The Physiology of
Electronic Fetal Monitoring

Emily Hamilton, MD CM
SVP Clinical Research
Featuring Emily Hamilton, MDCM
Senior Vice President of Clinical Research

An experienced obstetrician, Dr. Hamilton is currently an Adjunct Professor of Obstetrics and Gynecology at McGill University, as well as leading PeriGen’s clinical research team.

Dr. Hamilton is the inventor of the PeriCALM advanced fetal monitoring system, holding 32 US and international patents for her research work. She is an internationally-known clinical thought leader on the use of technology to improve obstetric outcomes. She presents her research regularly at obstetric conferences and in peer-reviewed journals.
1. Physiology of heart rate regulation

2. How scientists simulated labor stresses and determined deceleration mechanisms

3. Relevance to EFM usage in humans
Intrinsic

- Myocardium
- Pacemakers
- Conducting system
- Adrenergic Receptors $\alpha, \beta$
- Cholinergic Receptors

Extrinsic

- Neuronal connections to
- Sympathetic
- Parasympathetic systems
- Circulating catecholamines
Cardioacceleratory Center  Cardioinhibitory Center

Vasomotor Center

- Autonomic control of dilation and constriction of peripheral vessels

Circulating catecholamines
Mechanisms of late decelerations in the fetal heart rate.
A study with autonomic blocking agents in fetal lambs

C.B. Martin, Jr., J. de Haan *, B. van der Wildt, H.W. Jongma, A. Dieleman and T.H.M. Arts

Department of Obstetrics and Gynecology, Catholic University and St. Radboudziekenhuis, Nijmegen, The Netherlands

Obstetrics

The intrapartum deceleration in center stage: a physiologic approach to the interpretation of fetal heart rate changes in labor

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Clinical Opinion

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Fetal Heart Rate Monitoring

Fourth Edition

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PeriGen
Empowering Clinicians, Advancing Perinatal Systems
Animal Experimentation

1. Umbilical cord compression
   Mechanical devices to occlude the cord

2. Uteroplacental insufficiency
   Compression of arteries outside the uterus

3. Implanted devices for BP, acid base status
Decelerations from Uteroplacental Dysfunction

Mechanisms of late decelerations in the fetal heart rate. A study with autonomic blocking agents in fetal lambs

C.B. Martin, Jr., J. de Haan *, B. van der Wildt, H.W. Jongsma, A. Dieleman and T.H.M. Arts

Department of Obstetrics and Gynecology, Catholic University and Sint Radboudziekenhuis, Nijmegen, The Netherlands
pH 7.40 BE +1.3

BP

FHR

Occlusion
Phentolamine pH7.35  BE -0.7
Physiological Mechanisms

Decreased Uteroplacental Oxygen Delivery to Fetus

- Chemoreceptor
  - Sympathetic
    - α Adrenergic
      - Hypertension
        - Baroreceptors
          - Parasympathetic (vagal)

Deceleration
pH 7.00 BE 1.31
pH 7.14 BE -13.1
pH 6.96 BE -19.7
pH 6.96  BE -19.7

pH 6.96  BE -19.7 with Atropine
Recurrent Lates
Incidence and Outcome

Low risk women normal tracing on admission
- Incidence of sporadic Lates 5.5%
  - With normal variability 5.4%
- Incidence of recurrent Lates 1.8%
  - With normal variability 1.4%
  - With minimal/absent variability 0.36%

With low variability and recurrent Lates/PD on admission
- Incidence 0.43%
  - Died or developed CP 33%
  - pH < 7.0 42%
  - pH < 7.1 20% → pH < 7.1

Conclusions: Decelerations from Uteroplacental dysfunction “Late” deceleration

1. Uncommon pattern
2. Multiple pathways and shapes
3. Not all late decelerations indicate myocardial hypoxia or acidemia
4. Presence of very low variability with recurrent Lates on admission VERY concerning
25 year old P0 with an uncomplicated antenatal course, admitted in spontaneous labor at term.

Delivered spontaneously 12 hrs later, a 3100 gram baby

- No oxytocin, no meconium
- Labor progression normal

Would you deliver at point 1 or 2 or 3
Admission 03:30  2 cm /50%/ -3

05:00
Delivery intervention recommended at point
SVD, Apgar 8,9  pH 7.25  Base deficit 4 mmol/L
Case Presentation #2

G1P0

41+ weeks

Admitted in spontaneous labor

Spontaneous Delivery 4 hrs later, 3750 g

pH 6.91  BD 20.7

Apgars 3/6
Tracing on Admission
This tracing is highly concerning because of:

1. Abnormal EFM patterns on admission
2. Decelerations with almost every contraction
3. High and rising baseline with low baseline variability
4. Abnormalities persist for >4hrs
5. All of the above
Decelerations From Umbilical Cord Compression
Cord Compression


Diagram:

1. Cord Compression
2. Hypertension
3. Baroreceptors
4. Parasympathetic (vagal)
5. Deceleration
Repeated 1 min Cord Occlusion


**5 mins**

- First 30 min
- Mid 30 min
- Last 30 min

<table>
<thead>
<tr>
<th>Time</th>
<th>pH</th>
<th>BD</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.5 min</td>
<td>7.34 stable...</td>
<td>1.3 stable...</td>
</tr>
</tbody>
</table>

**2.5 min**

<table>
<thead>
<tr>
<th>Time</th>
<th>pH</th>
<th>BD</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.5 min</td>
<td>7.25</td>
<td>3.3</td>
</tr>
<tr>
<td></td>
<td>7.14</td>
<td>13.6</td>
</tr>
<tr>
<td></td>
<td>6.92</td>
<td>19.2</td>
</tr>
</tbody>
</table>
Non Vagal Component


FIGURE 1
Examples show the contribution of the parasympathetic system to bradycardia during 8 minutes of severe asphyxia that was induced by complete occlusion of the umbilical cord in near-term sheep fetuses.
Physiological Mechanisms

Cord Compression

Hypertension

Baroreceptors

Parasympathetic (vagal)

Deceleration

Hypoxemia/Acidemia

Myocardial Depression
Baseline Variability

1. **Heart**
   Myocardium and its pacemakers contribute to variability

2. **Central Nervous System**
   “Push Pull” effect of sympathetic and parasympathetic system
   Drugs, malformations, sleep

3. **Circulating catecholamines**
NICHD 2008 EFM Update Category I,II,III Defined

"Moderate FHR variability reliably predicts the absence of fetal acidemia at the time that it is observed."

How reliable?
How much acidemia?
Experimental Hypoxemia Increases FHR Variability initially

<table>
<thead>
<tr>
<th>Animal</th>
<th>Intervention</th>
<th>Variability change</th>
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</thead>
<tbody>
<tr>
<td>Ikenoue et al 1981</td>
<td>monkeys hypoxemia</td>
<td>Increased</td>
</tr>
<tr>
<td>Dalton, Dawes et al 1977</td>
<td>sheep hypoxemia</td>
<td>Increased</td>
</tr>
<tr>
<td>Murotsuki, Bocking et al 1997</td>
<td>IUGR sheep hypoxemia</td>
<td>Initial increase in variability After 21 days of hypoxemia variability decreased by 20%</td>
</tr>
<tr>
<td>Martin 1979</td>
<td>Sheep Hypogastric occlusion pH 6.96</td>
<td>Increased variability</td>
</tr>
<tr>
<td>Kozuma et al 1997</td>
<td>sheep severe acidosis (mean pH 6.92)</td>
<td>Decreased in 2/3 Increased in 1/3</td>
</tr>
</tbody>
</table>
## Low Variability in Humans

<table>
<thead>
<tr>
<th>Study</th>
<th>Outcome</th>
<th>Number</th>
<th>With Low variability close to birth</th>
<th>Normal Variability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Samueloff 1994</td>
<td>pH &lt;7.2</td>
<td>303</td>
<td>26%</td>
<td>74%</td>
</tr>
<tr>
<td>Cahill 2012</td>
<td>pH&lt;7.1</td>
<td>57</td>
<td>9%</td>
<td>91%</td>
</tr>
<tr>
<td>Williams and Galarneau 2003</td>
<td>BD&gt;12</td>
<td>36</td>
<td>42%</td>
<td>58%</td>
</tr>
<tr>
<td>Low 1999</td>
<td>BD&gt;16</td>
<td>71</td>
<td>68%</td>
<td>32%</td>
</tr>
<tr>
<td>Williams and Galarneau 2003</td>
<td>BD&gt;16</td>
<td>13</td>
<td>85%</td>
<td>15%</td>
</tr>
</tbody>
</table>
G1  40+wks labor X 12.5Hrs   3600g baby
pH 6.89  BD 21.4  Apgar 3/5
2.5 hours before birth
1 hour before birth
Last 30 minutes
pH 6.89  BD 21.4  Apgar 3/5
Baseline Variability

- Initial response to hypoxemia in experimental conditions is an increase in variability.
- Low variability appears with advanced acidemia.
- And/or with other “depressing” or “chronic” central nervous system factors.
EFM patterns are the result of many interacting physiological pathways that overlap and change in the presence of severe acidemia.

Important considerations for EFM interpretation
1. Decelerations from uteroplacental insufficiency and cord compression share common physiological pathways
2. Size, frequency of decelerations
3. Duration of EFM problem and its progression
4. Loss of baseline variability is a late change
5. Underlying fetal tolerance and cause
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Thank you for joining us