

Intrapartum Fetal Surveillance

Topic- based Uworld Questions

Block 1, 2, 7, 8

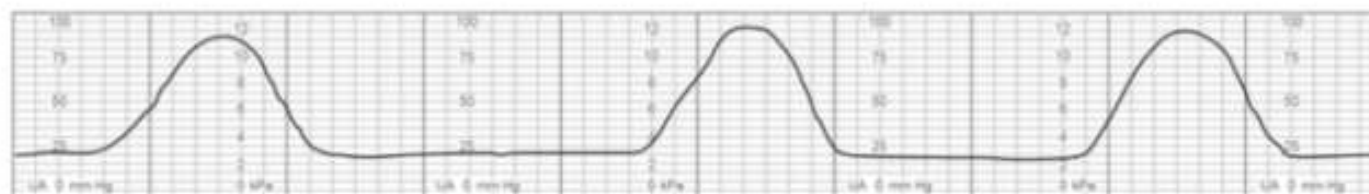
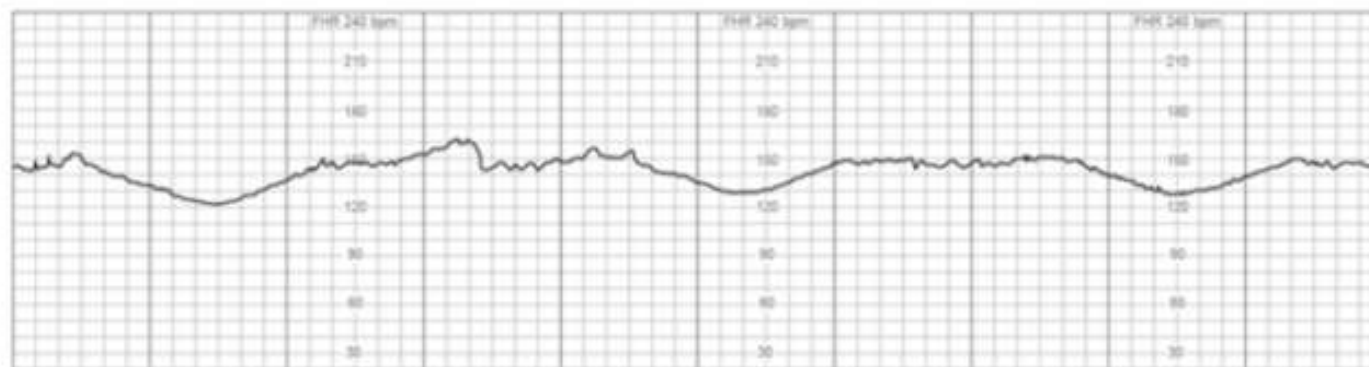


A 31-year-old woman, gravida 3 para 2, at 40 weeks gestation is in active labor. The patient has had a normal prenatal course. Her first pregnancy was an uncomplicated term vaginal delivery. Her second pregnancy ended with an emergent cesarean delivery for a nonreassuring fetal heart rate pattern. She has epidural anesthesia and is feeling mild pelvic pressure. Spontaneous rupture of the membranes occurred 15 minutes ago with blood-tinged amniotic fluid. Blood pressure is 124/76 mm Hg, pulse is 78/min, and respirations are 18/min. On pelvic examination, the cervix is 6 cm dilated and 80% effaced, and the fetal presenting part is at +1 station. The fetal heart rate tracing is shown in the [exhibit](#). Which of the following is the most likely cause of this fetal heart rate pattern?

- A. Fetal head compression
- B. Placental abruption
- C. Umbilical cord compression
- D. Uterine rupture
- E. Uteroplacental insufficiency

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- A. Fetal head compression (83%)
- B. Placental abruption (1%)
- C. Umbilical cord compression (9%)
- D. Uterine rupture (0%)
- E. Uteroplacental insufficiency (4%)

Omitted

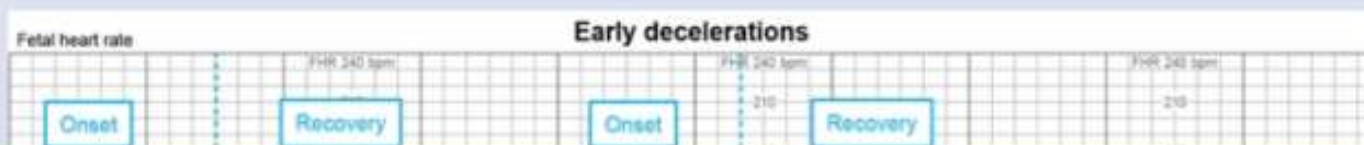
Correct answer
A

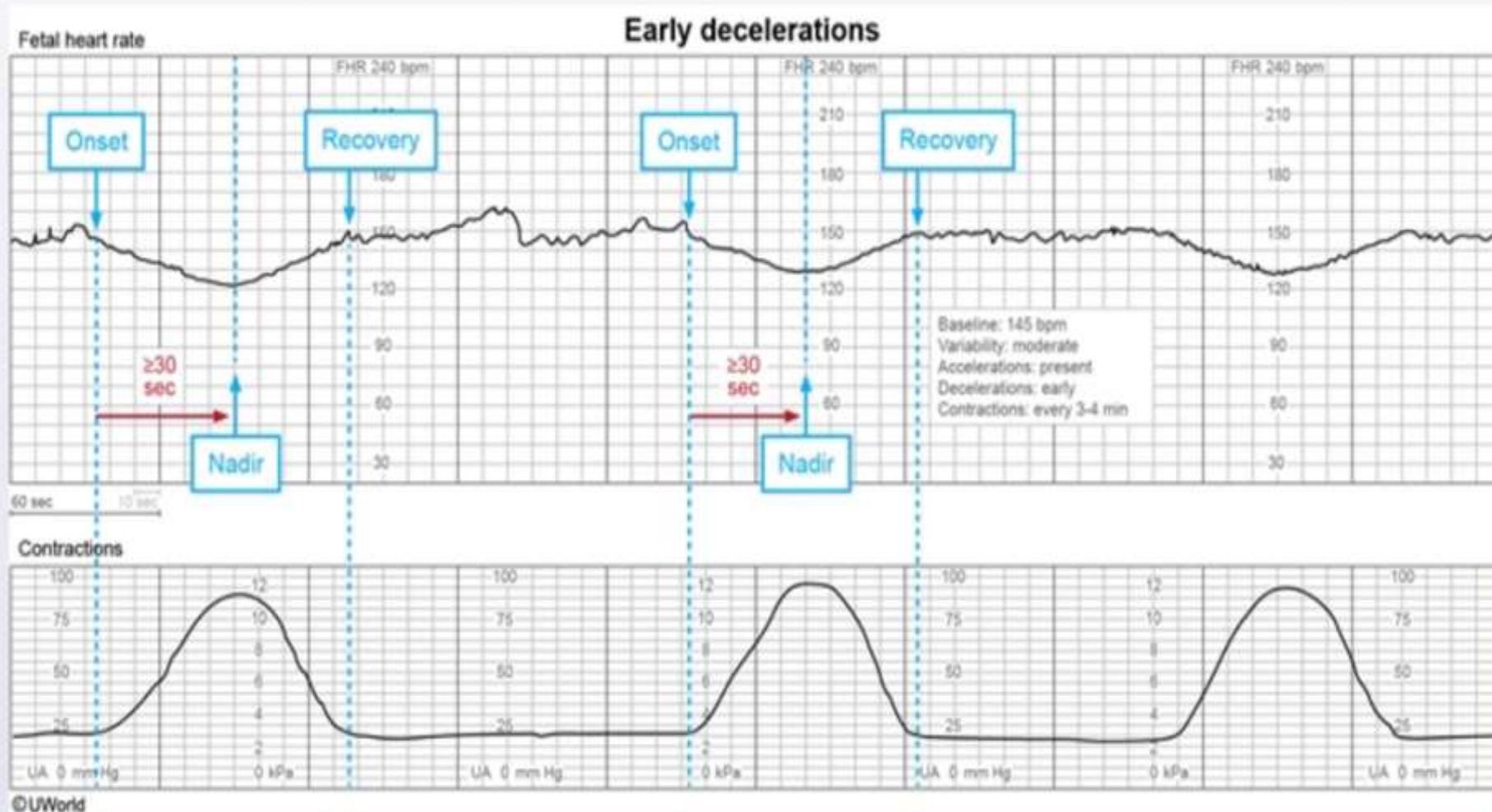
83%
Answered correctly

11 secs
Time Spent

04/18/2020
Last Updated

Explanation





This fetal heart rate tracing demonstrates moderate variability (average amplitude 6-25/min) and **early decelerations**. **Early decelerations** show onset (≥ 30 seconds from onset to nadir) and occur symmetrically with contractions. This results in the characteristic **uniform, shallow**

This fetal heart rate tracing demonstrates moderate variability (average amplitude 6-25/min) and **early decelerations**. [Early decelerations](#) have a **slow onset** (≥ 30 seconds from onset to nadir) and occur symmetrically with contractions. This results in the characteristic **uniform, shallow deceleration** with the **nadir at the peak of the contraction** and return to baseline at the end of the contraction.

Early decelerations are caused by **fetal head compression**; these occur when the fetal head descends closer to the cervix, which contracts and causes narrowing of the fetal anterior fontanelle. The narrowed anterior fontanelle causes a transient alteration in cerebral blood flow and stimulates a **vagal response**, which slows the heart rate. Early decelerations are a **benign, physiologic finding** and do not indicate fetal hypoxia; therefore, these decelerations do not require intervention.

(Choices B and E) Placental abruption and uteroplacental insufficiency typically cause late decelerations due to fetal hypoxia. Like early decelerations, late decelerations have a slow onset (≥ 30 seconds from onset to nadir); but unlike early decelerations, they are delayed compared to contractions. [Late decelerations](#) begin after the onset of the contraction, nadir after the peak of the contraction, and return to baseline after the contraction.

(Choice C) Umbilical cord compression results in [variable decelerations](#), which have an abrupt onset (< 30 seconds from onset to nadir) and a variable duration and depth with each deceleration. Variable decelerations are not always associated with contractions.

(Choice D) Uterine rupture may cause bradycardia, variable decelerations, and late decelerations due to concomitant fetal hypoxia or umbilical cord compression (which occurs through the ruptured uterine scar).

Educational objective:

Early decelerations are uniform, shallow decelerations with gradual onset that occur symmetrically with contractions. They nadir at the peak of the contraction and return to baseline at the end of the contraction. Early decelerations are a benign finding caused by fetal head compression, which leads to a physiologic vagal response.

References

- [A framework for standardized management of intrapartum fetal heart rate patterns.](#)
- [The 2008 National Institute of Child Health and Human Development workshop report on electronic fetal monitoring: update on definitions, interpretation, and research guidelines.](#)

Exhibit Display

Intrapartum fetal heart rate monitoring: early decelerations

Relationship to contraction:

- Symmetric to contraction
- Nadir of deceleration corresponds to peak of contraction
- Gradual (≥ 30 sec from onset to nadir)

Etiology:

- Fetal head compression
- Can be normal fetal tracing

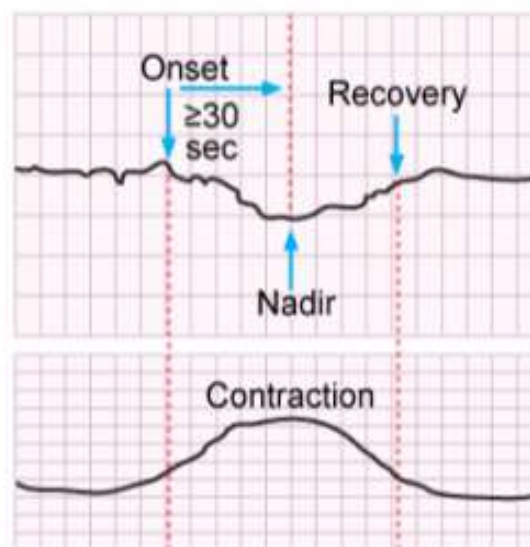


Exhibit Display

Intrapartum fetal heart rate monitoring: late decelerations

Relationship to contraction:

- Delayed compared to contraction
- Nadir of deceleration occurs after peak of contraction
- Gradual (≥ 30 sec from onset to nadir)

Etiology:

- Uteroplacental insufficiency

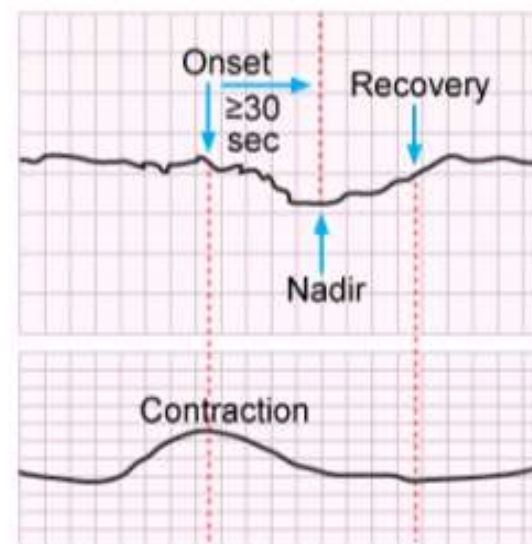


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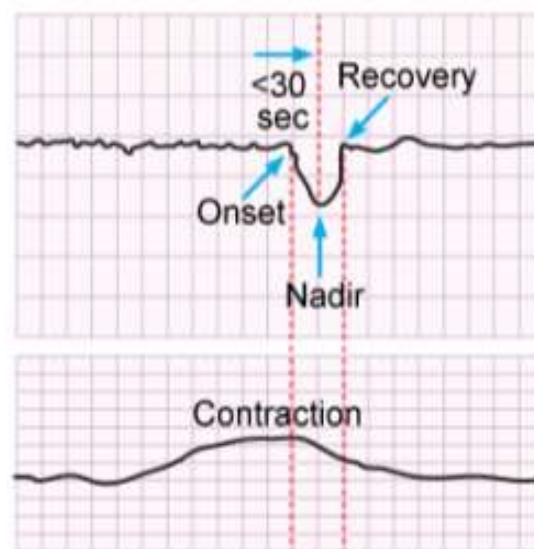
Intrapartum fetal heart rate monitoring: variable decelerations

Relationship to contraction:

- Not necessarily associated with contractions
- Abrupt (<30 sec from onset to nadir)
- Decrease ≥ 15 /min; duration ≥ 15 sec but < 2 min

Etiology:

- Cord compression
- Oligohydramnios
- Cord prolapse

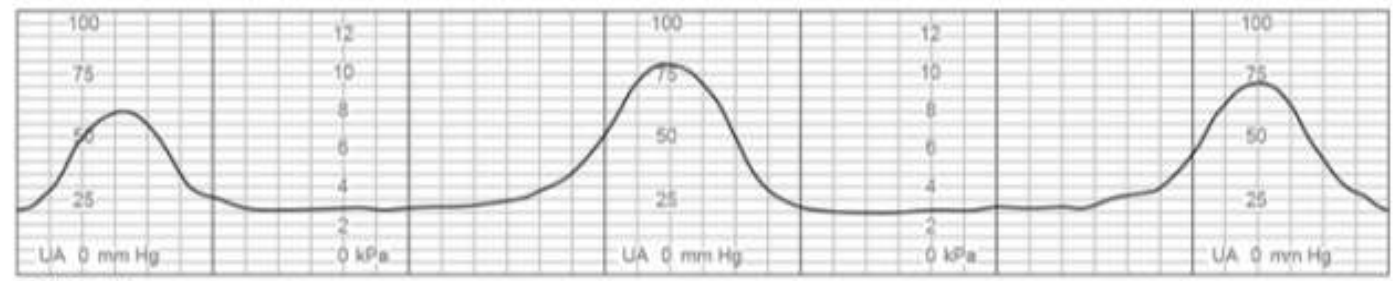
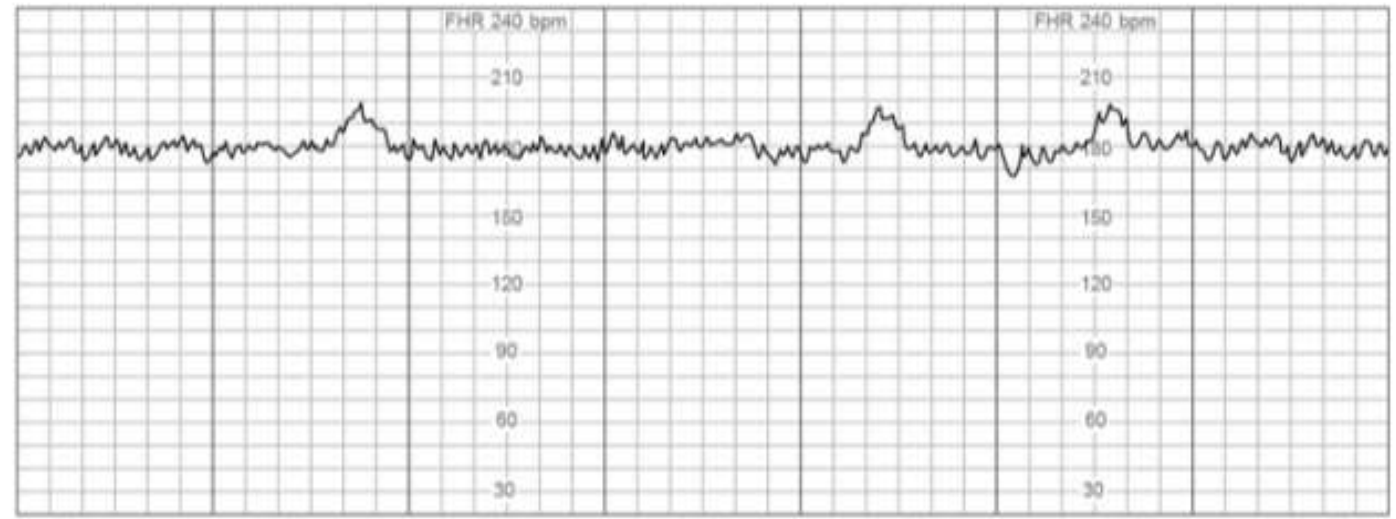


A 36-year-old woman, gravida 1 para 0, at 38 weeks gestation comes to the hospital due to increased leakage of fluid. She reports spontaneous rupture of membranes approximately 48 hours earlier, and contractions started 12 hours after rupture. She reports intermittent vaginal spotting and regular fetal movement. The patient was attempting a home birth but came to the hospital because her cervix has been 6 cm dilated for the past 5 hours. Temperature is 39.4 C (103 F), blood pressure is 120/80 mm Hg, and pulse is 108/min. Digital examination reveals the cervix to be 6 cm dilated and 70% effaced, with the fetal vertex at 0 station. The fetal heart tracing is shown in the [exhibit](#). Which of the following is most likely responsible for the findings on this patient's fetal monitoring?

- A. Fetal anemia
- B. Fetal head compression
- C. Fetal sleep cycle
- D. Intraamniotic infection
- E. Placental insufficiency
- F. Umbilical cord compression

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A 36-year-old woman, gravida 1 para 0, at 38 weeks gestation comes to the hospital due to increased leakage of fluid. She reports spontaneous rupture of membranes approximately 48 hours earlier, and contractions started 12 hours after rupture. She reports intermittent vaginal spotting and regular fetal movement. The patient was attempting a home birth but came to the hospital because her cervix has been 6 cm dilated for the past 5 hours. Temperature is 39.4 C (103 F), blood pressure is 120/80 mm Hg, and pulse is 108/min. Digital examination reveals the cervix to be 6 cm dilated and 70% effaced, with the fetal vertex at 0 station. The fetal heart tracing is shown in the [exhibit](#). Which of the following is most likely responsible for the findings on this patient's fetal monitoring?

- A. Fetal anemia (1%)
- B. Fetal head compression (2%)
- C. Fetal sleep cycle (2%)
- D. Intraamniotic infection (80%)
- E. Placental insufficiency (3%)
- F. Umbilical cord compression (8%)

Omitted

Correct answer
D80%
Answered correctly09 secs
Time Spent04/06/2020
Last Updated

Explanation

Intraamniotic infection (chorioamnionitis)

Intraamniotic infection (chorioamnionitis)	
Risk factors	<ul style="list-style-type: none"> • Prolonged rupture of membranes (>18 hours) • Preterm prelabor rupture of membranes • Prolonged labor • Internal fetal/uterine monitoring devices • Repetitive vaginal examinations • Presence of genital tract pathogens
Diagnosis	Maternal fever PLUS ≥ 1 of the following: <ul style="list-style-type: none"> • Fetal tachycardia (>160/min) • Maternal leukocytosis • Purulent amniotic fluid
Management	<ul style="list-style-type: none"> • Broad-spectrum antibiotics • Delivery
Complications	<ul style="list-style-type: none"> • Maternal: postpartum hemorrhage, endometritis • Neonatal: preterm birth, pneumonia, encephalopathy

This patient has a [fetal heart tracing](#) with moderate variability and **fetal tachycardia**, a sustained fetal baseline heart rate >160/min. Common causes of fetal tachycardia include maternal infection, poorly controlled maternal hyperthyroidism, medication use (eg, terbutaline), and [abruptio placentae](#). This patient has an **intraamniotic infection** (chorioamnionitis) of the uterine decidua, placenta, amniotic fluid, membranes, and umbilical cord that results from migration of vaginal or enteric flora through the cervix into the uterine cavity.

In the setting of **protracted or arrested labor** (slow labor progression) and **prolonged membrane rupture** (>18 hours), **maternal fever** suggests intraamniotic infection as the etiology of the fetal tachycardia. Additional clinical features of intraamniotic infection include maternal leukocytosis, purulent amniotic fluid, maternal tachycardia, and uterine fundal tenderness. Broad-spectrum antibiotics are administered to reduce the risk of

neonatal (eg, sepsis, encephalopathy) and maternal (eg, postpartum hemorrhage, endometritis) morbidity.

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(Choice A) Fetal anemia typically presents with a **sinusoidal fetal heart tracing**, a smooth, undulating waveform with no variability. This patient's tracing has moderate variability.

(Choice B) **Early decelerations** are shallow decreases in the fetal heart rate that occur as a mirror image with the uterine contraction. They are likely an autonomic response to alterations in intracranial pressure caused by fetal head compression during contractions. This fetal tracing has no early decelerations.

(Choice C) Fetal sleep cycles are commonly associated with nonreactive non-stress tests (no accelerations), but they do not cause fetal tachycardia.

(Choice E) Placental insufficiency causes late decelerations, which are smooth and subtle drops in the fetal heart rate following a contraction due to placental hypoperfusion during the contraction that results in transient fetal hypoxia. This fetal tracing has no late decelerations.

(Choice F) **Variable decelerations**, abrupt drops in the fetal heart rate of varying depth and duration, are likely due to compression of the umbilical cord causing transient fetal hypertension that triggers a parasympathetic response and slows the heart rate. Umbilical cord compression does not cause sustained fetal tachycardia.

Educational objective:

Intraamniotic infection, also known as chorioamnionitis, is a cause of fetal tachycardia (baseline heart rate >160/min). Intraamniotic infection is caused by migration of vaginal or enteric flora through the cervix and is associated with prolonged rupture of membranes and a protracted labor course.

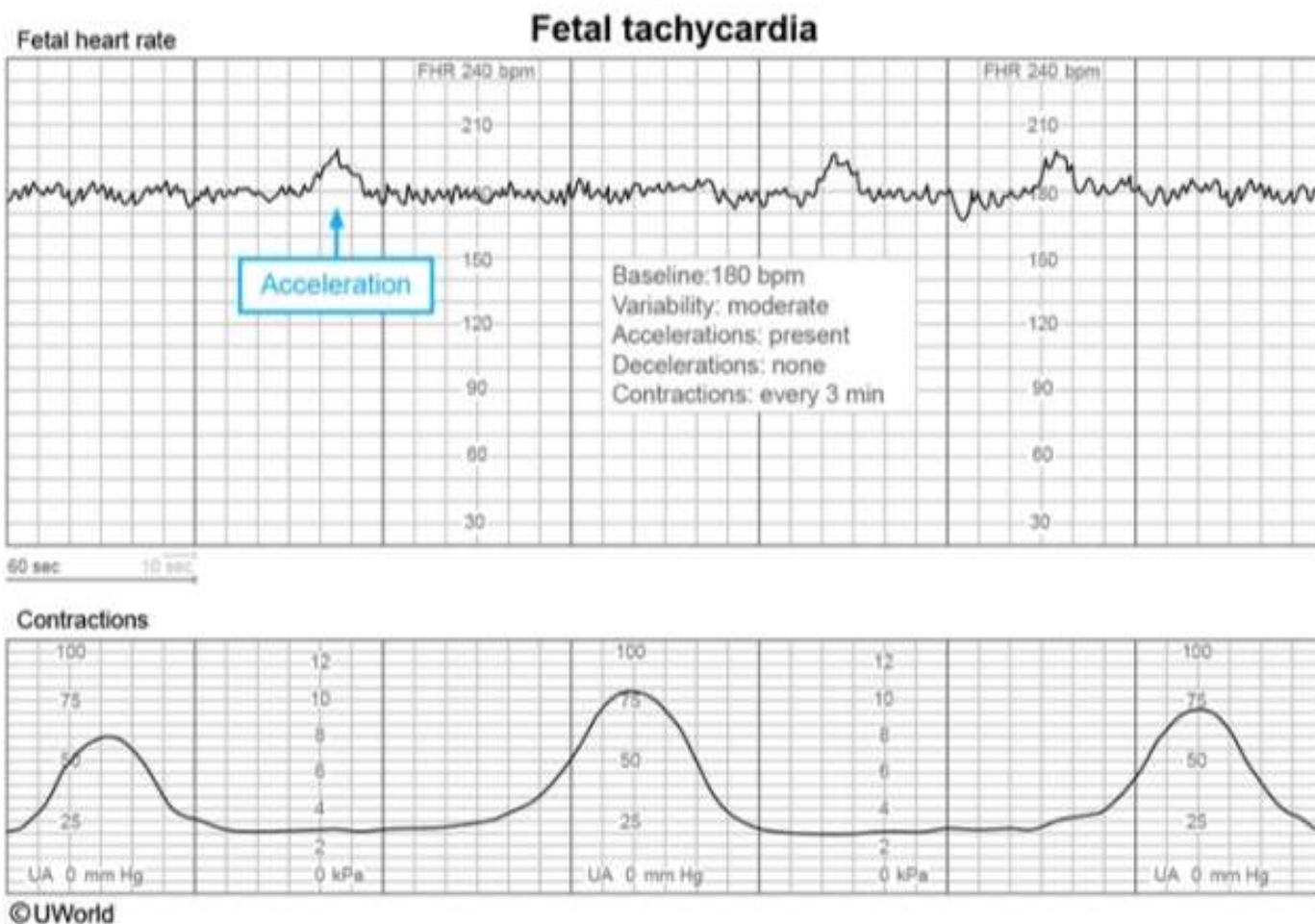
References

- [The 2008 national institute of child health and human development workshop report on electronic fetal monitoring: update on definitions, interpretation, and research guidelines.](#)

Diagnosis

- Fetal tachycardia (>160/min)

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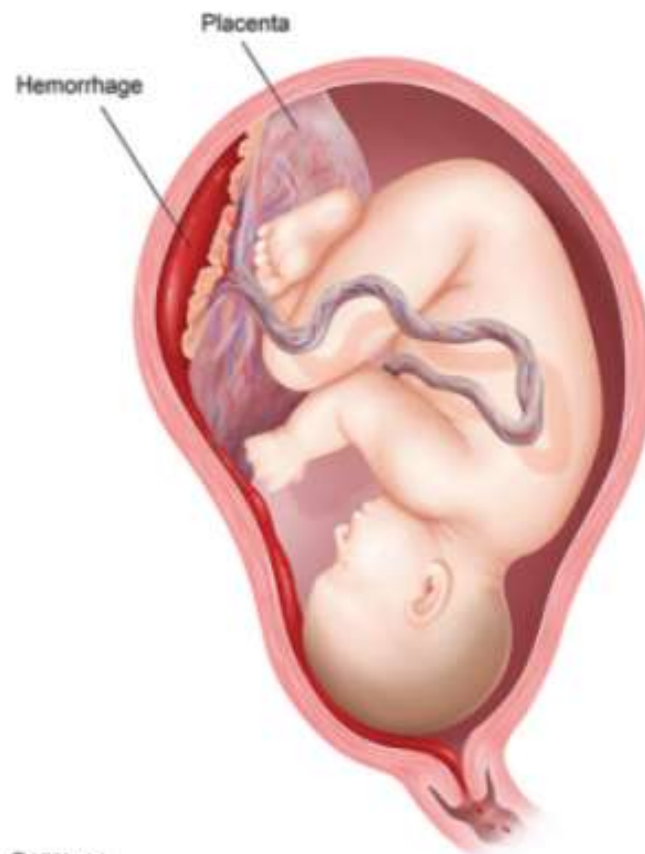
Placental insufficiency causes late decelerations, which are smooth and subtle drops in the fetal heart rate following a contraction due

Diagnosis

- Fetal tachycardia ($>160/\text{min}$)

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Placental abruption



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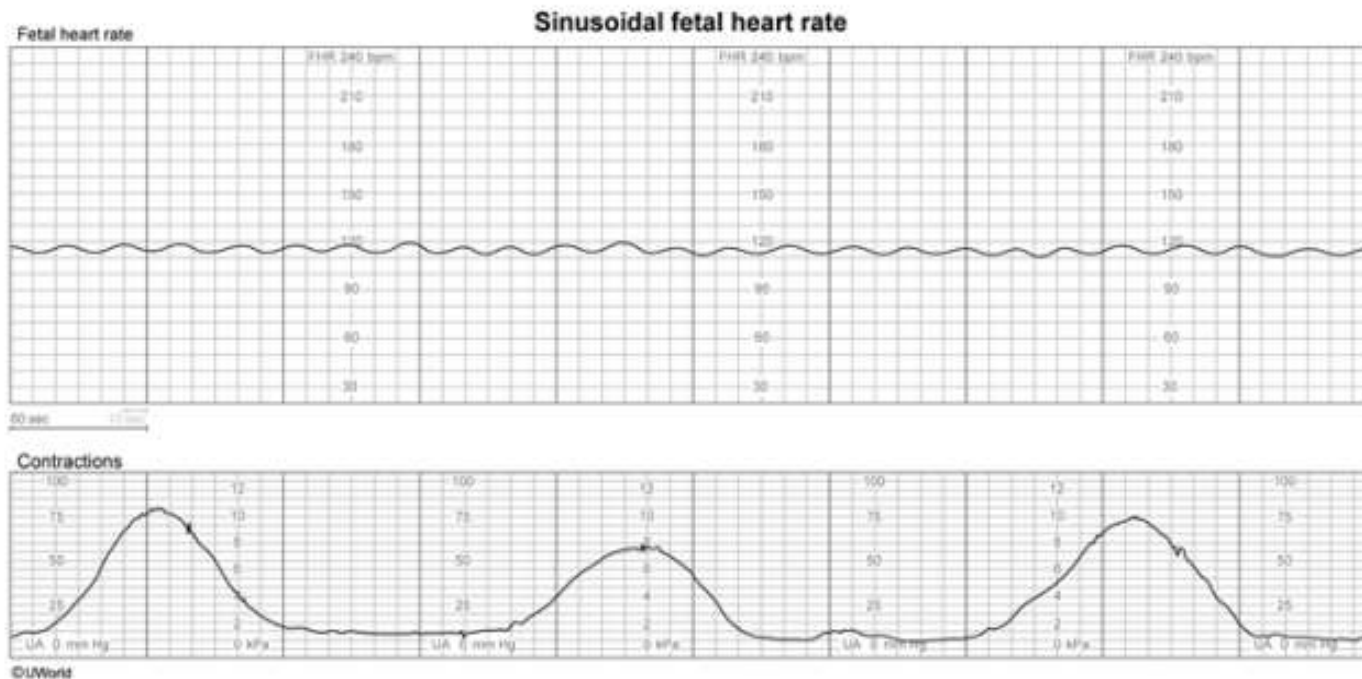
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(Choice 2) Placental insufficiency causes late decelerations, which are smooth and subtle drops in the fetal heart rate following a contraction and

Diagnosis

- Fetal tachycardia (>160/min)

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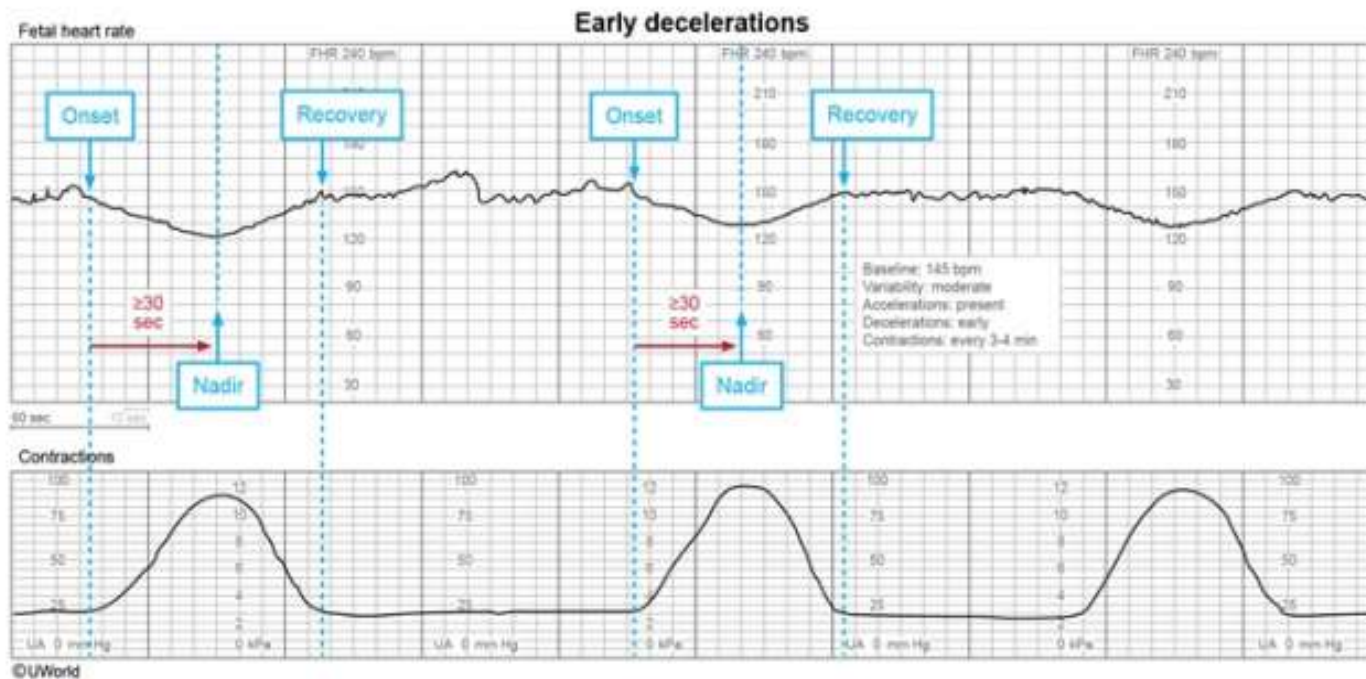
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Placental insufficiency causes late decelerations, which are smooth and subtle drops in the fetal heart rate following a contraction due

Diagnosis

- Fetal tachycardia (>160/min)

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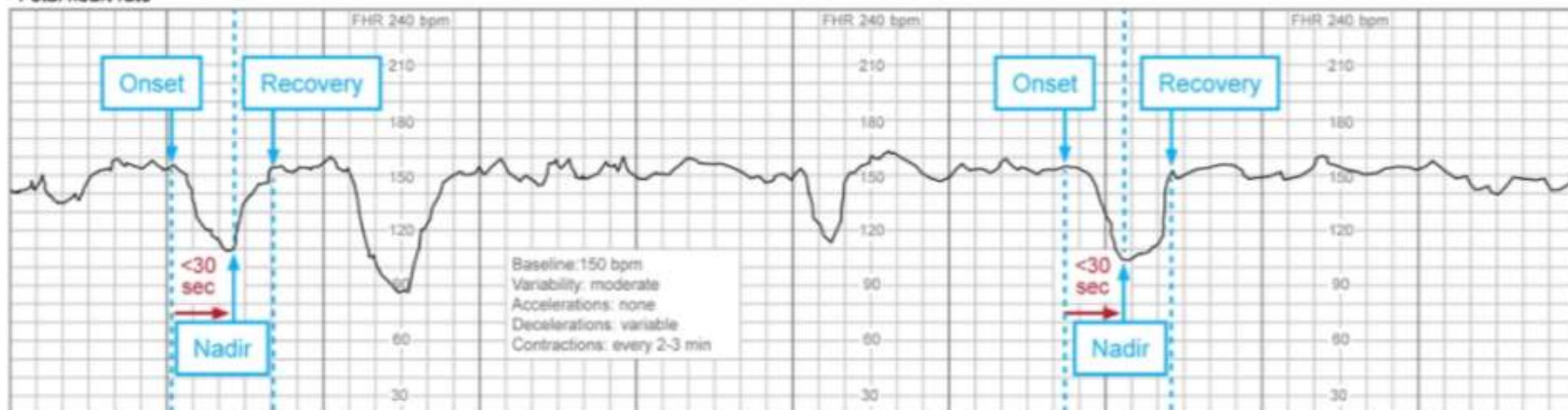
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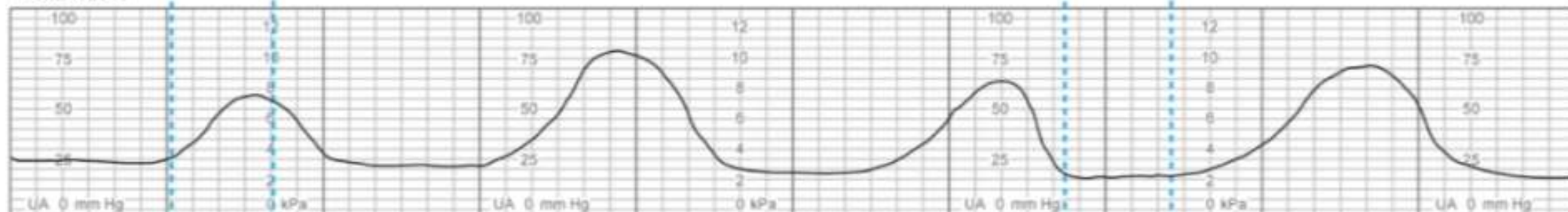
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Fetal heart rate



60 sec 10 sec

Contractions



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A 26-year-old woman, gravida 2 para 1, at 35 weeks gestation comes to the labor and delivery department due to regular, painful contractions over the past 3 hours. The patient has had no prenatal care during this pregnancy. She has no chronic medical conditions, and her only surgery was a low transverse cesarean delivery 2 years ago. Her cervix on admission is 7 cm dilated and 100% effaced with the fetal head at +2 station. Fetal heart rate tracing on admission is category 1. Epidural analgesia is administered, and the patient has pain relief from the contractions. Rupture of membranes results in bright-red amniotic fluid. Blood pressure is 130/80 mm Hg and pulse is 112/min. Current fetal monitoring is as seen in the [exhibit](#). Which of the following is the most likely cause of this fetal heart rate tracing?

- A. Fetal blood loss
- B. Intraamniotic infection
- C. Oligohydramnios
- D. Preterm gestation
- E. Umbilical cord compression

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- A. Fetal blood loss (68%)
- B. Intraamniotic infection (5%)
- C. Oligohydramnios (7%)
- D. Preterm gestation (5%)
- E. Umbilical cord compression (12%)

Omitted

Correct answer

A



68%

Answered correctly



02 secs

Time Spent



01/05/2020

Last Updated

Explanation

Fetal heart rate tracing patterns

Requires all the following criteria:

- Baseline 110-160/min

Fetal heart rate tracing patterns	
Category I	Requires all the following criteria: <ul style="list-style-type: none"> • Baseline 110-160/min • Moderate variability (6-25/min) • No late/variable decelerations • \pm Early decelerations • \pm Accelerations
Category II	<ul style="list-style-type: none"> • Not category I or III (indeterminate pattern)
Category III	≥ 1 of the following characteristics: <ul style="list-style-type: none"> • Absent variability + recurrent late decelerations • Absent variability + recurrent variable decelerations • Absent variability + bradycardia • Sinusoidal pattern

Fetal heart rate (FHR) monitoring in labor assesses fetal oxygenation and metabolic status to prevent fetal morbidity and mortality. A category I FHR tracing (reassuring) has a low risk of fetal hypoxemia and acidemia and is managed expectantly. In contrast, a category III FHR tracing has an increased risk of fetal compromise and requires urgent intervention to prevent fetal injury and demise.

This patient has a **sinusoidal fetal heart rate tracing**, defined as a **smooth, wave-like oscillation** (ie, rolling line), with fixed amplitude (5-15/min) and frequency (3-5 cycles/min). Sinusoidal tracings are a **category III tracing** and are considered ominous because they are associated with **severe fetal anemia**, suggested in this patient with likely fetal blood loss from ruptured **vasa previa** (eg, bright-red amniotic fluid). Due to rapid **fetal exsanguination and deterioration**, this patient requires **urgent cesarean delivery**.

(Choice B) Intraamniotic infection (chorioamnionitis) is usually associated with prolonged membrane rupture. FHR monitoring typically shows **fetal tachycardia** (>160/min) due to maternal fever and increased sympathetic drive.

(Choices C and E) Oligohydramnios (amniotic fluid index ≤ 5 cm) may cause **variable decelerations** due to umbilical cord compression. Variable

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(Choice B) Intraamniotic infection (chorioamnionitis) is usually associated with prolonged membrane rupture. FHR monitoring typically shows **fetal tachycardia** (>160/min) due to maternal fever and increased sympathetic drive.

(Choices C and E) Oligohydramnios (amniotic fluid index ≤ 5 cm) may cause **variable decelerations** due to umbilical cord compression. Variable decelerations occur because, as the uterus contracts, the umbilical cord is compressed, initially affecting the thin-walled umbilical vein and causing a transient reflex rise in the FHR. Continued cord compression eventually occludes the thick-walled umbilical artery, causing a rapid increase in blood pressure and subsequent abrupt drop in heart rate (ie, parasympathetic response). As the contraction releases, there is an equally rapid return to baseline.

(Choice D) Preterm (<37 week) gestation may be associated with an increased FHR baseline, reduced variability, and lower-amplitude accelerations. It does not cause sinusoidal rhythms.

Educational objective:

Sinusoidal fetal heart rate tracings are typically associated with severe fetal anemia. They are a category III tracing that reflects an increased risk of fetal compromise (eg, hypoxemia, acidemia) and requires urgent cesarean delivery.

References

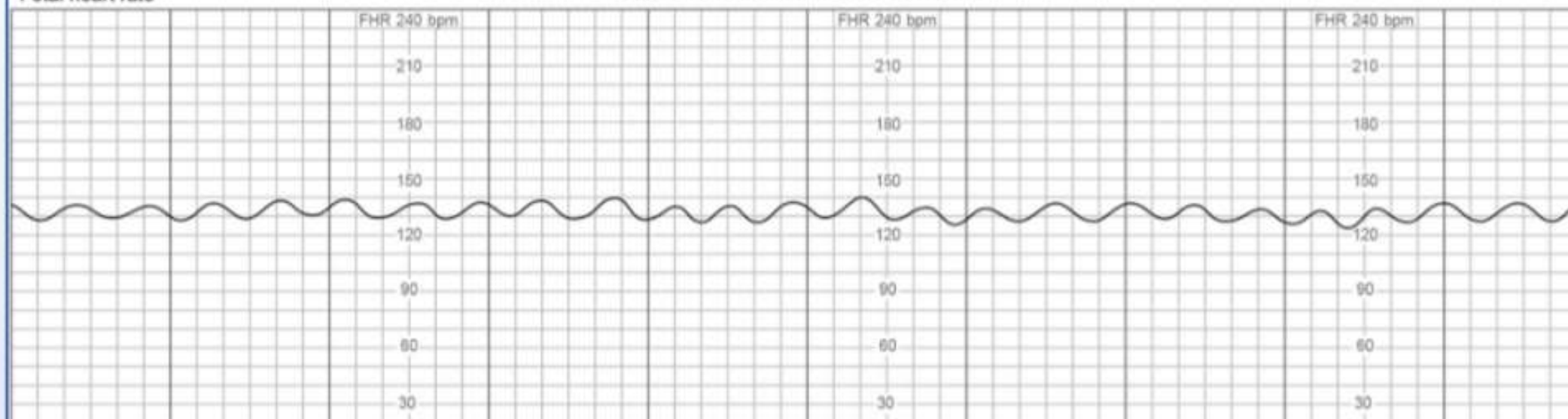
- [Continuous cardiotocography \(CTG\) as a form of electronic fetal monitoring \(EFM\) for fetal assessment during labour.](#)
- [Massive fetomaternal hemorrhage as a cause of severe fetal anemia.](#)
- [Antepartum high-frequency fetal heart rate sinusoidal rhythm: computerized detection and fetal anemia.](#)

- ± Early decelerations

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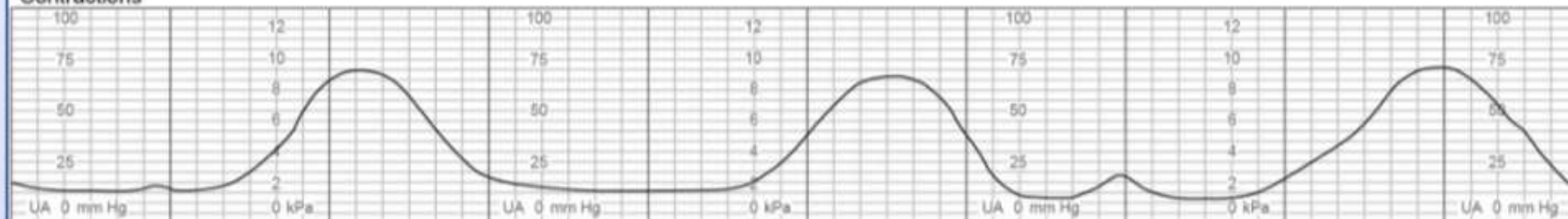
Fetal heart rate

Sinusoidal fetal heart rate



60 sec 10 sec

Contractions



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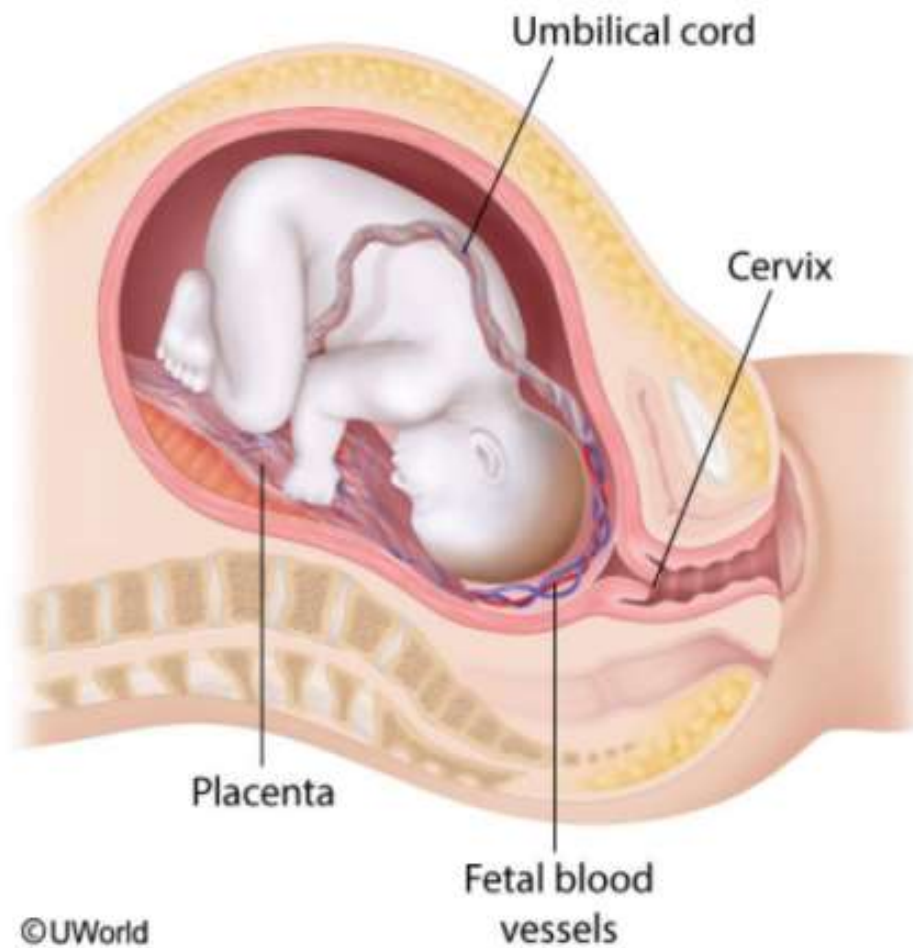
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- ± Early decelerations

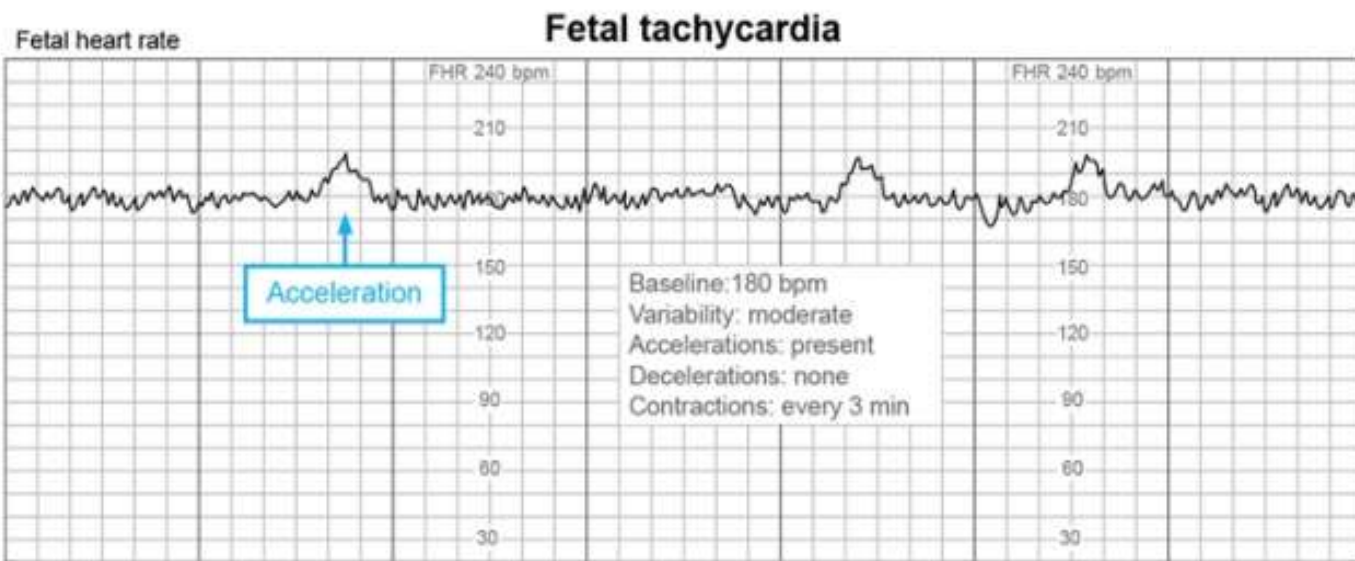
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Vasa previa

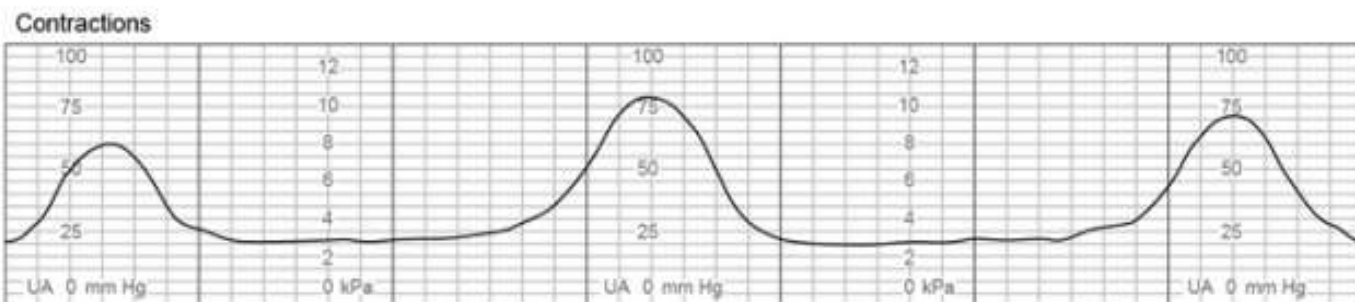


FHR tracing (reassuring) has a low risk of fetal hypoxemia and acidemia and is managed expectantly. In contrast, a category III FHR tracing has

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60 sec 10 sec



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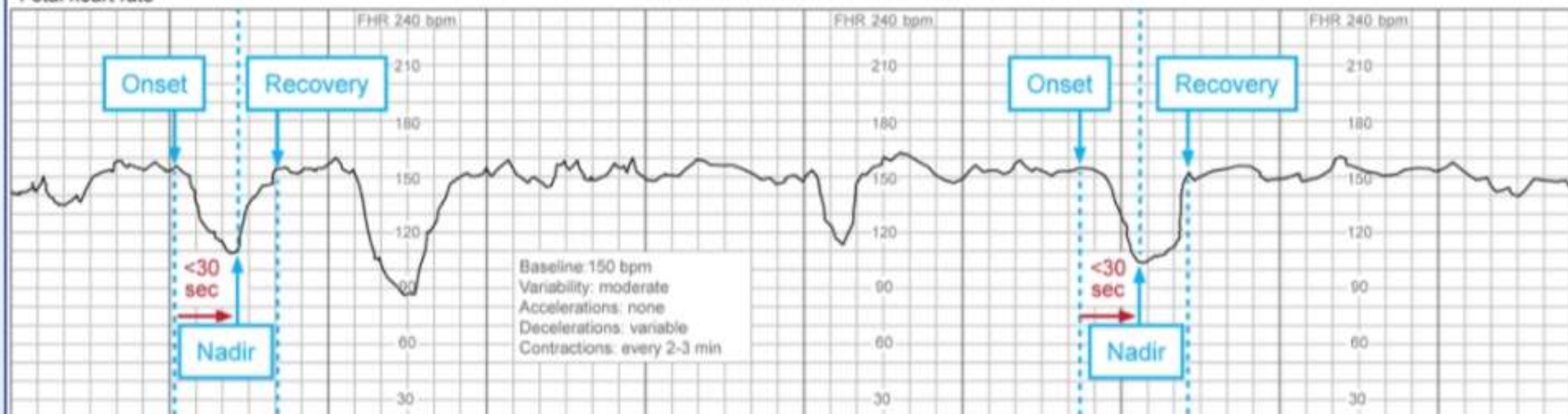
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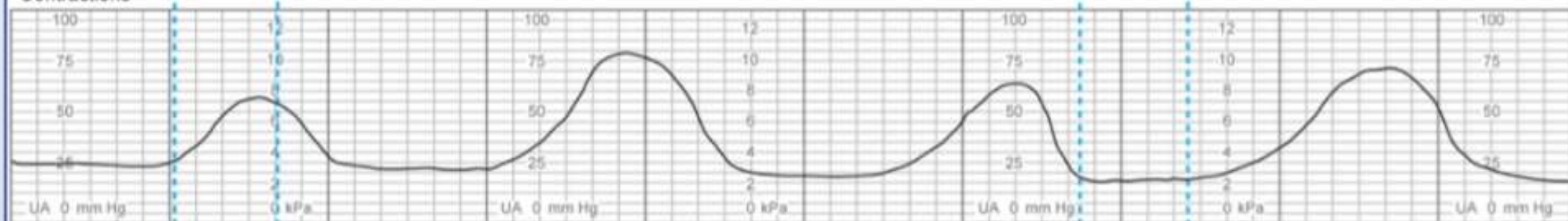
Variable decelerations

Fetal heart rate



60 sec 10 sec

Contractions



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A 28-year-old woman, gravida 1 para 0, at 41 weeks gestation comes to the hospital for antepartum fetal surveillance. She has had less fetal movement than usual but no abdominal pain, leakage of fluid, or vaginal bleeding. Blood pressure is 120/80 mm Hg. The patient's pre-pregnancy BMI was 40 kg/m², and she has gained 20 kg (44 lb) during the pregnancy. Her pregnancy has otherwise been uncomplicated. Nonstress test shows a baseline fetal heart rate of 140/min, moderate variability, and no accelerations for >40 minutes. Two contractions are observed on the tocodynamometer. An episode of fetal heart rate decrease to 130/min began after the peak of a contraction and lasted for a minute. Ultrasound shows a fetus in cephalic presentation, a loop of umbilical cord near the fetal neck, an estimated fetal weight at the 35th percentile for gestational age, and a single deepest pocket of amniotic fluid of 1 cm. The biophysical profile results are shown below.

Biophysical profile component	Score
Nonstress test	0
Amniotic fluid volume	0
Fetal movements	2
Fetal tone	2
Fetal breathing movements	0

Which of the following is the most likely cause of this patient's biophysical profile results?

- A. Fetal head compression
- B. Fetal sleep cycle
- C. Intraamniotic infection
- D. Nuchal cord
- E. Polyhydramnios

shows a fetus in cephalic presentation, a loop of umbilical cord near the fetal neck, an estimated fetal weight at the 35th percentile for gestational age, and a single deepest pocket of amniotic fluid of 1 cm. The biophysical profile results are shown below.

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- B. Fetal sleep cycle
- C. Intraamniotic infection
- D. Nuchal cord
- E. Polyhydramnios
- F. Uteroplacental insufficiency

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Biophysical profile component	Score
Nonstress test	0
Amniotic fluid volume	0
Fetal movements	2
Fetal tone	2
Fetal breathing movements	0

Which of the following is the most likely cause of this patient's biophysical profile results?

- A. Fetal head compression (3%)
- B. Fetal sleep cycle (4%)
- C. Intraamniotic infection (0%)
- D. Nuchal cord (17%)
- E. Polyhydramnios (0%)
- F. Uteroplacental insufficiency (72%)

Omitted

Correct answer



72%

Answered correctly



06 secs

Time Spent



05/17/2020

Last Updated

Biophysical profile*	
Component	Normal finding
Nonstress test	Reactive fetal heart rate monitoring
Amniotic fluid volume	Single fluid pocket $\geq 2 \times 1$ cm or amniotic fluid index >5
Fetal movements	≥ 3 General body movements
Fetal tone	≥ 1 Episodes of flexion/extension of fetal limbs or spine
Fetal breathing movements	≥ 1 Breathing episode for ≥ 30 seconds
Maximum score = 10: 0 = abnormal, 2 = normal for each component.	
*Performed continuous observation for ≥ 30 minutes.	

Decreased placental function is typically due to increased placental vascular resistance caused by age-related placental changes in **late-term pregnancies** (≥ 41 weeks gestation). Decreased placental function reduces placental perfusion during contractions, which can result in intermittent fetal hypoxemia. On nonstress test, intermittent hypoxemia presents as **late decelerations**, fetal heart rate decelerations that gradually nadir after the peak of a contraction.

Progressive placental dysfunction can lead to **chronic fetal hypoxemia** (ie, **uteroplacental insufficiency**), which causes central nervous system suppression and possible fetal demise. To prevent this suppression, blood is preferentially distributed to the brain rather than peripheral tissue, resulting in decreased fetal activity (eg, movement, breathing, muscle tone). In the fetal kidney, the decreased renal perfusion and urine production result in **oligohydramnios** (single deepest pocket of amniotic fluid < 2 cm).

A **biophysical profile (BPP)**, performed in patients at risk for uteroplacental insufficiency, includes a nonstress test and ultrasound assessment of amniotic fluid volume and fetal activity. Each BPP component is scored 0 or 2 and summed to a total score of 0-10. Normal BPP scores (8 or 10) exclude fetal hypoxemia. Chronic hypoxemia from uteroplacental insufficiency causes an **abnormal BPP** (particularly ≤ 4) and suggests **imminent**

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(Choice A) Fetal head compression can cause a physiologic parasympathetic response that presents as **early decelerations**, gradual heart rate decelerations that nadir at the peak of the contraction. Head compression does not result in hypoxemia and therefore does not cause an abnormal BPP.

(Choice B) Fetal sleep cycles can cause a nonreactive nonstress test (ie, no accelerations due to lack of movement); sleep cycles do not cause late decelerations or oligohydramnios.

(Choice C) Intraamniotic infection (chorioamnionitis) can present with oligohydramnios if associated with premature rupture of membranes (not seen in this patient). Fetal monitoring indicates tachycardia (>160 /min).

(Choice D) Nuchal cords are associated with **variable decelerations**, abrupt (≥ 15 /min) decreases in fetal heart rate below the baseline of varying depth and duration. Nuchal cords do not cause oligohydramnios or decreased fetal breathing movements.

(Choice E) Polyhydramnios, an amniotic fluid index ≥ 24 cm or a single deepest pocket ≥ 8 cm, does not affect the BPP score.

Educational objective:

A biophysical profile (BPP) is performed in patients at risk for uteroplacental insufficiency (eg, ≥ 41 weeks gestation). Chronic hypoxemia causes an abnormal BPP and suggests imminent risk of fetal demise; in such cases, delivery is typically indicated.

References

- Practice bulletin no. 146: management of late-term and postterm pregnancies.

Exhibit Display

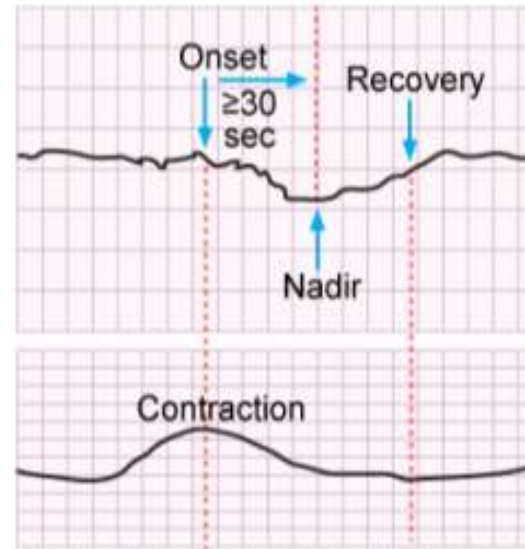
Intrapartum fetal heart rate monitoring: late decelerations

Relationship to contraction:

- Delayed compared to contraction
- Nadir of deceleration occurs after peak of contraction
- Gradual (≥ 30 sec from onset to nadir)

Etiology:

- Uteroplacental insufficiency



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Progressive placental dysfunction can lead to **chronic fetal hypoxemia** (ie, **uteroplacental insufficiency**), which causes central nervous system

Exhibit Display

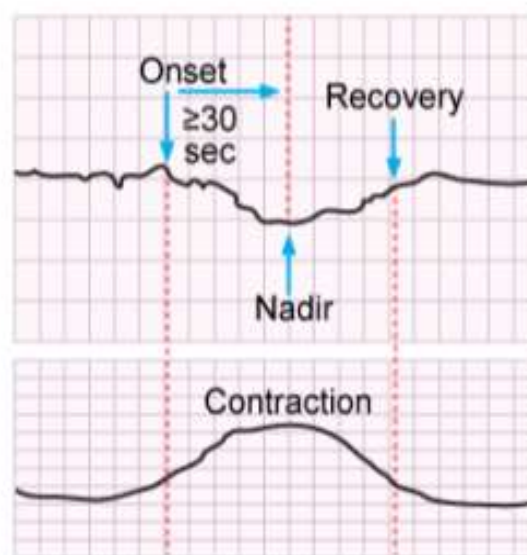
Intrapartum fetal heart rate monitoring: early decelerations

Relationship to contraction:

- Symmetric to contraction
- Nadir of deceleration corresponds to peak of contraction
- Gradual (≥ 30 sec from onset to nadir)

Etiology:

- Fetal head compression
- Can be normal fetal tracing



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Progressive placental dysfunction can lead to **chronic fetal hypoxemia** (ie, **uteroplacental insufficiency**), which causes central nervous system

Exhibit Display

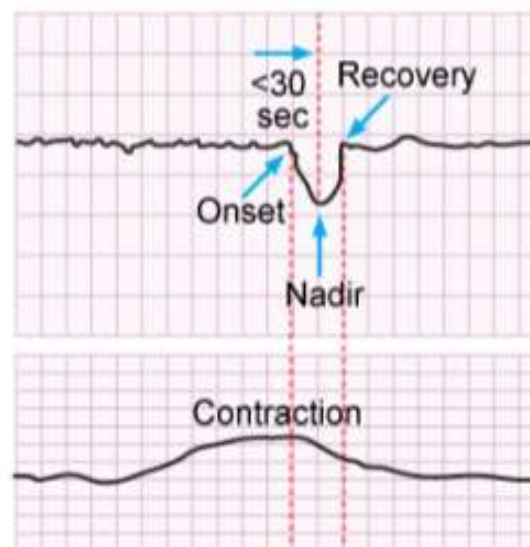
Intrapartum fetal heart rate monitoring: variable decelerations

Relationship to contraction:

- Not necessarily associated with contractions
- Abrupt (<30 sec from onset to nadir)
- Decrease ≥ 15 /min; duration ≥ 15 sec but <2 min

Etiology:

- Cord compression
- Oligohydramnios
- Cord prolapse



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