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Interventions for Intrapartum Fetal Heart Rate Abnormalities

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Abstract: Intrapartum fetal heart rate (FHR) decelerations may represent interrupted oxygen transfer to the fetus. In many cases, these interruptions are transient and do not result in progressive fetal acidemia with risk for asphyxia and neurological compromise. When significant FHR decelerations are present, reversible causes of reduced fetal oxygen delivery should be considered and corrective measures should be undertaken to optimize oxygenation. In this review, we describe potential intrapartum causes of reduced fetal oxygen delivery and the efficacy of common interventions for an abnormal FHR tracing.
Key words: fetal oxygenation, abnormal FHR tracing

Introduction

Clinically significant fetal heart rate (FHR) changes reflect interruptions in oxygen transfer to the fetus. In many cases, these interruptions are transient and do not result in progressive fetal acidemia with associated potential for asphyxia leading to hypoxic-ischemic encephalopathy and neurological compromise. However, consideration should always be given to addressing reversible causes of reduced fetal oxygenation with corrective measures to optimize oxygen delivery whenever possible. Interventions to improve oxygen delivery are warranted for significant or recurrent FHR decelerations (Table 1). If interventions fail to improve the FHR tracing such that fetal metabolic acidemia cannot be reasonably excluded, expedited delivery is indicated. Standardized algorithms for the management of indeterminate FHR tracings have been proposed.

Interruptions in oxygen delivery can occur at any level during its passage from the environment to the fetus. When FHR changes are observed, it is important to consider where in the pathway this interruption may be occurring to inform the most appropriate interventions. The etiologies range from subtle and nonprogressive to abrupt and catastrophic for mother and fetus. Maintaining situational

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TABLE 1. FHR Decelerations: Definitions

Type of Deceleration	NICHD Classification*	"Significant Deceleration" Definition†
Variable	Visually apparent *abrupt* decrease in the FHR An *abrupt* FHR decrease is defined from the onset of the decelerations to the beginning of the FHR nadir of <30 s. The decrease in FHR is calculated from the onset to the nadir of the deceleration The decrease in FHR is ≥15 bpm, lasting ≥15 s, and <2 min in duration When variable decelerations are associated with uterine contractions, their onset, depth, and duration commonly vary with successive uterine contractions	Lasting >60 s and falling >60 bpm below the baseline Lasting >60 s with a nadir <60 bpm
Late	Visually apparent usually symmetrical *gradual* decrease and return of the FHR associated with a uterine contraction A *gradual* FHR decrease is defined as from the onset to the FHR nadir of ≥30 s The decrease in FHR is calculated from the onset to the nadir of the deceleration The deceleration is delayed in timing, with the nadir of the decelerations occurring after the peak of the contraction In most cases, the onset, nadir, and recovery of the deceleration occur after the beginning, peak, and ending of the contraction, respectively	*Any late deceleration is significant*
Prolonged	Visually apparent decrease in FHR from the baseline that is ≥15 bpm, lasting ≥2 min, but <10 min	*Any prolonged deceleration is significant*

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FHR indicates fetal heart rate; NICHD, National Institute of Child Health and Human Development.

awareness to inform appropriate intervention is paramount in the care of the intrapartum patient.

In this review, we will consider potential sources of reduced oxygen transfer and targeted interventions to improve fetal oxygen delivery (Table 2).

MATERNAL LUNGS

Adequate maternal oxygenation is the first step in fetal oxygen delivery. Impaired maternal oxygenation can occur in the setting of chronic lung disease, most commonly asthma, and acute processes such as pneumonia, acute lung injury (acute respiratory distress syndrome), respiratory depression, recent pulmonary embolism, and pulmonary edema. Late FHR decelerations may occur as a consequence of maternal hypoxia. Maternal oxygen supplementation is appropriate in cases of primary maternal lung disease to optimize maternal oxygen delivery, while other directed corrective measures are underway to improve maternal oxygenation. These may include nebulized β agonist treatment for uncontrolled asthma, diuretics for pulmonary edema, antimicrobial therapy,

TABLE 2. Fetal Oxygen Delivery: Pathways, Interruptions, Interventions

Route of Oxygen Delivery	Potential Interruptions	Potential Interventions
Maternal lungs	Suboptimal maternal oxygenation Acute respiratory illness Underlying pulmonary disease	Optimize maternal oxygenation O_2 supplementation Respiratory support β agonist treatment
Heart/vasculature	Reduced cardiac output, uterine hypoperfusion Aortocaval compression Hypovolemia Hypotension Underlying cardiac disease	Optimize volume status, systemic vascular resistance Lateral positioning Fluid bolus Correction of hypotension
Uterine blood flow to placental bed	Uterine tachysystole Tachysystole Uterine rupture*	Reduce uterine contractions, optimize perfusion Reduce/stop oxytocin Tocolysis with terbutaline Alternative pushing strategy
Placenta (maternal-fetal interface)	Poor gas exchange Placental insufficiency Abruption*	Reduce uterine contractions, optimize perfusion Reduce/stop oxytocin Alternative pushing strategy
Umbilical cord	Compression Nuchal/body cords, knots Velamentous cord insertion Cord prolapse*	Measures to reduce cord compression Maternal repositioning Amnioinfusion

*When uterine rupture or cord prolapse is suspected or diagnosed, immediate expedited delivery is indicated. In cases of suspected placental abruption, the clinician should come to the bedside for evaluation as expedited delivery may be required.

and supportive care for infectious etiologies and naloxone for reversal of acute opiate-induced central respiratory depression. The use of supplemental maternal oxygen for suspicion of impaired fetal oxygen delivery in the *absence* of maternal hypoxemia has been a subject of recent controversy. If maternal oxygenation is impaired, supplemental oxygen is indicated to treat maternal hypoxia, which will, in turn, promote fetal oxygenation. In the third trimester, the maternal mean arterial oxygen content is 101 to 106 mm Hg compared with 93 mm Hg in a nonpregnant woman.[3] If maternal hypoxia is suspected or plausibly playing a role in suboptimal fetal oxygenation, then supplemental oxygen is indicated. The empiric use of oxygen for a category II FHR tracing will be addressed later in this chapter in more detail.

MATERNAL VASCULATURE

The ability of the maternal vasculature to deliver oxygen to the fetus depends on adequate maternal cardiac output, vascular tone, volume status, and preload to promote adequate uteroplacental perfusion. When maternal cardiac output is reduced, uterine perfusion can be comprised. This typically manifests as fetal late or prolonged decelerations, or, in severe or protracted cases, fetal bradycardia. Conditions that impair maternal cardiac output such as arrhythmia, cardiomyopathy, heart failure, and valvular disease may impair fetal oxygen delivery. Reduced maternal systemic vascular resistance and/or hypovolemia can have the same results. In the healthy parturient, a common etiology of intrapartum hypotension is reduced systemic vascular resistance following regional anesthesia

with resultant sympathectomy.[4] Aortocaval compression from the gravid uterus in the late third trimester can result in a 10% to 30% decrease in maternal cardiac output secondary to reduced venous return/cardiac preload.[5] Hypovolemia secondary to dehydration from the poor oral intake is not uncommon among women who present late in their labor course. The final common pathway amongst these is typically a maternal relative or absolute hypotension resulting in uteroplacental hypoperfusion.

Appropriate treatment of maternal hypotension requires careful consideration of the underlying etiology, particularly in a patient with known cardiac disease, but position change to relieve aortocaval compression, correction of hypovolemia with intravenous fluid administration, and pharmacologic blood pressure support for iatrogenic hypotension following regional anesthesia with resultant sympathectomy may all be appropriate.[6] In some cases, *relative* hypotension can be the source of uteroplacental hypoperfusion, such as in the severely hypertensive parturient after pharmacologic intervention with inadvertent overcorrection of blood pressure. In the setting of an antepartum or intrapartum hypertensive emergency, the goal is to maintain maternal systolic blood pressure <160 mm Hg and diastolic blood pressure <110 mm Hg. Care should be taken to titrate antihypertensive therapy to maintain maternal systolic pressure in the mild range (140 to 159 mm Hg and diastolic pressure 90 to 105 mm Hg) without overcorrection and relative hypotension that may result in impaired uteroplacental perfusion. When overcorrection is suspected based on the appearance of late decelerations in the FHR tracing in the context of a rapid pharmacologically mediated drop in maternal blood pressure, working closely with the anesthesiologist to address relative hypotension is essential.

UTERINE PERFUSION

Once maternal blood reaches the uterus, normal perfusion of the uteroplacental interface depends upon blood flow through the uterine vasculature. During uterine contraction, the myometrial pressure increases, and the small uterine arterioles are compressed, reducing perfusion of the maternal-fetal interface at the placental bed. The strength and frequency of uterine contractions thus determine the adequacy of placental perfusion and fetal oxygenation. The mean intrapartum maternal arterial blood pressure is 85 to 90 mm Hg. During normal, spontaneous labor, the intra-amniotic pressure typically reaches a peak of 80 to 100 mm Hg during a contraction in the second stage with a resting tone of 10 to 12 mm Hg.[7] The myometrial pressure during a contraction is estimated to be 2 to 3 times higher than the intra-amniotic pressure such that when intrauterine pressure reaches about 30 mm Hg during a contraction, myometrial pressure occludes the maternal spiral arteries and flow to the placental bed is transiently interrupted.[8] It takes at least 90 seconds for reperfusion of the placental bed between contractions.[9] In the setting of uterine tachysystole, characterized by >5 contractions in a 10-minute period averaged over 30 minutes, there is insufficient time for perfusion between contractions to allow optimal maternal-fetal oxygen-CO_2 exchange and this results in fetal oxygen debt and acid accumulation over time.[10] Tachysystole can occur during spontaneous or stimulated labor and is not always accompanied by FHR decelerations. When decelerations are present and when tachysystole is the result of overstimulation, interventions are indicated to reduce uterine activity and thus promote optimal oxygen delivery to the fetus. If tachysystole is occurring during stimulated labor, then oxytocin should be reduced or discontinued. Tachysystole resolves more quickly when cessation of

oxytocin is combined with lateral positioning and administration of a 500 mL intravenous fluid bolus.[11] When FHR changes are seen in association with tachysystole, treatment with a β2-adrenergic agent such as terbutaline often improves the FHR tracing.[12] Administration of a β2 agonist is most commonly used in the setting of acute and significant FHR changes, such as a prolonged deceleration or bradycardia, occurring in response to tachysystole. The efficacy of β2-adrenergic agents in terms of more meaningful outcomes such as the rate of cesarean delivery, Apgar scores, umbilical arterial pH, and neonatal morbidity is less clear.[13] It is worth noting that the use of tocolytic agents for an abnormal FHR tracing in the *absence* of tachysystole may improve the FHR tracing but has not been associated with improvement in neonatal outcome.[13] There is insufficient evidence to recommend tocolysis for an abnormal FHR tracing in the absence of uterine tachysystole.

In rare cases, acute uterine rupture can result in an abrupt and massive disruption in fetal oxygen transfer leading to catastrophic risk to mother and fetus. While uterine rupture can occur spontaneously (particularly after protracted or obstructed labor or in the setting of uterine overdistension), this rare complication is more common among parturients undergoing a trial of labor after one or more previous cesareans. When suspected, immediate intervention by emergent delivery is required.

PLACENTAL FUNCTION

Ultimately, oxygen transfer from mother to the fetus occurs at the maternal-fetal placental interface. Maternal oxygenated blood flows under low pressure into the maternal intervillous space where gas and nutrient exchange occurs across a single cell layer barrier between maternal blood and fetal chorionic villi. This delicate but reliable process can be interrupted chronically or acutely. Placental insufficiency with longstanding, diffusely poor gas exchange leads to a chronic fetal hypoxemic state that can acutely worsen intrapartum as uterine contractions further reduce uteroplacental perfusion. The typical FHR pattern observed in cases of preexisting uteroplacental insufficiency is late decelerations due to poor fetal tolerance of the added physiological stress of contraction-related reductions in uteroplacental blood flow. Placental abruption can lead to a loss of perfused placental surfaces for maternal-fetal exchange. Abruption may be chronic or acute, mild or catastrophic, depending on the extent of disruption at the uteroplacental interface. Abruption can cause an acute, sometimes catastrophic, interruption in fetal oxygen delivery. When abruption is suspected by the onset of vaginal bleeding, sudden exacerbation of labor pain, uterine tenderness with tetanic contraction, and/or a classic tocometric pattern of contractions every 1 to 2 minutes without return to normal resting tone between contractions, the managing physician or midwife should come to the bedside for an immediate evaluation of the patient and the FHR tracing. Preparations should be made in case an emergent delivery is required for maternal or fetal decompensation before vaginal delivery can be achieved.

UMBILICAL CORD

The umbilical cord is the final conduit of oxygen delivery from mother to fetus. Two umbilical arteries carry deoxygenated blood from the fetus to the placenta and one umbilical vein returns oxygenated blood to the fetal circulation. Fetal oxygen delivery can be reduced acutely by umbilical cord compression which can occur intrapartum due to oligohydramnios, nuchal/body cords, true knots, a paucity of protective Wharton jelly, and, of course, the dreaded umbilical cord prolapse below the fetal presenting part

through a dilated cervix and into the vagina. Variable decelerations are the pattern most typically seen in the setting of episodic cord compression. The new onset of significant variable decelerations following spontaneous or artificial rupture of membranes should prompt immediate evaluation with a vaginal examination to exclude cord prolapse, which requires elevation of the fetal head and emergent delivery. Of course, if cord compression is severe or unrelenting, such as with cord prolapse, then this can produce sudden bradycardia in a previously normal FHR tracing and immediate delivery is required. When nonprolapse-related cord compression is suspected, amnioinfusion may be undertaken in an effort to restore protective fluid cushioning to the cord. Intrapartum saline amnioinfusion has been shown to improve repetitive variable decelerations, increase the umbilical arterial pH, and reduce cesarean delivery among term laboring patients with suspected cord compression without increasing the risk of infection.[14,15] The success of amnioinfusion for relief of cord compression-associated variable decelerations is inversely correlated with the preinfusion amniotic fluid index.[16]

Alternative Pushing Strategies

A category II FHR tracing is more commonly encountered in the second stage of labor.[17] Expulsive efforts with Valsalva increases intrauterine pressure, maternal breath-holding reduces continuous oxygen delivery, most women have already experienced a rupture of membranes (spontaneous or artificial) with higher potential for cord compressive effects, and many women are encouraged to push in a supine position that can lead to aortocaval compression. As discussed above, all of these measures can reduce fetal oxygen delivery to the fetus. If recurrent FHR decelerations are observed, consideration should be given to alternative pushing strategies such as side-lying, squatting, pushing with every other contraction, and reduced duration of breath-holding to optimize fetal oxygenation.[18]

Abrupt Versus Evolving Interruption in Fetal Oxygen Delivery

During the evaluation of FHR changes indicative of impaired oxygen delivery, it is imperative that providers retain situational awareness to differentiate incremental reductions in oxygen delivery that may be amenable to conservative interventions to restore oxygenation and prevent the development of fetal acidemia over time from those that represent a catastrophic interruption in oxygen delivery requiring emergent intervention to prevent catastrophic oxygen deprivation and rapid fetal asphyxia. Examples of the latter include umbilical cord prolapse, uterine rupture, rupture of a vasa previa, and massive placental abruption.

Maternal Oxygen Supplementation for the Category II FHR Tracing

The normal maternal arterial partial pressure of oxygen (PaO_2) is ~100 mm Hg (dissolved, unbound oxygen in the blood) with >98% oxygen saturation (percentage of occupied oxygen-binding sites on hemoglobin), while the fetal venous partial pressure of oxygen (PO_2) is about 28 mm Hg with 60% to 70% oxygen saturation.[19] Oxygen diffuses from maternal arterial blood (which has the highest PO_2 in the placental circulation) into the intervillous space across the single-cell layer of the chorionic villi into the fetal arterial blood (which has the lowest PO_2 in the placental circulation) where it is carried back through the umbilical vein into the fetal circulation as dissolved oxygen and oxygen bound to fetal hemoglobin. The higher oxygen-binding capacity of fetal

hemoglobin combined with a higher absolute fetal hemoglobin level compensates for this lower PO_2 to create a high fetal venous total oxygen content.[19] The fetal venous PO_2 can never exceed maternal venous PO_2 based upon the principle of diffusion of gases along partial pressure gradients. The use of supplemental maternal oxygen to increase fetal oxygenation in anticipation that is will produce improvement in neonatal outcomes is a longstanding practice in obstetrics supported by relatively limited evidence. Several studies have attempted to further elucidate the impact of maternal oxygen supplementation on fetal oxygenation and acid-base status. Two small studies from the 1960s and 1970s suggested that maternal hyperoxia could reduce the frequency of late FHR decelerations.[7,20,21] Early work in an ovine model showed that maternal hyperoxia from oxygen supplementation produced only small increases in fetal arterial and venous oxygen content.[22,23] A fetal pulse oximetry study in normal, term laboring patients showed that maternal administration of 100% oxygen could produce small increases in fetal oxygen saturation while the use of 40% oxygen, such as might be expected in clinical practice with a typical face mask in the L&D setting, did not produce a significant increase in fetal oxygen saturation.[24] Similar studies focused on laboring patients with category II FHR tracings showed a modest improvement of 10% to 15% in fetal oxygen saturation as measured by transcutaneous fetal oximetry.[25] Indeed, the lower the starting fetal oxygen saturation, the higher the degree of observed improvement with maternal oxygen supplementation.[25] The most relevant question to address is how these modest improvements in fetal oxygenation from maternal hyperoxia impact meaningful perinatal outcomes. Some have raised the important question of whether maternal oxygen supplementation actually improves the fetal acid-base status or simply makes the FHR tracing more reassuring.[26] Two small randomized controlled trials among term, laboring patients with reassuring FHR tracings showed no apparent improvement in fetal acid-base status with the administration of maternal oxygen.[27,28] One of these studies demonstrated a higher incidence of umbilical arterial pH <7.2 among neonates born to women given supplemental oxygen before delivery compared with those on room air.[27] Two small randomized controlled trials compared supplemental oxygen to room air for laboring women with a category II FHR tracing and both showed no difference in umbilical arterial lactate, umbilical arterial pH, cesarean, or operative vaginal delivery.[29,30]

In the absence of distinct evidence for the improved perinatal outcome by the use of maternal oxygen supplementation, the potential risks must be considered carefully. Hyperoxia can lead to the increased production of oxygen-free radicals that are known to cause cellular damage to phospholipid membranes, lipoproteins, and DNA. Excess oxygen exposure and associated oxidative stress in premature neonates is associated with intraventricular hemorrhage, retinopathy of prematurity, and chronic lung disease.[31] In cases of perinatal asphyxia, the production of free radicals following neonatal reperfusion is thought to be one mechanism of neonatal asphyxia-related brain injury.[32–34] On the basis of considerable evidence that neonatal mortality is increased by resuscitation with 100% oxygen, the American Academy of Pediatrics and American Heart Association guidelines recommend the use of room air in newborn resuscitation.[35–38] Animal models have demonstrated that maternal administration of oxygen following a period of induced fetal hypoxia leads to increased markers of free radical activity in fetal blood.[39–41] Khaw et al[42] administered 60% oxygen to women before

planned cesarean delivery and found elevated levels of 3 markers of oxygen-free radical activity in cord blood samples. It is as yet unclear how this correlates to the clinical scenario of oxygen supplementation for the hypoxic fetus in utero. One study showed that while fetal hyperoxemia itself was not associated with neonatal morbidity, among neonates born with acidemia, those with venous umbilical hyperoxemia had a slightly increased incidence of neonatal morbidity compared with acidemic neonates with normal venous oxygen levels.[43] On the basis of our current understanding of the risks of high oxygen exposure in the neonate and the correlation of oxygen-free radicals with reperfusion injuries follow a period of hypoxia, we agree with others who have suggested reevaluation of the longstanding practice of routine maternal oxygen supplementation for an abnormal FHR tracing.[26] When the FHR tracing suggests impaired oxygen delivery but hypoxemia and metabolic acidosis can be reasonably excluded (there is moderate variability and the presence of accelerations), maternal supplemental oxygen is unlikely to have a significant impact on meaningful neonatal outcomes, may increase the production of harmful oxygen-free radicals, and may unnecessarily increases maternal anxiety. A more judicious approach to oxygen supplementation should be considered. In cases where fetal hypoxemia and acidosis are strongly suspected (category III FHR tracing), oxygen should be considered while preparations are being made for immediate delivery without delay based on available evidence that amongst truly hypoxic fetuses, maternal hyperoxia may improve fetal oxygenation.[25] For category II tracings, wherein fetal hypoxia and metabolic acidosis cannot be reasonably excluded (recurrent late or significant variable decelerations, prolonged decelerations, minimal to absent variability for >30 to 60 min), the plausible benefits of maternal hyperoxia to improve fetal oxygenation may also outweigh the uncertain efficacy and theoretical risks.[44]

Conclusions

FHR monitoring can be thought of as fetal brain oxygenation monitoring. FHR tracing abnormalities often reflect interruptions in oxygen transfer from mother to fetus. An understanding of maternal-to-fetal oxygen delivery enables the clinician to consider the underlying pathophysiology behind FHR decelerations and thus informs interventions to optimize fetal oxygenation in the management of an abnormal FHR tracing.

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