Intrapartum Fetal Heart Rate Monitoring

A Standardized Approach to Interpretation and Management
Lecture Objectives

At the end of this program, participants will be able to:

Review standardized FHR terminology proposed by the NICHD in 1997 and 2008

Describe 3 central concepts in standardized FHR interpretation that are evidence based and reflect consensus in the medical literature
Intrapartum FHR monitoring is the single most common obstetric procedure in the US, impacting the lives of almost 8 million mothers and babies every year.
For 4 decades, a lack of standardized training and competency testing in intrapartum FHR monitoring has led to:

Ill-defined, confusing terms

Unsubstantiated theories, myths, urban legends and folklore passed down from resident to resident and generation to generation

A communication crisis that jeopardizes the safety of our patients and the credibility of our entire profession
Intrapartum FHR monitoring was introduced with limited prospective evidence of efficacy and no consensus regarding terminology, interpretation or management...

...and the lack of consensus persisted for almost 40 years
However, since 1997 there have been several consensus statements that have reshaped the fetal monitoring landscape:

- NICHD – 1997
- International Cerebral Palsy Task Force – 1999
- ACOG-AAP Cerebral Palsy Task Force – 2003
- NICHD – 2008

In 2005 and 2006, ACOG, AWHONN and ACNM officially endorsed the standardized FHR terminology proposed by the NICHD in 1997.
Now, for the first time since intrapartum FHR monitoring was introduced, there is evidence-based consensus in the literature that allows us to:

1. Standardize the words we use to communicate FHR patterns (terminology)

2. Clear up 40 years of confusion surrounding the physiologic significance of intrapartum FHR patterns (interpretation)

3. Develop a standardized, practical, systematic, evidence-based approach to the management of intrapartum FHR patterns (management)
Joint Commission on Accreditation of Healthcare Organizations

Issue 30 - July 21, 2004

Identified “poor communication of abnormal FHR patterns” as a leading risk factor for preventable perinatal injury
Recommended that hospitals educate nurses, residents, nurse midwives, and physicians to use *standardized terminology* to communicate abnormal fetal heart rate tracings.
The commission further recommended that healthcare organizations develop clear guidelines for *interpretation* of FHR patterns…
2006 ACOG Survey on Professional Liability

10,659 survey respondents

2.6 medical malpractice lawsuits in a career

89% of ob-gyns sued at least once

Obstetric malpractice claims made up 62%
2006 ACOG Survey on Professional Liability

10,659 survey respondents

Primary allegations for obstetric claims:

“Neurologically impaired infant” 31%

“Stillbirth/neonatal death” 16%
# AMA Crisis States

**January, 2007**

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17 “Crisis States” with limited patient access to essential healthcare services because of litigation concerns
Do other industries face similar challenges? If so what can we learn from them?

Commercial Aviation  
Commercial Banking  
Nuclear Power Industry  
Healthcare

What do these industries have in common? They are all founded on “High Reliability Organizations”

High Reliability Organizations operate in complex, hazardous environments tolerating few mistakes over long periods of time with high public expectations for reliability

One of the central principles governing the function of these organizations is standardization
What can fetal monitoring really do?
Electronic FHR monitoring is most analogous to:

A. Pap
B. Cone biopsy
Electronic FHR monitoring is a screening test

It is not a diagnostic test

Except in the most extreme cases, it has never been capable of reliably diagnosing fetal injury or “impending injury”
Definitive intervention

Heightened surveillance
Conservative corrective measures if needed

Routine surveillance
However, much of the research in FHR interpretation has focused on using the technology as a diagnostic test for impending fetal injury.

And not unexpectedly, FHR monitoring has not performed well in this regard.
Inability to live up to early unrealistic expectations has led many to discount the technology as a failure and to abandon much needed efforts to standardize interpretation and management.

In the absence of clear standards for FHR interpretation and management, clinicians often find themselves at the mercy of opinions.
In reality, intrapartum FHR monitoring is not a failed technology. It is a success on at least three fronts:

1. Its introduction coincided with the virtual elimination of intrapartum fetal death

2. It is at least as effective as the previous “gold standard” intensive intermittent auscultation, the only alternative that has ever been studied in prospective trials

3. While not a reliable DIAGNOSTIC test, it is an exceptional SCREENING test. A normal intrapartum FHR tracing virtually precludes ongoing hypoxic injury at the time it is observed
The exceptional negative predictive value of intrapartum FHR monitoring can be used to construct a systematic, logical approach to standardized interpretation and management.
Where do we go from here?
FHR monitoring consists of three components:

- Intrapartum FHR Monitoring
- Terminology
- Interpretation
- Management
Or in common terms…

Terminology – *What do I call it?*

Interpretation – *What does it mean?*

Management – *What do I do about it?*
Terminology

Recent progress in the standardization of FHR definitions is reflected in the endorsement of the 1997 NICHD FHR definitions by:

- ACOG – May 2005
- AWHONN – May 2005
- ACNM – December 2006

The 1997 definitions were updated in 2008 by a second NICHD workshop and published in September, 2008

Terminology

Endorsement of the NICHD definitions represented the first time that physicians, nurses and midwives all agreed to use the same language...
Terminology

A brief review and update
A FHR tracing has the appearance of an irregular horizontal line.
What is that line?
What appears to be an irregular horizontal line actually is a series of closely-spaced, individual points.

Each point represents an individual heart rate calculated from the interval between two R waves in the fetal ECG.*
Along with uterine contractions, there are five essential components of a FHR tracing:

- Baseline rate
- Variability
- Accelerations
- Decelerations
- Changes or trends over time
Normal baseline rate 110-160 bpm

Mean FHR rounded to increments of 5 bpm in a 10-minute window, excluding accelerations, decelerations and periods of marked variability.

There must be at least 2 minutes of identifiable baseline in any 10-minute window (not necessarily contiguous), or the baseline for that period is indeterminate. In that case, it may be necessary to refer to the previous 10-minute window to determine the baseline.
Variability is determined in a 10-minute window, excluding accelerations and decelerations.

Calculated FHR (bpm) 138 139 138 137 136 137 138

Raw FHR data
Fetal ECG
Variability is defined as fluctuations in the baseline that are irregular in amplitude and frequency…

No longer “≥ 2 cycles per minute”

No distinction is made between short-term (beat-to-beat) variability and long term variability because in actual practice they are visually determined as a unit
The fluctuations are visually quantitated as the amplitude of the peak-to-trough in bpm

Amplitude range undetectable: *absent* variability
Detectable but $\leq 5$ beats/min: *minimal* variability
Range 6 to 25 beats/min: *moderate* variability
Range > 25 beats/min: *marked* variability
Acceleration
Abrupt increase (onset to peak < 30 sec) in the FHR from baseline

32 weeks and beyond – 15 x 15
Before 32 weeks – 10 x 10
Decelerations

Early
Late
Variable
Prolonged
Early Deceleration

Visually apparent usually symmetrical, gradual decrease and return of the FHR associated with a uterine contraction

In most cases the onset, nadir, and recovery of the deceleration are coincident with the beginning, peak, and ending of the contraction, respectively
Late Deceleration

Visually apparent usually symmetrical gradual decrease and return of FHR associated with a uterine contraction

Delayed in timing, in most cases, the onset, nadir, and recovery of the deceleration occur after the beginning, peak, and ending of the contraction, respectively
During a uterine contraction, decreased maternal perfusion of the intervillous space may cause the fetal arterial PO2 to fall below a critical threshold.
Decreased fetal PO2 (hypoxemia) during a uterine contraction is detected by chemoreceptors.

Chemoreceptors signal the medullary vasomotor center.

Sympathetic outflow results in peripheral vasoconstriction to redistribute oxygenated blood away from the extremities, gut and kidneys.

Blood flow to the brain, heart, adrenal glands and placenta is preserved or increased.

Peripheral vasoconstriction causes the blood pressure to rise.

Rising blood pressure is detected by baroreceptors.

Baroreceptors signal the medullary vasomotor center.

Parasympathetic (vagal) stimulation of the heart causes a gradual slowing of the FHR to reduce cardiac output and maintain normal blood pressure.

This combined chemo and baroreceptor mediated reflex is reflected in the fetal heart rate tracing as a late deceleration.
As the uterine contraction subsides, maternal perfusion of the intervillous space is reestablished.
Perfusion of the intervillous space with oxygenated maternal blood causes the fetal PO2 to rise above the critical threshold. Autonomic reflexes subside and the FHR returns to baseline.
## Hypoxemia

### Initial fetal response to hypoxemia in the lamb

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1. AJOG 1974;120:817-24
2. AJOG 1979;135:637-46
7. AJOG 1994;170:156-61
Variable Deceleration

Visually apparent abrupt decrease in FHR at least 15 bpm below the baseline, lasting at least 15 seconds and less than 2 minutes in duration
Variable Deceleration – Cord Compression

Venous compression
Decreased venous return
Relative hypovolemia
Reflex increase in FHR

Arterial compression
Increased SVR, elevated BP
Baroreceptor stimulation
Vagal outflow

Reverse
Decelerations

Evidence in the literature does not support assigning specific clinical significance to observations such as…

“Variable with a late component”
“Overshoot”
“Shoulders”
“Variability within the deceleration”
“W-shaped”, “V-shaped”, “U-shaped” variables
Decelerations

In addition, evidence in the literature does not support classification of decelerations as:

- Mild
- Moderate
- Severe
Prolonged Deceleration

A prolonged deceleration is a visually apparent decrease in FHR from the baseline with a depth of at least 15 bpm and a duration of at least 2 minutes, but less than 10 minutes.

A deceleration lasting 10 minutes or longer is a baseline change.
Interpretation

In 1997, the NICHD Workshop on electronic fetal monitoring limited recommendations to standardized terminology.

In 2008, the NICHD, ACOG and SMFM partnered to sponsor a 2-day workshop to revisit the standardized terminology proposed in 1997 and to address the issue of standardized interpretation of electronic fetal heart rate patterns.
Intrapartum FHR interpretation has become confusing and controversial. However, recent consensus has clarified some of the confusion.

Forty years of research in intrapartum FHR interpretation can be distilled into three central concepts that are evidence based, reflect consensus in the literature and are practical and teachable.
Intrapartum FHR monitoring interpretation is intended to assess fetal oxygenation during labor.
Fetal oxygenation involves the transfer of oxygen from the environment to the fetus...
And the subsequent fetal physiologic response if oxygen transfer is interrupted...

Environment

- Lungs
- Heart
- Vasculature
- Uterus
- Placenta
- Cord

Oxygen transfer

Fetus

- Hypoxemia
- Hypoxia
- Metabolic acidosis

Fetal response

Potential Injury

- Metabolic acidemia
What information can the FHR tracing provide regarding oxygen transfer?
Interruption of the pathway of oxygen transfer from the environment to the fetus caused by compression of the umbilical cord can result in a variable deceleration.
Interruption of the pathway of oxygen transfer from the environment to the fetus caused by a uterine contraction with reduced perfusion of the intervillose space of the placenta can result in a late deceleration.
Interuption of the oxygen pathway at any point can result in a prolonged deceleration.
Oxygen transfer can be interrupted at any of these points and can manifest as a FHR deceleration (variable, late, prolonged).

Potential Injury

- Hypoxemia
- Hypoxia
- Metabolic acidosis
- Metabolic acidemia

Fetal response
All clinically significant FHR decelerations (late, variable, prolonged) reflect interruption of the pathway of oxygen transfer from the environment to the fetus.
What do we know about the fetal response to interrupted oxygen transfer?
Oxygen transfer leads to the environment affecting the fetus. Sustained or recurrent interruption of oxygen transfer can eventually lead to injury. Fetal response includes hypoxemia, hypoxia, and metabolic acidosis. Potential injury can be further divided into metabolic acidosis and metabolic acidemia, indicating potential harm to the fetus.
Is there a point that must be reached before oxygen deprivation can cause injury?

Where is the injury threshold?

Potential Injury

- Hypoxemia
- Hypoxia
- Metabolic acidosis
- Metabolic acidemia
In 1999, the International Cerebral Palsy Task Force published a consensus statement defining the relationship between intrapartum events and neurologic injury.

Supporters included:

American College of Obstetricians and Gynecologists
American Gynecological and Obstetrical Society
Australian College of Midwives
Hong Kong Society of Neonatal Medicine
Institute of Obstetrics and Gynaecology of the Royal College of Physicians of Ireland
International Society of Perinatal Obstetricians
New Zealand College of Midwives
Paediatric Society of New Zealand
Perinatal Society of Australia and New Zealand
Royal Australasian College of Physicians, Paediatric Division
Royal Australian College of General Practitioners
Royal Australian College of Obstetricians and Gynaecologists
Royal College of Obstetricians and Gynaecologists
Royal College of Pathologists of Australasia
Royal New Zealand College of Obstetricians and Gynaecologists
Society of Obstetricians and Gynaecologists of Canada
In 2003, ACOG and the American Academy of Pediatrics (AAP) jointly published a monograph summarizing the medical literature regarding the relationship between neonatal encephalopathy and cerebral palsy.
The publication was endorsed by:

American College of Obstetricians and Gynecologists
American Academy of Pediatrics
Centers for Disease Control and Prevention
Child Neurology Society
March of Dimes Birth Defects Foundation
National Institute of Child Health and Human Development
Royal Australian and New Zealand College of Obstetricians and Gynecologists
Society for Maternal-Fetal Medicine
Society of Obstetricians and Gynaecologists of Canada
Acute intrapartum interruption of fetal oxygenation does not result in neurologic injury unless it progresses to the stage of:

1. Hypoxemia
2. Hypoxia
3. Metabolic acidosis
4. Metabolic acidemia
Intrapartum interruption of fetal oxygenation does not result in neurologic injury (cerebral palsy) unless it progresses to the stage of significant metabolic acidemia (umbilical artery pH < 7.0 and base deficit ≥ 12 mmol/L)
Clinically significant FHR decelerations (late, variable, prolonged) reflect interruption of the pathway of oxygen transfer from the environment to the fetus.

Acute interruption of oxygen transfer does not cause injury unless the fetal response progresses to the stage of metabolic acidemia.

Are there any FHR characteristics that reliably predict the absence of metabolic acidemia?
FHR accelerations reliably predict the absence of fetal metabolic acidemia at the time they are observed.
Moderate FHR variability reliably predicts the absence of metabolic acidemia at the time it is observed.
Distilling 40 years of research in FHR interpretation into three central concepts:

With respect to interrupted oxygenation, FHR interpretation can be summarized as…
Clinically significant FHR decelerations (late, variable, prolonged) reflect interruption of the pathway of oxygen transfer from the environment to the fetus.

Acute interruption of oxygen transfer does not cause injury unless the fetal response progresses to the stage of metabolic acidemia.

Moderate variability and/or accelerations reliably predict the absence of metabolic acidemia at the time they are observed.

Potential Injury:
- Hypoxemia
- Hypoxia
- Metabolic acidosis
- Metabolic acidemia
Intrapartum FHR Interpretation
Three Central Concepts

1. Variable, late or prolonged decelerations signal interruption of oxygen transfer

2. Moderate variability or accelerations reliably predict the absence of metabolic acidemia

3. Injury requires significant metabolic acidemia
G2P1 at 38 weeks. How would you interpret this tracing?

1. Decelerations signal interruption of oxygen transfer
2. Moderate variability excludes metabolic acidemia
3. Injury requires metabolic acidemia
4. All of the above
The 2008 NICHD Workshop Report on Electronic Fetal Monitoring

Update on Definitions, Interpretation and Research Guidelines

Obstet Gynecol 2008;112:661-6
“Three-Tier” Fetal Heart Rate Interpretation System

Category I – “Normal”

“Strongly predictive of normal fetal acid-base status at the time of observation”

Baseline rate: 110-160 bpm
Variability: Moderate
Accelerations: Present or absent
Decelerations: No late or variable decelerations (or prolonged)

“May be followed in a routine manner”

Obstet Gynecol 2008;112:661-6
“Three-Tier” Fetal Heart Rate Interpretation System

Category III – “Abnormal”

“Predictive of abnormal fetal acid-base status at the time of observation”

Absent variability with any of the following:
- Recurrent late decelerations
- Recurrent variable decelerations
- Bradycardia
- Sinusoidal pattern
“Three-Tier” Fetal Heart Rate Interpretation System
Category III – “Abnormal” - Requires prompt evaluation

“Depending on the clinical situation, efforts to expeditiously resolve the abnormal FHR pattern may include but are not limited to:

• Provision of maternal oxygen
• Change in maternal position
• Discontinuation of labor stimulation
• Treatment of maternal hypotension”

Obstet Gynecol 2008;112:661-6
“Three-Tier” Fetal Heart Rate Interpretation System

Category II...Everything else

“Not predictive of abnormal fetal acid-base status...indeterminate”

“Category II FHR tracings require evaluation and continued surveillance and reevaluation, taking into account the entire associated clinical circumstances.”
“Three-Tier” Fetal Heart Rate Interpretation System

“Categories” are summary terms

They do not replace qualitative and quantitative description of:

• Baseline rate
• Variability
• Accelerations
• Decelerations
• Changes or trends over time

Obstet Gynecol 2008;112:661-6
“Three-Tier” Fetal Heart Rate Interpretation System

The 2008 NICHD consensus report did NOT recommend specific management of any category…

Instead, the report clearly stated that management algorithms are the function of professional specialty societies (such as ACOG, SMFM etc)

Do not misinterpret the NICHD document to mean that Category II tracings never require intervention
2008 NICHD Recommendations
Key Points

Moderate variability reliably predicts the absence of metabolic acidemia at the time it is observed.

However, the converse is not true: Minimal-absent variability alone does NOT reliably predict the presence of metabolic acidemia.
2008 NICHD Recommendations
Key Points

Accelerations reliably predict the absence of metabolic acidemia at the time they are observed.

However, the converse is not true: The absence of accelerations alone does NOT reliably predict the presence of metabolic acidemia or hypoxemia.

Obstet Gynecol 2008;112:661-6
Standard terminology
We have achieved consensus in the United States on the terminology used to describe the five components of a FHR tracing

Standard interpretation
Three central concepts of FHR interpretation are evidence-based and reflect consensus in the literature
Intrapartum FHR Interpretation
Three Central Concepts

1. Variable, late or prolonged decelerations signal interruption of oxygen transfer

2. Moderate variability or accelerations reliably predict the absence of metabolic acidemia

3. Injury requires significant metabolic acidemia

Environment
- Lungs
- Heart
- Vasculature
- Uterus
- Placenta
- Cord

Fetus
- Hypoxemia
- Hypoxia
- Metabolic acidosis

Metabolic acidemia

Potential injury
Standardized management is the next challenge