

Evaluation of Fetal Arrhythmias

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Abstract

Fetal arrhythmias are not uncommon. The diagnosis of a fetal arrhythmia is challenging and normally requires referral for a detailed fetal echocardiogram. The first step in the ultrasound evaluation should be distinguishing whether the arrhythmia is an irregular rhythm, a bradycardia, or a tachycardia. This can be done by evaluating the arrhythmia using simultaneous atrial and ventricular M-mode or pulsed Doppler. Although the majority of fetal arrhythmias are self-limited and benign, some are potentially life-threatening for the fetus and for these cases a multidisciplinary approach to treatment may be required.

Learning Objectives

- Know the different types of fetal arrhythmias
- Understand how fetal arrhythmias are diagnosed
- Know which fetal arrhythmias require treatment

Keywords: Fetal arrhythmia, fetal echocardiogram, pregnancy complication.

INTRODUCTION

Fetal arrhythmias are common and occur in 1 to 3% of all pregnancies.¹ Up to 20% of referrals for a fetal echocardiogram are for the indication of an abnormal heart rhythm.² Arrhythmias can be divided into one of three types: Irregular heart rhythms; sustained tachycardia, defined as a persistent heart-rate greater than 180 beats per minute (bpm); and sustained bradycardia, defined as a persistent heart-rate less than 100 bpm. Fortunately, only 15% of fetal arrhythmias are life-threatening for the fetus. The management of potentially lethal arrhythmias often requires a multidisciplinary approach involving an obstetrician, maternal fetal medicine specialist, pediatric cardiologist, adult cardiologist, neonatologist and sometimes an electrophysiologist. The basic evaluation of a suspected fetal arrhythmia is outlined in this review.

EVALUATION OF FETAL ARRHYTHMIAS

The evaluation of a suspected arrhythmia should begin with a referral for a fetal echocardiogram performed by a physician with specialized training in the evaluation of fetal heart disease. The evaluation of a fetal arrhythmia is limited because with current technology the electrical cardiac activity of the fetus cannot be measured directly. Unlike an

electrocardiogram, ultrasound does not measure electrical signals. However, ultrasound can measure motion and the motion of the fetal heart can be used to interpret the fetal rhythm. The current standard of care for the evaluation of fetal arrhythmia is a fetal echocardiogram to evaluate cardiac anatomy, function and rhythm. Newer technology, such as fetal magnetocardiography, provide valuable information about the fetal heart rhythm, however its availability is currently limited to a limited number of referral centers. In the case of an arrhythmia it is important to confirm normal cardiac anatomy as this can significantly affect prognosis and management. In addition, an assessment of cardiac function is an important component of the evaluation of an arrhythmia. The standard technique for evaluation of a suspected fetal arrhythmia is with simultaneous atrial and ventricular M-mode or pulsed Doppler.

M-mode assessment of the fetal heart rhythm is achieved by interrogating the fetal heart from the apex or the base. The cursor is placed so that atrial and ventricular wall motion is recorded simultaneously (Figure 1). The atrioventricular (AV) valve motion is normally recorded as well. From an M-mode recording, the fetal heart-rate and rhythm can be determined. Often it is difficult to appreciate atrial contractions with M-mode because the wall motion of the atrium is relatively minimal.

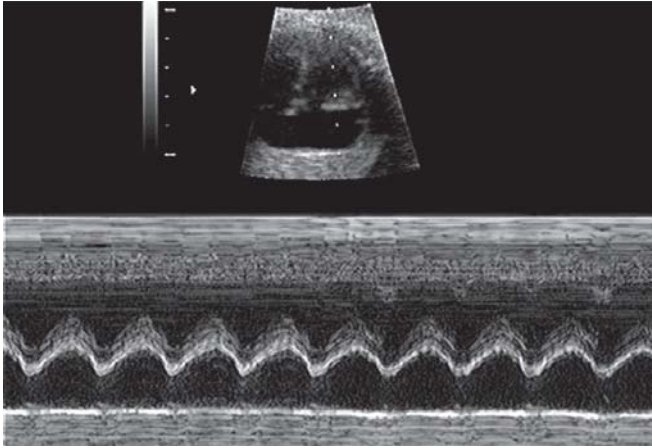


Figure 1: Simultaneous atrial and ventricular M-mode

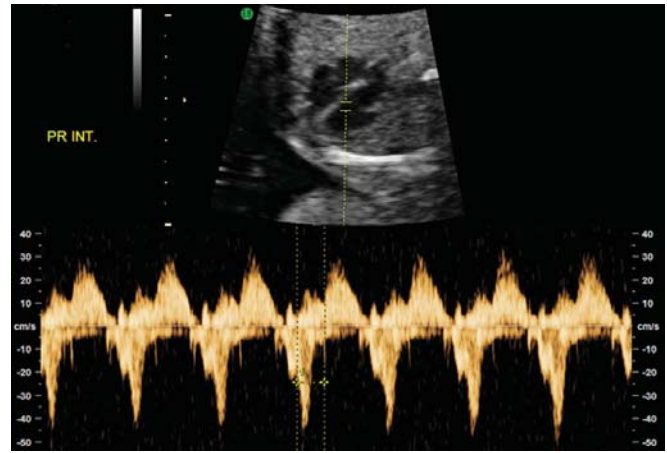


Figure 3: Pulsed Doppler of the mechanical PR interval

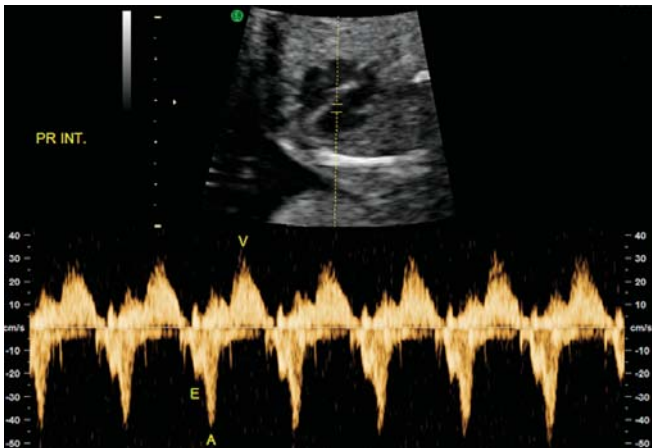


Figure 2: Pulse Doppler of mitral inflow and aortic outflow

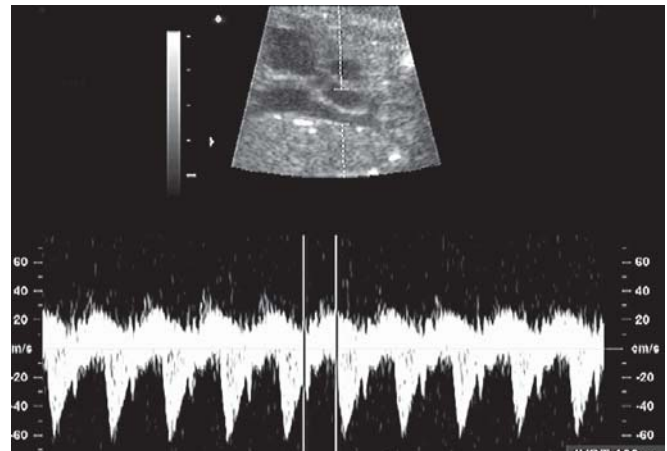


Figure 4: Pulsed Doppler of the aorta and superior vena cava

Pulsed Doppler measurement uses blood flow rather than wall motion to assess the fetal heart-rate and rhythm. This can be performed by simultaneously measuring mitral inflow and aortic outflow (Figure 2). Pulse Doppler measurements are normally performed from the apex or base of the heart using the five chamber view. Once the correct view is obtained, the pulse Doppler gate is placed in the left ventricle below the mitral and aortic valves. The resulting wave form displays the *e* wave, which represents passive ventricular filling as blood flows from the atrium to the ventricle, the *a* wave, which represents flow across the mitral valve during atrial systole, and the *v* wave, which represents ventricular systole as blood exits the ventricle through the aorta. In addition to the heart-rate and rhythm, the mechanical PR interval can be measured using this technique (Figure 3). The mechanical PR interval represents the time between atrial and ventricular contractions, and

this can be used to diagnose congenital heart block. Pulsed Doppler can also be used to assess the fetal heart rhythm from a sagittal view by placing the gate over the aorta and the superior vena cava (Figure 4). From this measurement, the *v* wave representing ventricular systole can be seen. The *a* wave is represented by a small reversal of flow in the superior vena cava during contraction of the right atrium. This view is particularly useful for determining the time interval between ventricular and atrial contractions in cases of supraventricular tachycardia (SVT).

Fetal arrhythmias can be divided into irregular rhythms, sustained tachycardia and sustained bradycardias.

IRREGULAR RHYTHM

The majority of fetal arrhythmias are irregular rhythms. Premature atrial contractions (PAC), premature ventricular contractions (PVC), and a sinus arrhythmia are all causes of an irregular rhythm in the fetus.

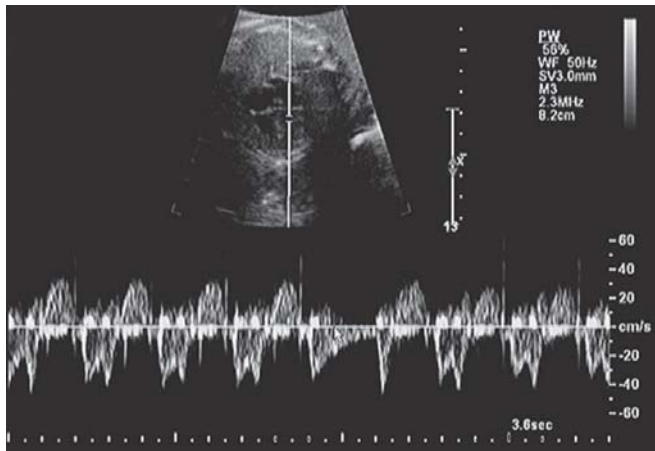


Figure 5: Pulsed Doppler of a nonconducted premature atrial contraction

PACs are the most common cause of an irregular heart rhythm in the fetus and are often diagnosed on routine office auscultation of the fetal heart-rate. PACs are caused by an ectopic electrical impulse originating in the atrium that occurs in advance of the sinoatrial node (SA). The early impulse can be blocked or conducted through the AV node. Isolated blocked or nonconducted PACs result in a skipped ventricular beat, a compensatory pause, and then an atrial contraction originating from the SA node. Using pulsed Doppler from the five chamber view of the fetal heart, a PAC does not have the biphasic *e-a* wave morphology across the mitral valve as is seen with a normal atrial contraction (Figure 5). Because the atrial contraction occurs prematurely, it is not preceded by passive filling of the left ventricle. Therefore, there is a single monophasic *a* wave, without a preceding *e* wave. By measuring the time between *a* waves, it can be seen that the premature *a* wave occurs earlier than the previous *a* wave. After the monophasic *a* wave, there is compensatory pause during repolarization of the conduction system. Because the left ventricle did not contract, the pressure remains elevated so that passive ventricular filling does not occur prior to the next atrial contraction. This is reflected in a second monophasic *a* wave that occurs after the blocked PAC. The atrial contraction is conducted to the ventricle as represented by a *v* wave and then there is a return to normal sinus rhythm with a biphasic *e-a* wave at the normal interval.

Similar to a blocked or nonconducted PACs, conducted PACs are reflected by an early atrial contraction that occurs before passive ventricular filling (Figure 6). This results in an early *a* wave without a preceding *e* wave. In contrast to

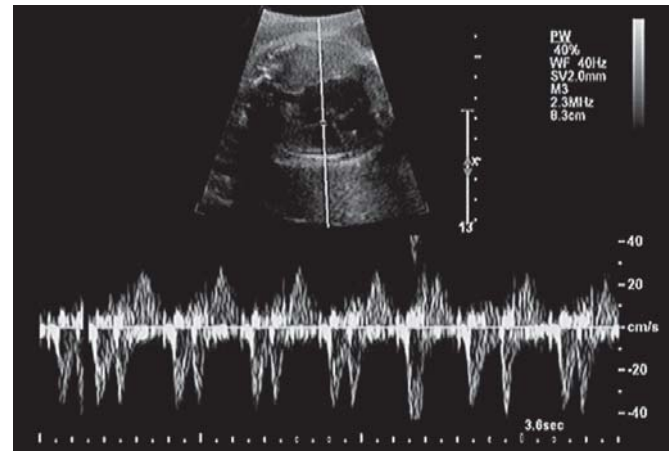


Figure 6: Pulsed Doppler of a conducted premature atrial contraction

a blocked PAC, a ventricular contraction follows a conducted PAC. Therefore a normal *v* wave is seen following the monophasic *a* wave. The ventricular contraction is then followed by passive filling of the ventricle, as represented by an *e* wave across the mitral valve. There is a brief compensatory pause prior to the *a* wave that represents repolarization of the conduction system. This is represented by a widening of the *e-a* wave following a conducted PAC and then a morphologically normal appearing *v* wave.

Isolated PACs are usually benign and resolve spontaneously without treatment. Importantly, 1% of fetuses with PACs have an associated cardiac abnormality, so a fetal echocardiogram should be considered when an irregular rhythm is diagnosed.³ In some cases a foramen ovale aneurysm is seen, and it is theorized that contact between the aneurysm and the wall of the left atrium results in a PAC⁴ (Figure 7). Also of concern is the propensity for PACs to convert to a tachyarrhythmia (Figure 8). This may be more common in fetuses with complex ectopy, such as atrial bigeminy and trigeminy.⁵ Patients with PACs are routinely instructed to avoid caffeine and sympathomimetic drugs, though a direct correlation has never been established. Patients could be counseled about the symptoms of a tachyarrhythmia, such as a decrease in fetal movement or a sudden increase in abdominal girth secondary to polyhydramnios. The monitoring could consist of weekly office auscultation of the fetal heart-rate to exclude the development of a tachyarrhythmia and for parental reassurance. A follow-up echocardiogram should be performed if a tachyarrhythmia is suspected, as a tachyarrhythmia places the fetus at risk of heart failure.



Figure 7: Foramen ovale aneurysm

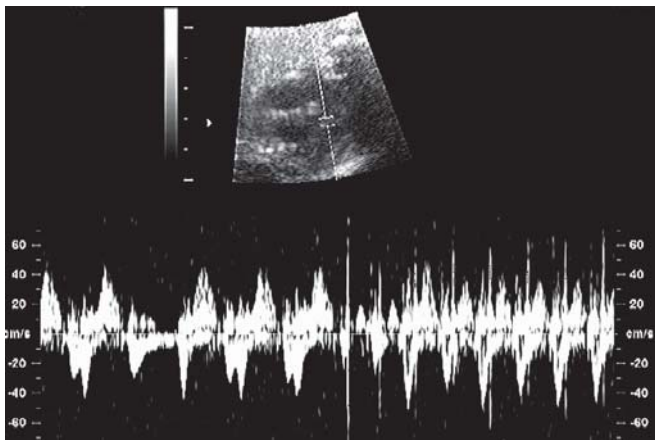


Figure 8: Pulsed Doppler of supraventricular tachycardia following a premature atrial contraction

PVCs are rare. A PVC is an early contraction of the ventricle without a preceding atrial contraction. With pulsed Doppler, this would be reflected in a v wave that occurs early without a preceding a wave.

BRADYCARDIA

A fetal bradycardia is defined as a persistent fetal heart-rate less than 100 bpm. Fetal bradycardias can be subdivided into 3 basic types: Sinus bradycardia, blocked atrial bigeminy, and second and third degree AV block.

In sinus bradycardia the atrial to ventricular conduction is 1:1, with a rate less than 100 bpm, that persists for 10 minutes or more. Causes of sinus bradycardia include fetal distress, long QT syndrome, structural cardiac anomalies, hypothyroidism and an ectopic atrial pacemaker. Distinguishing fetal distress from other causes of a fetal bradycardia

can be critical as the management of fetal distress is often delivery. Importantly, the clinician should not rush to deliver a fetus with a sustained bradycardia until other causes have been excluded. One to one conduction between the atria and ventricles distinguishes sinus bradycardia from blocked atrial bigeminy and atrioventricular block.

Blocked atrial bigeminy can result in a ventricular heart rate less than 100 bpm. It is caused by alternating blocked PACs and a normal sinus beat. Because every other atrial contraction is premature and blocked at the AV node, the rhythm appears similar to 2:1 AV block. The distinction can be made by measuring the time interval between each atrial contraction. With atrial bigeminy, each normally conducted atrial beat is followed by a PAC that occurs earlier than the subsequent atrial beat. In contrast, the atrial contractions in 2:1 AV block occur at a regular interval that does not vary from contraction to contraction. Blocked atrial bigeminy can be demonstrated by pulsed Doppler evaluation from the five chamber view (Figure 9). With this technique each blocked PAC is followed by a normally conducted atrial contraction, and the time interval between each atrial contraction can be measured. Blocked atrial bigeminy is considered to be a benign arrhythmia that is not associated with fetal compromise. Fetuses with blocked atrial bigeminy could be monitored to ensure fetal well-being and to exclude the development of a tachyarrhythmia which could result in fetal compromise. Fortunately blocked atrial bigeminy normally resolves spontaneously before delivery.⁶

Heart block can be diagnosed in a fetus with ultrasound. First degree AV block is diagnosed when the mechanical PR interval exceeds 150 msec⁷ (Figure 10). Measurement of the mechanical PR interval can be performed using pulsed Doppler in the five chamber view. The gate is placed so that flow across the mitral and aortic valves is recorded simultaneously. The distance between the start of the a wave to the start of the v wave is measured in time. Currently, frequent assessment of the mechanical PR interval is recommended to screen for the development of heart block in fetuses at risk because of maternal Sjorgen's antibodies (SSA/anti-Ro and SSB/anti-La). It is thought that these antibodies cross the placenta and result in immune-mediated inflammation and fibrosis of the fetal conduction system at the AV node. These antibodies may also cause cardiomyopathy, endocardial fibroelastosis and serositis. The peak onset of isolated AV block secondary to maternal antibodies is between 20 to 24 weeks, and it rarely develops

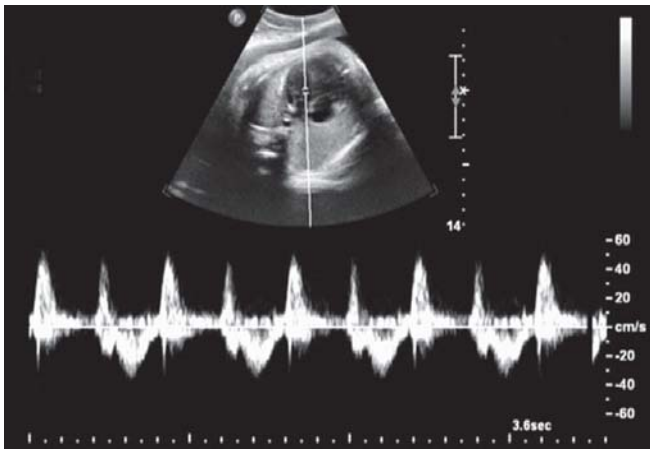


Figure 9: Pulsed Doppler of blocked atrial bigeminy

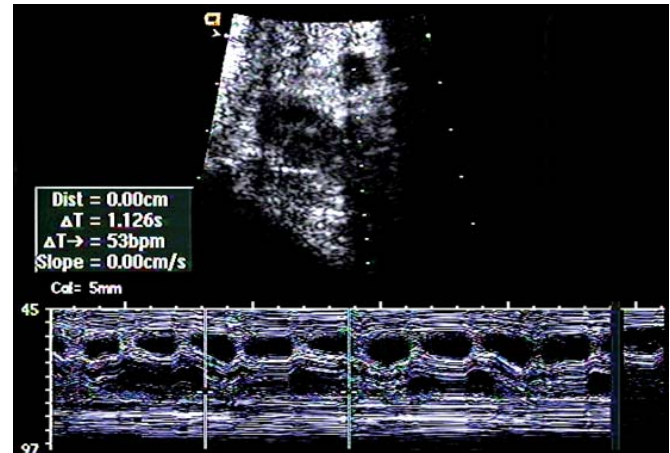


Figure 12: M-mode of complete heart block

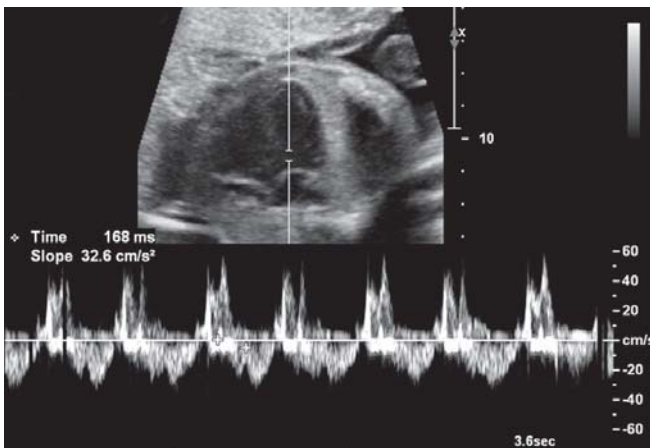


Figure 10: Pulsed Doppler of first degree heart block

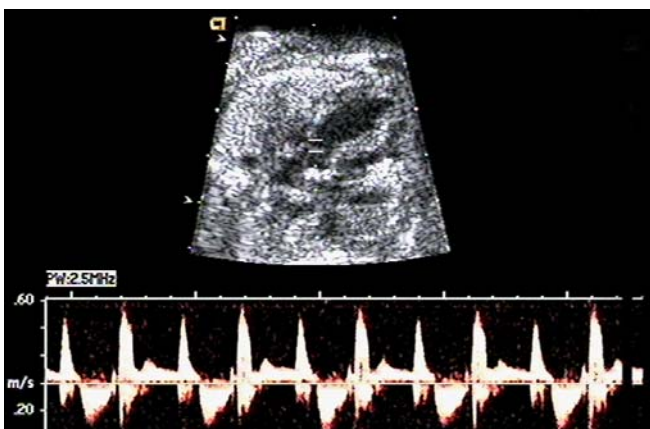


Figure 11: Pulsed Doppler of second degree heart block

beyond 34 weeks. If maternal serologies are positive, then the fetus has a 1 to 2% risk of developing complete heart block. If a previous pregnancy has been affected with complete heart block secondary to maternal antibodies, the

recurrence risk may be as high as 16 to 18%. It has been proposed that treatment of first degree heart block with dexamethasone may prevent the progression to complete heart block, however this has yet to be confirmed in a large study.⁸

In second degree heart block, there is 2:1 conduction with a ventricular rate that is normally 60 to 100 bpm (Figure 11). If the heart structure is normal and the antibody screen is negative, then the patient can be reassured. In third degree or complete heart block there is complete dissociation between the atria and ventricles. This can be seen using M-mode or pulse Doppler to demonstrate a normal atrial rate and a ventricular bradycardia (Figure 12). Complete heart block may be caused by maternal Sjogren's antibodies. Complex heart defects, such as heterotaxy (polysplenia/left atrial isomerism), L-transposition of the great vessels, or cardiac tumors, are associated with approximately 50% of cases of complete heart block.²

If a fetus is diagnosed with AV block, a fetal echocardiogram is indicated to determine if there is an associated structural heart defect. An assessment of maternal antibodies (SSA/anti-Ro and SSB/anti-La) is also recommended. Frequent follow-up with at least weekly ultrasounds should be performed to ensure fetal well-being and to exclude the development of heart failure. Transplacental treatment with dexamethasone should be considered for autoimmune mediated heart block, as it may prevent progressive heart block in fetuses with first or second degree block and it may prevent further damage to the myocardium in fetuses with third degree block.⁹

If persistent blocked atrial bigeminy or heart block is present during labor, some experts recommend a cesarean delivery because these arrhythmias limit the interpretation

of fetal heart-rate monitoring. Others have suggested that fetal well-being can be assumed if there is variability in the ventricular heart-rate and an absence of decelerations, or with periodic biophysical profiles during labor. For this reason the management of labor and delivery during blocked atrial bigeminy or heart block should be individualized based on a discussion about the limitations of ensuring fetal well-being.

TACHYCARDIA

Fetal tachycardia is defined as a sustained heart-rate greater than 180 bpm. Persistent tachycardia in the fetus can result in the development of hydrops within 24 to 48 hours depending on the gestational age. The causes of fetal tachycardia include sinus tachycardia, supraventricular tachycardia (SVT), atrial flutter, and ventricular tachycardia. Normally these can be distinguished by determining the rate and rhythm with M-mode or pulsed Doppler as previously outlined.

In sinus tachycardia the rhythm is 1:1 and the rate is normally 180 to 200 bpm. The onset and offset of the arrhythmia is usually gradual. Causes include hypoxia, maternal pain, maternal fever, intraamniotic infection, hyperthyroidism and drugs (beta-adrenergic or vagolytic drugs). When the heart-rate exceeds 200 bpm and there is AV synchrony, SVT should be considered. In SVT the rhythm is 1:1 and the rate is normally 240 to 280 bpm (Figures 13 and 14). Unlike sinus tachycardia, the onset and offset are usually abrupt. Fetal SVT is caused by an accessory conduction pathway, such as Wolff-Parkinson-White syndrome. In some cases it may be beneficial to distinguish between short ventriculoatrial and long ventriculoatrial tachycardia, as this may influence the choice of an antiarrhythmic agent.² The distinction can be made by placing the pulse Doppler gate over the superior vena cava (SVC) and the ascending aorta in a sagittal view of the fetus (Figure 4). Often this requires opening the pulse gate wide enough so that flow through the aorta and SVC can be measured simultaneously. From this view the *v* wave is represented by forward flow through the ascending aorta and the *a* wave is represented by a small retrograde wave form in the SVC during atrial systole.

Measurements of the *v-a* and *a-v* intervals are then made. In short ventriculoatrial tachycardia, the *v-a* interval is shorter than *a-v* interval, as is seen in atrioventricular re-entry tachycardia. In long ventriculoatrial tachycardia, the

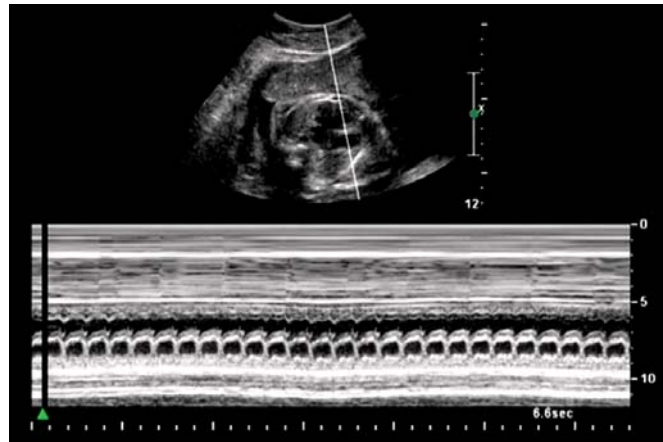


Figure 13: M-mode of supraventricular tachycardia

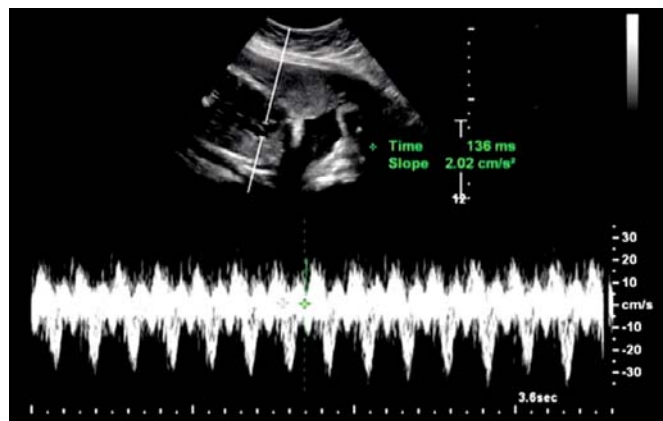


Figure 14: Pulsed Doppler of supraventricular tachycardia

v-a interval is longer than the *a-v* interval, which can be seen in atrial ectopic tachycardia or paroxysmal junctional reciprocating tachycardia. Again, some experts recommend choosing an antiarrhythmic medication based on the type of SVT. Atrial flutter can be distinguished from SVT by an atrial rate that exceeds the ventricular rate. Often the atrial rate is as high at 400 to 480 bpm and there is a varying degree of heart block that results in a slower ventricular rate (Figure 15). In contrast, ventricular tachycardia is characterized by a ventricular rate that exceeds the atrial rate. The ventricular rate in ventricular tachycardia is normally between 180 to 200 bpm and, because of AV dissociation, the atrial rate may be within the normal range of 120 to 160 bpm.

In cases of fetal tachycardia, a detailed fetal echocardiogram should be obtained to determine the type of tachycardia, evaluate the cardiac structure and function, and assess fetal well-being. Admission for 12 to 24 hours of continuous fetal monitoring is normally recommended

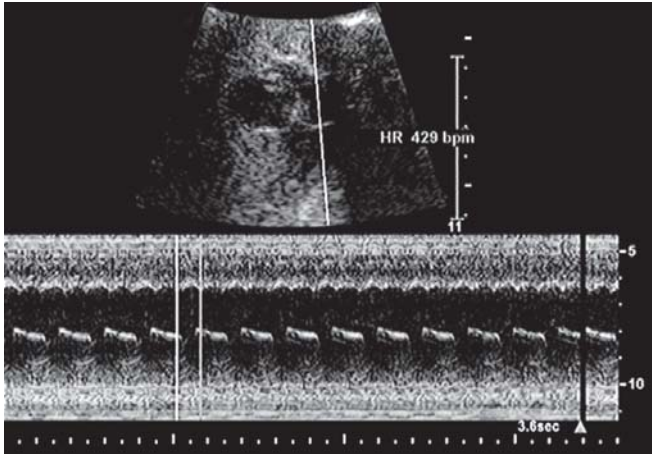


Figure 15: M-mode of atrial flutter

to determine if the tachycardia is sustained. Sustained tachycardia is defined as tachycardia present for more than 12 of 24 hours. Nonsustained tachycardia in the absence of hydrops can often be managed as an outpatient with fetal kick counts, nonstress tests to exclude the development of a sustained tachycardia and to ensure fetal well-being, and ultrasounds to exclude the development of hydrops. Importantly nonsustained tachycardia can become sustained.⁶

The management of sustained tachycardia usually involves admission for continuous fetal heart-rate monitoring and a multidisciplinary approach to management that usually involves a pediatric cardiologist, maternal fetal medicine specialist, neonatologist, obstetrician and sometimes an electrophysiologist. Again, the management of a sustained tachycardia goes beyond the scope of this review.

CONCLUSION

In conclusion, it is important to recognize a fetal arrhythmia on auscultation and, when an arrhythmia is heard, to refer

the patient for a detailed fetal echocardiogram. Distinguishing the different arrhythmias by ultrasound can be challenging. But, narrowing the arrhythmia to an irregular rhythm, a bradycardia, or a tachycardia, and evaluating the arrhythmia using pulsed Doppler of the mitral inflow and aortic outflow can simplify the diagnosis. Although the vast majority of fetal arrhythmias are self-limited and benign, several arrhythmias are potentially life-threatening and these often require a multidisciplinary approach to management.

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