Late Decelerations

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Late Decelerations

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LATE QUOTES

How did it get so late so soon?
Its night before its afternoon.
December is here before its June.
My goodness how the time has flown.
How did it get so late so soon?

Theodor Geisel
American Writer
1904 - 1991

Outline

• Acid Base Balance
• Definition
• Etiology
• Historical perspective
• RSA and late decelerations
• Management
• Late Decelerations and CP
**Aerobic Metabolism**

- **Glucose Metabolism**
  - 6 ATP
  - 36 kcal
  - 6 CO₂
  - 6 H₂O
  - HEAT (417 kcal)

Oxidative phosphorylation: Very efficient form of energy production. Each pyruvic acid is converted into 34 ATP.

**Anaerobic Metabolism**

- **Glucose Metabolism**
  - 2 Lactic Acid
  - 2 ATP
  - HEAT (32 kcal)

Glycolysis: Inefficient source of energy production; 2 ATP for every glucose; produces pyruvic acid.

**Anaerobic Metabolism**

- Occurs without oxygen
  - Oxidative phosphorylation can’t occur without oxygen
  - Glycolysis can occur without oxygen
  - Cellular death leads to tissue and organ death
  - Can occur even after return of perfusion
    - Organ or organism death
Inadequate Cellular Oxygen Delivery → Anaerobic Metabolism → Inadequate Energy Production → Metabolic Failure

Anaerobic Metabolism → Lactic Acid Production → Metabolic Acidosis

Ultimate Effects of Anaerobic Metabolism

**PARTIAL PRESSURE OF OXYGEN – pO2**

Inspired Air - 150 mm Hg

Alveolus

\[ \text{pO}_2 105 \text{ mmHg} \]

\[ \text{pO}_2 95 \text{ mmHg} \]

**Normal Fetal Arterial pO2**

Cordocentesis (N = 35) – 23 mmHg (14 – 32)

Elective cesarean without labor (N = 665) – 23 mmHg (± 14%)

Cesarean after labor (N = 1,609) – 18 mmHg (± 13%)

Vaginal delivery (N = 14,285) – 24 mmHg (± 15%)

Richardson Am J Obstet Gynecol 1998;178:572-9

Umbilical artery 16 mmHg (± 4.6)

Placental artery 16.5 mmHg (± 3.6)


5-minute Apgar score ≥ 7 (N = 15,073) – 17 mmHg (2SD 6-30)

Electronic fetal heart rate monitoring: Research guidelines for interpretation

[Clinical Opinion]

National Institute of Child Health and Human Development Research Planning Workshop.

“The purpose of the National Institutes of Health research planning workshops is to assess the research status of clinically important areas. This article reports on a workshop whose meetings were held between May 1995 and November 1996 in Bethesda, Maryland, and Chicago, Illinois. Its specific purpose was to develop standardized and unambiguous definitions for fetal heart rate tracings.”

“A late deceleration of the FHR is a visually apparent gradual (onset to nadir ≥ to 30 sec) decrease and return to baseline FHR associated with a uterine contraction.”

“Decelerations are tentatively defined as recurrent if they occur with ≥ 50% of uterine contractions in any 20-minute segment.”
### NIHCD Terminology

<table>
<thead>
<tr>
<th>Pattern</th>
<th>NICHD Definitions</th>
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</thead>
<tbody>
<tr>
<td>Acceleration</td>
<td>• Abrupt ↑ in FHR above baseline, peak 15 bpm above baseline, acceleration sustained for 15 sec (10 x 10 in fetus &lt; 32 wks)</td>
</tr>
<tr>
<td>Early Deceleration</td>
<td>• Gradual onset-to-nadir (&gt; 30 seconds) with nadir coinciding with peak of contraction</td>
</tr>
<tr>
<td>Late Deceleration</td>
<td>• Gradual onset-to-nadir (&gt; 30 seconds) with nadir occurring after peak of contraction</td>
</tr>
<tr>
<td>Variable Deceleration</td>
<td>• Abrupt onset-to-nadir (&lt; 30 seconds)</td>
</tr>
<tr>
<td>Prolonged Decelerations</td>
<td>• Deceleration ≥ 2 minutes but &lt; 10 minutes</td>
</tr>
<tr>
<td>Baseline FHR</td>
<td>Approximate mean FHR rounded to 5 bpm increments, 10 minutes. Minimum 2-minute segment</td>
</tr>
<tr>
<td>• Normal FHR</td>
<td>110-160 (10 minutes)</td>
</tr>
<tr>
<td>• Bradycardia</td>
<td>FHR &lt; 110 bpm for 10 minutes</td>
</tr>
<tr>
<td>• Tachycardia</td>
<td>FHR &gt; 160 bpm for 10 minutes</td>
</tr>
<tr>
<td>Variability</td>
<td>Fluctuations or variations in baseline FHR</td>
</tr>
<tr>
<td>• Absent</td>
<td>Amplitude range undetectable</td>
</tr>
<tr>
<td>• Minimal</td>
<td>&gt; undetectable but &lt; 5 bpm</td>
</tr>
<tr>
<td>• Moderate</td>
<td>6-25 bpm</td>
</tr>
<tr>
<td>• Marked</td>
<td>&gt; 25 bpm</td>
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</table>

### LATE DECELERATIONS

**Historical**

- Little known before EFM
- Description of FHR changes with UPI
  - Hon’s “Late deceleration”
  - Caldeyro-Barcia’s “Type II Dip”


### FETAL HEART RATE MONITORING

**Ed Hon**

LATE DECELERATION
Terminology

- Type
  - Reflex
    - Hypoxia – chemoreceptors – vagal discharge
    - Accompanied by normal FHR variability (normal CNS)
  - Nonreflex
    - Hypoxia – chemoreceptors – vagus - myocardial depression
    - Abnormal variability
    - Fetal CNS & CV decompensation

- Degree
  - Mild <15
  - Moderate
  - Severe >45

Parer & Nageotte, in Creasy & Resnik 5th Edition

Recurrence late decelerations suggest recurrent episodes of hypoxemia during contractions.

LATE DECELERATIONS & HYPOXIA

- Late decelerations with intact variability are mediated by vagal reflex, are unlikely to indicate fetal acidemia and are likely to respond to O2, fluids and position changes.

- Late decelerations with decreased or absent variability are caused by myocardial depression, likely associated with fetal acidemia and do not respond to conservative measures.


LATE DECELERATIONS
Vagus Nerve Mediated I

- Late deceleration FHR pattern produced in fetal sheep by periodic occlusion of the maternal common hypogastric artery for 30-60 s
  - Transient fetal hypertension occurred during the occlusions
- Alpha-adrenergic blockade with phentolamine
  - Eliminated or markedly reduced the hypertensive response
  - FHR deceleration character was greatly altered
- Parasympathetic blockade with atropine
  - Decelerations replaced by periodic FHR accelerations during occlusions
  - Accelerations eliminated by the beta-adrenergic blocker propranolol


LATE DECELERATIONS
Vagus Nerve Mediated II

- Combined parasympathetic, alpha- and beta-adrenergic blockade
  - FHR remained constant during occlusions in non-acidemic fetuses
  - FHR decelerations persisted after parasympathetic or total autonomic blockade when fetuses significantly hypoxic
  - BTBV persisted in the face of severe hypoxia and acidosis
- CONCLUSIONS...Reflex mechanisms are involved in acutely hypoxic fetus, direct myocardial depression a factor as hypoxic acidosis develops


Vagus Nerve + Chemoreceptor

- Studied initially normoxic chronically instrumented sheep fetuses after sudden occlusion of uterine blood flow (20s):
  - Late decelerations occurred only after significant decrease in fetal O2 consumption, O2 content, and O2 tension occurred
- No significant change in BP
- Vagal blockade led to a late acceleration which was eliminated by beta-blockade
- Late deceleration in initially normoxic sheep are vagally mediated and are due to chemoreceptor, rather than, baroreceptor activity


LATE DECELS & ACIDOSIS

- Nine chronically instrumented rhesus monkeys.
- After recovery, all the fetuses had shown accelerations and no late decelerations with spontaneous uterine contractions.
- Late decelerations were the first sign of fetal deterioration and occurred with a slight but significant decrease in fetal PaO2 without changes in pH, whereas accelerations in FHR were still present.
- The loss of FHR accelerations was a later phenomenon and was associated with significant reductions in fetal pH and PaO2.

Late Decels: Mechanism

- Studied normoxic and chronically hypoxic sheep fetuses after transient obstruction of uterine artery of ewe:
  - In initially normoxic fetuses a chemoreceptor reflex induced bradycardia and decreased LV output developed after the carotid PoO2 decreased to < 20 mmHg
  - This reaction was abolished by atropine (vagally mediated)
- In the chronically hypoxic fetuses there was a late deceleration composed of three stages:
  - Chemoreceptor induced, vagally mediated bradycardia
  - Baroreceptor induced bradycardia with slow and late recovery
  - Non-reflex bradycardia due to myocardial depression
- The time to onset depended, and the depth and duration of the deceleration depended on the initial state of fetal oxygenation


Vagus Nerve + Chemoreceptor

- Studied chronically hypoxic sheep fetuses after transient occlusion of maternal aorta (20s):
  - Late decelerations occurred
- Significant decrease in fetal BP and UA flow
- Vagal blockade with atropine before occlusion reduced but did not eliminate the late deceleration
- Late deceleration was associated with decreased myocardial O2 consumption:
- Conclusion: Late decelerations are caused by 2 mechanisms
  - Chemoreceptor vagal reflex
  - Myocardial depression


**LATE DECELERATION**

**Hypoxemia during contraction**
pO2 falls below critical threshold ~ 15 - 20 mm Hg near peak of contraction.

Chemoreceptors sense ↓ pO2 and trigger reflex peripheral vasoconstriction. Oxygenated blood is shunted away from non-essential vascular beds toward essential vascular beds in brain, heart and adrenals.

Blood pressure rises.

Increased BP is sensed by baroreceptors, triggering increased vagal outflow to reduce HR, reduce CO and return BP to normal.

- pO2 < 15-20 mmHg sensed by carotid and aortic chemoreceptors
- Medullary vasomotor center
- Peripheral Vasoconstriction (Gut, kidneys, limbs)
- "Central" Vasodilatation Brain, heart, adrenals

Combined effect → Increased BP
HYPOXEMIA
INITIAL FETAL RESPONSE TO HYPOXEMIA IN THE LAMB

<table>
<thead>
<tr>
<th>Reference</th>
<th>Min</th>
<th>Brain</th>
<th>Heart</th>
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<th>Carcass</th>
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Base Excess and Hypoxia

- Normal acid base balance in the fetus
  - Base Excess: -2.3 +/- 0.6 mmol/L
  - pH: 7.39 +/- 0.05 mmol/L
  - Base excess does not change with gestation

- BE after SNVD with 5 min Apgar > 7:
  - UA: -4 +/- 3 mmol/L
  - UV: -3 +/- 3 mmol/L
  - 2.5% for the UA was -11 mmol/L

Base Excess and Hypoxia

- A normal baby enters labor with a BE of -2 mmol/L and can be expected to decrease by an additional 3 mmol/L

- Expected normal decrease in BE during:
  - First stage is -1 mmol/L per 3 to 6 hours
  - Second Stage is -1 mmol/L per 1 hour

Base Excess and Hypoxia

- When there are repetitive decels for hours buffer base decreases 1mmol/L per 30 minutes

- When there is intrapartum hypoxia causing asphyxia buffer base decreases 1mmol/L per 6 to 15 minutes

- When there is terminal bradycardia from uterine rupture buffer base decreases 1mmol/L per 2 to 3 minutes
**Base Excess and Hypoxia**

- With BE $-12$ to $-16$ mmol/L about 10% have:
  - Moderate to severe encephalopathy
  - Respiratory complications

- Buffer base: Normal = 46 mmol/L
  - $< 34$ mmol/L = asphyxia (~BE $-12$)
  - Major/minor deficits 20%
  - Increased to 80% when buffer base $< 22$ mmol/L (~BE $-24$)

- UV BE represents the situation at the time initial cord compression

- A wide (> 6 mmol/L) UA/UV BE difference suggests short duration asphyxia and a narrow (< 6 mmol/L) difference suggests long term asphyxia

- No change in BE is likely in the immediate neonatal period:
  - immediate effective resuscitation with no bicarb
  - no sepsis
  - no severe hypoxia or hypotension

- BE is likely to continue to fall at about 1mmol/L per 2 minutes if resuscitation is ineffective

- Most neonates with a BE of $-12$ mmol/L do not exhibit encephalopathy

- Even with a BE $<-16$ mmol/L most babies will either die or survive normally, only a small proportion develop CP

- Most babies that do get CP have an underlying propensity to CP
Late Decelerations

• Experiment on monkey's brain showed only pH <7.00 for >30 minutes can cause hypoxic damage. Metabolic acidosis pH >7.00 lasting for several hours did not cause neurological injury. Myers RE 1977 In Gluck Yearbook, inc 37-97

• pH <7.00 (Base deficit >16mmol/L) is a realistic threshold below which brain dysfunction significantly increases. At this level systemic hypotension start to occur. Low 1993; Clinical OB&GYN 36:82

Acidemia and FHR

• Evaluate late decelerations to detect low pH (<7.1) in low-risk pregnancies
• Four units where 10,030 women delivered, of which 5522 were low-risk
• Last 2 hours of FHR patterns before delivery were interpreted (NICHHD)
• The correlation between the incidence of LD (occasional, < 50%, recurrent, >50%) and severely (reduced baseline FHR accelerations and variability) of LD, and low pH (< 7.1) were evaluated.
• Statistical analyses included a contingency table with ch2 and the Fisher test, and one-way analysis of variance with the Bonferroni/Dunn test. In the 5522 low-risk pregnancies, 301 showed occasional LD and 99 showed recurrent LD. Blood gases and pH values deteriorated as the incidence of LD increased and as baseline accelerations or variability was decreased.
• Positive predictive value for low pH (< 7.1) was exponentially elevated from 0% at no deceleration, 1% in occasional LD, and > 50% in recurrent LD with no baseline FHR accelerations and reduced variability. In low-risk pregnancies, information on LD combined with acceleration and baseline variability enables us to predict the potential incidence of fetal acidemia.


Acidemia and FHR

• Case-control study of 71 term infants with BD > 16 mmol/l versus 71 matched infants with BD < 8
• 10 minute segments were scored for the 4 hours before delivery in both groups
• Selected FHR patterns in the 1 hour before delivery
• Asphyxia was associated with absent/minimal BTBV and late and prolonged decelerations
• Most specific pattern for asphyxia was absent BTBV but identified only 17% of asphyxia
  • When additional less specific patterns were added to absent BTBV:
    • 93% sensitivity, 2.6 – 18% PPV, and 98.3 – 99.5% NPV
• Concluded:
  • If fetus with minimal variability and late or prolonged decels is delivered within 1 hour of birth asphyxial injury can be avoided in most cases
  • Many false positive cases

**Predictive value of FHR variables for fetal asphyxia**

- **METHODS:**
  - Asphyxia group of 71 term infants with UA BD >16 mmol/L
  - Control group of 71 term infants with UA BD <8 mmol/L
- **RESULTS:**
  - FHR variables associated with fetal asphyxia
    - absent and minimal baseline variability
    - late and prolonged decelerations
  - FHR patterns with absent baseline variability
    - Where the most specific but identified only 17%
    - Sensitivity increased to 93% with addition of less specific patterns
    - PPV (18.1% to 2.6%) NPV (98.3% to 99.5%)
- **CONCLUSION:**
  - A narrow 1-hour window of FHR patterns including minimal baseline variability and late or prolonged decelerations will predict fetal asphyxial exposure before decompensation.
  - Predictive FHR patterns can be a useful screening test.
  - Supplementary tests are required to confirm the diagnosis and to identify the large number of false-positive patterns to avoid unnecessary intervention.


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**Late Decels: Aortic Compression**

- 14% (126/902) laboring patients had late decelerations
- Of these 19% (24/126) only had lates when supine
- Associated with reduced maternal femoral BP and decreased pulse pressure in the big toe
- Conclusion: Supine hypotensive syndrome can cause late decelerations


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**Late Decels and Abruption**

- Case control study of 69 patients with severe early onset pre eclampsia with and without abruptio placentae
- Late decelerations: 58% abruption vs. 32% controls
- Only recognizable warning sign noted for abruption in preeclamptic women


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**Respiratory Sinus Arrhythmia**

- RSA is heart rate variability in synchrony with respiration.
- R-R interval is shortened during inspiration & prolonged during expiration.
- Used as an index of cardiac vagal function.
- Reflects respiratory-circulatory interactions universally among vertebrates.
- Efficiency of pulmonary gas exchange is improved by RSA, suggesting active physiologic role.
- The matched timing of alveolar ventilation and its perfusion with RSA within each respiratory cycle could save energy expenditure by suppressing unnecessary heartbeats during expiration and ineffective ventilation during the ebb of perfusion.
- Evidence of a possible dissociation between RSA and vagal control of the heart rate, suggesting differential controls between the respiratory modulation of cardiac vagal outflow and cardiac vagal tone.
- RSA or heart rate variability in synchrony with respiration is a biological phenomenon, which may have a positive influence on gas exchange at the level of the lung via efficient ventilation/perfusion matching.

RSA and Polyvagal Theory

• Model defining the role of the parasympathetic NS and particularly vagus N in mammalian stress reaction
• Phylogenetic shift in neural development of ANS has 3 stages:
  – Primitive unmyelinated visceral vagus – Immobilization, depressed metabolic activity
  – Sympathetic NS – increasing metabolic output, inhibit visceral vagus
  – Myelinated vagus – rapidly regulates cardiac output
• 2 primary medullary source nuclei of the vagus (X nerve)
  – Nucleus Ambiguus – myelinated (more developed)
  – Dorsal Motor Nucleus – unmyelinated (primitive)
• Both nuclei have fibers terminating on the SA node BUT only the NA fibers regulate RSA


RSA and Polyvagal Theory

• Neurophysiological model based on the Polyvagal Theory to interpret FHR patterns.
• Beat-to-beat heart rate data from 7 fetuses monitored during the first and second stages of labor were analyzed.
• Transitory FHR accelerations and reduced beat-to-beat variability reliably preceded FHR decelerations.
• Data are interpreted within the context of the Polyvagal Theory
  – Transitory FHR accelerations and the depression of the respiratory rhythm in the beat-to-beat heart rate pattern reflect a withdrawal of the vagal tone determined by myelinated vagal pathways originating in the nucleus ambiguus.
  – Withdrawal of vagal tone originating in the NA results in the cardiac pacemaker becoming vulnerable to sympathetic influences and to the more-primitive unmyelinated vagal pathways originating in the dorsal motor nucleus of the vagus, which may contribute to clinically relevant bradycardia.


Late Decels and Fetal Breathing

• 6 patients evaluated between 37 – 42 weeks
• "Late decelerations" in association with fetal breathing seen on US or EFM
• antepartum/intrapartum
• 3 "high risk" (diabetes, IUGR, postdates)
• New onset of "late decels" in previously normal tracings – shallow, increased BTBV
  – no change after position change and O2
  – normal rate and BTBV after deceleration
  – normal neonatal outcomes
• Suggested that this represents fetal breathing


Late Decels and AV Block

• ECG evaluated in 15 fetuses during decelerations during labor
• 6/15 cases had sinus bradycardia
• 7/15 had complete dissociation of p wave from QRS – complete heart block
• Most decelerations are vagally mediated but some may be due to hypoxia of the Bundle of His with reversible AV node dysfunction

CORRECTIVE MEASURES – FETAL “ABCs”

**Cardiac Output**
- Exclude hypotension (supine, epidural)
- Lateral decubitus position
- Hydration

**Oxygenation**
- Exclude pulmonary disease (asthma, PE, AFE)
- Supplemental oxygen
- Breathing techniques

**Uterine Blood Flow**
- Exclude uterine rupture, abruption, hyperstimulation
- Discontinue uterine stimulants
- Uterine relaxants as needed
- Anxiety regulation
- Modify pushing technique

**Umbilical Blood Flow**
- Exclude umbilical cord prolapse, vasa previa
- Amnioinfusion as needed
- Manual elevation of fetal vertex

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**Fetal Heart Rate Patterns**

<table>
<thead>
<tr>
<th></th>
<th>CP (n = 78)</th>
<th>No CP (n = 300)</th>
<th>Odds Ratio</th>
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<tbody>
<tr>
<td>Tachycardia</td>
<td></td>
<td></td>
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<tr>
<td>&gt;160 bpm</td>
<td>22</td>
<td>85</td>
<td>1.0 (0.6-1.7)</td>
</tr>
<tr>
<td>&gt;180 bpm</td>
<td>5</td>
<td>16</td>
<td>1.3 (0.4-3.4)</td>
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<tr>
<td>Bradycardia</td>
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<tr>
<td>&lt;100 bpm</td>
<td>27</td>
<td>75</td>
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<td>&lt; 80 bpm</td>
<td>13</td>
<td>35</td>
<td>1.5 (0.8-3.0)</td>
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<td>Multiple lates</td>
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<td>↓ variability</td>
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<td>12</td>
<td>3.9 (1.7-9.3)</td>
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<tr>
<td></td>
<td>13</td>
<td>21</td>
<td>2.7 (1.1-5.8)</td>
</tr>
</tbody>
</table>

Nelson et al NEJM 1996; 334:613-8

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**Cerebral Palsy**

- Nonprogressive motor deficit of early onset
- One or more limbs with paralysis, spasticity or problems of motor control
- Type is determined by location of the lesions(s), nature of timing of its occurrence, and the ability of the CNS to compensate

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**LATE DECELERATIONS**

**Summary**

- Know and use NICHD EFM definitions!
- Lates mediated by multiple mechanisms
  - Old News - Hypoxemia, hypertension, myocardial depression
  - New News - RSA and conduction abnormalities
- By understanding physiology, you will
  - Know appropriate treatments
  - Positively affect some outcomes
“Better never than late.”

George Bernard Shaw  
1856-1950  
Irish Essayist

A late deceleration is a protective reflex that is triggered when fetal arterial pO2 falls below a critical threshold (hypoxemia).

**Question:** What is the normal fetal umbilical arterial pO2?

A. 85 – 95 mmHg  
B. 70 – 80 mmHg  
C. 55 – 65 mmHg  
D. 35 – 45 mmHg  
E. 15 – 25 mmHg

Fetal hypoxemia leads to all of the following, except:

A. Stimulation of aortic chemoreceptors  
B. Increased blood flow to the brain  
C. Decreased blood flow to the extremities  
D. Decreased blood flow to the kidneys  
E. Transient hypotension

**Question:** A late deceleration is the result of fetal

A. Hypoxemia  
B. Hypoxia  
C. Acidemia  
D. Acidosis  
E. Asphyxia