Physiology of FHR Baseline, Variability, Accelerations & Decelerations

EFM for the Experienced Nurse

FHR Baseline

Physiology of FHR Baseline

- Intrinsic cardiac pacemakers – SA Node and AV node
- Conduction pathways – AV & SA nodes
- Autonomic nervous system
  - Parasympathetic – reduce the baseline
  - Sympathetic – increase the baseline
- Humoral factors
  - Catecholamines – increase the baseline
- Extrinsic Factors
  - Medications
- Local Factors
  - Calcium
  - Potassium

Physiology of FHR Baseline Continued

- Sympathetic innervation and plasma catecholamines ↑ the fetal heart rate baseline and cause vasoconstriction
- Parasympathetic innervation ↓ the fetal heart rate baseline

Physiology of FHR Baseline Continued

- Autonomic nervous system regulates the FHR in response to:
    1. Fluctuations in PO2 and PCO2
    2. Blood pressure which is detected by baroreceptors and chemoreceptors located in the aortic arch and the carotid arteries
        - Miller, Miller, & Cypher (2022) p. 112
        - Miller, Miller, & Cypher (2027) p. 106

Potential Causes of Fetal al Tachycardia

- Maternal fever, infection
- Medications
  - Sympathomimetics
  - Parasympatholytics
  - Caffeine
  - Theophylline
  - Cocaine
  - Methamphetamine
- Fetal anemia
- Maternal hyperthyroidism
- Arrhythmia
  - Sinus Tachycardia
  - Supraventricular tachycardia
  - Atrial fibrillation
  - Atrial flutter
  - Ventricular arrhythmia
- Metabolic acidemia

- Miller, Miller & Cypher (2027) p. 108

Conditions Associated with Fetal Bradycardia

- Medications
  - Sympatholytics
- Cardiac conduction abnormalities
- Heart block
- Heterotaxy Syndrome
- Structural cardiac defects
- Viral infections – CMV
- Sjogren's antibodies
- Fetal heart failure
- Maternal hypoglycemia
- Maternal hypothermia
- Interruption of fetal oxygenation

Miller, Miller & Cypher (2027) p. 110

FHR Variability

- *"Fluctuations in the fetal heart rate baseline which are both **irregular** in amplitude and frequency"*

Miller, Miller & Cypher (2027) p. 113

Physiology of FHR Variability

- Many factors interact and regulate the FHR variability
- Regulated by intrinsic cardiac pacemakers – SA Node and AV node
- Conduction pathways
- Autonomic nervous system
  - Parasympathetic – reduce the baseline
  - Sympathetic – increase the baseline and cause vasoconstriction

Physiology of FHR Variability Continued

- Humoral factors
  - Catecholamines
- Extrinsic Factors
  - Medications
- Local Factors
  - Calcium
  - Potassium

Physiology of FHR Variability Continued

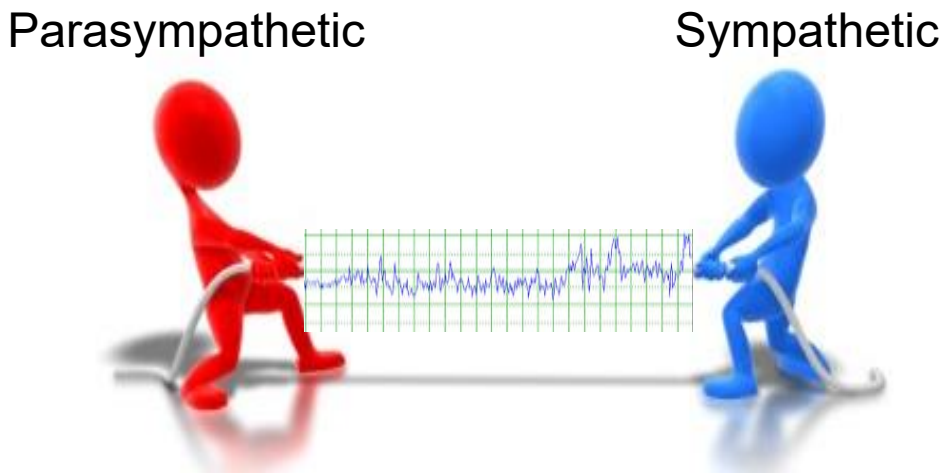
1. Fluctuation in PO_2 and PCO_2 and blood pressure are detected by chemoreceptors and baroreceptors in aortic arch and carotid arteries
2. These signals are processed in the medullary vasomotor center, possibly with regulatory input from higher centers in the hypothalamus and cerebral cortex
3. Sympathetic and parasympathetic signals from the medullary vasomotor center modulate the FHR in response to moment to moment:
   - Changes in fetal PO_2, PCO_2 and BP

Purpose of this Process is to:

- Try and optimize fetal cardiac output to maximize distribution of oxygen to the fetal tissue
    - Miller, Miller & Cypher (2027) p. 110

Physiology of FHR Variability

The Push and Pull of the autonomic nervous system (parasympathetic and sympathetic) results in FHR variability



Keep In Mind……

- “Moderate variability reliably predicts the absence of fetal metabolic acidemia at the time it is observed”
  - However, the opposite is not true. Meaning, absent or minimal variability cannot confirm fetal metabolic acidemia

Conditions associated with minimal-absent FHR variability and the absence of accelerations:

- Sleep cycle
- Fetal tachycardia
- Medications
  - Narcotics, Barbiturates, Phenothiazines, Tranquilizers, General anesthesia, Atropine
- Prematurity
- Congenital anomalies
- Fetal anemia
- Fetal cardiac arrhythmias
- Infection
- Preexisting neurologic injury
- Fetal metabolic acidosis

- Miller, Miller & Cypher (2027) p. 112

Variability Continued

- NOTE: most studies in the literature do not differentiate between minimal and absent variability. “In the setting of persistent minimal variability without the presence of accelerations, the fetal heart rate tracing alone, cannot reliably exclude metabolic acidemia”.
    - Miller, Miller & Cypher (2027) p. 112

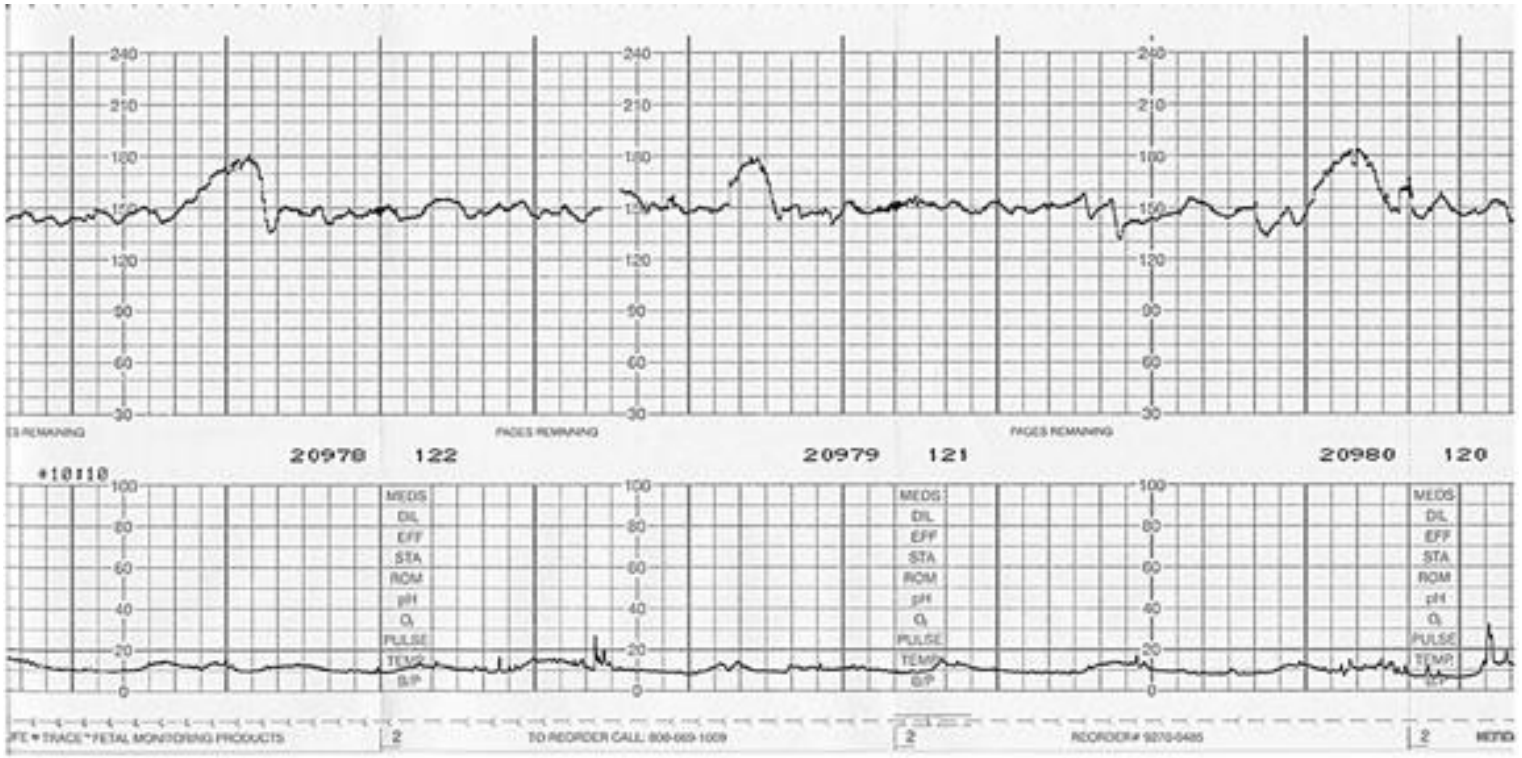
Marked Variability

- Significance of marked variability is unclear
- May be plausible that marked variability reflects autonomic perturbation in the setting of early hypoxemia
  - Science is limited
    - Miller, Miller & Cypher (2027) p. 116

Sinusoidal Pattern

- Defined as smooth, wave like undulating pattern in the FHR baseline with a cycle of 3 to 5 beats per minute that last 20 minute or greater.
- Not included in the definition of FHR variability
- Exact pathophysiology is unknown but has been associated with severe fetal anemia, chorioamnionitis, fetal sepsis and the administration of narcotic analgesia.
- “Fluctuations in the fetal heart rate baseline which are both **regular** in amplitude and frequency”
    - Miller, Miller & Cypher (2027) p. 116-117

FHR Accelerations



Physiology of FHR Accelerations

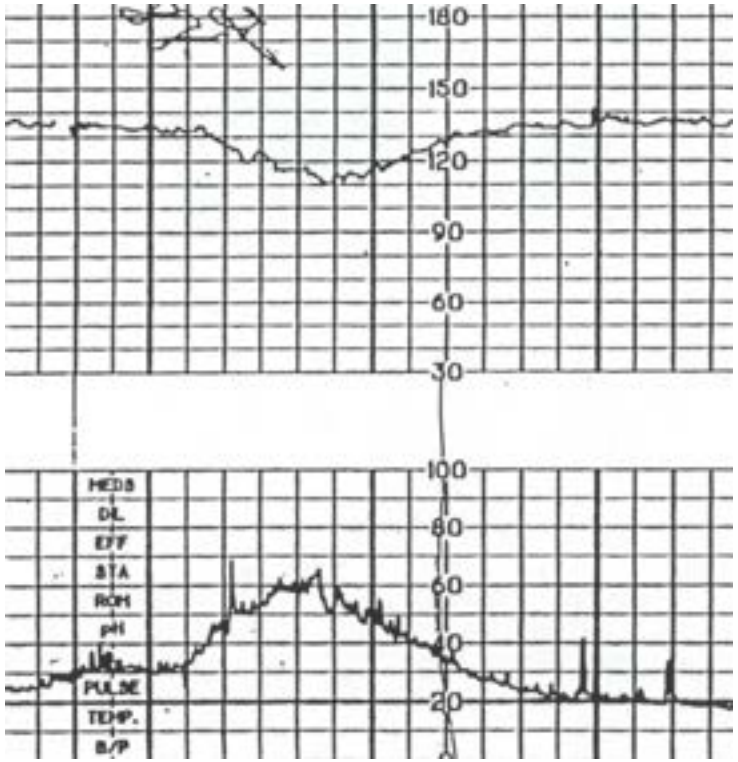
1. Occurs in association with fetal movement
2. Possibly as a result of stimulation of peripheral proprioceptors
3. Increased catecholamine release, and autonomic stimulation of the heart
4. Transient compression of the umbilical vein which results in decreased fetal venous return and a reflex rise in fetal heart rate
    - In the absence of spontaneous accelerations, fetal scalp stimulation and or vibroacoustic stimulation during the fetal baseline may elicit an acceleration
        - Miller, Miller & Cypher (2027) pgs. 117-118 & 123

Similar to variability:

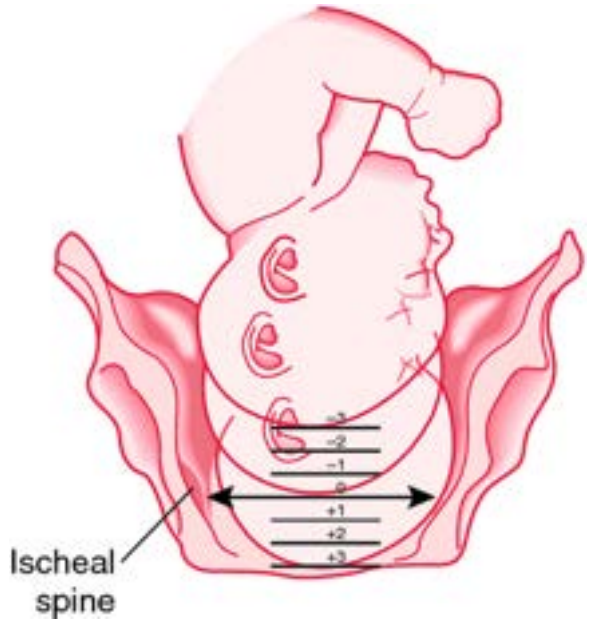
- “Accelerations reliably predict the absence of fetal metabolic acidemia at the time it is observed”
  - However, the lack of accelerations cannot confirm fetal metabolic acidemia

Decelerations

Physiology of Early Decelerations



Early Deceleration Interpretation



http://medical-dictionary.thefreedictionary.com/station

During a contraction

1. Transient compression to the fetal head
2. Altered intracranial pressure and or cerebral blood flow
3. Reflex parasympathetic outflow – [**Vagal Reflex**] Gradual slowing of the fetal heart rate results in early deceleration
4. When compression is relieved, the autonomic reflex subsides, and the fetal heart rate returns to baseline

Same stuff different language

- Thought to represent ***a vagal response*** to a cerebral redistribution of blood flow caused by compression of the fetal head. Early decelerations are not associated with interruption in fetal oxygenation and are thought to be benign.
- When the contraction occurs, the fetal head is subjected to pressure which stimulates the Vagus nerve. The heart rate begins to decrease at the onset of the contraction when the head compression begins and returns to the baseline at the end of the contraction when the head is no longer compressed. Hence giving a “mirror” effect of the deceleration in relation to the contraction.
- Early decelerations are not associated with fetal hypoxia, acidosis or low Apgar Scores
  - » AWHONN (2021) pg. 130

Be Advised or Warned…..

- Theory regarding head compression
  - Based upon hypothesis – extracranial pressure during contractions or maternal pushing efforts can result in neurologic injury well after the neonatal period
  - CCIE – Cranial Compression Ischemic Encephalopathy
  - Theory has no valid scientific base
    - Miller , L. (2017) pg. 297

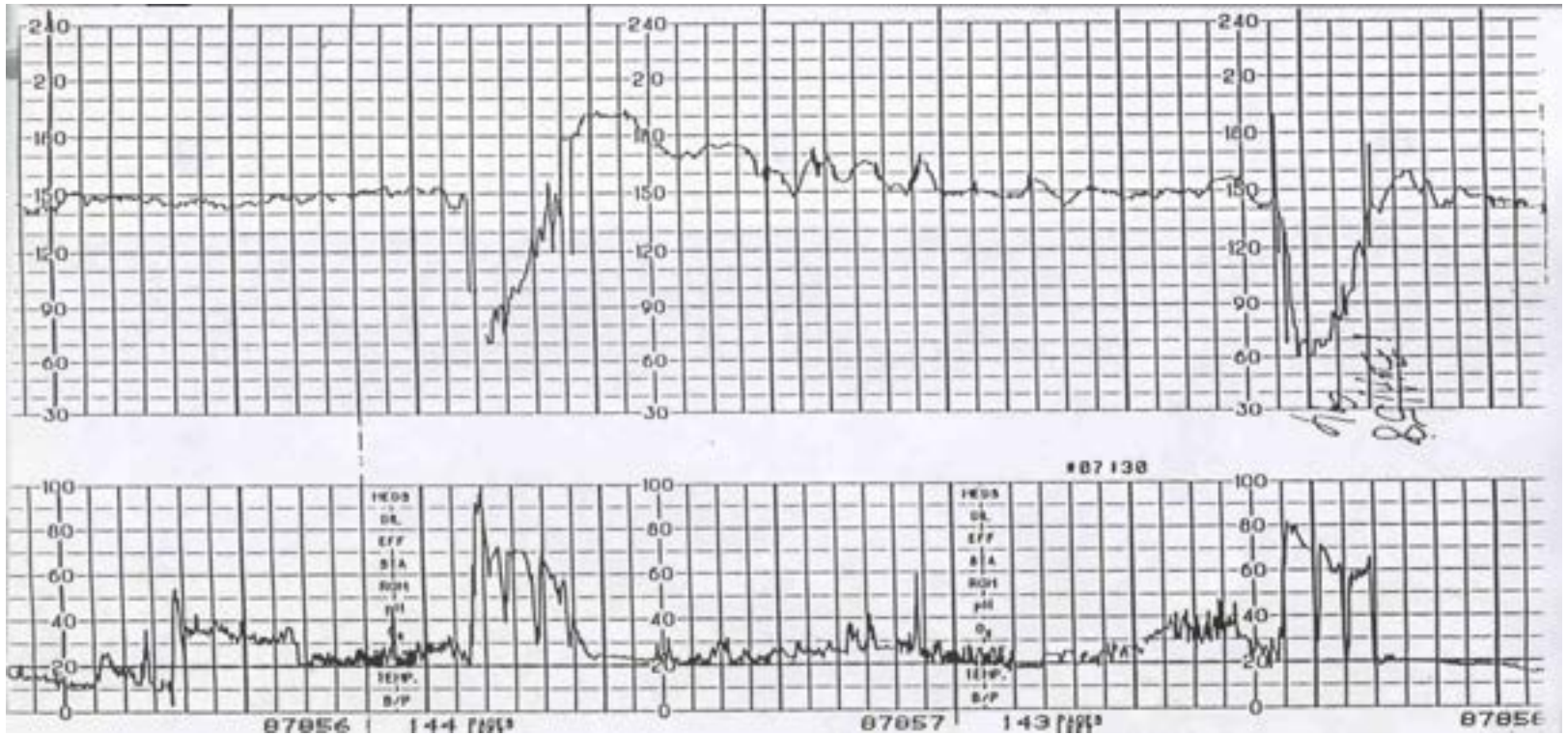
Resources to Debunk the Theory

- Risk management, providers and nurses are aware of lack of scientific proof
- All staff should be able to discuss normal physiology of labor and articulate uterine activity during the different phases and stages of labor – uterine activity related to fetal acid base balance
- Multidisciplinary fetal monitoring education should include mechanisms of various decelerations types and the oxygen pathway – staff should be able to explain this in simple language

Resources to Debunk the Theory – cont.

- 2nd stage pushing efforts should be well documented and open glottis pushing should be encouraged
- If litigation occurs – the defense should retain experts who are familiar with the theory and the evidence against the theory
  - Heyborne KD a systematic review of intrapartum fetal head compression: what is the impact on the fetal brain? Am J. Perinatol Rep. 2017,7:e79-e85
  - https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5406232/
    - Miller (2017) pg. 298

Variable Decelerations

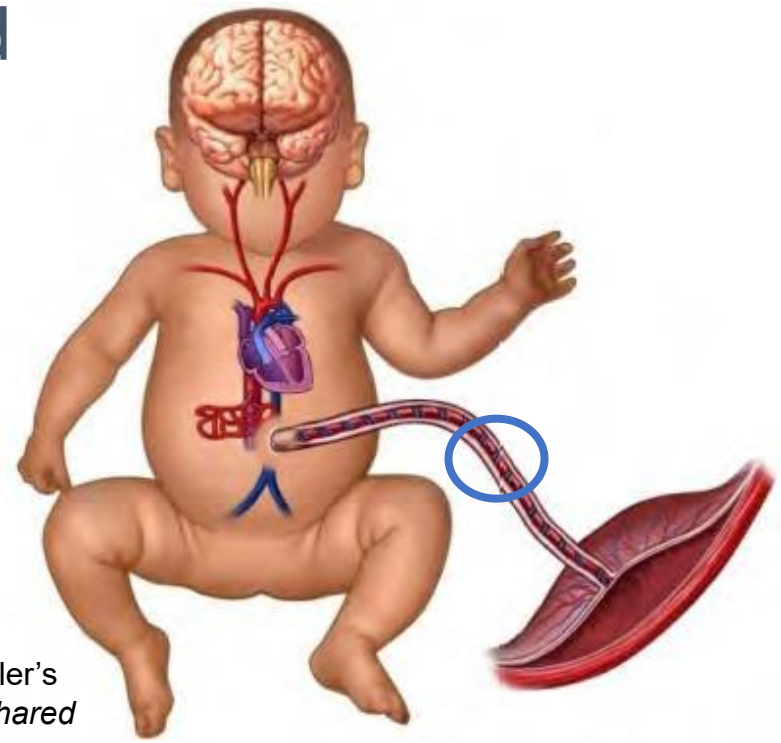


Interpretation of Variable Decelerations

- Reflects a fetal autonomic response to transient mechanical umbilical cord compression
- A variable deceleration alone, is a transient occurrence.
  - However, recurrent variable decelerations may lead to fetal hypoxemia, hypoxia, metabolic acidosis and metabolic acidemia resulting in a rise in baseline, absent to minimal variability and absent accelerations
    - Miller, Miller, and Cypher (2027, pp. 123-125)

Variable Decelerations

The complete occlusion of the umbilical cord causes the blood pressure to RISE



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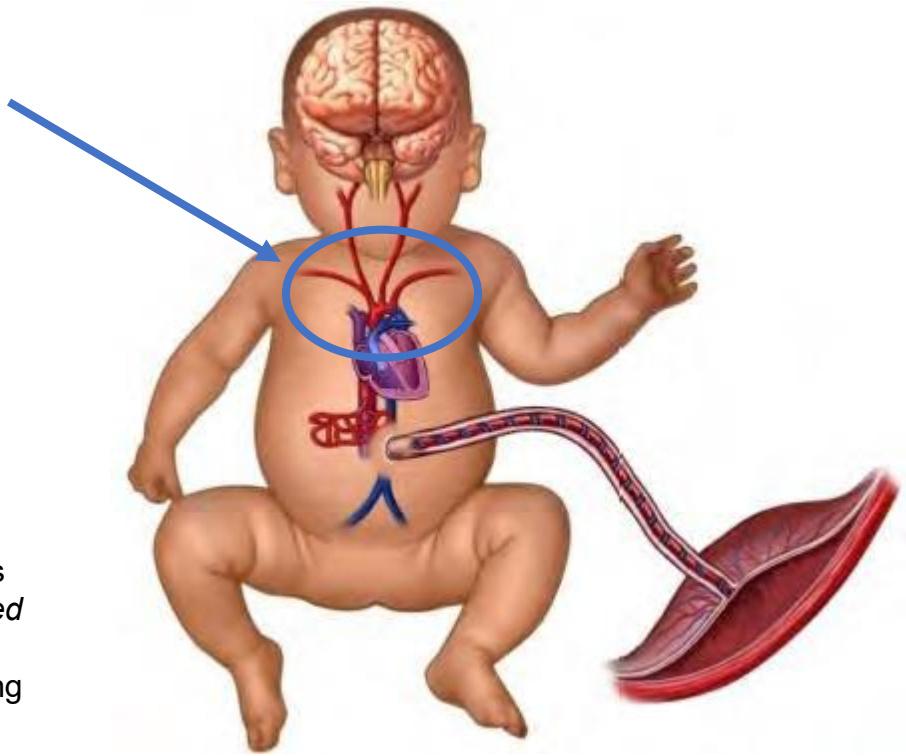
Variable Decelerations Cont.

Rising blood pressure is detected by baroreceptors



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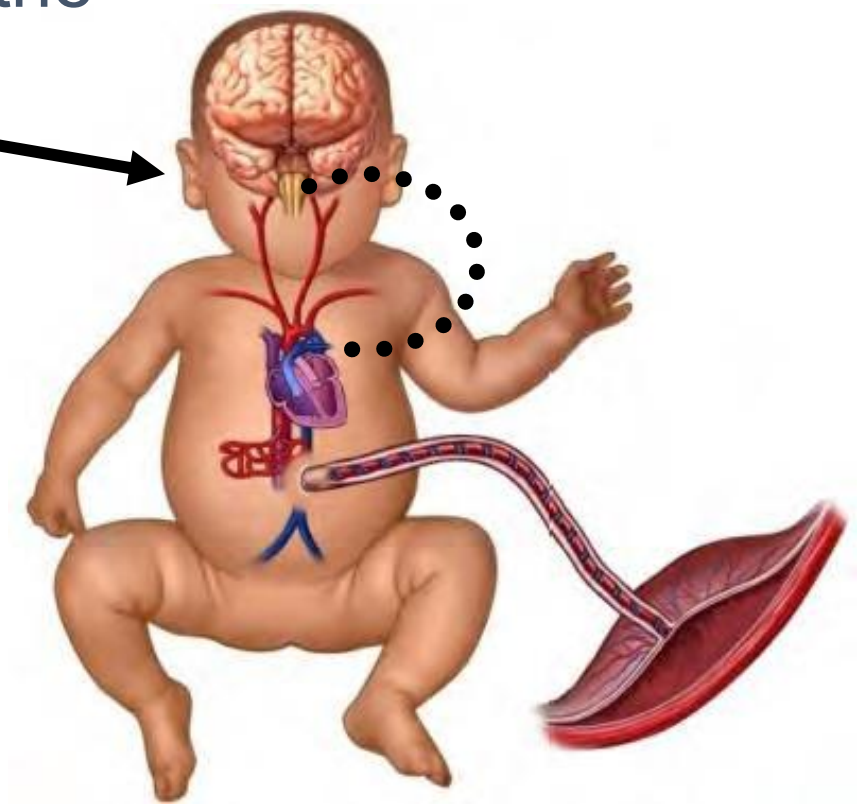
Variable Decelerations Cont.

Baroreceptors signal the brain stem



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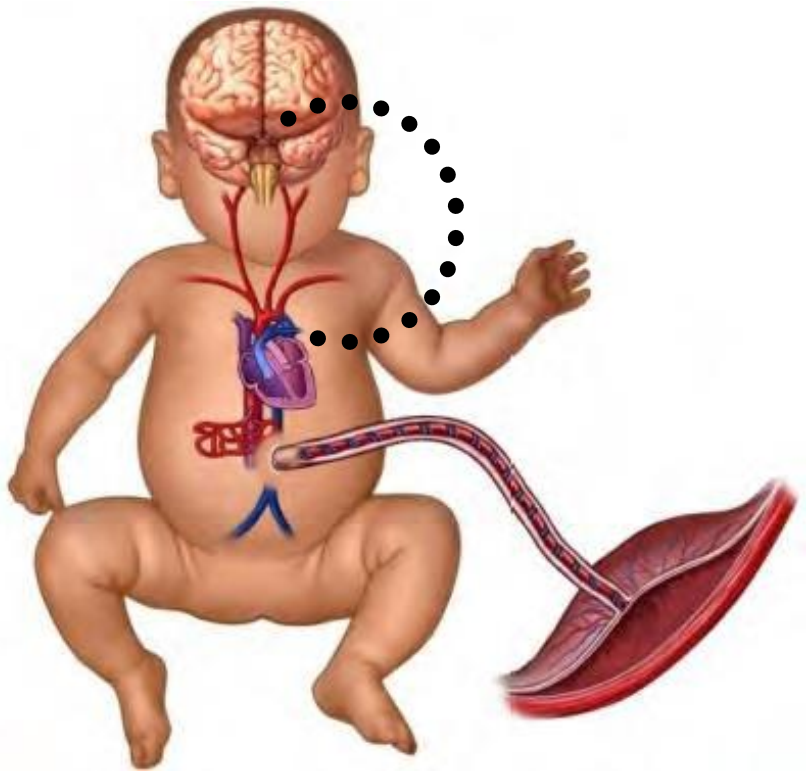
Variable Decelerations Cont.

Parasympathetic (vagal) outflow slows the FHR to reduce cardiac output and lowers blood pressure



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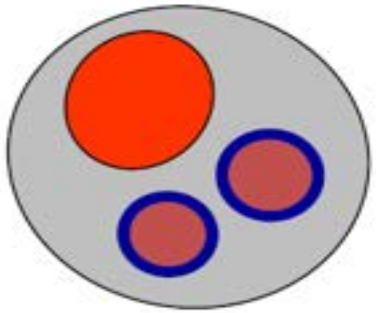
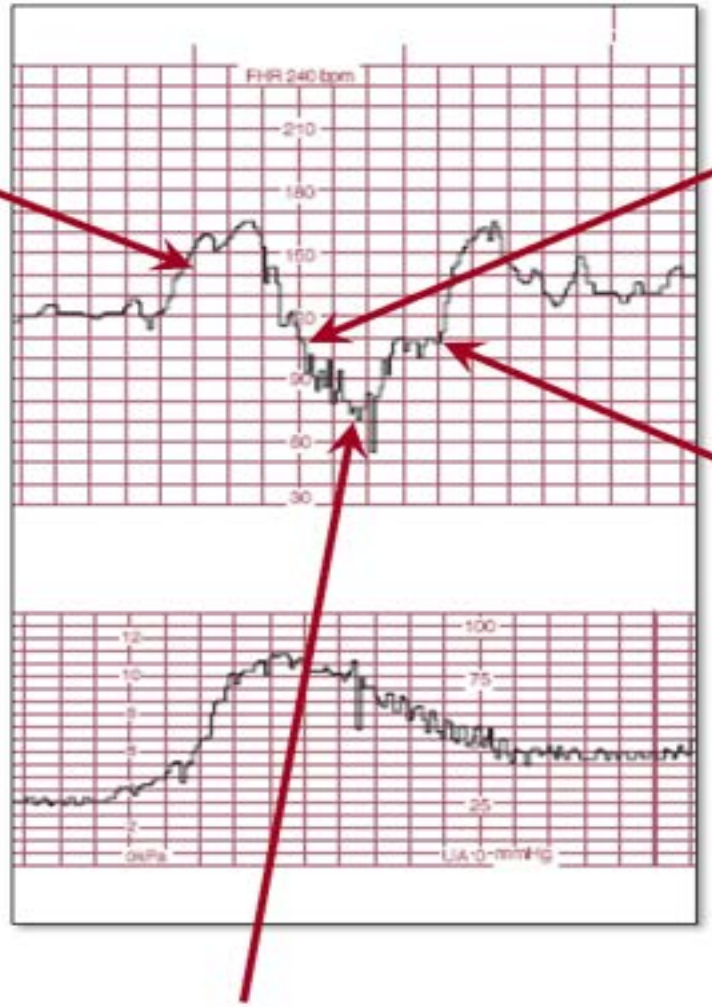
VARIABLE DECELERATION

VENOUS COMPRESSION
Decreased venous return
Relative hypovolemia
Reflex increase in FHR





ARTERIAL COMPRESSION
Increased SVR, elevated BP
Baroreceptor stimulation
Vagal outflow

Reverse

Junctional / Idioventricular Rate

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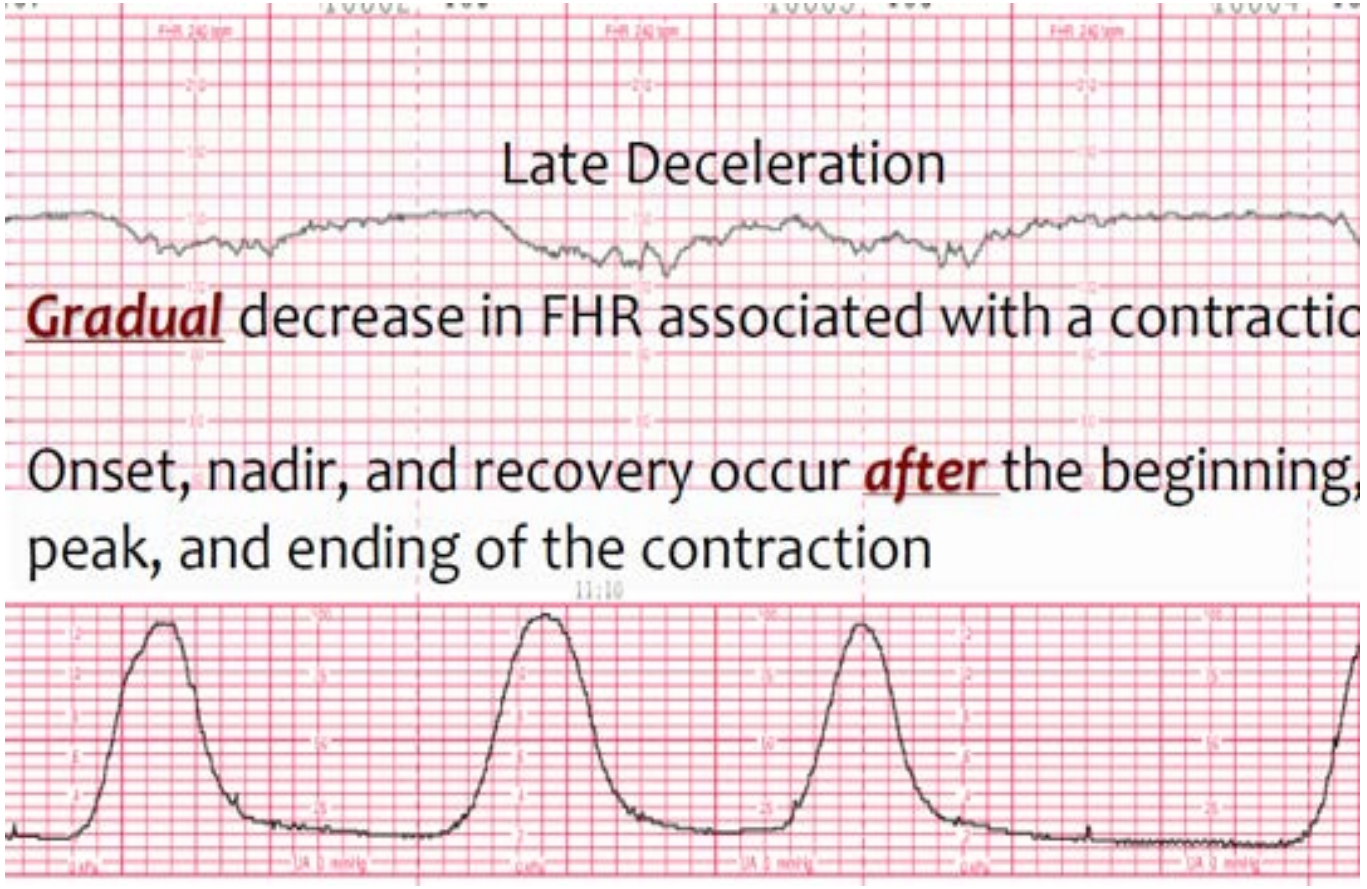
Physiological Mechanisms of Variable Decelerations

- Umbilical cord compression
- Initial compression of the thin-walled **umbilical vein**
- Transient decreased fetal venous return
- Transient reduction in fetal cardiac output and blood pressure which stimulates the **Baroreceptors** to increase the FHR due to a decrease in cardiac output and **B**lood Pressure
- **Umbilical arteries** are compression
- Abrupt rise in fetal peripheral resistance and BP
- **Baroreceptors** are stimulated again due to the rise in BP which stimulates a parasympathetic response
- **Abrupt** slowing of the fetal heart rate resulting in the variable deceleration

Physiological Mechanisms of Variable Decelerations Continued

- Once the umbilical cord compression is relieved, this process occurs in reverse
    - Miller, Miller & Cypher (2027) pgs. 124-125

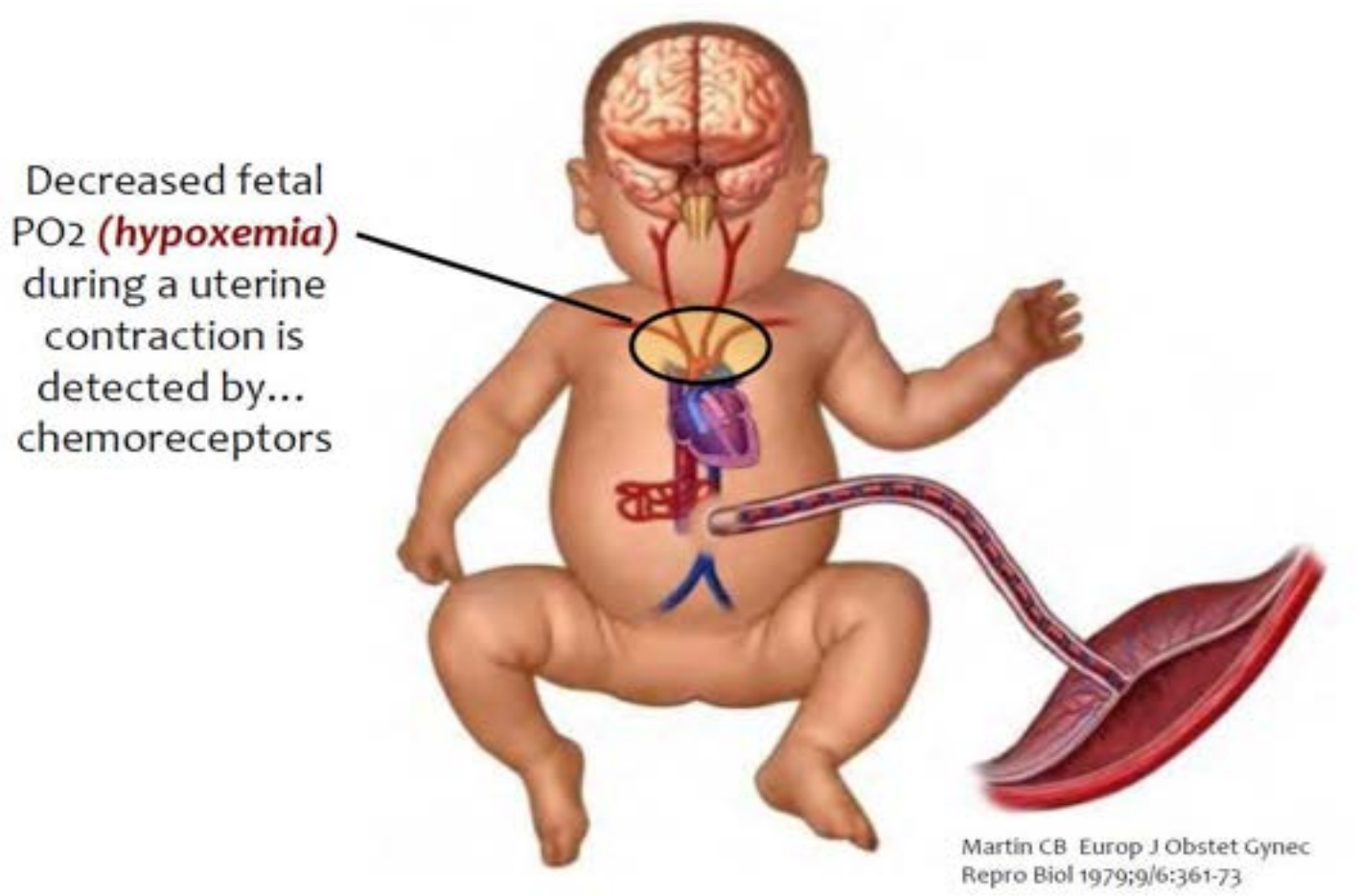
Late Decelerations



Interpretation of Late Decelerations

- “A late deceleration reflects transient interruption of oxygen transfer from the environment to the fetus during a uterine contraction, resulting in transient fetal hypoxemia”
  - Miller, Miller, & Cypher (2027) pgs. 120-122

Late Decelerations



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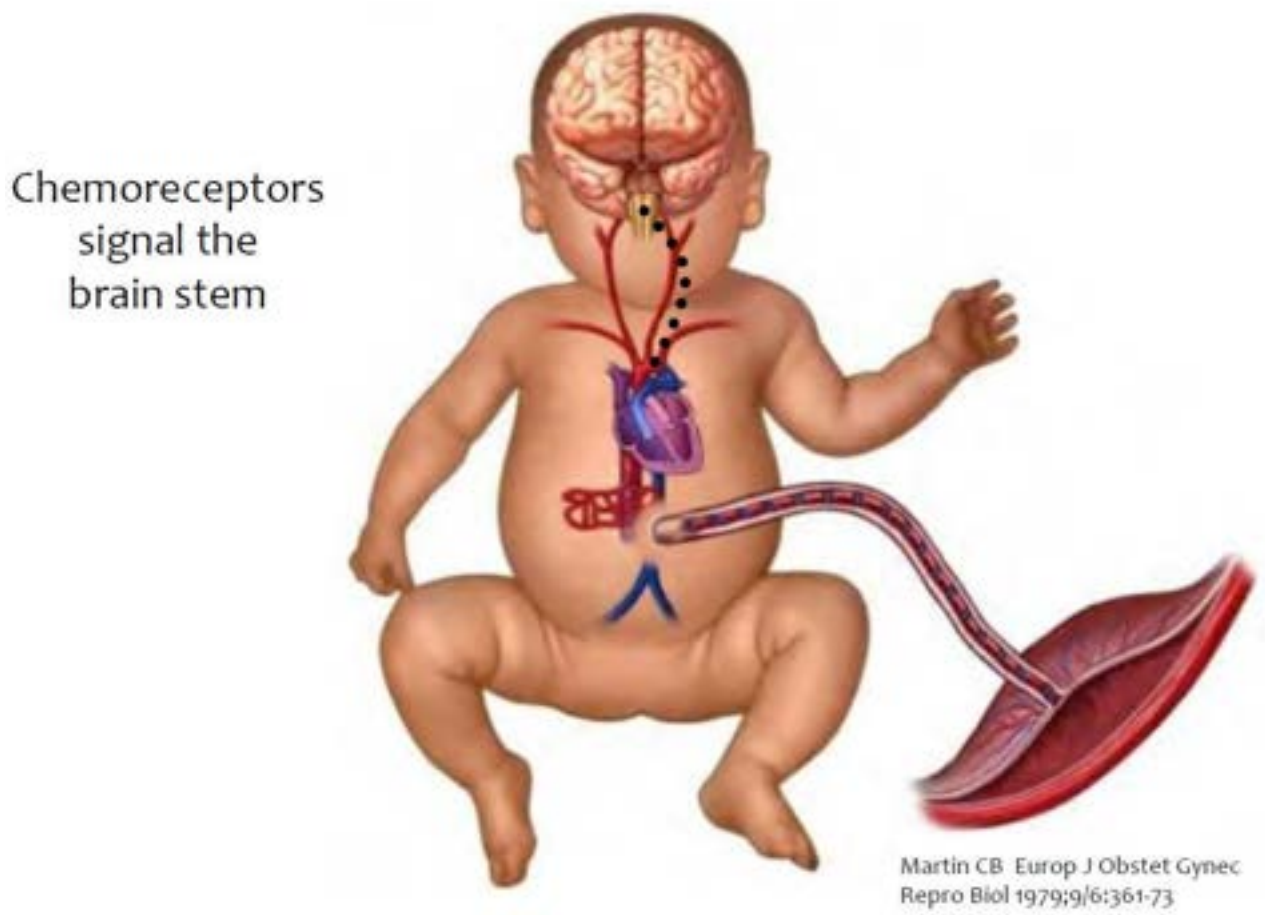
Late Decelerations Cont.



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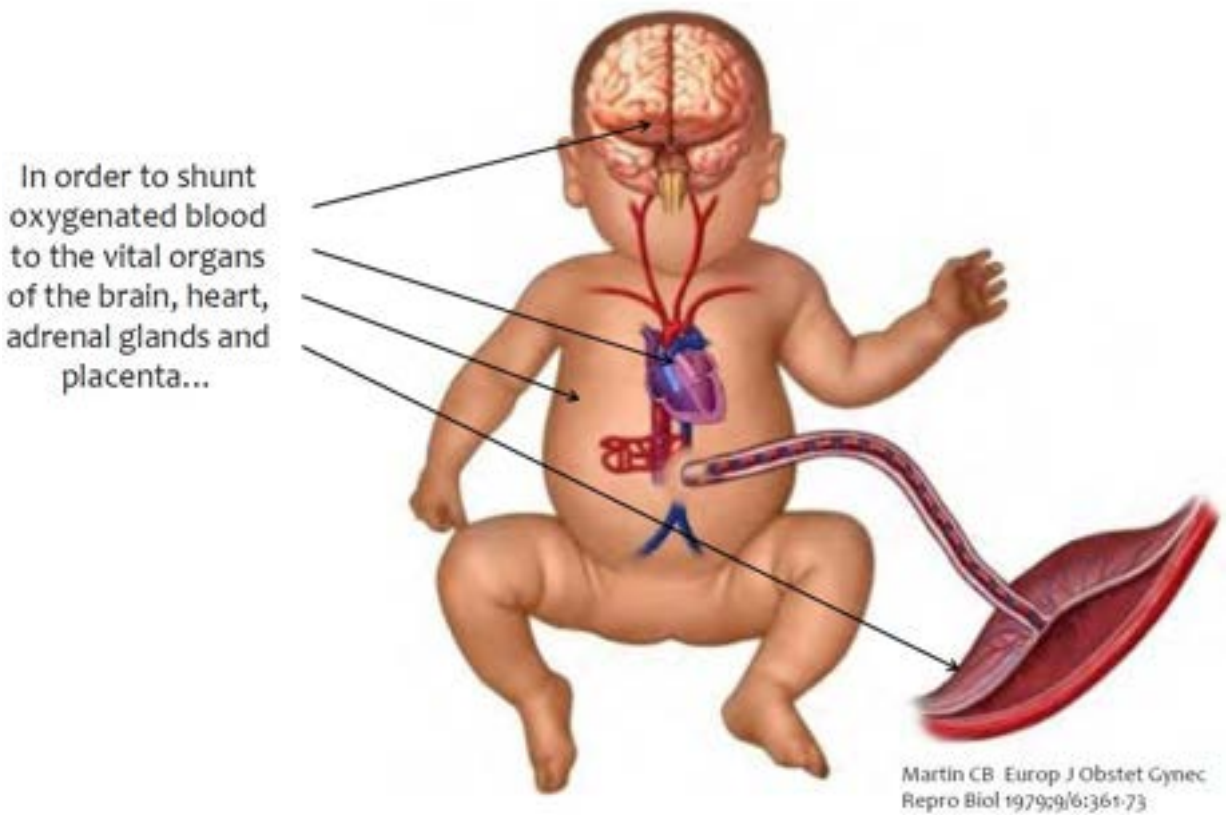
Late Decelerations Cont.



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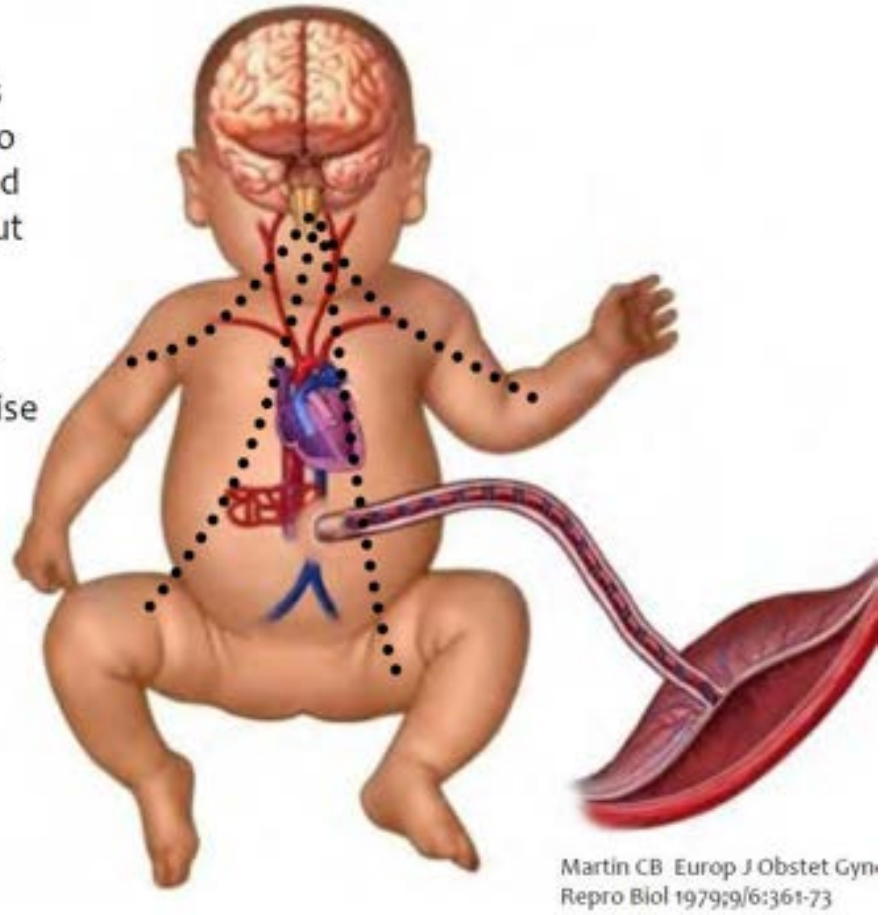
Late Decelerations Cont.

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

Peripheral vasoconstriction causes the blood pressure to rise



Martin CB Europ J Obstet Gyn
Repro Biol 1979;9/6:361-73

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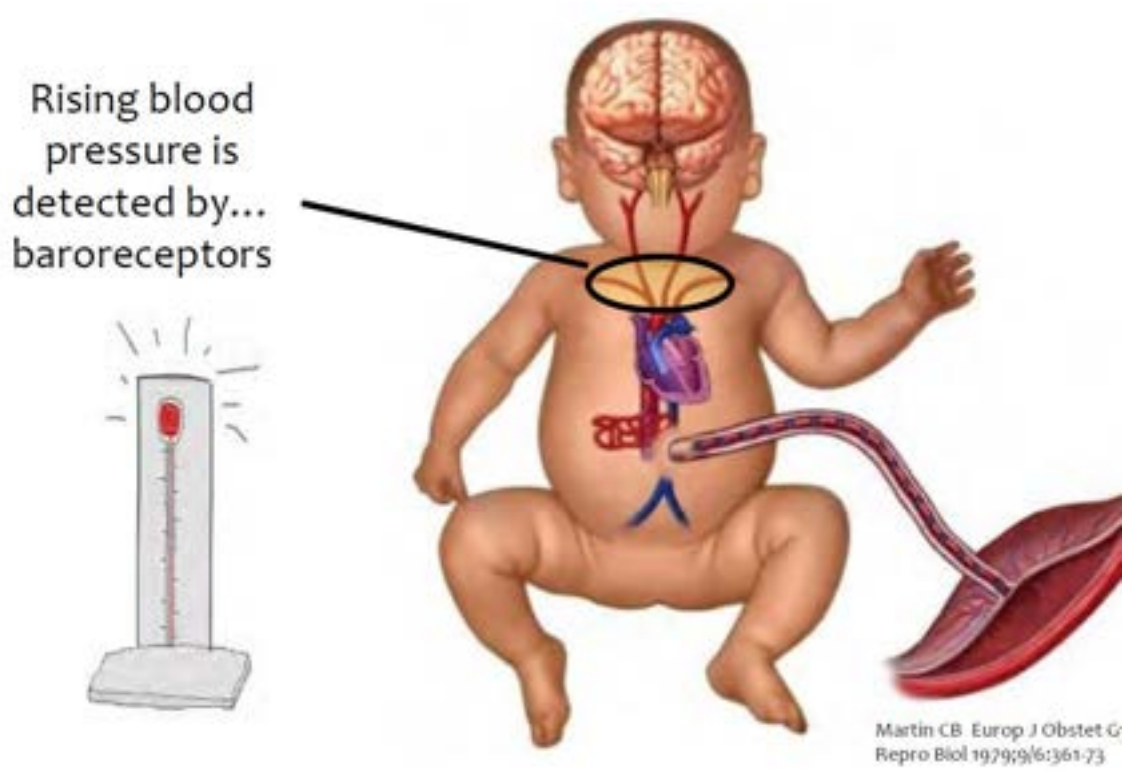
Late Decelerations Cont.



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Late Decelerations Cont.

Baroreceptors signal the brain stem

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Late Decelerations Cont.

Parasympathetic (vagal) outflow slows the FHR to reduce cardiac output and lower blood pressure

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Physiology of Late Decelerations Continued

- Reflex response to hypoxemia during a contraction:
    - Can compress the blood vessels which transverse the uterine wall and decrease maternal perfusion of the intervillous space of the placenta
    - This decrease results in decreased diffusion of oxygen into the fetal capillary blood in the chorionic villi which leads to a decrease in fetal PO2
- When the fetal PO2 is < 15-25mmHg
    - Chemoreceptors detect the change and signal the medullary vasomotor centers to initiate a protective reflex response

Physiology of Late Decelerations Continued

- Sympathetic outflow results in ***peripheral vasoconstriction***, preferentially ***shunting*** oxygenated blood away from the peripheral tissues and toward central vital organs of the fetus which are
  - ***Brain, Heart, Adrenal Glands***

Physiology of Late Decelerations Continued

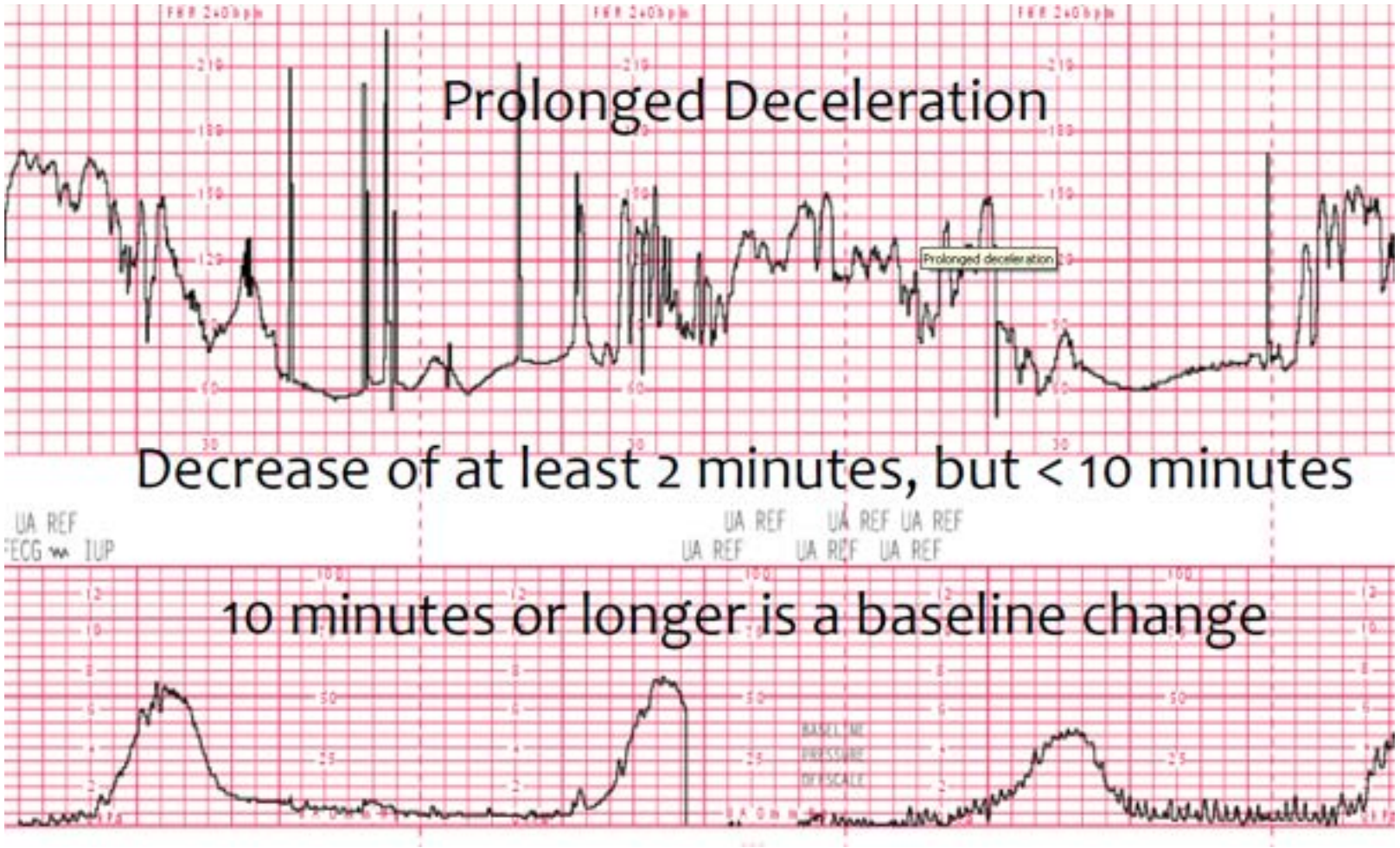
- The increase in fetal peripheral resistance and blood pressure then causes a Baroreceptor stimulation and a reflex parasympathetic outflow which causes a gradual slowing of the FHR
- And the Late deceleration occurs .
- After the contraction is over the reflex subsides

- Miller, Miller, & Cypher (2027) pg. 122

Physiology of Late Decelerations Continued

- Note: In the presence of fetal metabolic acidemia, transient hypoxemia may result in myocardial hypoxia and a late deceleration secondary to direct myocardial depression
    - Miller, Miller, & Cypher (2027) p. 121

Prolonged Deceleration



Prolonged Decelerations

- If the physiologic mechanisms responsible for late or variable decelerations persist, a deceleration can last long enough to be defined by NICHD as a prolonged deceleration.
  - However, if the prolong decelerations lasts greater than 10 minutes it should not be interpreted as a “benign baseline change”.
- Disruption of oxygen to the fetus at one or more points along the oxygen pathway
- Examples:
  - Lungs – maternal seizure
  - Heart – maternal cardiac arrhythmia
  - Vasculature – maternal hypotension
  - Uterus - tachysystole or uterine rupture
  - Placenta – abruption
  - Cord – prolapse

Miller, Miller & Cypher (2027) pgs. 125-126

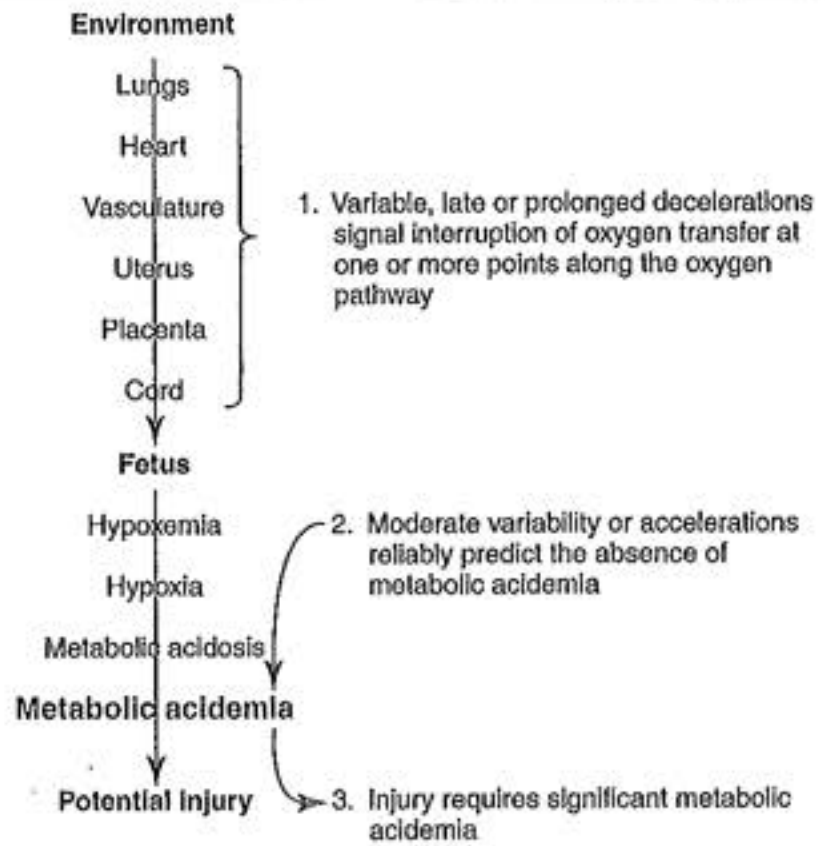
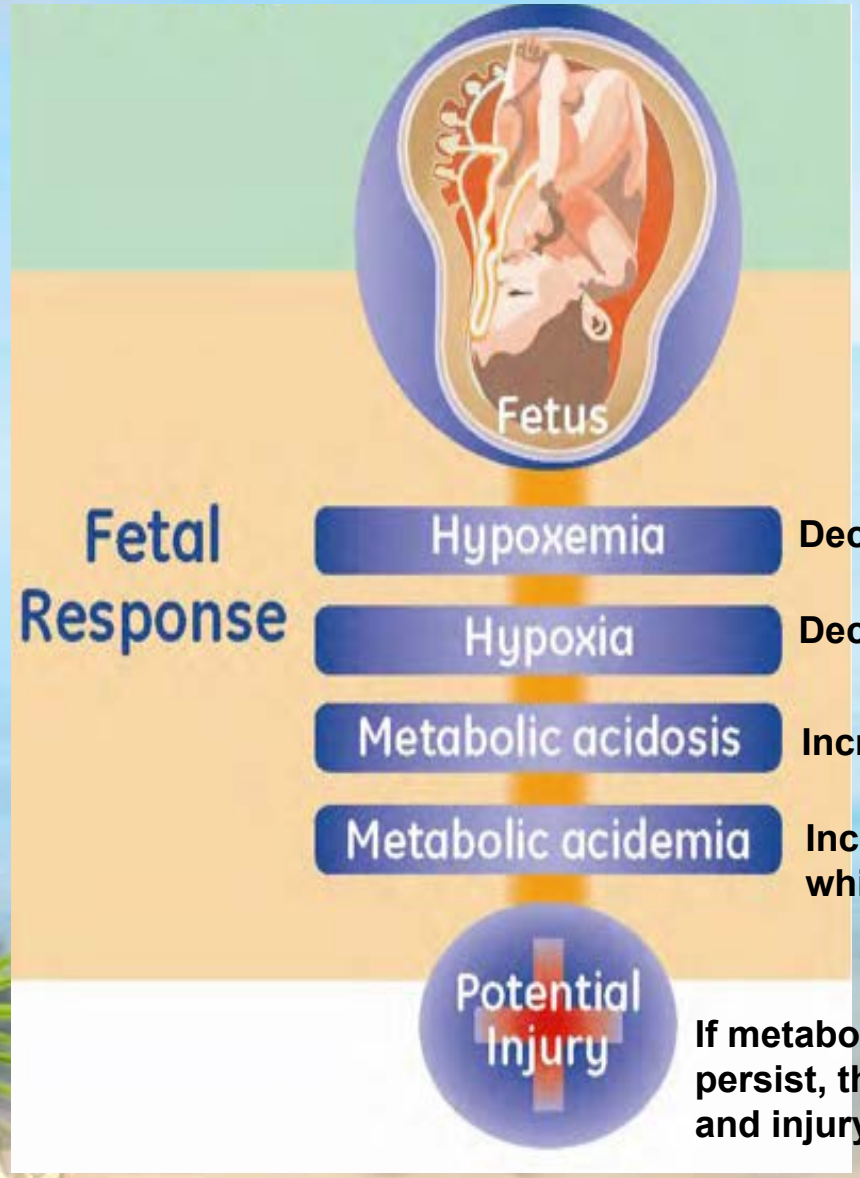


FIGURE 5-26 Three central principles of intrapartum fetal heart rate interpretation.

Miller et al (2013) p. 144

Intrapartum disruption of fetal oxygenation does not cause neurologic injury (cerebral palsy) without first causing *significant** metabolic acidemia

* (umbilical artery pH < 7.0 and base deficit ≥ 12 mmol/L)



Decreased oxygen in the blood

Decreased oxygen in the tissues

Increase in lactic acid in the tissues

Increase in H+ ions in the blood stream which lowers the pH

If metabolic acidemia and fetal hypotension persist, the fetus is at risk for cellular damage and injury

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The End