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NCC EFM EXAM (2022/2023) RATED A+

FHR Reflects: ✓✓Fetal Oxygenation

Obtaining a "category 1" or "Normal" FHR Rules Out: ✓✓Metabolic Acidemia

Extrinsic Fetal Oxygenation Pathway Factors Include: ✓✓1. Maternal oxygenation

2. Uterine Blood Flow

3. Placental Exchange

4. Umbilical Blood Flow

Intrinsic Fetal Oxygenation Pathway Factors Include: ✓✓1. Fetal Circulation

2. Oxygenation of Tissues

3. FHR Regulation

Well-Oxygenated Maternal Blood is Dependent Upon 2 Main Things: Also What Conditions

Could Hinder Each? ✓✓1. Adequate Hgb Concentration & Saturation (O2 Carrying Capacity)

Problem could arise with severe maternal anemia

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Sample EFM Questions | Verified EFM Answers

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NCC Certified - Electronic Fetal Monitoring Sample Questions (Q20-Q25):

NEW QUESTION # 20

The tracing shown is a:



- A. Category II
- B. Category I
- C. Category III

Answer: A

Explanation:

Comprehensive and Detailed Explanation From Exact Extract-Based NCC C-EFM References (No URLs):

Interpretation of fetal heart rate (FHR) tracings in the NCC C-EFM exam follows the standardized NICHD three-tier classification, which is fully adopted in NCC's content outline and recommended references such as AWHONN Fetal Heart Monitoring Principles & Practices, Miller's EFM Pocket Guide, Menihan, Simpson's Perinatal Nursing, and Creasy & Resnik.

Baseline:

The tracing demonstrates an FHR baseline around 145-150 bpm, which falls within the normal range of 110-160 bpm. NCC references define baseline as the mean FHR rounded to increments of 5 bpm over a 10-minute window.

Variability:

The strip shows minimal variability, with amplitude fluctuations approximately 0-2 bpm.

According to NCC-aligned definitions:

* Moderate variability: 6-25 bpm

* Minimal variability: 1-5 bpm

* Absent variability: undetectable amplitude

This tracing shows minimal variability, not moderate, so it cannot be Category I.

Accelerations:

No accelerations are present. Lack of accelerations alone does not classify the tracing as Category III.

Decelerations:

There are no recurrent late decelerations, no recurrent variable decelerations, and no prolonged decelerations. Without these, and with minimal variability, the tracing does not meet Category III criteria.

Category III criteria (per NICHD/NCC):

Must include at least one of the following:

* Absent variability with recurrent late decelerations

* Absent variability with recurrent variable decelerations

* Absent variability with bradycardia

* Sinusoidal pattern

None of these are present.

Category II criteria (per NICHD/NCC):

Category II includes tracings that are not Category I or III.

Examples specifically listed include:

* Minimal variability

* Absent accelerations after fetal stimulation

* Tachycardia

- * Bradycardia without absent variability
- * Variable or late decelerations occurring intermittently

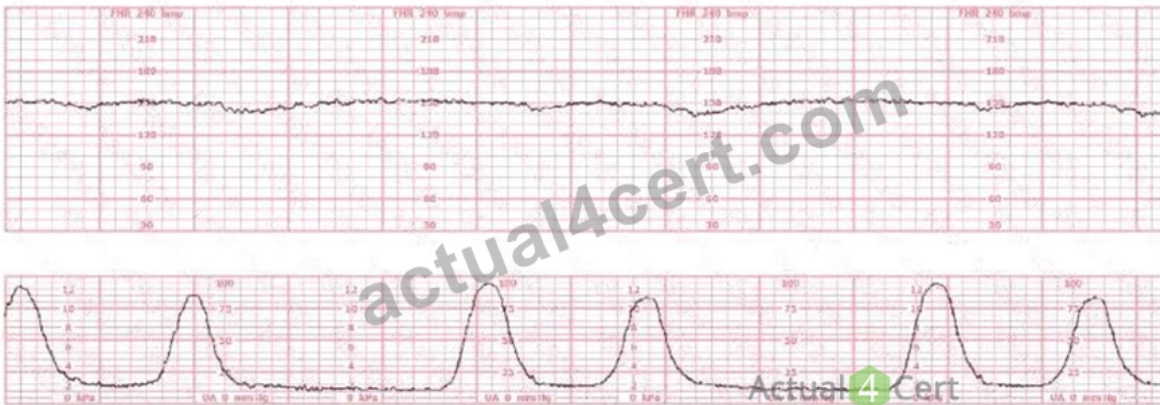
Because this tracing shows minimal variability, a normal baseline, no accelerations, and no recurrent decelerations, it fits squarely into Category II.

Therefore, the correct classification is Category II.

References: NCC C-EFM Candidate Guide and Content Outline (2025); AWHONN Fetal Heart Monitoring Principles & Practices; Miller's Fetal Monitoring Pocket Guide; Menihan Electronic Fetal Monitoring; Simpson & Creehan Perinatal Nursing; Creasy & Resnik Maternal-Fetal Medicine; NICHD Three-Tier FHR Interpretation System.

NEW QUESTION # 21

A woman is being induced with oxytocin. The tracing shown is representative of 20 minutes. Based on this tracing, the next step would be to:



- A. Place a spiral electrode
- B. Proceed to operative birth
- C. Discontinue oxytocin

Answer: C

Explanation:

Comprehensive and Detailed Explanation From Exact Extract-Based NCC C-EFM References:

Evaluation of a tracing during oxytocin induction requires analysis of fetal status (baseline, variability, accelerations, decelerations) and uterine activity, with attention to tachysystole and fetal intolerance. NCC, AWHONN, Miller, Menihan, Simpson, and the NICHD guidelines all emphasize that oxytocin must be adjusted based on fetal response and contraction frequency.

Baseline:

The fetal heart rate baseline is approximately 150 bpm, which is within the normal range of 110-160 bpm.

Variability:

The tracing shows minimal variability (approximately 1-4 bpm amplitude). Minimal variability for a sustained period is categorized as a Category II pattern under NCC/NICHD classification.

Accelerations:

No accelerations are present during the 20-minute representative segment.

Decelerations:

There are no recurrent variable, no recurrent late, and no prolonged decelerations.

Uterine Activity:

The tracing shows very frequent contractions—approximately every 1½ to 2 minutes, which meets the NCC definition of tachysystole when averaged over 10 minutes (more than 5 contractions in 10 minutes).

According to NCC and AWHONN standards, when tachysystole is present with minimal variability, oxytocin must be reduced or discontinued even in the absence of late decelerations.

Clinical decision-making (per NCC principles):

NCC emphasizes that management of Category II patterns during induction starts with intrauterine resuscitative measures, including decreasing or stopping oxytocin when uterine activity is excessive or fetal response is suboptimal. Minimal variability with tachysystole requires correction of uterine stimulation before escalating to invasive monitoring or considering operative birth. Option B (place a spiral electrode) is not indicated because the pattern is clearly visible and the priority is correcting uterine overstimulation, not refining the tracing.

Option C (operative birth) is not indicated; there is no Category III pattern or recurrent decelerations.

Option A (discontinue oxytocin) is the correct first-line action according to NCC-aligned guidelines when tachysystole and minimal

variability occur.

References:

NCC C-EFM Candidate Guide (2025); NCC Content Outline; NICHD Three-Tier FHR Interpretation System; AWHONN Fetal Heart Monitoring Principles & Practices; Miller's Fetal Monitoring Pocket Guide; Menihan Electronic Fetal Monitoring; Simpson & Creehan Perinatal Nursing; Creasy & Resnik Maternal-Fetal Medicine.

NEW QUESTION # 22

Interventions undertaken to address fetal tachycardia are targeted at maximizing

- A. uteroplacental perfusion
- B. maternal circulation
- C. sympathetic autonomic tone

Answer: A

Explanation:

Comprehensive and Detailed Explanation From Exact Extract NCC-Recommended Sources Fetal tachycardia is typically caused by maternal fever, dehydration, hypoxia, medications, infection, or fetal stress. AWHONN and Simpson & Creehan emphasize that management focuses on improving oxygen delivery across the placenta, which is governed by uteroplacental perfusion.

Menihan's EFM text states that "interventions for fetal tachycardia must address oxygen transfer by optimizing uteroplacental blood flow," including hydration, reducing uterine activity, maternal repositioning, and treating maternal fever.

Increasing maternal circulation alone is insufficient unless it improves placental blood flow. Enhancing fetal sympathetic tone is not a clinical goal and would worsen tachycardia.

Creasy & Resnik highlight that fetal heart rate abnormalities resolve when uteroplacental perfusion is restored, confirming this as the primary target of intervention.

References:

AWHONN - Fetal Heart Monitoring Principles & Practices
Simpson & Creehan - Perinatal Nursing
Menihan - Electronic Fetal Monitoring
Creasy & Resnik - Maternal-Fetal Medicine
Miller's Pocket Guide

NEW QUESTION # 23

A woman in active labor at 8 cm experiences spontaneous rupture of membranes and acute bright red vaginal bleeding. The uterus is soft and nontender to palpation. The fetal monitor tracing has been normal and now shows tachycardia followed by bradycardia with minimal variability. The maternal blood pressure is 130/76 mm Hg, and the pulse is 86 beats per minute. The most likely cause of these findings is:

- A. Abruptio placenta
- B. Ruptured vasa previa
- C. Placenta previa

Answer: B

Explanation:

Comprehensive and Detailed Explanation From Exact Extract-Based NCC C-EFM References:

When bright red vaginal bleeding occurs at the moment of membrane rupture, accompanied by an acute, severe fetal heart rate deterioration, NCC sources emphasize considering conditions causing fetal hemorrhage rather than maternal instability.

The key features in this scenario:

* Timing: Bleeding occurs immediately with spontaneous rupture of membranes—this is classic for vasa previa rupture, where fetal vessels traverse membranes and are torn when the membranes rupture.

* Bleeding characteristics: Bleeding is acute, bright red, and sudden. In vasa previa, the blood observed vaginally is fetal blood, not maternal blood.

* Uterine exam: The uterus is soft and nontender, which strongly argues against abruptio placenta, where the uterus is typically firm, rigid, or painful.

* Maternal vital signs: Maternal blood pressure and pulse are normal, indicating no maternal hypovolemia. In placental abruption or placenta previa with significant maternal bleeding, maternal vitals are often abnormal. Here, the mother is stable, meaning the blood is not maternal-supporting fetal vessel rupture.

* Fetal heart rate pattern:

* Initial tachycardia, followed by

* Bradycardia with minimal variability. Such a pattern is consistent with acute fetal blood loss, which rapidly leads to fetal hypovolemia and hypoxia.

* Differential based on NCC-aligned physiology:

A). Abruptio placenta - NOT supported

Typically presents with:

* Painful bleeding

* Firm, tender uterus

* Maternal tachycardia

* Uterine irritability None of these are present.

B). Placenta previa - NOT supported

Classically painless bright red bleeding before or early in labor, not triggered by membrane rupture.

Fetal compromise is less sudden unless maternal shock occurs, which is not the case here.

C). Ruptured vasa previa - CORRECT

Defined by:

* Painless, sudden bright red bleeding at ROM

* Normal maternal vital signs

* Rapid fetal deterioration (tachycardia # bradycardia # minimal variability)

* Soft, nontender uterus This fits the scenario exactly.

Therefore, the most likely cause is ruptured vasa previa, a recognized obstetric emergency described across AWHONN, NCC C-EFM references, and maternal-fetal physiology texts such as Menihan and Creasy & Resnik.

References: NCC C-EFM Candidate Guide (2025); NCC Content Outline; AWHONN Fetal Heart Monitoring Principles & Practices; Miller's Fetal Monitoring Pocket Guide; Menihan Electronic Fetal Monitoring; Simpson & Creehan Perinatal Nursing; Creasy & Resnik Maternal-Fetal Medicine.

NEW QUESTION # 24

Tachysystole can have a negative effect on fetal oxygenation during labor by

- A. increasing maternal blood pressure
- B. blocking active transport of oxygen to the fetus
- C. interfering with reperfusion of the intervillous space

Answer: C

Explanation:

Comprehensive and Detailed Explanation From Exact Extract NCC-Recommended Sources NCC-recommended physiology references (AWHONN, Simpson & Creehan, Menihan, Creasy & Resnik) consistently state that the primary mechanism by which tachysystole affects fetal oxygenation is reduced uteroplacental perfusion, specifically through impaired intervillous space reperfusion. During a normal contraction cycle, the fetus receives oxygen between contractions, when the uterus relaxes and maternal blood re-enters the intervillous space. AWHONN's Fetal Heart Monitoring Principles & Practices explains that tachysystole-defined as more than five contractions in 10 minutes averaged over 30 minutes-shortens or eliminates the relaxation phase, preventing adequate placental reoxygenation.

Simpson & Creehan highlight that "tachysystole decreases uteroplacental blood flow and interferes with replenishment of oxygenated maternal blood in the intervillous space." Menihan emphasizes that fetal hypoxemia in tachysystole results from interrupted perfusion, not from altered oxygen transport or maternal hemodynamic changes. Creasy & Resnik confirm that uterine overactivity reduces intervillous perfusion during contractions and impairs fetal oxygen exchange.

Thus, the physiologic problem is failure of the intervillous space to reperfuse, which compromises fetal oxygenation.

References:

AWHONN - Fetal Heart Monitoring Principles & Practices Simpson & Creehan - Perinatal Nursing Menihan - Electronic Fetal Monitoring Creasy & Resnik - Maternal-Fetal Medicine Miller's Pocket Guide

NEW QUESTION # 25

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