Power and Passenger: Understanding Uterine Activity

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Emily Hamilton MDCM
Featuring Michelle Flowers, RNC-OB
Program Manager

Michelle Flowers serves as a PeriGen Program Manager & Clinical Consultant. Her 20+ year career also includes experience as a Clinical Quality Coordinator, Educator for obstetric services, and L&D charge nurse.

Her more recent experience has focused on designing and executing OB documentation workflows and training programs. Her education credentials have included AWHONN Intermediate/Advanced Fetal Monitoring Instructor, ICEA Approved Trainer and Certified Childbirth Educator and AAP NRP Instructor.
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.

PeriGen
Advanced Perinatal Systems
Objectives

1. Review definitions related to uterine activity
2. Review etiology and physiology of uterine tachysystole
3. Analyze impact of UT on the fetal heart rate
Uterine Contractions

Quantified as the number of contractions in 10 minutes averaged over 30 minutes.

- Uterine activity terminology used to assess and describe contractions:
  - **Frequency**
    - Beginning of contraction to beginning of next one
  - **Duration**
    - Length of contraction from beginning to end
    - Measured in seconds
  - **Intensity**
    - Strength of contraction
    - Assessed via palpation or mmHg (Montevideo units)
  - **Resting Tone**
    - Intraterine pressure when uterus is not contracting
    - Assessed via palpation or mmHg
Uterine Palpation

Palpation of uterine contractions is one of the key components of labor assessment.

<table>
<thead>
<tr>
<th>Uterus</th>
<th>Feels Like</th>
<th>Contraction Intensity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Easily Indented</td>
<td>Tip of Nose</td>
<td>Mild</td>
</tr>
<tr>
<td>Slightly indented</td>
<td>Chin</td>
<td>Moderate</td>
</tr>
<tr>
<td>Cannot indent</td>
<td>Forehead</td>
<td>Strong</td>
</tr>
</tbody>
</table>
Contractions

• Generate intrauterine pressure
• Cause retraction, effacement and dilation of the cervix
• Facilitate descent and birth of the fetus
• Affect maternal blood flow through the myometrium
• Normal contraction pattern
  – 5 Contractions or less in 10 minutes
Resting Tone

- Measured by palpation or I UPC

- Normal
  - Assists in maintaining adequate fetal oxygenation

- Hypertonus (elevated resting tone)
  - Palpation: uterus that doesn’t return to soft
  - I UPC: >20-25 mmHg
Montevideo Units

- Two methods of measurement

- Measured in 10 minute segments
  - Measure peak intensity, or amplitude, in mmHg for each contraction and add together
  - Subtract baseline uterine tone from peak contraction pressure for each contraction and add together
Other Contraction Measurements

Physiology

Contractions
Contraction Physiology

• The uterine muscle is composed of upper and lower uterine segments
  – Upper segment: active and contains pacemaker sites
  – Lower segment: passive zone

• At the start of labor, changing levels of various hormones cause myometrial cells to be more excitable.
  – (corticotrophin-releasing hormone, progesterone, estrogen, prostaglandin and oxytocin)

• Myometrium advances from a quiet to an active state.

• Constructions result when there is coordinated propagation of myometrial contraction usually beginning in the fundus
Contractions
Intramyometrial Pressure Effect on Blood Flow
Fleisher 1987  Brar 1988  Janbu 1987

- Maternal flow reduced
- Directly related to pressure
- Spiral arteries compressed when pressures >35-60 mmHg
Compensatory Mechanisms

Healthy fetus has twice the reserve required to navigate the birth process.

- **Extrinsic factors impacting FHR Patterns:**
  - Maternal Influences
  - Utero-placental perfusion
  - Umbilical circulation
  - Amniotic fluid characteristics

- **Intrinsic factors impacting FHR Patterns:**
  - Placental transfer capacity
  - Fetal adaptive compensatory mechanisms

Diagram:
- Redistribute blood flow
- Increase O₂ extraction
- Fetal reserves
- Reduce O₂ need
Uterine Tachysystole
Uterine Tachysystole

> 5 contractions per 10 minutes averaged over 30 minutes

Mathematical equivalent:
>15 contractions in 30 minutes
Causes of Tachysystole

- Known complication of induction/augmentation
- Spontaneous
  - Intrauterine infection
    - “Irritation” of the myometrium by bacterial toxins
  - Placental abruption
- Cocaine use
- Unknown etiology
Physiology

Uterine Tachysystole
Timing

Figure 1. Fetal heart rate (I), fetal tcPO$_2$ (II), relative local perfusion (III), and intrauterine pressure in a patient in labor. Shaded area = tcPO$_2$ integral during a contraction period. Arrows = onset and end of corresponding contraction period. Recording paper moves from right to left.
Repeated 1 min Cord Occlusion

5 mins

First 30 min

Mid 30 min

Last 30 min

pH 7.34 stable...

BD 1.3 stable ...

2.5 min

pH 7.25 7.14 6.92

BD 3.3 13.6 19.2
Fetal Experience

1. Amount of oxygen available correlated with contraction strength, duration and intercontraction interval.

2. Reperfusion time important to restore oxygen levels in the intervillous space before next contraction begins.
<table>
<thead>
<tr>
<th>Reference</th>
<th>Year</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bakker et al 2007</td>
<td></td>
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<tr>
<td>Rice Simpson et al 2008</td>
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<tr>
<td>Hamilton et al 2012</td>
<td></td>
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<tr>
<td>Kunz et al 2013</td>
<td></td>
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<tr>
<td>Smith et al 2013</td>
<td></td>
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<td>Ahmed et al 2016</td>
<td></td>
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</tbody>
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UT lasting >1hr 4.2%
% DEP low even with UT >60min

DEP = Neonatal Depression
Umbilical artery BD ≥ 10 mmol/L or 5 minute Apgar ≤ 6

<table>
<thead>
<tr>
<th></th>
<th>No uterine tachysystole #</th>
<th>Any uterine tachysystole ##</th>
<th>Uterine tachysystole 30-60 minutes *</th>
<th>Uterine tachysystole Over 60 minutes **</th>
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<tbody>
<tr>
<td>Number</td>
<td>5095</td>
<td>1139</td>
<td>879</td>
<td>260</td>
</tr>
<tr>
<td>n (%) Neontal Depression</td>
<td>66 (1.3%)</td>
<td>11 (1.0%)</td>
<td>6 (0.7%)</td>
<td>5 (1.9%)</td>
</tr>
</tbody>
</table>

# vs. ## P=0.45  # vs. *P= 0.17  # vs. **P=0.56  * vs. ** P=0.14
% UT: Higher with Oxytocin

<table>
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<tr>
<th>Uterine Tachysystole</th>
<th>Induction *</th>
<th>Augmentation **</th>
<th>Neither #</th>
<th>(P^* \text{ vs.} ^{**})</th>
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<td>Uterine Tachysystole</td>
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<td>18.7%</td>
<td>232</td>
<td>23.1%</td>
<td>427</td>
<td>16.0%</td>
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<td>34</td>
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<td>17</td>
<td>1.7%</td>
<td>26</td>
<td>1.0%</td>
</tr>
<tr>
<td>DEP In UT n (%)</td>
<td>5/480</td>
<td>1.0%</td>
<td>4/232</td>
<td>1.7%</td>
<td>2/427</td>
<td>0.5%</td>
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\(P^* < 0.0001, P^{**} = 0.498, P^* \text{ vs.} ^{**} = 0.0035\)
% DEP: low and similar

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<td>0.012</td>
<td>&lt;0.0001</td>
<td>0.0035</td>
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<td>0.104</td>
<td>0.50</td>
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<td>DEP in UT n (%)</td>
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<td>4/232 1.7%</td>
<td>2/427 0.5%</td>
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% DEP in UT: low and similar

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More Decelerations

![Graph showing the average number of decelerations per 10 minutes for NeoDep and Controls groups. The graph indicates a statistically significant difference (P<0.05) for the NeoDep group at certain contraction rates.]
Greater Exposure to UT

UT Rates Over Time

Higher rate of #deceleration/#contractions
HIE babies highest ratio
Clinical Summary

1. UT is common (usual range ~11-26%)

2. How a baby responds to UT depends upon the balance between contraction-related decreases in perfusion and compensatory capacity of baby

3. Compensatory capacity is finite

4. Red flag: Deceleration response to any level of contractions

5. Red flag: What is causing the UT?
Impact of UT on the Fetal Heart Rate

Case Presentations
G3P1  39 5/7 wks  
NVD  pH 7.35  BD 0.6  Apgar 8/9  3800g
Compensation capacity is finite
4 hrs before birth
G3P0 40 3/7 wks
NVD pH 7.04  BD 13.3  Apgar 8/9  4100g  9363
G3P1 39 3/7 wks
NVD  ph 7.05 BD 16.2  Apgar 8/9  3500g
G1P0 39 0/7 wks
NVD  pH 6.6 BD 27  Apgar 0/0  3300g  101018
Medicolegal Perspective
Uterotonics and Adverse Outcomes

45% of brain injuries - Excessive use of contraction stimulating drugs

71% of births with severe asphyxia seeking legal action - incautious use of oxytocin
Berglund S, et al. BJOG. 2008 Feb;115(3):316-323

47% of babies born with metabolic acidosis have oxytocin misuse

>70% of closed legal claims with oxytocin use showed a failure to appreciate and act on severity of EFM abnormalities. 75% resulted in a settlement.
CMPA Perspective, March 2014
UT in perspective

28,486
>34 wks, vaginal births 1994-2004
routine with umbilical artery gases

161
Severe metabolic acidosis   UA pH <7.05 and BD ≥12 mmol/L

Excluded
Elective CS
Catastrophic OB events (abruption, cord prolapse, eclampsia)

UT => 6/10 x 20 minutes

Metabolic Acidosis

- Oxytocin misuse: 47%
- No oxytocin misuse: 53%
Metabolic Acidosis

- Oxytocin misuse without UT: 28%
- UT and Oxytocin misuse: 19%
UT without Oxytocin Misuse

- UT without Oxytocin misuse: 7%
- Oxytocin misuse without UT: 28%
- UT and Oxytocin misuse: 19%
- UT without Oxytocin misuse: 46%
UT Normal

- UT and Oxytocin misuse: 3.2%
- UT without Oxytocin misuse: 3.7%
- Oxytocin misuse without UT: 10.0%
Conclusions: Uterine Tachysystole

1. Fetal tolerance demonstrated by changes in FHR

2. Uterine tachysystole and/or failure to intervene for abnormal FHR is very common in OB litigation

3. Avoid it, Fix it, Can’t defend it!
References


Managing the Risks of Labour Induction. CMPA Perspective March 2014
References


