FHR Decelerations: Pathophysiology and Management

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FHT Monitoring Objectives

- Understand pathophysiology of variable decelerations
- Understand pathophysiology of late decelerations
- Be able to list types of variability
- List 2 patterns with high association with fetal metabolic acidosis

NICHD Definitions 1997

- Baseline FHT
  - evaluate over 10 minutes
    - minimum must be at least 2 minutes
  - 110’s - 160 normal
  - > 160 for at least 10 minutes: tachycardia
  - < 110 for at least 10 minutes: bradycardia

Variability

- Absent
- Decreased (minimal): amplitude > undetectable but < 6 bpm
- Normal (moderate): amplitude 6 - 25 bpm
- Increased (marked): amplitude > 25 bpm

NICHD, 1997
EFM as Screening Test

- Strength of test is in the normal response
  - Way < 1% chance of metabolic acidosis
- Weakness of test is in the abnormal response
  - Not that high association with metabolic acidosis except in 4 patterns

Variable Decelerations

- Abrupt decrease: onset to nadir < 30 seconds
- ≥ 15 bpm below baseline
- Lasts ≥ 15 seconds but < 2 minutes from onset to return
- Nothing about contraction association in definition

Late Decelerations

- Gradual decrease: onset to nadir ≥ 30 seconds
- Associated with contraction
- Delayed in timing with onset, nadir, recovery occurring after beginning, peak, ending of contraction in most cases
Late Decelerations

- Late deceleration with normal variability
- Not as worrisome as lates with decreased or absent variability
Prolonged Deceleration

- > 15 bpm below baseline lasting 2 - 10 minutes
- Bradycardia: < 110 bpm lasting > 10 minutes

NICHD, 1997

Physiology of FHT

- Rate controlled by vagus (parasympathetic)
  - Stimulate vagus: bradycardia
- Variability controlled by vagus, midbrain and cortex
  - Normal variability requires intact pathway between cortex, midbrain, vagus, cardiac conduction system

Normal Variability

- Strongest indicator adequately oxygenated cortex
- If present fetus does not have a metabolic acidosis
Interpretation of EFM

- True positive: abnormal equals metabolic acidosis
- False positive: abnormal but gas is normal
- True negative: normal tracing and normal gas
- False negative: normal tracing and metabolic acidosis

DeLee, 1947

- Contractions begin FHT beats faster
- During height of pain FHT are slow
- More pronounced after rom
- Blood driven out of placenta causes stimulation carotid sinus and depressor nerves of aorta
- When child in danger: FHT very fast, very slow, or irregular

Pathophysiology of Variables

- Response to cord compression
- UAs compresses first, reduces blood flow from fetus to intervillus space
- Fetal baroreceptors triggered by increase in TPR
- Vagus stimulated
- Immediacy of response reflected in shape, timing
Pathophysiology of Lates

- Response to uteroplacental insufficiency, decreased uterine blood flow, exchange O2, CO2
- Chemoreceptors fire if threshold for PO2 or PCO2 reached
- Takes time to reach threshold to fire chemoreceptor explaining timing and shape of late decel

Pattern Evolution

- Variability becomes decreased before significant fetal acidemia present
- Variability as long as brain well oxygenated
  » reflex lates
- Decrease in to absent variability as oxygenation of brain worsens
  » seen as lates with decreased to absent variability
- Usually change occurs gradually

How Much Time Do You Have?

- Most difficult question
- Really no data to help except in extremes
  » normal FHT
  » 4 asphyxial patterns
- Continuum
G2P1 38 wks labor

To OR

Forces placed on
Interventions for Variant Patterns

- Think pathophysiology
- Always try to make any recurrent decelerations go away

Patterns with Highest Association with Metabolic Acidosis

- Absent variability with repetitive decelerations
- Bradycardia with absent variability
- Bradycardia < 80
2nd Stage FHT and Metabolic Acidosis

- 601 low risk singletons
- Initially normal tracing
- Acidosis defined as pH < 7.2 and BE ≤ -12
  - 28/601 (5%)

Frequencies of Pathologic FHTs

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<tr>
<th>Factor</th>
<th>n</th>
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Sheiner, 2001

Sheiner, 2001 2nd stage
### PH <7.2  BE ≥ 12

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N=57, pH n=28 BE

### Pathologic FHT In 2nd Stage

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pH < 7.2, BE of 12 (n = 28).

Multivariate, sheiner, 2001

34 wk preeclampsia
Variables

- **Cause**
  - cord compression
  - head compression second stage
- **Interventions**
  - position change
  - amnioinfusion
  - push every other contraction

Parer, king; 1999
Lates

- **Cause**
  - decreased uterine blood flow to trigger threshold
- **Interventions**
  - position change
  - IV fluid
  - maternal hyperoxia
  - decrease contraction frequency, tocolytic
  - increase maternal blood pressure
  - Effectiveness dependent on variability

G4P2 2 prior stillbirths 29 wks decreased FM
G5P1 30 wks IUGR, oligo, breech, reverse diastolic flow
Bradycardia

- **Cause**
  - hypotension, hyperstimulation
  - terminal
- **Interventions**
  - position change
  - IV fluids
  - correct maternal blood pressure
  - decrease contractions
  - deliver

34 wks, dudenal atresia, 3 cm
Documentation

- Variability
- Baseline FHT
- Accelerations
- Decelerations
  - type, repetitiveness, severity
- Pattern evolution
- Clinical associations
- Urgency: any notification

Conclusions

- Purpose FHT monitoring
  - Reduce asphyxial damage
  - Reduce metabolic acidosis
  - Without doing unnecessary cesareans
- Requires
  - Collaborative approach (RN, CNM, MD)
  - Excellent communication
  - Trust

Avoid terms such as nonreassuring or fetal distress unless you are moving asap to delivery
- Describe
- If variant pattern emphasize what is reassuring