FHR Monitoring: Maternal Fetal Physiology

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Goals

• Review the basic physiology and adaptive responses that regulate the FHR

• Review oxygen delivery to the fetus, potential disruptions, and route to injury

• Correlate physiologic fetal adaptations to stress with FHR deceleration patterns
Goal of Intrapartum Monitoring

Assess the adequacy of intrapartum fetal oxygenation

- Reduce perinatal morbidity and mortality
  - Perinatal death
    - Intrapartum and neonatal
  - Birth asphyxia
    - Long-term neurological impairment
The Basic Assumption

Fetal adaptive responses to progressive hypoxemia and acidosis are detectable

FHR monitoring = fetal brain oxygenation monitoring
Basic Heart Rate Physiology

- Cardiorespiratory Center (medulla oblongata)
- Determines FHR baseline, variability, pattern
- Coordinates input from intrinsic influences
  - Parasympathetic nervous system
  - Sympathetic nervous system
  - Baroreceptors (aortic arch, carotid)
  - Chemoreceptors (central and peripheral)
  - Endocrine system (hormones)
  - Sleep-wake cycle
  - Breathing/Pain/Sound/Temperature
Parasympathetic Influence

- Vagus (CN X) nerve originates in CRC
  - Innervates SA and AV node
    - Acetylcholine mediated inhibitory influence slows HR
  - Determines baseline fetal heart rate
  - Responsible for beat-to-beat variability
    - Anticholinergic block (atropine) leads to loss of variability
    - Presence of moderate variability reasonably excludes significant metabolic acidosis
  - Vagal influence can rapidly decrease fetal heart rate
    - Responsible for variable decelerations
Sympathetic Influence

- Sympathetic nerve fibers throughout the myocardium and vasculature
  - Catecholamine (epinephrine, norepinephrine) mediated stimulatory influence
    - Sympathetic nerve terminals, adrenal medulla
  - Increased heart rate, contractility
    - Produces *accelerations* in response to stimulus
    - Produces *fetal tachycardia*
  - Vasoconstriction, hypertension
    - Preserve central/brain perfusion in times of stress
Baroreceptor Influence

- Pressure detecting sensory nerve fibers
  - Stretch receptors
  - Aortic arch, carotid bifurcation
- Stimulation leads to increased vagal outflow
  - Lowers heart rate, reduces cardiac output
    - Think homeostasis!
- Key role in *variable decelerations* from cord compression and *decelerations/bradycardia* in response to fetal hypertension
Chemoreceptor Influence

- Chemosensitive cells
  - Detect $\downarrow$PaO$_2$, $\uparrow$H$^+$, $\uparrow$PaCO$_2$

- Central (medulla oblongata)
  - Stimulates reflex increase in sympathetic outflow
    - *tachycardia and hypertension*

- Peripheral (aortic arch, carotid bodies)
  - Increased parasympathetic outflow
    - *hypoxemia related decelerations (late and variables with associated hypoxia)*
    - *prolonged decelerations, bradycardia*
Hormonal Regulation

- Adrenal medulla
  - Stress induced catecholamine release
    - Epinephrine, norepinephrine
  - Sympathetic nervous system responses
    - Increased HR, vasoconstriction, cardiac contractility
      - *Fetal tachycardia*
  - Peripheral vasoconstriction, central shunting to brain/heart/adrenals for self-preservation
Homeostatic Interactions

- Maintenance of Acid-Base Status
  - Determined by blood supply and oxygen delivery by the placenta
  - Bicarbonate buffering system
  - Placenta is the fetal organ for respiration
    - Provides oxygen
    - Removes CO₂
Extrinsic Influences

- Medications
  - Magnesium, beta blockers, betamethasone, opioids, anesthetic agents
- Local electrolyte levels
  - Calcium, potassium
Back to the Forest
FHR Baseline

- Initiated at the SA node
  - Modulated by intrinsic and extrinsic factors

- Normal range 110-160 bpm
  - Mean FHR in a 10 min window rounded to the nearest 5 bpm, excluding accels/decels
    - A two minute window of consistent baseline is required to establish the baseline rate

- Determined by *parasympathetic tone*
  - Increase in vagal tone leads to the 10 bpm decrease in FHR baseline between 28w and term
What is the baseline of this tracing??

130 BPM
FHR Variability

- Changes in FHR around the baseline rate over short (seconds) and extended (minutes) time
  - Peak to trough measurement
  - Utilize same 10 min window as baseline assessment
- Results from *interplay of parasympathetic and sympathetic nervous systems*
- Normal variability means:
  - Functioning neuromodulation of FHR
  - Normal cardiac responsiveness
  - Normal acid-base status
What is the variability? Moderate ~25 bpm
Absent/Undetectable Variability

Minimal Variability ≤5 bpm

Moderate Variability 6-25 bpm

Marked Variability >25 bpm
FHR Accelerations

- Abrupt increase in FHR to peak within 30 sec
- After 32 weeks “15 x 15”
  - At least 15 bpm x 15 sec, lasting <2 min
- Before 32 weeks “10 x 10”
  - At least 10 bpm x 10 sec, lasting <2 min
- Sympathetic nervous system mediated
  - Periodic with contractions
    - Scalp stimulation, mild recurrent venous cord occlusion
  - Non-Periodic (the majority)
    - fetal activity, acoustic/scalp stimulation, mild cord compression (venous)
FHR Acceleration
The Pathophysiology of Decels

Decelerations represent an adaptation to interrupted oxygen transfer*

* Early decelerations are the exception, representing head compression and increased vagal outflow during a contraction
Early Deceleration

Context:
• Head compression during UC

Etiology:
• Vagal stimulation
  • Reflex slowing of HR during compression

http://perinatology.com/images/earlyex.gif
Oxygen Transfer to the Fetus

- Environment
- Lungs
- Heart
- Vasculature
- Uterus
- Placenta
- Umbilical cord

Oxygen transfer can be interrupted at any point along this pathway.

http://www.stanfordchildrens.org/content-public/topic/images/67/281867.jpeg
## Interrupted Oxygen Transfer

<table>
<thead>
<tr>
<th>Oxygen Transfer Pathway</th>
<th>Potential Cause of Interrupted Transfer</th>
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<td>Respiratory Depression</td>
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<td>Asthma</td>
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<td>Heart/Vasculature</td>
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<td>Uterine stimulant effect</td>
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<td></td>
<td>Uterine rupture</td>
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<tr>
<td>Placenta</td>
<td>Abruption, fetal-maternal hemorrhage,</td>
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<tr>
<td></td>
<td>Insufficiency (poor gas exchange)</td>
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<tr>
<td>Umbilical Cord</td>
<td>Cord compression (nuchal/body cord, knot,</td>
</tr>
</tbody>
</table>
Oxygen and Energy Generation

**Aerobic**

GLUCOSE

\[ \text{O}_2 \]

ATP + H\textsubscript{2}O + CO\textsubscript{2}

Placental diffusion
CO\textsubscript{2} rapidly blown off through maternal respiration

**Anaerobic**

\[ \not\text{O}_2 \]

ATP + Lactic Acid

**Fetal Acidemia**

Impaired metabolic function, cell injury and cell death
Pathway to Fetal Injury

Hypoxemia → Reduced oxygen in fetal BLOOD

Hypoxia → Reduced oxygen in fetal TISSUE

Metabolic acidosis → Increased lactic acid in TISSUE

Metabolic acidemia → Increased lactic acid in BLOOD

Reduced peripheral vascular smooth muscle contraction

Reduced peripheral vascular resistance

Hypotension and POTENTIAL FOR INJURY
Fetal Response to Hypoxemia

Hypoxemia

Chemoreceptor Reflex

- Reduced oxygen in fetal BLOOD
- Sympathetic outflow catecholamines

Increase heart rate, blood pressure

Baroreceptor Reflex

- Parasympathetic outflow vagus nerve

FHR Deceleration
Specific Deceleration Pathways

- **Variable**
  - Abrupt decel ≥ 15 bpm below baseline, rapid onset to nadir ≤ 30 sec, lasting ≥ 15 sec but < 2 min
  - Varied/inconsistent relationship to contractions

- **Late**
  - Gradual in onset (>30 sec), typically smooth and symmetric
  - Delay in timing with onset, nadir and resolution after the beginning, peak, and end of a contraction, respectively

- **Prolonged**
  - Decel ≥ 15 bpm below baseline lasting >2 min but < 10 minute (if >10 min considered baseline change or bradycardia if <110 bpm)
Variable Decelerations

Context:
- Cord compression

Etiology:
- Alterations in fetal hemodynamics causing baroreceptor reflexes
- Persistent/prolonged eventually lead to hypoxemia and then chemoreceptors engage

http://perinatology.com/images/variableex.gif
Umbilical vein occlusion
- Reduced preload, CO, NP
- Reduced vagal tone, may see rise in FHR

Umbilical artery occlusion
- Sudden increase in afterload, BP
- Rapid baroreceptor response
- Increased vagal outflow, sudden drop in FHR

Resolution of arterial occlusion
- Sudden drop in afterload, BP
- Relax baroreceptor response
- Sudden rise in FHR

Resolution of venous occlusion
- Return to baseline
Early compression, venous occlusion
- Reduced cardiac preload
- Reduced cardiac output
  - Reduced aortic arch pressure
  - Reduced vagal outflow
    - Transient increase in FHR ("shoulder")

Compression relieved, the process reverses
- Relief of arterial occlusion first

Full compression, arterial and venous occlusion
- Increased afterload
  - Increased aortic arch pressure
  - Baroreceptor reflex, vagal outflow
    - Sudden drop in FHR
Late Deceleration

Context:
- Uterine contractions
- Reduced uterine perfusion

Etiology:
- Response to reduced fetal PaO2
- *Chemoreceptor* and *baroreceptor* mediated

http://perinatology.com/images/lateex.gif
- Uterine Contraction
  - Intervillous space compression
  - Reduced gas exchange, lower fetal PaO₂

- Chemoreceptors engage
  - Sympathetic outflow
  - Vasoconstriction to shunt flow to vital organs
  - Fetal hypertension

- Baroreceptors engage
  - Vagal nerve mediates slowing of FHR
  - Deceleration occurs

- Uterine relaxation
  - Intervillous perfusion reestablished
  - PaO₂ increases, process reverses
Prolonged Deceleration/Bradycardia

Context:
- Sudden and prolonged reduction in oxygen delivery
  - Acute events
    - Benign (rapid change/descent)
    - Serious (abruption, cord prolapse)
  - Poor fetal reserve with evolving metabolic acidosis

Etiology:
- Chemoreceptor/Baroreceptor
  - Akin to Late but there is delayed resolution
- Direct myocardial depression
  - Late stages of acidosis
Decels vs. Metabolic Acidosis

- Decelerations indicate interruption of oxygen transfer *not necessarily* metabolic acidosis.
- Transient interruption of oxygen delivery is tolerable for *most fetuses*.
- Assessment of apriori fetal risk is critical to evaluation and management of FHR tracings.
  - Maternal and fetal risk factors for poor fetal or placental reserve:
    - IUGR, maternal diabetes, hypertension, etc.
FHR Tracings and Acidemia

- Moderate variability predicts pH > 7.15
  - Negative predictive value 98%
- Minimal/absent variability AND decels associated with pH < 7.15
  - Though predictive value still poor (23%)
- Likelihood of acidemia increases with depth of recurrent decelerations
  - Especially late and with min/absent variability

Three Key Concepts

1. Significant FHR decelerations (variable, late, prolonged) represent interruptions in fetal oxygen transfer

2. Disrupted oxygen transfer does not cause injury unless there is progression to metabolic acidemia

3. The presence of FHR variability and/or accelerations predict the ABSENCE of metabolic acidosis*

* The converse is not always true…
Crystal Clear, Right?

Questions???