I'm not robot

How to read contraction monitor

How do i read a contraction monitor. How to read contraction monitor at hospital. How to read contraction monitor paper. How to read contraction monitor toco. What should contraction monitor read. How to read baby contraction monitor. How to read contraction monitor numbers. How to read contraction monitor with 3 lines. How to read contraction monitor screen.

Accurate fetal heart rate assessment is crucial for determining the status of the fetus and guiding management decisions for specific conditions. To accurately interpret fetal heart rate patterns, healthcare providers should consider five key areas: baseline rate, variability, accelerations, decelerations, and trends over time. The National Institute of Child Health and Human Development and the American College of Obstetrics and Gynecology have established standardized terminology for describing these patterns. When interpreting a fetal monitoring tracing, it's essential to follow a systematic approach. This involves evaluating the recording quality, identifying the type of monitor used, determining the baseline heart rate and variability, detecting accelerations or decelerations, and identifying trends over time. Additionally, observing uterine contraction patterns can provide valuable information about labor progress and potential effects on the fetus. A typical EFM display and sample strip are provided below to illustrate these concepts. The baseline heart rate is defined as the mean FHR rounded to increments of 5 beats per minute during a 10-minute segment, excluding periodic changes or marked variability. A minimum of 2 minutes of stable baseline is required for any 10-minute segment. The normal FHR baseline range is between 110 and 160 beats per minute, with the baseline set by the atrial pacemaker and governed by autonomic nervous system balance. As the central nervous system matures, the baseline FHR gradually drops, and post-term fetuses may have a lower heart rate. Fluctuations in FHR variability can provide valuable insights into fetal physiologic status. Variability in fetal heart rate (FHR) is a crucial indicator of fetal well-being, particularly during labor. The amplitude of peak-to-trough fluctuations in beats per minute (BPM) can be categorized into four levels: minimal, moderate, marked, and absent. * **Minimal Variability**: Characterized by an amplitude range detectable but 5 BPM or fewer. This is considered a normal variation and does not necessarily indicate any underlying issue. * **Moderate Variability**: Defined as an amplitude range of 6-25 BPM, which is typically seen in healthy fetuses with a normally functioning central nervous system. A moderate variability is generally considered reassuring and indicative of adequate oxygenation. * **Marked Variability**: Involves an amplitude range greater than 25 BPM, often referred to as a "saltatory pattern." This can be a sign of acute hypoxia or mechanical compression of the umbilical cord, especially during the second stage of labor. When coupled with decelerations, this pattern is considered non-reassuring and warrants further investigation. * **Absent Variability**: Characterized by an amplitude range undetectable, which can indicate some dysfunction in one or both of the sympathetic and parasympathetic nervous systems. Absent variability may be seen in conditions such as hypoxemia, fetal sleep cycles, or due to the effects of certain drugs. Causes of decreased or absent variability include: * Hypoxemia/acidosis * Fetal sleep cycles * Drugs (analgesics, barbiturates, tranquilizers, phenothiazines, para-sympatholytics, anesthetics) * Prematurity * Arrhythmias * Fetal tachycardia * Preexisting neurological abnormality * Congenital anomalies Causes of marked (increased or saltatory) variability include: * Fetal stimulation * Mild, transient hypoxemia * Drugs The fetal heart rate (FHR) pattern can be analyzed in different circumstances to determine if the fetus is at risk. One example is a fetus with severe anemia, which may exhibit a sinusoidal pattern due to absent short-term variability but present long-term variability. Accelerations are visually apparent increases in FHR that last for at least 15 seconds and meet certain criteria based on gestational age. These can be periodic (associated with contractions) or episodic (not associated with contractions). Most accelerations are episodic, and they may be triggered by fetal movement, scalp stimulation, or other external stimuli. A nonstress test (NST) is a method used to assess fetal well-being based on the presence of accelerations. A reactive NST indicates that the fetus is at low risk for hypoxia. Fetal bradycardia, or slower than normal heart rate, can be associated with hypoxemia, but it may also have other causes such as maternal hypotension, hypoglycemia, or congenital heart block. Decelerations are visually apparent decreases in FHR that last for at least 15 seconds and meet certain criteria based on gestational age. Early decelerations occur simultaneously with uterine contractions and may be associated with mild cord compression. Late decelerations occur after the contraction has ended and may indicate fetal distress or hypoxemia. It's worth noting that FHR patterns can change over time, and a single abnormal pattern does not necessarily indicate a problem. However, a consistent pattern of abnormalities can suggest that the fetus is at risk and requires further evaluation. The fetal heart rate (FHR) can experience a decrease, either early or late, as a result of uterine contractions. Early decelerations are benign and caused by vasovagal responses to fetal head compression, while late decelerations may indicate uteroplacental insufficiency, which can lead to decreased oxygen transfer to the fetus. Early decelerations occur simultaneously with the contraction's peak and resolve as the pressure resolves. They are commonly seen during active labor and have not been linked to fetal hypoxemia or acidosis. In contrast, late decelerations appear after the contraction's onset, nadir, and end, and can be classified into reflex and non-reflex types. Reflex late decelerations occur with normal FHR variability, while non-reflex decelerations are associated with diminished or absent variability. Uteroplacental insufficiency is a classic cause of late decelerations, resulting from problems with uterine perfusion, uterine activity, or placental function. These issues can lead to decreased maternal-fetal oxygen transfer, fetal hypoxemia, and lactic acidosis secondary to anaerobic metabolism. Increased sympathetic stimulation leads to high blood pressure, causing a vagally mediated heart rate decrease. This dual response may explain why heart rates often slow after contractions. Reflex late decelerations are associated with normal heart rate variability due to an intact central nervous system, whereas nonreflex late decelerations indicate decreased or absent variability and are linked to greater hypoxemia and fetal acidosis. Late decelerations without reflexes signify the fetus's inability to compensate for relative hypoxemia. This is often caused by placental dysfunction rather than uterine issues or maternal conditions like hypertension, diabetes, or heart disease. Other causes include premature rupture of membranes, placenta previa, intrauterine growth restriction (IUGR), and maternal fever. Fetal tachycardia can result from various factors, including maternal fever, infection, fetal distress, or medications affecting the nervous system. It's generally associated with increased sympathetic tone or decreased parasympathetic tone, indicating reduced variability in heart rate. However, if a deceleration is followed by an increase in heart rate, it may be a sign of hypoxemia, possibly due to the fetus trying to compensate for low oxygen levels. A prolonged deceleration can also be a concern, as it signifies a sustained decrease in heart rate below baseline, often lasting more than 2 minutes. This phenomenon is visually apparent and associated with various maternal conditions, including hypertension, preeclampsia, or infection, which may lead to fetal distress or complications. Prolonged fetal heart rate (FHR) decelerations can occur due to various mechanisms that normally cause periodic or episodic decelerations but fail to resolve. This is often associated with hypoxia. Factors such as cord compression, maternal hypotension or hypoxemia, tetanic uterine contractions, or head compression during labor may lead to prolonged decelerations. A FHR below 100 beats/min is concerning, and a drop below 60 beats/min requires immediate action due to potential fetal hypoxia. Variable decelerations are characterized by an abrupt decrease in FHR, with a sudden drop followed by a rapid return to baseline. This pattern may appear as a V, U, or W shape on the monitoring strip. These decelerations can be periodic or episodic and often include small accelerations before and after the main event, referred to as shoulders. The classic cause of variable decelerations is umbilical cord compression, which leads to occlusion of the vein and then the artery, causing reflex tachycardia followed by a protective reflex slowing of the heart rate. Severe variable decelerations are those lasting more than 60 seconds, falling below 70 beats/min, or showing a drop of 60 beats/min. While umbilical cord compression is typically responsible during labor, head compression during the second stage may also cause this pattern. Variable decelerations can be seen in various settings, including antepartum, early labor with oligohydramnios, after membrane rupture, and may indicate cord prolapse if membranes ruptured earlier. The presentation part's engagement during labor is crucial. During the descent phase, typically between 8-10 cm cervical dilation, factors like nuchal cords causing cord stretch or compression, or head compression due to rapid descent and maternal valsalva, might occur. Variable decelerations with normal FHR variability don't signify hypoxia but repetitive severe variable decelerations with diminished or absent FHR variability could indicate fetal distress. Uterine contractions can provide two key pieces of information: quantifying uterine activity (contraction strength) and contraction patterns (frequency, duration). Qualitative analysis through external monitoring or internal catheters assesses contraction patterns. These include regular contractions, polysystole, tachysystole, paired contractions, skewed contractions, tetanic contractions, and hypertonus. Normal labors exhibit contractions every 2-5 minutes lasting 30-60 seconds with gradual ascent and descent. Quantitative methods like Montevideo units (MVUs), Alexandria units, Active Planimeter units, Total planimeter units, and average rate of rise measure contraction strength and frequency. MVUs are most commonly used outside research protocols, defined as the sum of each contraction's intensity in a 10-minute period (in mmHG). Adequate uterine activity is above 200 MVUs, sufficient for 90% of labors to progress. Studies show more than 40% of women in spontaneous labors have MVUs > 300 mmHG. Baseline pressure refers to the resting tone or uterine pressure while relaxed, defined as greater than 25 mmHg or MVUs above 400. Tachysystole is six or more contractions in 10 minutes without fetal distress. Hypertonus is an abnormally high resting tone or excessive contraction strength. Tetanic contractions or polysystolic patterns occurring within a minute can be a sign of rapid and intense uterine activity. The most common triggers for this pattern are oxytocin and prostaglandins. Other types of contractions include paired ones, where consecutive contractions are followed by a prolonged period of inactivity before the next set begins. This pattern is sometimes linked to cephalopelvic disproportion. A skewed contraction occurs when the rise and fall of the uterine muscle fibers don't mirror each other exactly. While this phenomenon's significance isn't fully understood, it may be related to uneven relaxation of uterine muscle fibers.