.

$$H_2O + CO_2 \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3^-.$$

Interruption in oxygen transfer from the maternal to the fetal circulation can be interrupted at any point along the pathway. Figure 1 details some of the ways in which maternal, placental, and fetal factors may contribute to this reduction in oxygen transfer.

Figure 2 illustrates the general pathway to fetal injury, which progresses from hypoxemia, through hypoxia, metabolic acidosis, metabolic acidemia, and finally to potential injury.

Hypoxemia, a reduction on the oxygen content of fetal blood, is the initial phase of oxygen deficiency. Cell and organ function remain intact and there is enhanced uptake of oxygen from the circulating blood. Clinical manifestations may include a reduction in the level of fetal

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Oxygen Transfer Pathway	Potential Cause of Interrupted Transfer
Lungs	Respiratory Depression Asthma PE, Pulmonary edema Pneumonia/ARDS, Seizure
Heart / Vascular	Reduced cardiac output Hypovolemia, IVC Compression Regional anestheia, medications Arrhythmia
Uterus	Tachysystole Uterine stimulant effect Uterine rupture
Placenta	Abruption, fetal-maternal hemorrhage, Insufficiency (poor has exchange)
Umbilical Cord	Cord compression (nuchal/body cord, knot oligohydramnios, prolapse)

FIGURE 1. Interrupted oxygen transfer. ARDS indicates acute respiratory distress syndrome; IVC, inferior vena cava. full color online

activity and decelerated fetal growth, both as a means for decreasing energy requirement and need for oxygen. Hypoxemia may continue for an extended period of time (days to weeks).

Hypoxia, a reduction in the oxygen content of fetal tissue, takes only hours to result in fetal injury. Stress hormones surge and decrease peripheral blood flow in favor of maintaining perfusion of the brain, heart, and adrenals. Anaerobic metabolism occurs in the peripheral tissues.

The final and most acute phase of the injury process is asphyxia affecting the central organs. At this point, fetal adaptations to reduced oxygen fail. Maximal activation of the sympathetic nervous system results in massive amounts of stress hormones. Anaerobic metabolism occurs in the central organs as the fetus uses glycogen stores from the liver and heart. The end result is major organ failure and damage, which occurs in a matter of minutes (Fig. 3).

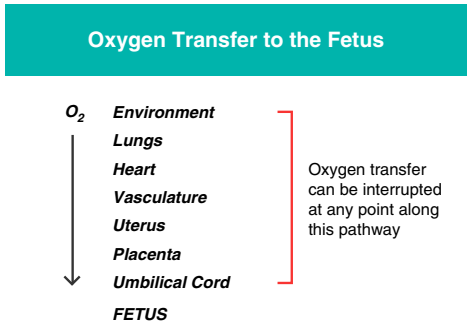

FIGURE 2. The general pathway to fetal injury, which progresses from hypoxemia, through hypoxia, metabolic acidosis, metabolic acidemia, and finally to potential injury. full color online

OXYGENATION IN NORMAL LABOR

Normal fetal-maternal gas exchange depends on perfusion of the placental intervillous spaces. Normal contractions decrease perfusion and thus gas exchange. Individual contractions have been associated with a decrease in maternal uterine artery blood velocity of up to 73% and compression of maternal spiral arteries.[1] Strong contractions (over 40 mm Hg) interrupt perfusion of the placental base completely, and up to 90 seconds are needed between contractions to fully re-

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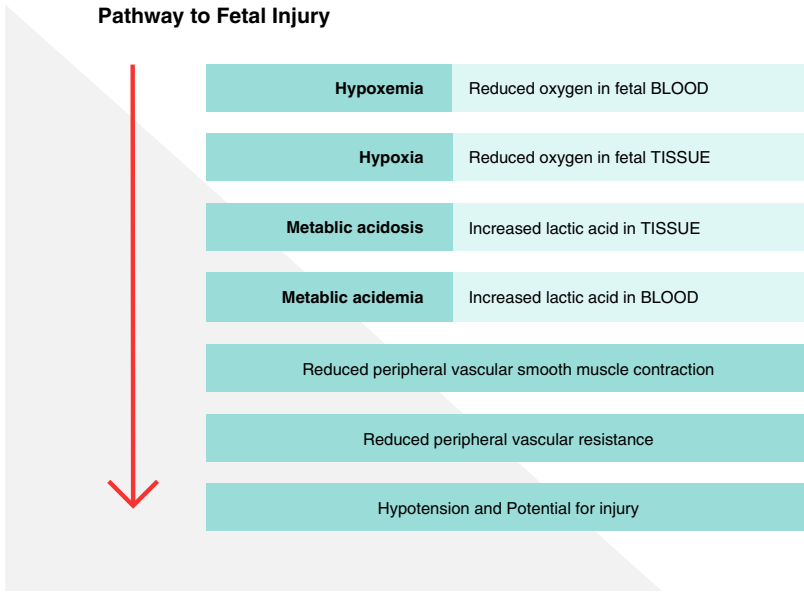

FIGURE 3. Fetal response to hypoxia. full color online

plenish the oxygen reserve. Thus, even normal labors are associated with periods of impaired maternal-fetal gas; a small but consistent fall in pH and oxygen tension; and a rise in carbon dioxide tension, base deficit, and lactate.[2–4] These changes occur even in the absence of umbilical cord compression or pathologic placental dysfunction.

If fetal oxygenation and acid-base status are normal at the onset of labor and fetal adaptive mechanisms are intact, the changes described over the course of labor are unlikely to result in adverse outcomes or even manifest changes in the FHT.

BASICS OF FETAL ADAPTIVE RESPONSES

The fetal cardioregulatory center (CRC) is located in the medulla oblongata and is responsible for maintaining homeostasis and optimizing oxygen delivery, the loss of which leads to cellular damage and death via the aforementioned mechanisms. The CRC is the central source that determines the FHR baseline, variability, and pattern (Fig. 4).

The CRC receives input from afferent nerves, which, in turn, receive input from different receptor types in the peripheral nervous system. Receptor types include:

- Baroreceptors: Located in the aortic arc and carotid bifurcation, these are stretch receptors that detect changes in fetal blood pressure. An *increase* in fetal blood pressure generally results in a *decrease* in the FHR and vice versa. These receptors play a key role in variable decelerations, late decelerations, and bradycardia, which will be further discussed in later sections.
- Chemoreceptors: These chemosensitive cells detect oxygen saturation and pH. They are mainly responsive to hypoxemia, acidosis, and hypercarbia. They can be further divided into central chemoreceptors located in the medulla oblongata and peripheral receptors located in the aortic arch and carotid bodies. Stimulation of central chemoreceptors generally results in increased sympathetic output, tachycardia, and hypertension. In contrast, stimulation of peripheral receptors has the opposite

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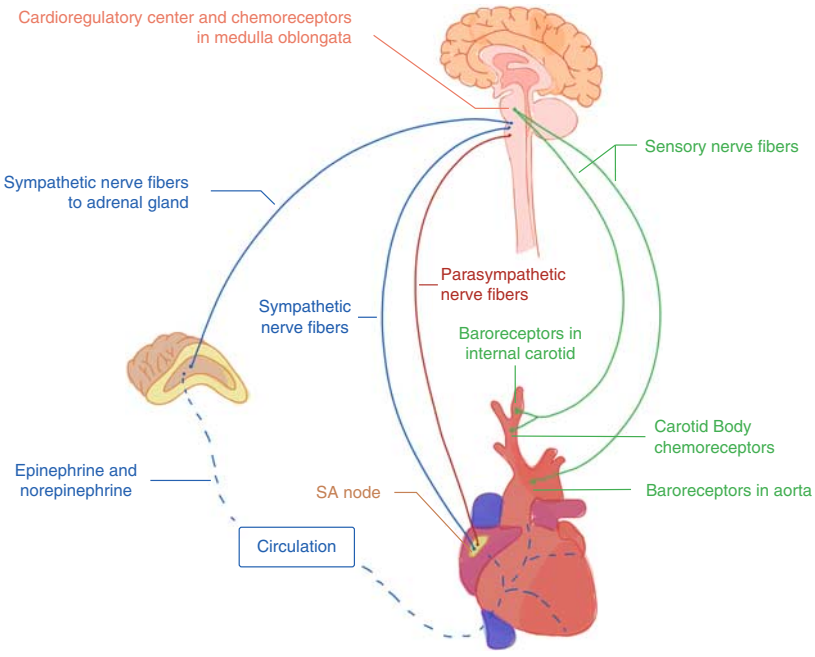

FIGURE 4. Anatomy of fetal heart rate regulation. SA indicates sinoatrial. full color online

effect, resulting in increased parasympathetic output.
- Acoustic receptors.
- Vibratory receptors.
- Temperature receptors.

The output from the CRC is via efferent nerves. The parasympathetic output is primarily through the vagus nerve, which originates in the CRC. The vagus nerve innervates the sinoatrial and atrioventous nodes of the heart and mediates a slowing of the FHR. Parasympathetic input partially determines the baseline rate and variability of the FHR. It is also involved in bradycardia and decelerations, as will be further detailed below. The sympathetic nervous system has nerve fibers that extend throughout the myocardium and vasculature and works to preserve brain perfusion in times of stress via vasoconstriction, hypertension, and increased cardiac output (mainly via an increase in FHR). Sympathetic nerve terminals also exist in the adrenal medulla and result in catecholamine (epinephrine, norepinephrine) release. The sympathetic nervous system also plays a role in baseline characteristics of the FHR, accelerations, and fetal tachycardia, which will be further discussed below.

Physiology of Specific Characteristics

BASELINE CHARACTERISTICS

Baseline characteristics of the FHT include rate and variability. The normal range for the FHR is 110 to 160 beats/min. The parasympathetic nervous system matures during gestation, resulting in a higher baseline in earlier gestation which falls as the pregnancy progresses.

Fetal tachycardia (baseline rate over 160 beats/min) is generally a physiological response, as in the nonfetal organism, to a demand for greater cardiac output, an increase in sympathetic tone, or a decrease

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in parasympathetic tone. When it results from oxygen debt, tachycardia tends to be a later development as central chemoreceptors detect hypoxemia and attempt to maintain central perfusion via in increased heart rate mediated by an increase in sympathetic tone and increased catecholamine release. Tachycardia may also be a manifestation of fever/infection, hypovolemia, or fetal arrhythmia. While an increase in heart rate can partially compensate for an oxygen debt, it can also accelerate the development of that debt as myocardial oxygen consumption increases.

Bradycardia (baseline <110 beats/min) can be a manifestation of several physiological and pathologic states. As with all characteristics of the FHT, it must be interpreted in the clinical context and in conjunction with other findings on the tracing. In an otherwise reassuring tracing with a baseline rate of over 90 to 95 beats/min, it may be a normal variant. In a third-degree heart block, there is complete interruption of the atrioventricular conduction pathway and disassociation of the atrial and ventricular rates. The ventricular rate is typically 50 to 80 beats/min. Acute and severe hypoxemia will also result in bradycardia, as in cases of cord prolapse, uterine rupture, and abruption.

The term "terminal bradycardia" is often used to describe bradycardia immediately before delivery. It may be caused by prolonged vagal stimulation from rapid descent of the fetal vertex and, in such cases, rarely exceeds 10 minutes. It may also be a final manifestation of prolonged hypoxia and acidosis. As noted above, the initial response of tachycardia further increases the cardiac oxygen requirement. If that need is not met, cardiac hypoxia results leading to myocardial depression manifesting as terminal bradycardia.

The variability of the baseline FHR results from the interplay between the sympathetic and parasympathetic nervous system. In the absence of pathology, the peak to trough measurement will exhibit a variation of 6 to 25 beats/min, aka "moderate variability." This reflects functioning neuromodulation of the FHR, normal cardiac responsiveness, and normal acid-base status. A decrease in variability is seen as the fetus responds to progressive hypoxia. Responses designed to maximize oxygen delivery overwhelm the usual interplay of the sympathetic and parasympathetic systems and result in decreased variability. Other factors that may decrease the variability of the FHR include medications (general anesthesia, opioids, magnesium, and betamethasone), fetal sleep cycles, congenital anomalies including neurological anomalies, and arrhythmias.

PERIODIC CHANGES

Periodic changes to the FHR include accelerations and decelerations. Accelerations are mediated by the sympathetic nervous system and result from activity, sensory stimulation (often acoustic or tactile), or a short spontaneous increase in sympathetic activity. Accelerations reliably indicate the absence of severe hypoxia or acidosis/acidemia. However, the absence of accelerations does not indicate the presence of these pathologic states. Accelerations may be absent during fetal sleep, arrhythmia, exposure to certain medications, and extreme prematurity.

Decelerations are usually characterized by their temporal relationship to contractions and maybe early, late, variable, or prolonged. The definitions of each are covered in the next chapter. The physiological basis for each will be discussed below.

Early Decelerations

The physiology of early decelerations is not well characterized. Classic teaching states that fetal head compression, either by the uterine wall or the pelvic floor, leads to nonpathologic decreases in the heart rate via the vagus nerve. However, the mechanism whereby this response occurs has not been adequately elucidated. Lear et al[5] suggest that it may be

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mediated via the Cushing response. The Cushing response is a decrease in heart rate in response to an intracranial pressure so high that cerebral blood flow is reduced. This process is terminal in the nonfetal state and thus can hardly be described as benign or nonpathologic. Nevertheless, some older animal data support this hypothesis and describe early decelerations in fetal sheep when manual head compression was marked enough to reduce cerebral and carotid blood flow.[6–9]

The Cushing response physiology of early decelerations stands in contrast to the most recently proposed interpretations.[10] Thus, other mechanisms have been proposed such as a response to pressure by scalp receptors. Understanding the mechanism of early decelerations is further muddled by the fact that all labor results in some degree of fetal head compression and yet early decelerations remain relatively uncommon. The exact neural events and pathways of early declarations thus afford multiple opportunities for future research.

Variable Decelerations

The classic teaching regarding the etiology of variable decelerations via umbilical cord compression is as follows: initial partial obstruction of the umbilical cord leads to partial or complete occlusion of the umbilical vein which reduces blood flow from the placenta to the fetus, fetal hypovolemia, and a compensatory increase in the FHR manifesting as the "shoulder" of the variable deceleration; further cord compression leads to occlusion of both the umbilical vein and arteries, leading to a marked increase in peripheral vascular resistance and a resulting abrupt decrease in the heart rate; finally, the entire process reverses as pressure on the umbilical cord abates. These responses would be mediated via peripheral baroreceptors responding to changes in fetal peripheral vascular resistance.

Animal studies partially support the aforementioned explanation. In 1983, Itskavoitz et al[11] found that once umbilical blood flow was reduced by at least 50%, the FHR decreased. However, in their study and the study by Giussani et al,[12] no significant changes in the arterial pressure were seen, suggesting that the deceleration is not mediated by baroreceptors. Other studies have found an increase in mean arterial pressure with complete occlusion of the cord. However, the initial increase in arterial pressure was small and a sustained and marked increase in pressure did not occur until *after* the FHR decrease. In these studies, initial cord occlusion resulted in a rapid decrease in FHR, a slower but still substantial fall in cerebral oxygenated hemoglobin, and a later increase in systemic vascular resistance, further calling baroreceptor mediated decrease in FHR into question.[5] Furthermore, animal studies have also shown that administration of phenylephrine results in an increase in blood pressure in fetal sheep. Once again, however, the heart rate response was a slow deceleration that took up to 30 seconds to develop.[13] This shape is not characteristic of the classic teaching about variable declarations, which nadir much more quickly. In fact, by definition, variable decelerations take 30 seconds or less to reach nadir. Physiologists have noted that while the baroreflex is active during fetal life, it is relatively immature. Thus, alternative mechanisms have been proposed.

One other proposed mechanism of variable decelerations via cord occlusion suggests that compression of the umbilical vein alone would reduce venous flow from the placenta to the fetus to such a degree as to reduce central blood volume and cardiac filling, thus triggering a vagal deceleration. This response is called the Bezold-Jarisch reflex. However, it is also known to be immature at birth (and thus, presumably, during fetal life).[14] Furthermore, fetal sheep studies[15] have shown an increase in blood flow in the vena cava and ductus venosus during partial cord occlusion,

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supporting the idea that preferential shunting maintains central circulation and atrial filling pressures and refuting the hypothesis that cord occlusion results in FHR decelerations through this mechanism.

Another proposed mechanism is that central hypoxia caused by umbilical cord compression leads to the abrupt decelerations via chemoreceptors. Animal models have shown that cerebral oxygenated hemoglobin decreased quickly in response to complete umbilical cord occlusion and that this decrease correlates temporally with abrupt decelerations in the FHR.[5] It has been hypothesized that this abrupt fall in FHR may be an adaptive mechanism to reduce myocardial work during periods of impaired gas exchange. The bottom line is that while umbilical cord compression likely results in variable decelerations, as per traditional teaching, the mechanism whereby this occurs may differ from classic explanations.

Late Decelerations

Late decelerations are most consistently associated with a response to a reduction in fetal oxygenation. Each time the uterus contracts, compression of the intervillous space leads to reduction in gas exchange and thus lower fetal oxygenation (Fig. 5). The normally oxygenated fetus will tolerate this brief reduction well. In contrast, when oxygen tension is already low or borderline, the loss of oxygen tension leads to vasoconstriction in an attempt to shunt blood (and thus oxygen) to vital organs. Baroreceptors recognize this increase in fetal blood pressure and instigate a lowering of the FHR, mainly via the vagal nerve.

It is not known why some compromised fetuses exhibit late decelerations, while others do not. It is also not understood why some noncompromised fetuses show late decelerations in the heart rate. Some experts have suggested that there are different categories of late decelerations: those truly due to placental insufficiently (which occur in the context of other nonreassuring findings in the FHR and result from an already compromised fetus unable to tolerate even small interruptions in gas exchange) and those that occur without other evidence of fetal compromise. The latter is suggested to be actually a variable deceleration that is late in timing and thus not associated with fetal compromise.[16,17]

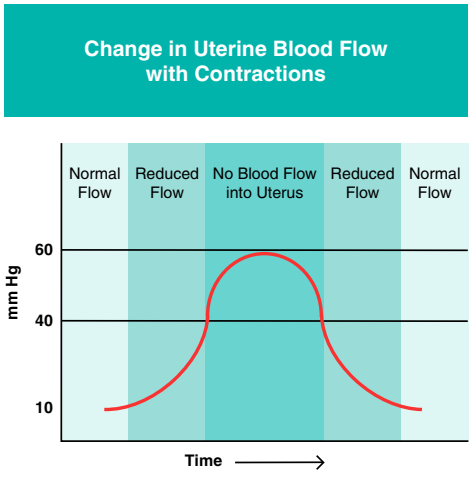

FIGURE 5. Uterine contractions and fetal oxygen status. full color online

Prolonged Decelerations

The final type of deceleration is the prolonged deceleration, defined as >2 minutes in duration (but <10 min, which would constitute a baseline change). The most likely mechanism in this type of deceleration is a sudden and prolonged reduction in oxygen delivery. The initial stages are likely to be vagal in origin—akin to the mechanism described for either variable or late decelerations above. As gas exchange becomes further impaired, direct myocardial depression can also manifest in a prolonged deceleration. The timing at which myocardial hypoxia plays a larger role in the deceleration than the peripheral chemoreflex is not known, nor is the exact mechanism whereby myocardial hypoxia triggers FHR decelerations. Experts speculate that

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the decrease in FHR is an attempt to conserve oxygen in cases of severe debt.

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Conclusions

In summary, the FHTs are controlled by a host of interwoven factors that, when properly understood, improves interpretation of the FHTs in labor. Proper interpretation can lead to improved outcomes.

Further chapters will detail terminology, interpretation, and courses of action.

While some of the physiology is well described, other areas remain controversial or even completely unknown. Thus, although practically challenging to study, this area of medicine presents multiple opportunities for further research.

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