Basic Fetal Monitoring Course

Instructor: Jonie Johnson, RNC

History of FHM

1968

Commercially available

1972

Fundamentals of FHM

Equipment

How can we capture the necessary data?
Fundamentals

- The Equipment
  - Monitor make and model
  - Types of transducers & techniques of monitoring
    - FHR: external & internal
    - Uterine activity: external & internal
  - The FHM strip: paper or electronic

Fundamentals: Makes of Monitors

Fundamentals: Transducers

External

Internal
Fundamentals: Transducers

External

Internal

Uterine Activity

• Methods of Assessing UA
  – Palpation
  – Tocodynamometer (TOCO)
  – Intrauterine Pressure Catheter

Palpation

Can obtain a general indication of frequency, duration, intensity and resting tone.
Palpation

• Benefits
  – Noninvasive
  – Hands on; human touch
  – Mobility of mother
  – No equipment necessary

Use with all other methods of monitoring uterine activity to verify accuracy of information

• Limitations
  – Maternal size can limit ability to palpate contractions
  – Subjective
  – No hard copy generated

Tocodynamometer (TOCO)

• Pressure sensitive button on TOCO detects external changes in the contour of the abdomen that occur with uterine contractions

• Can assess relative frequency and duration

• Palpate to obtain a general indication of intensity and resting tone

TOCO

• Benefits
  – Minimally invasive
  – Does not require ROM
  – Tracing generated

• Limitations
  – Does not objectively measure intensity and resting tone
  – Maternal size can interfere with ability of TOCO to sense changes in abdomen
  – Location sensitive; placement can lead to false information
  – Limits maternal mobility
TOCO

- Troubleshooting
  - UA Reference
  - Palpate fundus to find point of maximum intensity
  - Apply TOCO firmly to abdomen
  - May need to apply lower for preterm patient

Intrauterine Pressure Catheter (IUPC)

- Can assess frequency, duration, intensity and resting tone

- Indications:
  - IF YOU NEED MORE INFORMATION
  - Dystocia
  - VBAC
  - Inability to obtain accurate assessment of UA with administration of oxytocin
  - More accurate timing of FHR changes with UCs
  - Amnioinfusion
  - Withdrawal of amniotic fluid for testing
IUPC

• Contraindications:
  – ROM not desired
  – Maternal infection with risk of vertical transmission
  – Vaginal bleeding
  – Placenta previa or low lying placenta

IS THE RISK OF IUPC PLACEMENT WORTH THE BENEFIT OF THE INFORMATION GENERATED?

IUPC

• Benefits
  – Objective measurement of frequency, duration, intensity and resting tone in mmHg or MVUs
  – More accurate correlation of FHR changes with UA
  – Tracing generated
  – Amnioinfusion

• Limitations
  – Requires ROM and cervical dilatation
  – Invasive procedure
  – Increased risk of uterine infection, perforation or placental separation
  – Limits maternal mobility

IUPC

• Troubleshooting
  – Have patient cough to verify placement
  – Palpate to confirm presence of contractions
  – Check for possible displacement of catheter
  – Rotate catheter 180 degrees
  – Re-zero transducer per manufacturer’s instructions
Fetal Heart Rate

• Methods of Assessing Fetal Heart Rate
  – Fetoscope or Hand Held Doppler
  – Ultrasound Transducer
  – Spiral Electrode

Fetoscope

• True method of auscultation
• Detects sounds of fetus’ heart beats
• Can assess fetal heart rate, rhythm, increases or decreases

Fetoscope

• Benefits
  – Widespread application
  – Noninvasive
  – Patient comfort and mobility
  – Increased hands on care with 1:1 nurse/patient ratio
  – May verify dysrhythmias—no machine error

• Limitations
  – Difficult to count
  – Cannot assess variability
  – May miss an event when not listening
  – Difficult to assess FHR with movement, obese mother or during contraction
  – No tracing generated
  – 1:1 nurse/patient ratio difficult
  – Requires practice and skill
Auscultation

- Count FHR for 6 sec, multiply by 10 to get bpm.
- Continue counting for 6 sec increments for 1 min to determine if the FHR has decels or accels.
- Auscultate during and after contractions so decels could be detected.
- Evaluate FHR by auscultation at same frequencies as stated in policy for stage of labor.
- If unsure of fetal status after auscultation, place patient on continuous EFM to assess further.

Ultrasound Transducer

- Sound waves detect fetal heart movement
- Assess fetal heart baseline rate, rhythm, variability, accelerations and decelerations

Ultrasound Transducer

- Benefits
  - Noninvasive
  - Does not require ROM
  - Provides a permanent record
- Limitations
  - Restricts maternal movement
  - Difficulty transmissions with maternal and/or fetal movement, maternal obesity, fetal position
  - Monitor may half/double count with tachycardia or bradycardia
Ultrasound Transducer

• Troubleshooting
  – Apply snugly to abdomen
  – Reposition
  – Apply gel
  – Palpate maternal pulse

Spiral Electrode

• Detects electrical activity of fetus’ heart
• Assess baseline rate, rhythm, variability, accelerations and decelerations
• Indicated when information obtained with other methods is not adequate
• Contraindicated with some maternal infections or fetal coagulopathies

IS THE RISK OF FSE PLACEMENT WORTH THE BENEFIT OF THE INFORMATION GENERATED?

Spiral Electrode

• Benefits
  – Continuous detection of FHR
  – Allows for more freedom of movement for patient than does U/S

• Limitations
  – Requires ROM, adequate cervical dilatation, appropriate fetal presenting part
  – Potential for transmission of maternal infection
  – Potential for fetal hemorrhage/injury
  – May record maternal HR with fetal demise
  – Potential for electronic interference and artifact
Spiral Electrode

- Troubleshooting
  - Check all connections
  - Replace SE and/or monitor part
  - Confirm fetal HR w/ fetoscope
  - Palpate maternal pulse while validating FHR

Fundamentals

<table>
<thead>
<tr>
<th>Frequency of Assessment:</th>
<th>Recommended Assessment and Documentation</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Latent Phase</td>
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<tr>
<td>ACOG</td>
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<tr>
<td>Low Risk</td>
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<td>High Risk</td>
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<td>AWHONN</td>
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<td>Low Risk</td>
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<td>High Risk</td>
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<tr>
<td>SOGC</td>
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<tr>
<td>Regularly after rupture of membranes or other clinically significant change</td>
<td>Every 15 minutes</td>
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FHR Assessments

<table>
<thead>
<tr>
<th>Assess FHR before:</th>
<th>Assess FHR after:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Initiation of labor enhancing procedures (amniotomy)</td>
<td>• Admission of patient</td>
</tr>
<tr>
<td>• Ambulation</td>
<td>• Artificial or spontaneous rupture of membranes</td>
</tr>
<tr>
<td>• Administration of medications</td>
<td>• Vaginal examinations</td>
</tr>
<tr>
<td>• Administration or initiation of analgesia or anesthesia</td>
<td>• Ambulation</td>
</tr>
<tr>
<td>• Transfer or discharge of patient</td>
<td>• Recognition of abnormal uterine activity patterns</td>
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<tr>
<td></td>
<td>• Administration of medication</td>
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Physiologic Basis for FHM Interpretation

Accurate FHM assessment is based on understanding the physiology and pathophysiology of fetal heart response to the intrauterine environment. These responses are indirect indicators of fetal oxygenation.

Intrinsic factors include the fetal mechanisms of FHR control and related fetal cardiovascular anatomy and physiology. Extrinsic factors include the fetal environment, maternal cardiovascular and uterine anatomy and physiology, and placental and umbilical cord structure and function.

Intrinsic Influences

• The fetal heart rate, like the adult heart, has an intrinsic rate which is determined by the dominant pacemaker or sinoatrial (SA) node. The average heart rate range is 110-1160 (bpm) in the healthy near-term fetus. The heart rate in a healthy fetus is rarely static. Normal baseline variations, or variability, as well as more dramatic changes in the rate are present. These changes occur due to the autonomic nervous system. Let's review the two branches.
Intrinsic Influences
Parasympathetic Nervous System

• Stimulation slows SA node rate of firing, producing a decrease in FHR.
• Action occurs via release of acetylcholine.
• Tone increases as gestation advances and produces downward effect on baseline rate.
• Effect on FHR may be exaggerated during hypoxemia.
• Blocking produces increased FHR and loss of variability.

Intrinsic Influences
Sympathetic Nervous System

• Stimulation produces increase in strength of myocardial contraction and increase in FHR.
• Action occurs through release of norepinephrine
• Blocking the sympathetic system, as with maternal medication, produces a decrease in the baseline FHR.
• Effect on the FHR may be stimulated during hypoxemia.

Intrinsic Influences
Chemoreceptor Influences

• Respond to the changes in O2 and CO2 tensions, and pH levels in blood or cerebrospinal fluid.
• Stimulation due to mild increases in CO2 or mild decreases in O2 produces an increase in fetal blood pressure and FHR; more severe changes produce bradycardia.
• Chemoreceptors are located in the aortic arch, carotid bodies, and medulla oblongata.
Intrinsic Influences

Baroreceptor Influences
- Respond rapidly to changes in fetal blood pressure.
- Increase in fetal blood pressure produces a decrease in the FHR, which decreases fetal cardiac output and blood pressure.
- Decrease in fetal blood pressure results in sympathetic stimulation to increase FHR.
- Baroreceptors are located in the aortic arch and the carotid sinuses.

Central Nervous System Influences
- Responsibility for variations in FHR and variability in response to fetal sleep state and body movements.
- Integrative center for central and peripheral neural influences that produces variability and net increase or decrease in baseline FHR.
- Located in the cerebral cortex and medulla oblongata.

Hormonal Influences
- Catecholamines
  - Facilitate hemodynamic changes in response to hypoxemia; facilitate adaptational changes in a neonate at birth.
  - Epinephrine
    - Secreted by the adrenal medulla (in significantly smaller amounts than norepinephrine).
    - Increases FHR and blood flow to skeletal muscle.
Intrinsic Influences
Hormonal Influences

- Norepinephrine
  - Predominant hormone secreted by adrenal medulla, also secreted by sympathetic nerves.
  - Associated with initial increase in FHR.
  - Increases blood flow to vital organs (brain, heart, adrenals), and away from nonvital organs (gastrointestinal tract and periphery).
  - The above hemodynamic changes elevate blood pressure and may cause a parasympathetic response that is reflected by a decreased FHR. Norepinephrine cannot overcome this parasympathetic response.
  - Secreted in greater amounts than that found in a resting adult.

- Vasopressin
  - Secreted by pituitary; increased release during hypoxemia and hemorrhage
  - Helps regulate blood pressure.
  - Produces a rise in blood pressure by increasing peripheral vascular resistance and decreasing FHR.
  - Decrease blood flow to nonvital organs.

- Renin
  - Secreted by kidneys; increased release in response to hemorrhage (hypovolemia)
  - Angiotensin II
  - Secreted by kidneys; increased release in response to hemorrhage and hypoxemia.
  - Exerts tonic vasoconstricting effect on peripheral vascular bed resulting in maintenance of systemic arterial blood pressure and umbilical-placental blood
Intrinsic Influences
Hormonal Influences

• Angiotensin II (cont)
  • Increased release produces marked increased in blood pressure with an initial decrease in FHR followed by an increase to higher than the previous FHR. Increased release produces increased cardiac output and blood flow to the heart.
  • Decreases renal blood flow.

Extrinsic Influences on the FHR

• Maternal Influences
  • Baseline maternal arterial oxygen is the source for oxygen transported to and used by the fetus.
    – Acute maternal hypoxemia compromises maternal arterial oxygen saturation and tension and reduces oxygen available to the fetus.
    – Acute or chronic maternal respiratory disease may reduce oxygen tension and result in compromised fetal or placental growth development.

Extrinsic Influences on the FHR

• Maternal smoking results in a lowered oxygen saturation despite adequate oxygenation because carbon monoxide molecules displace oxygen on maternal and fetal hemoglobin.
• Maternal hypoventilation due to breath holding during pushing may transiently decrease oxygen availability.
Extrinsic Influences on the FHR

• Maternal oxygen-carrying capacity depends on sufficient hemoglobin to transport oxygen.
  – Maternal blood volume increases by approximately 45% during pregnancy due to an increase in plasma volume and an increase in erythrocytes of approximately 30%; Maternal hematocrit actually decreases due to a dilutional effect.
  – Maternal anemia that is due to iron deficiency, hemoglobinopathies, or hemorrhage reduces available hemoglobin for oxygen transport.

Extrinsic Influences on the FHR

• Adequate blood flow to the uterus determines the availability of oxygen for placental perfusion.
  – Maternal cardiac output increases during pregnancy by approximately 40% in response to the increased volume of the circulatory system. This increased volume is accompanied by peripheral vasodilation and the development of the large vascular bed in the placenta.
  – Normal uterine blood flow is determined by adequacy of maternal arterial blood pressure. Supine positioning can significantly reduce uterine blood flow by decreasing venous return and uterine arterial blood pressure. Maternal diseases and drugs that produce significant vasoconstriction reduce uterine blood flow.

Extrinsic Influences on the FHR

• Conduction anesthesia may cause a systemic hypotension, which markedly reduces uterine arterial pressure and therefore, uterine blood flow.
  • Maternal hyperventilation also may increase catecholamine production, exaggerate the mild compensated respiratory alkalolemia of pregnancy, and reduce uterine blood flow.
Extrinsic Influences on the FHR

- Uterine contractions influence fetal oxygen availability.
  - The myofibrils of the uterus consist of two layers, one circular and one longitudinal. Biochemical and physiologic changes occur near term and during parturition, enabling the uterus to contract in a coordinated, efficient manner. Brief, weak uterine contractions occur early in pregnancy and are replaced by stronger, more regular contractions near term. Progression into labor contractions may be gradual or abrupt.

Extrinsic Influences on the FHR

- Baseline tonus is a characteristic of the pregnant uterus at rest and represents intrauterine pressure between contractions; pressures greater than 20-25 mmHg usually are considered hypertonus.
- Uterine arteries and veins conducting maternal blood to and from the placental circulation pass through the myometrium and are compressed during uterine contractions. During labor, oxygen/carbon dioxide exchange occurs primarily between uterine contractions when blood flow is unimpeded. Uteroplacental blood decreases at intrauterine pressures greater than 35mmHg and may cease at 50-60 mmHg; therefore, some degree of reduction in maternal-fetal exchange will occur during most uterine contractions.
Extrinsic Influences on the FHR

• Placental influences
  • The placental structure and ability to function both affect the availability of oxygen for fetal use, and in turn affect FHR. Key placental influences include the placental structure, the placental function, and the placental blood flow.

• Placental structure
  • Functional placental surface area is the amount of placental-fetal interface surface available for the exchange of nutrients, elimination of fetal waste, and production of hormones and steroids.
  • Functional placental surface area depends on adequate maternal nutrients and maternal-uterine blood flow.

Extrinsic Influences on the FHR

• Placental Function
  • Adequate placental function provides for the transport of oxygen to the fetus at levels above fetal basal needs
  • Compromised placental growth and development results in decreased placental function, and may, depending on the degree and timing of compromise, result in fetal growth restriction and, possibly, inadequate oxygenation.
  • Decreased placental function impairs the fetal ability to withstand the normal stresses of labor and birth.

Extrinsic Influences on the FHR

• Depending upon the degree of loss of placental function, additional hypoxic stresses usually tolerated by the healthy fetus may result in a rapid decompensation.
  • Compromised placental function also may be associated with a reduction of amniotic fluid volume, limiting protections of the fetus and umbilical cord.
Extrinsic Influences on the FHR

- Placental blood flow and blood oxygen content affect oxygen delivery to the fetus.
- Approximately 70-90% of uterine blood flow reaches the placenta. This percentage directly reflects the amount of oxygen available for maternal-fetal exchange.
- Substances cross the placenta by several mechanisms; oxygen is believed to cross the placenta predominantly by simple passive diffusion at a rate directly proportional to placental area and to the differences in concentration and pressures of oxygen on either side.

Extrinsic Influences on the FHR

- Fetal gas exchange occurs in the placental villi contained within the cotyledons (normally 15-30 in number) and depends on the structural integrity of the placenta and the related placental blood flow. Placental structural integrity may be compromised by damage to the cotyledons (infarcts) as seen in maternal conditions such as inadequate nutrition, diabetes, smoking or pre-eclampsia.
Extrinsic Influences on the FHR

- Placental aging, partial abruption, and structural abnormalities, such as circumvallate placenta, also may compromise placental integrity, blood flow, and oxygen delivery. These conditions may be observed and evaluated by ultrasound scan.
- Uteroplacental vessels have a marked capacity for constriction in response to either maternal sympathetic nervous system activation or vasconstrictor drugs. The constricted vessels may result in reduced placental blood flow and reduced fetal gas exchange even in the presence of normal maternal hemoglobin and arterial oxygen saturation.

Extrinsic Influences on the FHR

- Umbilical Cord is the vascular connection between the placenta and fetus. The cord’s contribution to fetal oxygenation and subsequent FHR responses may be considered as either extrinsic or intrinsic factors.
- Vascular abnormalities of the cord, such as true knots, strictures, or hematomas may cause an acute or chronic impairment in blood flow.

Fetal Heart Monitoring Interpretation

What is this code the fetus is sending out?
Fetal Heart Monitoring Interpretation

• Fetal assessment relies on the premise that the FHR reflects fetal oxygenation
  – It is a good predictor of normal outcome
  – It is not a good predictor of poor outcome

Fundamentals:
Assessment of FHR & UA Characteristics

Fetal Heart Rate:
The 4 Elements
• Baseline
• BL variability
• Accelerations
• Decelerations

Uterine Activity:
The 4 Elements
• Frequency
• Duration
• Intensity
• Resting tone

Consistency of Process: The
Same way EVERY time

Basic Pattern Interpretation

• Systematic method to evaluate every tracing:
  – FHR baseline
  – FHR baseline variability
  – Periodic or episodic changes
  – Uterine activity
  – Category
  – Pattern evolution
  – Accompanying clinical characteristics
  – Probable cause of the changes present
  – Normalcy vs. Urgency
Basic Pattern Interpretation

• The 2008 NICHD Report of Fetal Heart Rate Monitoring:
  – Defined standard fetal heart rate nomenclature
  – Identified three categories for fetal heart rate interpretation
  – Proposed future research
    • Endorsed by ACOG, AWHONN, ACNM, AAFP

Uterine Activity

• Frequency
  – How often are the contractions occurring?
  – Usually assessed in ½ minute or whole minute intervals - count from the beginning of one contraction to the beginning of the next.
  – Document range
  – Avoid “occasional” or “irregular”

Uterine Activity

• Frequency
  – Normal: ≤ 5 contractions in 10 minutes, averaged over 30 minutes
  – Tachysystole: > 5 contractions in 10 minutes, averaged over 30 minutes
  – Coupling & tripling contractions
• **Duration**
  
  – Usually assessed in ten second intervals—count from when contraction starts to when it ends
  – Document range
  – How long is too long?
Uterine Activity

- Intensity
  - How strong are they?
  - Assessed by palpation or IUPC
  - With palpation, document as mild, moderate, or strong
  - With IUPC, document in mmHg or MVUs

Calculating MVUs

Connection forces are usually reported in Modified Units (MVUs), which represent the total of the intensity of each contraction in a 10-minute period. MVUs > 200 are adequate for 90% of labors to progress.

MVUs = \( \frac{75+60+50+45}{4} = 57.5 \) MVUs

(Note: the baseline pressure was subtracted from each reading)
Uterine Activity

• Resting Tone
  – Uterine tone between contractions
  – Palpation (with TOCO): soft or firm
  – IUPC: mmHg
    • Usual ≤ 20mmhg

Fetal Heart Rate

• Baseline Fetal Heart Rate
  – Normal range is 110-160 bpm
  – Mean FHR over a 10 minute period rounded to increments of 5 bpm, excluding accelerations and decelerations and periods of marked FHR variability
  – In any 10-minute window, the baseline must last for at least a 2 minute period (not necessarily contiguous), otherwise the baseline is indeterminate. You may need to refer to the previous 10-minute window to determine the baseline.
Fetal Heart Rate

- **Tachycardia**
  - Sustained baseline FHR greater than 160 bpm for more than 10 minutes
  - Causes can be either maternal or fetal

- **Bradycardia**
  - Sustained baseline FHR less than 110 bpm for more than 10 minutes
  - Causes can be either maternal or fetal

Fetal Heart Rate

- **Baseline FHR variability**
  - Fluctuations in the baseline FHR that are irregular in amplitude and frequency
  - Amplitude range is **visually** quantified as follows:
    - **Absent** FHR variability = Undetectable amplitude range
    - **Minimal** FHR variability = > undetectable ≤ 5 bpm
    - **Moderate** FHR variability = 6-25 bpm amplitude range
    - **Marked** FHR variability = >25 bpm amplitude range
Fetal Heart Rate

- Baseline FHR Variability
  - Reflects fetal oxygen reserve
  - Minimal variability
    - Sleep, sedation, hypoxic stress
  - Absent variability

Fetal Heart Rate

- Periodic Changes
  - Associated with contractions
    - Recurrent if occur with ≥ 50% of contractions in a 20-minute window.
    - Intermittent if < 50% of contractions in 20 min
- Episodic Changes
  - Not associated with contractions
### Fetal Heart Rate

- **Periodic Changes**
  - Late decelerations
  - Early decelerations
  - Variable decelerations
  - Accelerations

- **Episodic Changes**
  - Accelerations
  - Variable decelerations

### Fetal Heart Rate

- **Accelerations**
  - Abrupt (onset to peak in < 30 sec) increases in FHR above the baseline
  - Can be periodic or episodic
  - In fetus ≥ 32 weeks should be at least 15 beats above the baseline and last for at least 15 seconds (15X15 rule)
  - In fetus < 32 weeks, can be acceptable if 10X10

- **Indicate a well-oxygenated fetus with an intact CNS**
- If present, can exclude fetal acidemia at that time
Fetal Heart Rate

• Decelerations
  – Decrease from the baseline FHR
  – Gradual or abrupt decline
  – Periodic or episodic
  – May be recurrent

Fetal Heart Rate

• Four types
  – Early
  – Late
  – Variable
  – Prolonged
Fetal Heart Rate

• Early Decelerations
  - Gradual decrease (onset to nadir in ≥ 30 seconds) in the FHR from the baseline
  - Usually symmetrical
  - The lowest point (nadir) occurs with the peak of the contraction
  - Associated with head compression
  - Thought to be a benign response to head compression, but decide if they are occurring in the usual circumstance

Fetal Heart Rate

• Variable Decelerations
  - Abrupt decline (onset to beginning of nadir in ≤ 30 seconds) from baseline FHR with usual abrupt return also
  - Decrease is ≥ 15 bpm, lasting ≥ 15 seconds, and < 2 min
  - Can be periodic or episodic
  - Associated with cord compression
  - Significance depends on duration and persistence and other parameters of the clinical picture, such as baseline FHR, variability, presence/absence of accelerations. Look at entire clinical picture to determine fetal tolerance
Fetal Heart Rate

• Late Decelerations
  – Gradual decrease (onset to nadir in ≥ 30 seconds) and return to baseline with nadir occurring after the peak of the contraction
  – Usually symmetrical
  – At end of contraction, FHR will not have returned to baseline (delayed in timing)
  – Associated with utero-placental insufficiency (UPI)
  – Determine significance by assessing if you can “fix” the cause—and by their recurrence
  – Fetal tolerance determined by accompanying FHR baseline, variability and presence or absence of other periodic or episodic changes
Fetal Heart Rate

• Prolonged Deceleration

  – Deceleration lasting ≥ 2 minutes and < 10 minutes
  – What just happened?
  – Fix the cause!
  – Usually will return to pre-deceleration state if interventions relieve the cause
Fetal Heart Rate

• Sinusoidal FHR Pattern
  – Smooth, sine wave-like undulations with a cycle frequency of 3-5/minute lasting ≥ 20 minutes

Arrhythmias

– Irregularity of cardiac rhythm
– Machine can halve or double FHR to "correct" the baseline
– Verify with US fetal echocardiogram or fetoscope

*Listen! Believe your ears until proven otherwise*
Interpretation

• NICHD Three Tier FHR System
  
  – Category I
  – Category II
  – Category III

Interpretation

• Category I – Normal fetal acid-base status
  
  – Includes ALL of the following:
    • Baseline FHR: 110-160 bpm
    • Baseline variability: moderate
    • Late or variable decelerations: absent
    • Early decelerations: present or absent
    • Accelerations: present or absent
Category I

Interpretation

• Category II – Indeterminate fetal acid-base status
• Examples:
  – Bradycardia not accompanied by absent variability
  – Tachycardia
  – Minimal variability
  – Absence of induced accelerations after fetal stimulation
  – Prolonged decelerations
  – Recurrent late decelerations with moderate variability

Category II
Category II

Interpretation

• Category III – Predictive of abnormal fetal acid-base status
• Include EITHER
  – Absent FHR variability and
  – Bradycardia or recurrent late or recurrent variable decelerations
• OR
  – Sinusoidal pattern

Category III
Category III

FHR Occurs Across a Continuum

NICHD Categories

- Nonhypoxic reflex response
  green light “go” Category I
- Compensatory mechanism to hypoxemia and/or hypoxia
  yellow light “warning” Category II
- Impending decompensation
  red light “stop” Category III
General Management Principles

- **Category I**: predictive of normal acid-base status; follow in routine manner; no action required

- **Category II**: indeterminate of fetal acid-base status
  - Require heightened surveillance
  - Clinical interventions vary to circumstances
  - Consider birth options in context of labor progress & evolution of pattern

- **Category III**: predictive of abnormal fetal acid-base status
  - Clinical interventions vary to circumstances
  - If not quickly resolved, expedite delivery

FHM Case

Gina is a G3, P2002 at 39 6/7 weeks’ gestation
She came to L&D with c/o FM for 24 hr.
Prenatal course without complication and all labs WNL
NST was non-reactive and BP156/98, P88, R 18, T 98.8
15 min. repeat BP 150/96
Provider notified, ordered oxytocin induction

US/TDCO
2 hr. later. Oxytocin at 8mu/min. SVE 3/80%/2, BP 154/96

BL, variability, decs, category?
155, mod, variable, II
What FHR characteristic indicates the presence or absence of fetal oxygen reserve?
   a. Depth of deceleration pattern
   b. Duration of deceleration of pattern
   c. Presence of variability

Interventions?
   • Reposition
   • Fluid bolus

Maybe DC Oxytocin (protocol?)
Moderate variability/one variable deceleration O2 not indicated

FSE/IUPC
40 min. later: ROM, clear fluid. BP 162/98, c/o HA
Magnesium Sulfate Infusion Initiated. Labetalol 20 mg IV SVE 4/90%/1

100, minimal, variable and late, II
Contractions?
3, 90-100, 45-60 peak, 10 rt

What is a possible physiologic extrinsic influence causing Gina's tracing?
   a. Increased uterine tone
   b. Decreased placental blood flow
   c. Fetal growth restriction

• Oxytocin was discontinued
FSE/IUPC
1 hr. later. Epidural in place. BP 155/96
Magnesium Sulfate at 2 gm/hr. SVE 9/100%/0
BL, variability, decs, category?
150, min-mod, variable & late, II
Contractions?
2/3/2/2, 90-130, 55-70 peak, 10-15 rt

What is a possible physiologic rationale for the FHR pattern?

a. Compromised placental and umbilical perfusion
b. Head compression and Magnesium Sulfate
c. Late decelerations and fetal acidosis

- Gina gave birth 40 min. later- pushed for 20 min. on her side with every other contraction, O2 per mask.
- SCN was at delivery.
- Baby girl did not require resuscitation. APGARs of 7/9 (1 off color, tone, reflex irrit.), wgt. 6#14.
- Pt continued on Mag for 24 hr.
- Discharge BP 144/90, home on Labetalol p.o., f/u in 3 days
Interventions

How do I fix this problem?

Interventions

• Physiologically based

• Follow the nursing process
  — Assess – Interpret – Diagnose – Intervene – Evaluate

• What is the underlying cause?
• Can I fix it?

• If not, interventions should promote oxygenation of mother and fetus

Interventions

Five goals of interventions

  • Maximize uterine-placental blood flow
  • Maximize umbilical circulation
  • Maximize available oxygen
  • Maintain appropriate uterine activity
  • Support maternal coping and labor progress
Interventions
• Maximize uterine-placental blood flow
  – Position laterally
  – Intravenous hydration
  – Medication
  – Reduce pain/anxiety

Interventions
• Maximize umbilical circulation
  – Maternal position change
  – Elevation of presenting part
  – Amnioinfusion

Interventions
• Maximize available oxygen
  – Position laterally
  – Give maternal oxygen
  – Guide maternal breathing techniques
  – Correct or treat underlying disease
Interventions

• Maintain appropriate uterine activity
  – Maternal lateral position
  – Reduce/discontinue oxytocin or other uterotonic drugs
  – IV fluid bolus
  – Administer tocolytics

Interventions

• Support maternal coping and labor progress
  – Provide a comforting presence
  – Maintain situational awareness
  – Evaluate mother’s expectations

Interventions

FHR Baseline Changes?
  – Tachycardia, Bradycardia, ↓ variability
• What is the cause?
  – Do I need further information?
• How can I correct the problem?
• Did my interventions fix it?
Interventions

Variable Decelerations
• What is the cause?
• Goal: Maximize umbilical circulation
• How?
• Did my interventions fix it?

Interventions

Late Decelerations
• What is the underlying cause?
  – ↓ maternal BP
  – ↓ maternal O2
  – Decreased placental functional area
  – Tachysystole

Interventions

Late deceleration Goals:
  – Maximize uterine-placental blood flow
  – Maximize available oxygen
  – Maintain appropriate uterine activity
• How?
• Did my interventions fix it?
  – If not repeat nursing process

This is an Ongoing, Dynamic Process!!
Review of FHR Strips

Group Practice

- Reading FM strips takes time and lots of practice
- Class is only an introduction to concepts that it is based on
- Reviewing strips in the context of labor with an expert mentor is the best way to learn
- It is a process of constant practice and updating
Communication & Responsibility

Communication

- In 2014 ineffective communication figured into 70% of all sentinel events.
- Perinatal sentinel events
  - Maternal injury or death near 46%
  - Neonatal injury or death near 65%

The Joint Commission, Sentinel Event Data Q2, 2014

Communication is the response you get from the message you sent regardless of its intent

- Author Unknown
Communication

• http://youtu.be/87HsUukEj4Y

Communications

• Foundations of Effective Communication
  – Complete
  – Brief
  – Clear/ Specific
  – Timely

• Challenges to Effective Communication
  – Distractions
  – Workload
  – Physical proximity
  – Language barriers
  – Personalities or conflict
Communication

- Teamwork Communication Strategies
  - Call Outs
  - Closed loop Communication
  - Handoff
  - Using SBAR (Situation-Background-Assessment-Recommendation)

Communication

- **Situation:** Mrs. Smith’s FHR is a category II due to a baseline of 190 with minimal variability for ~ 25 min. Her temperature is 38.1.
- **Background:** She is a G2 P1001 at 37 weeks who came in with SROM. Her membranes have now been ruptured for ~ 10 hours. Previous FHR baseline has been ranging from 140-150 with mod-min variability.

Communication

- **Assessment:** I think she may be developing chorioamnionitis.
- **Request:** Would you like me to collect cultures, order antibiotics and an antipyretic?
Communication

Physician Report

- Patient's name
- GPTAL
- EDC
- Patient age
- Reason for coming to hospital
- Vital signs
- FHR
- Uterine Activity
- SVE
- Is the patient in labor? Or what do you want the doctor to do?

Communication

Should conflict occur try to resolve 1 to 1 with individual

If that can’t happen then:

- Chain of Command
  - Know your hospital’s chain of command policy
  - Initiate only when you cannot resolve a safety issue (ie. a patient may be harmed)
  - Should be a rare occurrence
  - Document all attempts at conflict resolution

Responsibility

- Act within scope of practice
- Seek support and guidance
- Work within organizational standards
- Duty of care to the woman and employer
- Maintain knowledge and skills
- Be prepared to explain ones practices
Responsibility

• Standards set by:
  – Nurse Practice Act: Established to protect the public by regulating nursing practice.
  – Regulating bodies: TJC, State Health Dept., Centers for Medicare/Medicaid Services, CDC, OSHA, FDA,
  – Professional organizations: AWHONN, AORN,
  – Policies & Procedures: Your institution’s guidelines

Know who to go to if you are not sure about a specific nursing practice.

Tests of Fetal Well Being

• Scalp stimulation: do SVE, rub finger on fetus’ scalp. If response is an acceleration, normal fetal blood pH. If no accelerations occur, provide in utero resuscitative measures. Evaluate further.
• Auditory stimulation: hold acoustic stimulator over fetal back for 3-5 seconds to stimulate fetus. If response is acceleration- normal fetal blood pH.

Tests of Fetal Well Being

• Non-stress test: evaluates fetal well-being by assessing fetal heart rate accelerations that occur with fetal activity, in a normally resting woman.
• Reactive NST: occurrence of two FHR accels in a 20 min observation period, is reassuring of fetal well being.
• Nonreactive NST: fewer than 2 accels lasting 15 seconds for 15 beats above baseline after 32 weeks gestation in 20 min observation period. Can use acoustic stimulator or manual abdominal stimulation of mother to stimulate the fetus. If get a FHR accel, the NST is reactive, if no accel it is nonreactive. A nonreactive NST is a warning sign and fetus needs further evaluation.
Tests of Fetal Well Being

• Contraction stress test: FHR is monitored while contractions are induced with oxytocin or nipple stimulation. Self-stimulation of patient’s nipples by gentle massage or warm packs for two minutes each side (not both together) may induce contractions. Or may use oxytocin similar to induction just until contractions occur.
• Negative CST: at least 3 contractions in 10 minutes and no late decels in FHR indicates adequate uteroplacental function at time of test.
• Abnormal CST: repetitive late decels in FHR with contractions. This indicates inadequate uteroplacental function at time of test and fetus needs to be delivered.
• Inconclusive CST: occurs if 3 contractions did not occur within 10 minutes, if there were late decels but did not occur with consecutive contractions or the late decels occurred with tachysystole. Should be repeated within 24 hours in this case.

Tests of Fetal Well Being

• Biophysical Profile: assesses fetal well-being by evaluating five different factors, 4 of which are done by ultrasound:
  • Non-stress test
  • Fetal movement
  • Fetal muscle tone
  • Fetal respirations
  • Amniotic fluid volume
Each component gets a score of 0 or 2.
Score of 8 or 10: reassuring, fetal well-being
Score of 6: repeat the test in 4-24 hours, fetus is at-risk
Score of 4 or lower: fetus is sick and should be delivered immediately.

Questions?

Remember to complete the evaluation of this course at http://opqic.org/opnf/bootcamp/evaluations/

References:
2008 NICHD report on Electronic Fetal Monitoring