Critical Thinking with Complex FHR Tracings

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Objectives

1. Discuss underlying physiology for complex EFM tracings.

2. Outline management for Category I, II and III EFM tracings.

3. Differentiate maternal vs. fetal heart rate with signal ambiguity.
A full description of an EFM tracing requires a qualitative and quantitative description of:

- Uterine contractions
- Baseline fetal heart rate
- Baseline FHR variability
- Presence of accelerations
- Periodic or episodic decelerations
- Changes or trends of FHR patterns over time

Mancones, 2008
Management of Intrapartum Fetal Heart Rate Tracings

Intrapartum electronic fetal monitoring (EFM) is used for most women who give birth in the United States. As such, clinicians are faced daily with the management of fetal heart rate (FHR) tracings. The purpose of this document is to provide obstetric care providers with a framework for evaluation and management of intrapartum EFM patterns based on the new three-tiered categorization.
Normal (Category 1) Tracing

Baseline 130
Moderate variability
No late, variable or prolonged decelerations
Does not require the presence of accelerations
Normal (Category 1) Tracing

Baseline 140
Moderate variability
Early decelerations
Normal (Category 1) Tracing

Baseline 135
Moderate variability
Accelerations
Category 2
Indeterminate tracings

- Includes **all** FHR tracings not categorized as Category 1 or 3.
- Represent about 80% of those encountered in clinical care
- Multiple basic science investigations and clinical trials have been published but this body of work has primarily served to raise more questions than it has answered.
OBSTETRICS

Intrapartum management of category II fetal heart rate tracings: towards standardization of care

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Interpretation and management of fetal heart rate (FHR) patterns during labor remains one of the most problematic issues in obstetrics. Multiple basic science investigations and clinical trials have been published since the introduction of this technique in the late 1950s. Unfortunately, this body of work has primarily served to raise more questions than it has answered—as a medical community, we seem to know less than we thought we did 30 years ago regarding the utility of this ubiquitous technique.

In recent years, several specific issues relating to the interpretation and management of FHR patterns have received considerable attention in the medical literature. These include the lack of agreement in interpretation even among recognized experts, the role of FHR patterns in fact prevents cerebral palsy or other types of neurologic impairment. Against this background, however, there remains in many of us suspicion (albeit based primarily upon anecdotal experience and the original basic science investigations) that the FHR monitor is a medical device that was introduced into clinical practice without an instruction manual, without the now common premarket testing to support the unrealistic expectations of efficacy, and without clearly defined key words: fetal heart rate monitoring, neonatal encephalopathy, patient safety.
Category 2 Management (Opinion paper)

- Category II patterns identify fetuses that may potentially be in some degree of jeopardy but are either not acidemic, or have not yet developed a degree of hypoxia/acidemia that would result in neonatal encephalopathy.

- Must also avoid unnecessary intervention, and encourage vaginal delivery in women whose FHR patterns suggest minimal risk of significant deterioration prior to delivery.

What are we doing?

Deciding to Wait

Clinical judgment

Waiting to Decide

Procrastination

Hope is NOT a plan
Algorithm for management of category II fetal heart rate tracings

Moderate variability or accelerations

- Yes
- No

Significant decelerations with ≥50% of contractions for 1 hour*

- Yes
- No

*Significant decelerations with ≥50% of contractions for 30 minutes*

- Yes
- No

Latent Phase
- Normal labor progress
  - Yes: Cesarean
  - No: Observe

Active Phase
- Normal progress
  - No: Cesarean or OVD
  - Yes: Observe

Second Stage
- Normal progress
  - Yes: Observe
  - No: Cesarean or OVD

Observe for 1 hour
- Yes
- No

Persistent pattern
- Yes: Manage per algorithm
- No: Observe

OVD, operative vaginal delivery.

*That have not resolved with appropriate conservative corrective measures, which may include supplemental oxygen, maternal position changes, intravenous fluid administration, correction of hypotension, reduction or discontinuation of uterine stimulation, administration of uterine relaxant, amnioninfusion, and/or changes in second stage breathing and pushing techniques.

Baseline 175, moderate variability, recurrent late decelerations

- Position change, fluid bolus may be helpful
- Per algorithm, if continuing to make progress, observe
• Per algorithm, the presence of accelerations are indicative of a non-acidemic fetus.
• Umbilical cord compression and placental insufficiency could, over time, lead to significant acidemia.
• Per algorithm, with normal progression in active phase or second stage, careful observation and intrauterine resuscitation would be appropriate. If the fetus is remote from delivery, delivery would be appropriate.
• Medication effect has been excluded.
• While fetus may have experienced prelabor CNS injury, absence of late decelerations excludes ongoing hypoxia in a neurologically intact fetus.
• Since such fetuses may not tolerate labor without sudden deterioration and demise, CD or OVD would be appropriate, per algorithm, if pattern persists 1 hour.
• Minimal variability may be an indication of evolving *fetal response to acute or chronic hypoxia* when associated with late, prolonged or variable decelerations.
• This may be due to an attempt by the fetus to increase perfusion by increasing cardiac output or it may be due to increased catecholamine activity from the adrenal medulla in response to the stress of hypoxemia, when associated with a deceleration, and loss of vagal tone.
• CD or OVD would be appropriate, per algorithm, if pattern persists 1 hour.
Abnormal EFM (Category 3)

Include:

Absent baseline FHR variability AND any of the following:
- Recurrent late decelerations
- Recurrent variable decelerations
- Bradycardia

OR

Sinusoidal Pattern

25% are acidotic

Highly FALSE POSITIVE
But you cannot be certain; therefore intrauterine resuscitation measure are warranted.
Baseline 150, absent variability, recurrent late decelerations

Per algorithm, expedited delivery is indicated regardless of labor progress.
What are we doing?

- Reasonable management decisions simply cannot be based on the results of a test that is *virtually always wrong*.
- The *negative predictive value* of intrapartum FHR monitoring is nearly 100%.
- Trying to use intrapartum FHR monitoring to diagnose neurologic injury is a recipe for failure.
- In contrast, relying on the presence of moderate variability or accelerations, or both, to confirm adequate fetal oxygenation allows the clinician to formulate and articulate a rational, evidence-based plan of management that reflects consensus in the literature.

EFM and Critical Thinking

- Is the fetus acidemic?
- Underlying cause?
- In-utero resuscitation helped?
- Labor progress?
- Well communicated plan?
How long to acidemia?

• The estimate of the time until the onset of metabolic acidemia and potential injury is guided by limited data suggesting that metabolic acidemia usually does not appear suddenly, but can evolve gradually over a period of approximately 60 minutes.

• This general statement applies only to FHR tracings that are normal initially and subsequently develop minimal to absent variability with recurrent decelerations and no acute events.

• It does not constitute a “safe harbor.”

How long to acidemia?

• One of the most common preventable errors at this stage of FHR management is to postpone a difficult but clinically necessary decision in the hope that the situation will resolve on its own. Despite the difficulty, the standard of care mandates that a decision must be made using the best information available.

• If a decision is made to expedite delivery, the rationale should be documented, and the plan should be implemented as rapidly and safely as feasible.

• If a decision is made to continue to wait, the rationale and plan should be documented, and the decision should be revisited after a reasonable period of time, usually in the range of 5 to 15 minutes in the second stage of labor.

Neurological damage?

- No single quantitative value of fetal arterial pH serves to define a point of hypoxia-induced damage applicable to all fetuses.

- However, the literature is consistent in its demonstration that for any individual fetus, baseline variability and accelerations will reliably be depressed before the pH has reached a level of acidemia associated with neurologic injury for that fetus, regardless of its quantitative value.

Oxygen pathway

Mother → environment

lungs

heart

vasculature

uterus

placenta

Umbilical cord

Fetus

hypoxemia

hypoxia

acidemia

acidosis

Maternal factors

Fetal response
Baseline deviation: Potential underlying causes:

<table>
<thead>
<tr>
<th>Bradycardia:</th>
<th>Tachycardia:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal heart signal</td>
<td>Maternal fever or fetal infection</td>
</tr>
<tr>
<td>Maternal hypotension</td>
<td>Intraamniotic infection</td>
</tr>
<tr>
<td>Chronic / Acute hypoxia</td>
<td>Maternal dehydration</td>
</tr>
<tr>
<td>Congenital heart block</td>
<td>Effect of medications (terbutaline)</td>
</tr>
<tr>
<td>Vagal response</td>
<td>Maternal heart rate increase with nervousness, anxiety, pain</td>
</tr>
<tr>
<td></td>
<td>Fetal tachyarrhythmia</td>
</tr>
<tr>
<td></td>
<td>Maternal hyperthyroidism</td>
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<td></td>
<td>Fetal anemia</td>
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</tbody>
</table>
Variability deviation: Potential underlying causes:

**Absent:**
Absent variability without decelerations may represent preexisting central nervous system injury with marked metabolic acidemia.

Is a hallmark sign of deep central asphyxia

Look for evidence of maternal factors (abruption, tachysystole, hypertension)

**Marked:**
- Increase in alpha-adrenergic activity
- Acute hypoxemia
- Maternal ephedrine administration
- Decreased uteroplacental perfusion or cord compression

**Minimal:**
- Drug induced (e.g., MgSO4, narcotics, general anesthesia)
- Sleep state of fetus (normal)
- Precursor to absent?
- Fetal anomalies, especially of the CNS
- Preterm fetus
- Fetal sepsis
- Recent administration of steroids
Late Deceleration: Potential underlying causes:

**Late Deceleration:**
Believed to reflect the fetal response to transient or chronic uteroplacental insufficiency.
Chemoreceptor response – delay in timing relative to the uterine contraction.

**Causes:**
- Placental causes
  - Post maturity
  - Abruption
  - Malformed placenta
- Hypertension
- Preeclampsia / gestational or chronic
- Hypotension
- Supine positioning
- Regional anesthesia
- Maternal trauma or hemorrhage
- Cocaine or methamphetamine
- Cardiac disease, anemia, smoking
O\textsubscript{2} and CO\textsubscript{2} exchange occurs in the intervillous space.

Resting tone

Contraction

Tachysystole can lead to increased potential for acidemia.
**Late Deceleration Physiology**

Oxygen mediated pathway

**Chemoreceptors** sense low pO2

- **Stimulates medullary vasomotor center**
  - **Peripheral Vasoconstriction** (Oxygenated blood is shunted away from nonessential vascular beds in gut, kidneys)
  - **“Central” Vasodilation** (Blood is shunted toward Essential organs – brain, heart, adrenals)

**Sympathetic discharge**

- **Blood pressure rises**

**Baroreceptors** sense elevated BP

Parasympathetic slows FHR

Recovery to baseline
Management
Recurrent late deceleration

Utilize maneuvers to promote uteroplacental perfusion:

- Lateral positioning,
- IV fluid bolus,
- Maternal oxygen administration and
- Evaluation for tachysystole

Category II:

- Intrauterine resuscitation and reevaluation to determine whether an adequate improvement in fetal status has occurred
- If late decelerations continue with minimal variability, the presence of fetal acidemia should be considered and the potential need for expedited delivery should be evaluated.

Category III (absent variability): deliver
Variable Deceleration

**Variable:**
- Due to umbilical cord compression
- Baroreceptor response
- Abrupt onset
- Decrease $>15\text{bpm} / >15\text{ sec}$

**Management:**
- Maternal positioning
- Vaginal exam to palpate for a prolapse
- Consider amnioinfusion during stage 1
- Adjunctive measures to improve oxygenation also may be useful depending on the severity and duration of the recurrent variable decelerations
- IV bolus
- Maternal O2
- Reduce uterine contraction frequency
- Modified pushing techniques
Variable Decelerations

Deceleration is one that is caused primarily by changes in systemic blood pressure in the fetus and is mediated through baroreceptors.

- Transitory umbilical cord compression
- Stimulation of the vagus nerve
- Abrupt decrease in fetal heart rate
- Recovery with compression release
Intrauterine Resuscitation

Change maternal position
Decrease uterine activity
IV fluid bolus
Correct maternal hypotension
Oxygen administration
Amnioinfusion
Alteration in 2\textsuperscript{nd} stage maternal pushing efforts
If prolapsed umbilical cord, elevate fetal presenting part while moving toward Cesarean

The effect should be apparent \textit{within 30 minutes} of application

Intrauterine Resuscitation: Position change

**Lateral positioning**

- May be used to correct umbilical cord compression
- Will decrease the frequency of uterine contractions
- Improves maternal cardiac return and cardiac output are maximized
- Blood flow to the uterus is optimal
- Either side is more favorable when compared with supine position
- May modify or eliminate late decelerations if the etiology is decreased uterine blood flow secondary to supine positioning

Laboring in the supine, as opposed to the lateral position is associated with lower femoral arterial blood pressure, and lower fetal scalp capillary pH.
Intrauterine Resuscitation: Decrease uterine activity

- Attention should be given to the prompt elimination of excessive uterine activity including tachysystole or prolonged contractions, especially when uterine stimulants (oxytocin or prostaglandin-containing agents) are being applied.
- Oxytocin infusion should be decreased or discontinued in the presence of excessive uterine activity and a persistent category II FHR pattern.
- Oxygen given for intrauterine resuscitation and continued infusion of oxytocin contradict each other.
- Consider administration of subcutaneous terbutaline 0.25mg.
Tachysystole management

[Diagram of Tachysystole management process]

ACOG Practice Bulletin 116, 2010
Tachysystole (if averaged over 30 minutes)

- Even if the FHR is normal, action must be done
- Applies to both spontaneous and stimulated contractions
Resumption of oxytocin after tachysystole resolves

- If oxytocin has been discontinued for less than 20–30 minutes, the FHR is normal, and contraction frequency, intensity, and duration are normal:
  - resume oxytocin at no more than half the rate that caused the tachysystole and
  - gradually increase the rate as appropriate based on unit protocol and maternal-fetal status.

- If the oxytocin is discontinued for more than 30–40 minutes:
  - resume oxytocin at the initial dose ordered.
Resolution of tachysystole

**INTERVENTION**

- Oxytocin discontinuation alone
- Oxytocin discontinuation + IV fluid bolus of at least 500mL LR
- Oxytocin discontinuation + IV fluid bolus of at least 500mL LR + Change to lateral position

**TIME TO RESOLUTION**

- 14.2 minutes
- 9.8 minutes
- 6.1 minutes

Simpson & James, 2008
Intrauterine Resuscitation: Fluid bolus

- Fluid bolus: Administer 500–1,000 mL Lactated Ringer’s solution IV over 20 min
- Glucose containing IV fluids should not be used for volume expansion
- Thought to improve uteroplacental perfusion through expansion of maternal intravascular volume. Positive effects continue for about 30 minutes
- Caution when increasing IV fluids or giving repeated IV fluid boluses.
- Some clinical situations (e.g. preeclampsia, corticosteroids) carry an increased risk for pulmonary edema
- Oxytocin has an antidiuretic effect (prolonged use can lead to fluid overload)
Intrauterine Resuscitation: Correct maternal hypotension

- Hypotension: defined systolic <100
- Lateral positioning
- IV fluid bolus of at least 500mL of Lactated Ringers
- Consider ephedrine: 10-15mg IV bolus

Conduction anesthetics/analgesics produce a sympathetic blockade, increasing the risk of decreased placental blood flow with or without overt maternal hypotension.
Intrauterine Resuscitation: Oxygen

- Administer 10 L/min of O2 by nonrebreather face mask for 15-30 min, then evaluate response.
- Prolonged oxygen administration should be avoided.
- Fetuses with lower oxygen saturation appear to benefit most from maternal oxygen administration.
- Even though healthy women have nearly 100% SpO2, increasing inspired oxygen increases blood oxygen tension and results in more oxygen delivered to the fetus and the fetal cerebral tissues (fetal Hg affinity).
Intrauterine Resuscitation: Amnioinfusion

• May be helpful in treatment of recurrent variable decelerations during first stage labor that have not resolved with maternal position changes.

• **Does not:**
  - effect late decelerations or patterns with minimal to absent variability
  - reduce risk of moderate or severe meconium aspiration syndrome

• Prophylactic for oligo is not necessary and does not seem to prevent variable decelerations

• Administration
  - Room temperature normal saline or LR via IUPC (may be warmed with blood/fluid warmer if preterm)
  - 250-500mL over 20-30 minutes
  - Continuous infusion 120-180ml/hr
  - Max amount infused is 1000 mL
  - Weigh chux
  - If uterine resting tone is > 40mmHg, temporarily discontinue
Intrauterine Resuscitation: Alter maternal pushing efforts

• Stopping pushing temporarily or pushing with every other or every third contraction based on the fetal response can be effective at allowing the fetus to recover and maintain adequate reserves

• Delaying active pushing until the woman feels the urge to push can minimize fetal stress.
Additional measures to consider if clinically appropriate:

- Cervical check to assess the progress of labor
- Fetal scalp stimulation to assess for FHR acceleration
  - Digital scalp stimulation may be performed by vigorously rubbing the fetal scalp for 15 sec using an examining finger while fetus is at baseline.

![Graph showing fetal heart rate monitoring](image)
What is the plan?

• **Standardization** has long been recognized as an essential element of patient safety, and a growing body of contemporary evidence confirms that standardization can reduce adverse outcomes and malpractice claims.

• After appropriate conservative measures have been implemented and obstacles to rapid delivery have been cleared away, it is sensible to take a moment to *estimate the time needed to accomplish delivery* in the event of a sudden emergency.

• This step should be addressed by the clinician who is ultimately responsible for performing operative delivery, should it become necessary.
Maternal vs. Fetal Heart Rate

• Fetal monitoring technology cannot detect a difference between a fetal and maternal signal source
  • Coincidence alarm
  • Notation on strip

• Should palpate maternal radial pulse or apply pulse oximetry for a minimum of 1 minute while listening to the device sounds.
Heart detection with IUFD

• With fetal death, the maternal ECG signal may be amplified through the fetal scalp electrode and be displayed spuriously as the FHR
  • Spiral electrode application is not helpful in confirming fetal life, because the printed MHR may be misinterpreted as an FHR

• An additional instance that may cause confusion is the mother with a cardiac pacemaker. If the transmitted maternal pacemaker pulse is at a higher voltage than the fetal R wave, the scalp electrode may record the pacemaker signal
  • Maternal electrocardiogram (ECG) signal is approximately 5–10 times stronger than the fetal one
Whose baseline?

• Maternal heart rate is usually significantly lower than the mean baseline FHR throughout all stages of labor and delivery and rarely exceeds 110 beats per minute between contractions.

• Maternal heart rate may be increased with chorioamnionitis
Whose variability?

- Mean ‘maternal variability’ of the baseline heart rate is significantly greater than that of the FHR baseline during the first and second stages of labor.

- However, most ‘maternal variability’ values fall within the moderate range for FHR variability (6–25 beats per minute), and thus this difference may not be large enough to be discriminatory.
Whose accelerations?

- Very similar to FHR accelerations during the latent phase of the first stage but, as labor progresses, maternal ‘accelerations’ become more frequent, have higher amplitudes, and longer duration.

- Cardiac output increases progressively during labor by about 35% at complete dilatation due to
  - Increased venous return to the heart leading to ↑ stroke volume
  - Increased heart rate
  - Increase in catecholamine
  - Valsalva with pushing.

The maternal heart rate usually accelerates with uterine contractions and bearing-down efforts in stage 2.
G1P0 induction at 41 weeks 14 hours in labor. Complete / O station
Maternal pulse oximetry applied

Fetal
With Valsalva, oxygen and blood flow is decreased in intervillous spaces. The physiologic fetal response is a decrease in fetal heart rate (not an acceleration).
Baseline 140, minimal variability, prolonged decelerations
Outcome – near miss
Summary

• Connect the underlying physiology and apply critical thinking to the pattern seen
  • Always consider what is the etiology of the pattern and not just the pattern
• Indeterminate patterns require surveillance and reassessment
• Acidemia usually evolves over a period of time
• Intrauterine resuscitation techniques should provide a response within 30 minutes
Summary

• Communication is key
  • With documentation, interpretation of EFM assessment, chain of command, handoffs, plan of care

• *Hope is not a plan*

• Fetal monitoring technology cannot detect a difference between a fetal and maternal signal source