FHR Case Presentation #2 Fetal Tachycardia

Expires Monday, July 31, 2023 Nursing Craig V. Towers , M.D., FACOG

Objectives

1. List the potential etiologies for fetal tachycardia.;
2. Categorize the various types of fetal tachycardia based on maternal versus fetal origin and state their differences. ;
3. Discuss the importance of the examination and other aspects of the fetal monitor tracing in helping to differentiate between the various causes for fetal tachycardia.

Article

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Fetal Tachycardia

Author: Craig V. Towers, M.D.

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2. Categorize the various types of fetal tachycardia based on maternal versus fetal origin and state their differences.
3. Discuss the importance of the examination and other aspects of the fetal monitor tracing in helping to differentiate between the various causes for fetal tachycardia.

Case Presentation:

A 22-year-old female Gravida 4 Para 1 entered labor and delivery with a complaint of discomfort related to regular uterine contractions. The patient was 39 weeks by her last menstrual period, but her prenatal care had been intermittent and she had no primary obstetrician that was on staff at the hospital. A history was obtained and she denied any major medical disorders, was on no medications except for vitamins, and stated that she had not had any problems during the pregnancy. She also confirmed that there had been good fetal movement. Her past obstetrical history was significant for two prior first trimester abortions completed by D & C and a prior term vaginal delivery of a 2750 gram male infant that was alive and well. Her vital signs were obtained revealing a blood pressure of 130/84, pulse of 112, and a temperature of 36.9^0C. When she arrived, she was placed on an external monitor that showed a tracing seen in Strip #1. A vaginal examination was performed that found her to be 5 centimeters dilated and completely effaced. The audible fetal heart rate from the monitor was consistent with the values being recorded. Her membranes were artificially ruptured revealing a small amount of amniotic fluid that was 3 to 4 plus meconium stained (Strip #2). Therefore, an amnioinfusion was initiated. By this time, an IV line had been placed and the patient was positioned on her left side.

Discussion:

Fetal tachycardia is defined by the baseline of the fetal heart rate. A normal fetal heart rate ranges between 110 beats per minute (bpm) on the lower end up to 160 bpm at the upper end (see ref. 1). When the fetal heart rate baseline is sustained above 160 bpm, it is considered to be tachycardic. A range of 160 to 180 bpm is considered to be a mild tachycardia by most authorities, whereas a sustained fetal heart rate above 180 bpm is considered to

be severe. For completion purposes, a sustained fetal heart rate baseline below 110 bpm is considered to be a bradycardia. Several etiologies exist as potential causes for fetal tachycardia, and these can be categorized into maternal and fetal. There are 4 basic maternal causes and these are:

- Maternal fever/infection
- {Chorioamnionitis}
- Maternal drug usage
- Maternal hyperthyroidism

There are 5 basic fetal causes and these are:

- Fetal hypoxia
- Fetal anemia
- Fetal infection/sepsis
- Fetal tachyarrhythmia
- Normal variant

Many of the causes for fetal tachycardia are the result of an increase in sympathetic and/or a decrease in parasympathetic tone, which will often decrease the variability and may eliminate fetal heart rate accelerations. By way of review, an "acceleration" in a pregnancy at or beyond 32 weeks gestation is an increase in the heart rate that peaks at least 15 beats per minute above the baseline and lasts for at least 15 seconds from the time the heart rate leaves the baseline to when it returns to the baseline. An "acceleration" in a pregnancy less than 32 weeks gestation is an increase in the heart rate that peaks at least 10 beats per minute above the baseline and lasts for at least 10 seconds from the time the heart rate leaves the baseline to when it returns to the baseline

The most common cause behind the development of fetal tachycardia is maternal fever. In the presence of a fever, the heart rate, in most cases, will increase somewhat. This phenomenon is also seen with the fetal heart rate when a mother is febrile. Thus, this is a normal physiologic response and is not harmful to the fetus. However, fevers are almost always associated with infection, whether viral or bacterial. Therefore, when a mother is febrile, an evaluation for a source of infection needs to occur. The most common sources for infection in a mother of child bearing age, who is not in labor, are viral in origin and include the common cold or flu, intestinal, or other viruses, such as herpes, Epstein-Barr, hepatitis, cytomegalovirus (CMV), parvovirus B-19, coxsackievirus, etc. (the full list is too extensive for this report). Common bacterial sources are pneumonia, urinary tract infections, appendicitis, and cholecystitis. However, it is extremely important to note that a basic urinary tract infection (UTI) or cystitis is very unlikely to produce a fever, because the infection is not systemic. If the urinary tract is the source of the infection, then the patient most likely has pyelonephritis and will probably need intravenous antibiotics. The bacterial infections will usually have distinctive clinical findings such as abnormal lung auscultation findings for pneumonia, flank pain for pyelonephritis, and abdominal pain for appendicitis or cholecystitis.

Chorioamnionitis is an infection, but it is listed separately because it directly involves the pregnancy. Chorioamnionitis is the most common cause for fever in a laboring patient, especially if the membranes are ruptured. In fact, two large series on laboring patients identified an incidence of chorioamnionitis of 7% (see ref. 2 & 3). Therefore, when a laboring patient (who initially was afebrile) develops a fever, the cause is chorioamnionitis until proven otherwise. To further emphasize this point, if a patient suddenly develops a fever while in labor, (who when admitted was completely asymptomatic from an infection standpoint) it is very unlikely that some viral infection has developed and it is also unlikely that she has suddenly developed bacterial pneumonia, pyelonephritis, appendicitis, or cholecystitis. If she has a Foley catheter in place or has been catheterized several times, she could develop a bladder infection, but again, this will not make her febrile. The most likely source is going to be the intrauterine cavity – i.e. chorioamnionitis. Even though the first clinical sign in chorioamnionitis is usually a maternal fever, and the cause for the fetal tachycardia is probably the fever, it is listed separately, because chorioamnionitis can lead to maternal and or fetal sepsis, if left untreated. Thus, it is recommended by the American College of Obstetrics & Gynecology that the mother receive intravenous broad-spectrum antibiotic therapy (see ref. 4). The fetal monitor tracings that are seen with maternal fever will often show moderate to minimal variability, but not absent variability. In addition, there often are no accelerations (though accelerations can occur in some cases), but there should not be any decelerations either.

Another common maternal cause for fetal tachycardia is drug usage. There are numerous drugs that might produce a fetal tachycardia. Some of the more common ones include over-the-counter decongestant medications (and other cold remedies), caffeine, asthma medications (beta-sympathomimetic agents such as terbutaline – a drug that is also used to treat premature contractions), parasympathetic blocking drugs (such as atropine, scopolamine, and some phenothiazines) and stimulant drugs of abuse (such as amphetamines or cocaine). The fetal tachycardia that is seen with maternal drug usage is similar to that seen with maternal fever. The tracing will often show moderate to minimal variability, but not absent variability. In addition, because of the increase in sympathetic and/or decrease in parasympathetic tone (depending on the drug), there often are no accelerations. However, again, there should not be any decelerations related to these agents either.

Maternal hyperthyroidism is an uncommon finding, but when present, may be responsible for fetal tachycardia. The actual etiology is believed to be related to an antibody that women may have in certain cases of hyperthyroidism. As a general rule, thyroid hormone does not cross the placenta and therefore, the fetus is usually unaware of the mother's level (whether it is elevated, normal, or low). However, some cases of hyperthyroidism are caused by an antibody, called thyroid-stimulating antibody, which attacks the thyroid gland and causes an increase in thyroid hormone production. This antibody is commonly found in patients with Graves's disease, but it can also be found in other thyroid disorders. This antibody has been documented to cross the placenta. The fetal monitor tracings in the few cases reported to date often appear normal with accelerations and moderate or normal variability (see ref. 5). Again, decelerations should not be present.

Regarding fetal causes for tachycardia, a fetal tachyarrhythmia usually has a rate that exceeds 200 bpm (and often will exceed 240 bpm) (see ref. 6). In these situations, the fetal heart rate will not record on the strip or the logic within the monitor itself will artificially halve the value. Therefore, it is possible to obtain a recording of 100 to 200 bpm on the strip when the actual fetal heart rate is 200 to 400 bpm. For example, if the true fetal heart rate is 280, the logic in the monitor may halve this and print out a rate of 140. The distinction comes in the difference between the audible heart rate sound and what is recorded. In addition, in this situation, because the monitor is artificially changing the rate, the recorded heart rate usually has absent variability. Furthermore, it is very unlikely that there will be any fetal movement noted. An area that needs to be addressed is if the fetal tachyarrhythmia is occurring at a rate between 220 and 320, and the monitor is artificially halving the rate. In this situation, the recorded value on the tracing will fall between 110 and 160 and would not actually exceed 160 (the definition level of fetal tachycardia). Fetal tachyarrhythmias can include atrial flutter, atrial fibrillation, and paroxysmal atrial tachycardia or supraventricular tachycardia. Atrial flutter and atrial fibrillation, though extremely rare, will produce very fast atrial rates (usually greater than 300 bpm). These arrhythmias are usually seen with some form of atrioventricular block so that the ventricular rate is slower. The most common tachyarrhythmia is supraventricular tachycardia (SVT), which is believed to be most often caused by a re-entry of electrical activity (thereby causing a circular motion of cardiac conduction). The main concern with tachyarrhythmias is that if sustained (for numerous hours), the fetus can develop cardiac failure leading to non-immune hydrops. If SVT or some other tachyarrhythmia occurs at a preterm gestation, the mother may be treated with such medications as digoxin, calcium channel blockers, quinidine, or beta-blockers, in hopes of causing inutero cardioversion.

Fetal tachyarrhythmias are rarely concerning from an acute management point of view, which is in contrast for 3 of the remaining 4 fetal causes (fetal hypoxia, anemia, and infection/sepsis). If a sustained fetal heart rate above 160 bpm is due to hypoxia, this tachycardia should be preceded by repetitive late or variable decelerations. This is an important issue to understand. When repetitive decelerations occur, over a period of time a fetus may lose its reserve and begin to decompensate. Fetal heart rate accelerations will no longer occur and eventually the baseline can begin to rise and become tachycardic. If the hypoxia continues, the variability will decrease and eventually become absent. By the time this occurs, fetal acidosis is usually present and if allowed to continue without delivery, the strip may progress to a blunted smooth pattern with an unstable baseline that then becomes agonal leading to inutero death.

Fetal anemia is uncommon but when present can also produce a fetal heart rate that is sustained above 160 bpm. Fetal anemia can occur from an increase in fetal red cell destruction (as seen with Rh sensitization or other red blood cell antibodies), a lack of red cell production (as seen with parvovirus B-19, CMV, and other viral infections), and fetal bleeding (as seen with a fetal-maternal hemorrhage or a ruptured fetal blood vessel – such as a bleeding vasa previa). Many of these causes can be determined from laboratory testing, such as a positive antibody screen that detects a red blood cell antibody or a positive Kleihauer-Betke test in the case of a fetal-maternal hemorrhage. When a fetal anemia develops slowly (over several days), the fetal monitor tracing will often demonstrate a sinusoidal pattern, rather than a tachycardia. If the anemia is sudden in onset (for example from a

sudden large fetal-maternal hemorrhage or fetal bleeding from a ruptured vasa previa), a transient fetal tachycardia may develop, but if the problem is not rapidly corrected, the fetal heart rate will drop into a prolonged deceleration / bradycardia pattern. The fetal tachycardia seen with fetal anemia will often show minimal to absent variability, no accelerations, and there might be intermittent or repetitive decelerations. In many cases, the final diagnosis may not occur until after delivery when anemia is found in the newborn based on laboratory testing.

The third acutely concerning fetal cause for tachycardia, is fetal infection/sepsis. When a patient is septic, they are often febrile (which by itself can increase the heart rate), and multiple organs are usually affected. When the cardiac system becomes affected, it often responds with a tachycardia (which is a compensatory mechanism that tries to overcome the failing cardiac muscle contractility). Again, this same phenomenon occurs when a fetus is septic. Most cases of fetal sepsis come from an ascending infection in cases after the membranes have ruptured. Though fetal sepsis in the presence of intact membranes has been reported, it is an extremely rare disorder (such organisms as Listeria, Group B Streptococcus, and E. Coli have been identified). The fetal heart rate pattern in cases of fetal sepsis is usually similar to that of anemia with minimal to absent variability, no accelerations, and there might be intermittent or repetitive decelerations.

The final etiology is a normal variant. This category was left for last because it is also rare and needs to be a diagnosis of exclusion. Though the normal fetal heart rate baseline is 110 to 160, a sustained baseline above 160 bpm may be normal for some babies, but usually this will not exceed 180 bpm. In this case, the fetal heart rate pattern should otherwise appear normal showing moderate variability with accelerations and no decelerations.

Case Presentation Outcome and Summary:

Over the next 2 hours, the patient progressed in labor and received an epidural for pain management. She remained afebrile but was slightly tachycardic in the 105 to 115 bpm range. When she reached complete dilation, the amnioinfusion was discontinued and she was moved to the delivery room. The fetal tracing prior to delivery is seen in Strip #3 & Strip #4. She went on to spontaneously deliver a 2600-gram female infant with Apgar scores of 6 and 9 for 1 and 5 minutes, respectively. The neonate was DeLee suctioned on the perineum once the head delivered but prior to delivery of the body. The neonatal team was in attendance because of the tracing, as well as the meconium, and no meconium was found below the vocal cords after the delivery. However, the newborn was noted to be "jittery" and thus was taken to the nursery under observation.

In looking at the first 2 tracings, we see a steady fetal heart rate of about 180 with no accelerations or decelerations, despite frequent contractions. The overall variability is minimal, but not absent. She was afebrile with intact membranes and she denied any issues of decreased fetal movement. In looking at the differential, she did not have a fever and maternal hyperthyroidism is unlikely. Likewise, a fetal tachyarrhythmia was ruled out based on auscultation and it is also unlikely to be hypoxia since there are no decelerations. It is also not likely to be a normal fetal variation. In looking at the 4 remaining choices, chorioamnionitis and fetal infection/sepsis are possible, but not at the top of the list because she had intact membranes and was afebrile. Fetal anemia is a possibility but maternal drug usage is much more common. The management at this point would be to follow the labor course and proceed with vaginal delivery, if she progressed and no significant decelerations developed. Tracings #3 and #4 continue to depict a mild tachycardia, but the variability is improved and there are no decelerations. Fetal tachycardia by itself is not an indication for cesarean section. However, the pediatric team should be notified so that an evaluation post delivery can occur.

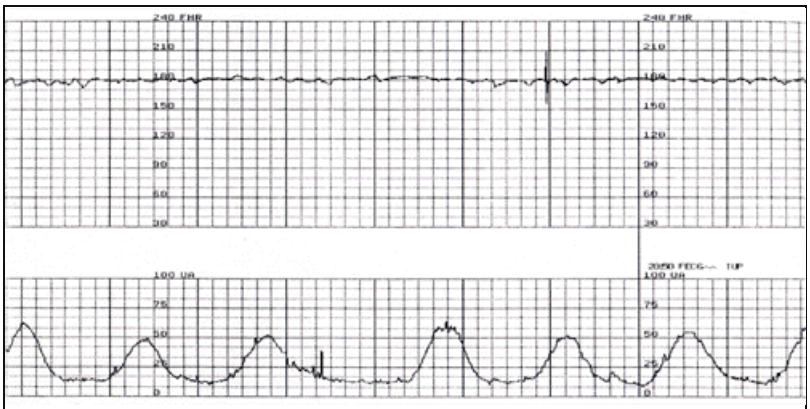
Because the child continued to be "jittery and fussy", a drug screen was obtained that returned positive for amphetamines. In further questioning of the mother, she no longer had custody of her first child because of drug abuse related issues and she admitted to using amphetamines shortly before she arrived in labor and delivery.

In review, the differential for fetal tachycardia involves 4 maternal causes (fever, chorioamnionitis, medications or drugs, and hyperthyroidism) and 5 fetal causes (tachyarrhythmia, hypoxia, anemia, infection/sepsis, or a normal variant). When presented with a case of fetal tachycardia on admission, the etiology can be narrowed by evaluating the patient's history (including fetal movement and medication usage), her vital signs, and evaluating the strip for the quality of the variability and the presence or absence of accelerations and decelerations.

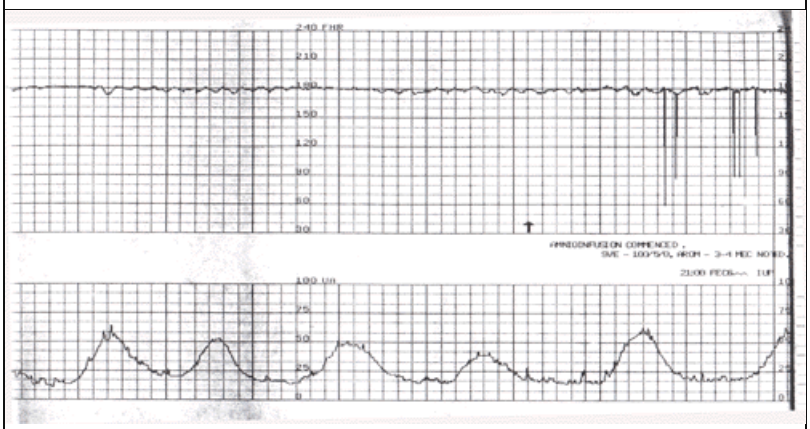
From a clinical point of view, most cases of fetal tachycardia will develop during the course of labor and this will

effectively rule out maternal hyperthyroidism, fetal tachyarrhythmia, and a normal fetal variant. The most common cause will be fever, which is often related to developing chorioamnionitis, especially if the membranes are ruptured. Fetal hypoxia should be watched for closely if there have been repetitive late or variable decelerations. If there is any question regarding whether or not the tachycardia is related to fetal hypoxia, an arterial cord blood gas at the time of delivery can be helpful. Any medications used during labor will be noted by the healthcare providers. Thus, if fetal tachycardia develops, the timing can be compared to the time of drug administration. The final two (fetal anemia and infection/sepsis), though not common, will remain on the list but in a secondary capacity.

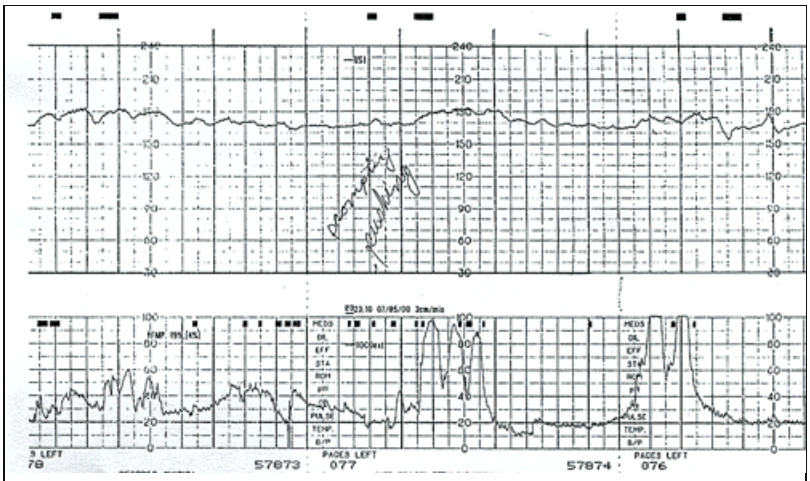
As previously stated, it would be prudent to notify the newborn healthcare providers when fetal tachycardia develops. Though the most common cause is fever (often related to chorioamnionitis), most neonates delivered in this situation do not become infected and therefore are essentially unaffected. However, there are several laboratory studies that can be performed on the neonate to help further delineate (rule in or out) four of the causes for fetal tachycardia. These include a hemoglobin and hematocrit for anemia, blood gases for hypoxia, blood cultures for fetal infection, and drug screens for maternal drug usage. At the conclusion of the birth, if the mother has no thyroid issues, was never febrile, and chorioamnionitis did not occur, and if a fetal tachyarrhythmia was not present, and the newborn lab work was all negative, then the cause for the fetal tachycardia is most likely a normal variant – i.e. a diagnosis of exclusion.



Strip #1



Strip #2



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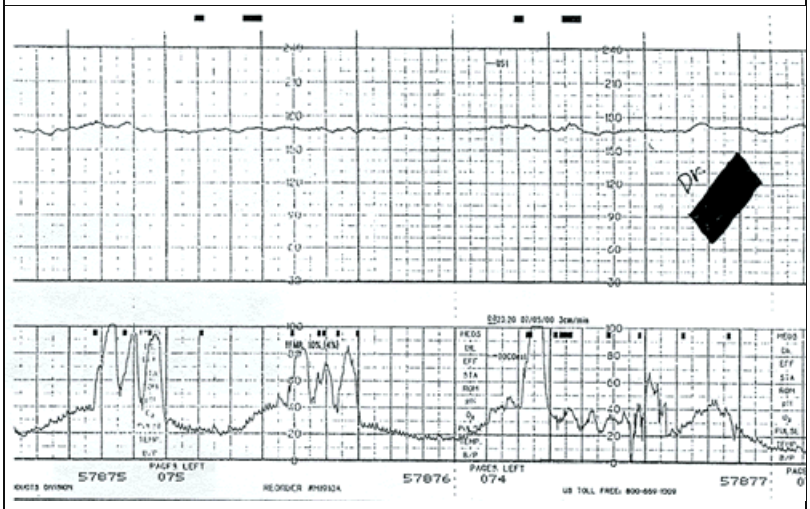

Strip #4

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About the Author(s)

Dr. Towers is currently Professor and Vice Chair of the Department of Obstetrics & Gynecology at University of Tennessee Medical Center Knoxville in the Division of Maternal-Fetal Medicine. He is still clinically active managing numerous high-risk pregnancies. He is also actively involved in research with over 90 publications in major medical journals. Though his research has spanned many areas in obstetrics, he has primary interests in drugs in pregnancy, infections in pregnancy, fetal heart monitoring, bleeding in pregnancy, and fetal lung maturity.

He has authored a book for consumers regarding the safety of over-the-counter medications that are used in treating the common cold entitled “I’m Pregnant & I Have a Cold – Are Over-the-Counter Drugs Safe to Use?” published by RBC Press, Inc. He is also one of the new Editors of the reference book for clinical care providers entitled “Drugs in Pregnancy and Lactation, published by Wolters & Kluwer.