Physiologic Basis for Fetal Heart Rate Monitoring
Physiologic Basis for Fetal Heart Rate Monitoring

The objective of intrapartum fetal heart rate (FHR) monitoring is to prevent fetal injury that might result from interruption of fetal oxygenation during labor.

The underlying assumption is that the FHR tracing can reveal specific information regarding fetal oxygenation.

Understanding the physiologic basis for electronic FHR monitoring requires a realistic appraisal of this basic assumption.
Goal of Fetal Heart Rate Monitoring

Intrapartum fetal heart rate monitoring is intended to assess the adequacy of fetal oxygenation during labor.

Fetal oxygenation involves:

- The transfer of oxygen from the environment to the fetus
- The fetal response to interrupted oxygen transfer

Certain FHR patterns provide reliable information regarding both of the basic elements of fetal oxygenation.
Transfer of Oxygen from The Environment To The Fetus

Oxygen is carried from the environment to the fetus by maternal and fetal blood along a pathway that includes the maternal lungs, heart, vasculature, uterus, placenta and umbilical cord.

The “Oxygen Pathway”
Maternal Lungs

Inspiration carries oxygen from the external environment to the distal air sacs of the lung, the alveoli.

Interruption of oxygen transfer from the environment to the alveoli can result from airway obstruction (for example asthma) or from interruption of breathing caused by depression of central respiratory control (narcotics, magnesium sulfate, seizure).
Maternal Lungs

From the alveoli, oxygen diffuses across a thin barrier into the pulmonary capillary blood.

 Interruption of oxygen transfer from the alveoli to the pulmonary capillary blood can be caused by factors such as ventilation-perfusion mismatch and diffusion defects (such as pneumonia or pulmonary embolus).
Interruption of oxygen transfer at the level of the lungs

In an obstetric population, pulmonary causes of interrupted oxygenation include:

- Respiratory depression
- Medications
- Seizures
- CNS depression
- Pulmonary embolus, amniotic fluid embolus
- Pulmonary edema
- Pneumonia, adult respiratory distress syndrome
- Asthma, atelectasis
Maternal blood

After diffusing from the pulmonary alveoli into maternal blood, more than 98% of oxygen combines with hemoglobin in maternal red blood cells.

Approximately 1-2% remains dissolved in the blood and is measured by the partial pressure of dissolved oxygen (PaO2).

A normal adult PaO2 value of 95-100 mmHg results in hemoglobin saturation of approximately 95-98%, indicating that hemoglobin is carrying 95-98% of the total amount of oxygen it is capable of carrying.
Interruption of oxygen transfer at the level of the maternal blood

Interruption of oxygen transfer from the environment to the fetus due to abnormal maternal oxygen carrying capacity can result from severe anemia or from hereditary or acquired abnormalities affecting oxygen binding (hemoglobinopathies, methemoglobinemia).

In an obstetric population, reduced maternal oxygen carrying capacity rarely causes acute interruption of fetal oxygenation.
Maternal heart

From the lungs, pulmonary veins carry oxygenated maternal blood to the heart.

Blood enters the left atrium with a PaO2 of approximately 95-100 mmHg.

Oxygenated blood passes from the left atrium, through the mitral valve into the left ventricle and out the aorta for systemic distribution.
Interruption of oxygen transfer at the level of the heart

Interruption of oxygen transfer from the environment to the fetus at the level of the maternal heart can be caused by any condition that reduces cardiac output, including:

- Altered heart rate (arrhythmia)
- Reduced preload (hypovolemia, compression of the IVC)
- Impaired contractility (ischemic heart disease, cardiomyopathy)
- Increased afterload (hypertension)
Interruption of oxygen transfer at the level of the heart

In addition, structural abnormalities of the heart and/or great vessels may impede the normal ability to pump blood (valvular stenosis, valvular insufficiency, pulmonary hypertension, coarctation of the aorta)

In a healthy obstetric patient, the most common cause of reduced cardiac output is reduced preload (hypovolemia, compression of the inferior vena cava).
Maternal vasculature

Oxygenated blood leaving the heart is carried by the systemic vasculature to the uterus

The path includes the aorta, iliac vessels and the uterine arteries

From the uterine artery, oxygenated blood travels through the arcuate arteries, the radial arteries and finally the spiral arteries before exiting the maternal vasculature and entering the intervillous space of the placenta
Interruption of oxygen transfer at the level of the maternal vasculature

Hypotension
- Regional anesthesia
- Hypovolemia (dehydration, hemorrhage)
- Impaired venous return
- Impaired cardiac output
- Medications

Vasoconstriction of distal arterioles in response to endogenous vasoconstrictors or medications
Uterus

Between the maternal uterine arteries and the intervillous space of the placenta, the arcuate, radial and spiral arteries traverse the muscular wall of the uterus.
Interruption of oxygen transfer at the level of the uterus

Interruption of normal oxygen transfer from the environment to the fetus at the level of the uterus commonly results from uterine contractions that compress intramural blood vessels and impede the flow of oxygenated maternal blood into and out of the intervillous space.

Less common causes include uterine rupture or trauma.

The evaluation of uterine activity is reviewed in a separate module.
Placenta

The placenta is the maternal-fetal interface that facilitates the exchange of gases, nutrients, wastes and other molecules (for example antibodies, hormones, medications) between maternal blood in the intervillous space of the placenta and fetal blood in the villous capillaries.
Placental causes of interrupted oxygenation

Many conditions can interfere with the normal transfer of oxygen across the placenta.

Clinically, the most common cause of acute interruption of oxygen transfer at level of the placenta is separation of the placenta from the uterine wall (abruption, bleeding placenta previa).

Fetal-maternal hemorrhage and vasa previa should be considered in the appropriate clinical setting.
Fetal blood

After oxygen has diffused from the intervillous space across the placental “blood-blood” barrier and into fetal blood, the PaO2 in the umbilical vein returning to the fetus is in the range of 35 mmHg and fetal hemoglobin saturation is between 50 and 70%
Umbilical cord

After oxygen combines with fetal hemoglobin in the villous capillaries, oxygenated blood returns to the fetus by way of a single umbilical vein within the umbilical cord.

Interruption of the normal transfer of oxygen from the environment to the fetus at the level of the umbilical cord most often results from mechanical cord compression.

Other uncommon causes may include vasospasm, thrombosis, atherosclerosis, hypertrophy, hemorrhage, inflammation or a “true knot”.
### Summary

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Fetal oxygenation

Oxygen transfer from the environment to the fetus represents the first component of fetal oxygenation.

The second component of fetal oxygenation involves the fetal physiologic response to interrupted oxygen transfer.
Fetal response to interrupted oxygen transfer

Depending upon frequency and duration, interruption of oxygen transfer at any point along the oxygen pathway may result in progressive deterioration of fetal oxygenation.

The cascade begins with hypoxemia, defined as decreased oxygen content in the blood.

At term, hypoxemia is characterized by an umbilical artery PaO2 below the normal range of 15-25 mmHg.
Fetal response to interrupted oxygen transfer

Recurrent or sustained hypoxemia can lead to decreased delivery of oxygen to the tissues and reduced tissue oxygen content, or hypoxia.

Normal homeostasis requires an adequate supply of oxygen and fuel in order to generate the energy required by basic cellular activities.
Fetal response to interrupted oxygen transfer

When oxygen is readily available, aerobic metabolism generates energy efficiently in the form of ATP.

By-products of aerobic metabolism include carbon dioxide and water.

When oxygen is in short supply, tissues may be forced to convert from aerobic to anaerobic metabolism, generating energy less efficiently and resulting in the production of lactic acid.
Fetal response to interrupted oxygen transfer

Accumulation of lactic acid in the tissues results in metabolic acidosis

Lactic acid accumulation can lead to utilization of protective buffer bases (primarily bicarbonate) to help stabilize tissue pH

If the buffering capacity is exceeded, the blood pH may begin to fall, leading to metabolic acidemia

It is critical to distinguish between metabolic acidemia and respiratory acidemia
Fetal response to interrupted oxygen transfer

Metabolic acidemia is caused by accumulation of lactic acid in the setting of anaerobic metabolism.

Respiratory acidemia is caused by accumulation of CO2, a byproduct of aerobic metabolism that than increase in the blood when placental gas exchange is suboptimal.

In contrast to metabolic acidemia, isolated respiratory acidemia has no known association with adverse outcome.
Fetal response to interrupted oxygen transfer

Regardless of the specific cause of interrupted fetal oxygenation, recurrent or sustained tissue hypoxia and acidosis can lead to loss of peripheral vascular smooth muscle contraction, reduced peripheral vascular resistance and hypotension.
Fetal response to interrupted oxygen transfer

If interrupted oxygen transfer progresses to the stage of metabolic acidemia and hypotension, multiple organs and systems (including the brain and heart) can suffer hypoperfusion, reduced oxygenation, lowered pH and reduced delivery of fuel for metabolism.

Interruption of normal cellular metabolism can lead to cellular dysfunction, tissue dysfunction and even death.
Injury threshold

We have reviewed fetal oxygenation in detail, including each step of oxygen transfer from the environment to the fetus and each stage of the fetal physiologic response to interrupted oxygenation.

We have reviewed the mechanisms of injury in the setting of recurrent or sustained interruption of oxygenation.

The precise relationship between interrupted fetal oxygenation and neurologic injury is complex and incompletely understood.
Electronic FHR monitoring was introduced with the expectation that it would significantly reduce the incidence of neurologic injury (specifically cerebral palsy) caused by intrapartum interruption of fetal oxygenation.

In recent years, it has become apparent that most cases of cerebral palsy are unrelated to intrapartum events and therefore cannot be prevented by intrapartum FHR monitoring.

Nevertheless, a significant minority of such cases may be related to intrapartum events and might be preventable.
In 1999, the International Cerebral Palsy Task Force published a consensus report regarding the relationship between intrapartum interruption of fetal oxygenation and subsequent neurologic injury.

Injury threshold

Agencies and professional organizations that reviewed and endorsed the ACOG-AAP report include:

American College of Obstetricians and Gynecologists
American Academy of Pediatrics
Centers for Disease Control
The Child Neurology Society
March of Dimes Birth Defects Foundation
National Institute of Child Health and Human Development
Royal Australian and New Zealand College of Obstetricians and Gynecologists
Society for Maternal-Fetal Medicine
Society of Obstetricians and Gynaecologists of Canada
Injury threshold

The consensus report established four essential criteria defining an acute intrapartum event sufficient to cause cerebral
Essential criteria that define an acute intrapartum event sufficient to cause cerebral palsy (must meet all four)

1. Umbilical cord arterial blood pH < 7 and base deficit ≥ 12 mmol/L

2. Early onset of severe or moderate neonatal encephalopathy in infants born at 34 or more weeks of gestation

3. Cerebral palsy of the spastic quadriplegic or dyskinetic type

4. Exclusion of other identifiable etiologies such as trauma, coagulation disorders, infectious conditions or genetic disorders
Essential criteria that define an acute intrapartum event sufficient to cause cerebral palsy

The first criterion indicates that intrapartum interruption of fetal oxygenation does not result in neurologic injury unless it progresses at least to the stage of significant metabolic acidemia (umbilical artery pH < 7 and base deficit ≥ 12 mmol/L)

It is important to note that fetal injury is uncommon even when metabolic acidemia is present

It is also important to understand that respiratory acidemia is not a recognized risk factor for fetal injury
Essential criteria that define an acute intrapartum event sufficient to cause cerebral palsy

The second criterion highlights an equally important point: intrapartum interruption of fetal oxygenation does not result in cerebral palsy without first causing moderate-severe neonatal encephalopathy.

The report further clarified that neonatal encephalopathy has many possible causes. “Hypoxic-ischemic” encephalopathy resulting from intrapartum interruption of fetal oxygenation represents only a small subset of the larger category of neonatal encephalopathy.
Essential criteria that define an acute intrapartum event sufficient to cause cerebral palsy

The third criterion emphasizes that different subtypes of cerebral palsy have different clinical origins

Spastic quadriplegia is associated with injury to the parasagittal cerebral cortex and involves abnormal motor control of all four extremities

The dyskinetic subtype of cerebral palsy is associated with injury to the basal ganglia and involves disorganized, choreoathetoid movements
Essential criteria that define an acute intrapartum event sufficient to cause cerebral palsy

The report concluded that these are the only two subtypes of cerebral palsy associated with term “hypoxic-ischemic” injury

Specifically, spastic diplegia, hemiplegia, ataxia and hemiparetic cerebral palsy are “unlikely to result from acute intrapartum hypoxia”
Essential criteria that define an acute intrapartum event sufficient to cause cerebral palsy

The report further concluded that other conditions, including epilepsy, mental retardation and attention deficit hyperactivity disorder do not result from “birth asphyxia” in the absence of cerebral palsy
Essential criteria that define an acute intrapartum event sufficient to cause cerebral palsy

The fourth criterion emphasizes that intrapartum “hypoxic-ischemic” injury is a potential factor in only a small subset of all cases of cerebral palsy.

Most cases of cerebral palsy are unrelated to intrapartum events.

The report identified four additional criteria that can help establish the timing of injury, emphasizing that these criteria are “nonspecific to asphyxial insults”
Criteria that collectively suggest the event occurred within 48 hours of birth

1. A sentinel hypoxic event immediately before or during labor

2. A sudden and sustained fetal bradycardia or the absence of FHR variability in the presence of persistent late or variable decelerations, usually after a hypoxic sentinel event when the pattern was previously normal

3. Apgar scores of 0–3 beyond 5 minutes

4. Onset of multisystem involvement within 72 hours of birth
Summary

The physiology of fetal oxygenation involves the sequential transfer of oxygen from the environment to the fetus and the subsequent fetal response if oxygen transfer is interrupted.

Interruption of normal oxygen transfer can occur at any point along the oxygen pathway.

Recurrent or sustained interruption of normal oxygen transfer can lead to progressive deterioration of fetal oxygenation and eventually to potential fetal injury.
Summary

However, significant metabolic acidemia (umbilical artery pH < 7.0 and base deficit ≥12 mmol/L) has been identified as an essential pre-condition to intrapartum hypoxic injury.

With respect to the relationship between fetal oxygenation and potential injury, there is consensus in the literature that interrupted oxygenation does not result in fetal injury unless it progresses at least to the stage of significant metabolic acidemia.
Summary

The physiologic basis of FHR monitoring can be summarized in a few key concepts.

The objective of intrapartum FHR monitoring is to assess fetal oxygenation during labor.

Fetal oxygenation involves the transfer of oxygen from the environment to the fetus and the subsequent fetal response if oxygen transfer is interrupted.
Oxygen is transferred from the environment to the fetus by maternal and fetal blood along a pathway that includes the maternal lungs, heart, vasculature, uterus, placenta and umbilical cord.

Summary

Environment
↓
Lungs
↓
Heart
↓
Vasculature
↓
Uterus
↓
Placenta
↓
Umbilical cord
↓
Fetus
Summary

The fetal response to interrupted oxygen transfer involves a sequential physiologic progression:

Hypoxemia → Hypoxia → Metabolic acidosis → Metabolic acidemia

Fetal injury due to interrupted oxygenation does not occur unless this process has progressed at least to the stage of significant metabolic acidemia (umbilical artery pH <7.0 and base deficit ≥ 12 mmol/L)