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Late Decelerations

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Continuing Education Activity

Late decelerations are one of the precarious decelerations among the three types of fetal heart rate decelerations during labor. They are caused by decreased blood flow to the placenta and can signify an impending fetal acidemia. Late decelerations are defined as a visually apparent, gradual decrease in the fetal heart rate typically following the uterine contraction. A late deceleration typically follows a uterine contraction meaning, the onset, nadir and the return of the deceleration will follow the onset, peak, and the return of a uterine contraction. This activity reviews the clinical significance of late decelerations and highlights the role of the interprofessional team in its management.

Objectives:

- Review the causes of late decelerations.
- Describe the evaluation of late decelerations in a pregnant patient.
- Summarize the treatment of late decelerations.
- Explain the importance of improving care coordination among interprofessional team members to improve outcomes for patients affected by late decelerations.

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Introduction

Late decelerations are one of the precarious decelerations among the three types of fetal heart rate decelerations during labor. They are caused by decreased blood flow to the placenta and can signify an impending fetal acidemia.

Definition

Late deceleration is defined as a visually apparent, gradual decrease in the fetal heart rate typically following the uterine contraction. The gradual decrease is defined as, from onset to nadir taking 30 seconds or more. A late deceleration typically follows a uterine contraction meaning, the onset, nadir and the return of the deceleration will follow the onset, peak, and the return of a uterine contraction.[1]

Typically, late decelerations are shallow, with slow onset and gradual return to normal baseline. The usual cause of the late deceleration is uteroplacental insufficiency.

Etiology

The primary etiology of a late declaration is found to be uteroplacental insufficiency. Decreased blood flow to the placenta causes a reduced amount of blood and oxygen to the fetus.

Some of the maternal and fetal conditions which can cause late decelerations are maternal dehydration, anemia, hypoxia, hypotension from epidural analgesia, uterine tachysystole, and placental abruption.[2] Any condition which predisposes to decreased uteroplacental blood flow can cause late decelerations.[3][4]

Epidemiology

Late decelerations are relatively common and correlate with uteroplacental insufficiency.

Pathophysiology

The central pathophysiology behind late deceleration involves uterine contraction constricting blood vessels in the wall of the uterus which decreases blood flow through the intervillous space of the placenta, reducing the diffusion of oxygen into fetal capillaries causing decreased fetal PO2. When fetal PO2 decreases, chemoreceptors initiate an autonomic response in the fetus causing intense vasoconstriction with increased blood pressure. The elevated blood pressure is perceived by the baroreceptors which ultimately stimulate the parasympathetic system to decrease the fetal heart rate, causing late deceleration.[2][4] The whole process reverses after the contraction is completed, with a relaxation of the uterine muscles allowing for increased blood flow to the placenta and resulting in the fetal heart rate returning to normal.

History and Physical

Late declarations are visually apparent, with gradual decreases in the fetal heart rate typically following the uterine contraction. A late deceleration usually follows a uterine contraction meaning that the onset, nadir, and the return of the deceleration will follow the onset, peak, and recovery of a uterine contraction.

Evaluation

Late decelerations are the most precarious decelerations among all types. Persistent and recurrent late decelerations need immediate, meticulous assessment to evaluate the cause and to rule out fetal acidemia.

The three-tier fetal heart rate tracing system is one of the valuable means in classifying the severity of the fetal oxygenation status.[5][6] It subdivides as follows:

- **Category I** is defined as with a baseline rate of 110 to 160 beats per minute, moderate baseline fetal heart rate (FHR) variability (amplitude 6 to 25 bpm), accelerations and early decelerations may be either present or absent, and no late or variable decelerations
- **Category III** is defined as fetal heart rate pattern with absent variability, recurrent variable, and late decelerations, bradycardia, or sinusoidal pattern
- **Category II** any other FHR pattern that does not qualify for Category I or III

Foremost, the entire fetal heart rate tracing requires evaluation, which includes assessing the uterine activity for tachysystole, presence or absence of variability, and accelerations.[7] The fetal heart rate tracing categorizes into I, II, or III depending upon the criteria as mentioned above. The presence of moderate variability and accelerations rules out acute fetal acidemia.[7] In the absence of variability and accelerations with persistent late decelerations, immediate attention is necessary as there may be ongoing fetal hypoxia resulting in metabolic acidosis.[8][9][4] Attention is also necessary to the full assessment of maternal status; this includes continuous monitoring of maternal

vital signs, prevention of dehydration and maintaining adequate intravascular volume, evaluation for ongoing bleeding, awareness of recent medication administration, and assessment of effects from epidural anesthesia. Any maternal hypotension should be addressed immediately.

Treatment / Management

The principal goal of management of late decelerations is to:

- Replenish uteroplacental blood flow by correcting the underlying cause
- Increase fetal PO2
- Prevention or correction of fetal acidemia

After completing a detailed assessment of the etiology of late declaration, immediate intrauterine resuscitative measures are initiated to prevent fetal acidemia and to decrease fetal morbidity and mortality. The resuscitative measures include[10][1]:

- Maternal repositioning to left lateral position, right lateral or knee-chest position, which will relieve the vena cava compression by the gravid uterus - this allows for an increased venous return and cardiac output, which subsequently increases uteroplacental blood flow
- Intravenous hydration: hypotension secondary to epidural anesthesia can also be corrected by giving the mother an intravenous fluid bolus or by alpha-adrenergic agonists by the anesthesiologist - correcting maternal hypotension will increase intravascular volume, increase maternal PO2, and eventually will increase uteroplacental blood flow and PO2
- Administering supplemental oxygen: there is conflicting data about maternal O2 administration for Category II and Category III fetal heart tracings - some observational studies report that O2 administration can improve fetal oxygenation and decrease decelerations, but overall data is conflicting; the standard of practice when fetal heart tracing abnormality observed remains to administer supplemental oxygen
- Discontinuing uterotonics: by discontinuing uterotonics, there is a relaxation of the uterine muscles which has been proven to increase uteroplacental blood flow - the frequency of uterine contractions can also be decreased by using tocolytics; tocolysis has proven to improve uterine relaxation by decreasing the frequency of uterine contractions and increased uteroplacental blood flow
- Expeditious operative vaginal delivery or cesarean delivery: if the above mentioned intrauterine resuscitative measures are not improving the fetal heart tracings and there are persistent late decelerations with loss of variability, then expeditious steps must be taken towards delivering the fetus.

Differential Diagnosis

- Increasing fetal acidemia
- Intrauterine umbilical cord compression
- Maternal/fetal poisoning
- Nuchal cord entanglements

- Oligohydramnios
- Pushing efforts during the second stage of labor
- Umbilical cord prolapse
- Uterine fetal acidemia
- Uterine tachysystole

Prognosis

The scenario of recurrent late decelerations can be precarious as they can become hazardous if not promptly evaluated. Evaluating the entire fetal heart tracing and maternal status is essential. Consideration is necessary to the degree of variability and presence, or absence of accelerations, and the underlying cause of the deceleration must be addressed immediately. If the late decelerations are recurrent with minimal or no variability and have not improved with intrauterine resuscitative measures, then expeditious steps must be taken towards delivering the fetus.

Complications

If recurrent late decelerations with no variability (Category III) persist or not promptly evaluated and treated, this can lead to increased fetal morbidity and mortality. Complications associated with Category III tracings include fetal acidemia with low APGAR scores, low umbilical cord pH, increased risk of neonatal intensive care unit admission after deliver, neonatal encephalopathy, and cerebral palsy.

Enhancing Healthcare Team Outcomes

The management of labor and delivery is usually done by an interprofessional team that includes an obstetrician, a labor and delivery nurse, and a midwife. During some deliveries, one may note recurrent late declarations which can become hazardous if not promptly evaluated. Evaluating the entire fetal heart tracing and maternal status is critical. The level of variability and presence or absence of accelerations merits strong consideration. Prompt intrauterine resuscitative measures must commence with concurrent correction of the underlying cause of the late deceleration. If the fetal heart tracing is not improved and late decelerations persist, then expeditious steps must be taken towards delivering the fetus. The outcomes for babies treated promptly are excellent. But if there is a delay, the fetus can suffer a severe anoxic injury to the brain.[11] [Level 5]

Review Questions

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