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The Use of Sonography to Diagnose Heart Arrhythmias in the Fetus

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An arrhythmia is a disturbance in the normal rhythm of the heart. Heart rhythms are generated by the sinoatrial (SA) node and travel through the heart's conduction system, causing the atrial and ventricular myocardium to contract and relax at a regular rate. If there is a disturbance in the electrical system of the heart, it can be detected in utero by using M-mode sonography. It is important to diagnose a fetal arrhythmia early in pregnancy. If the arrhythmia persists, it can lead to congestive heart failure and hydrops.

Key words: M-mode, PAC, PVC, tachycardia, bradycardia

Normal cardiac rhythm in the fetus can generally be assumed in the presence of a regular heart rate ranging from 110 to 180 bpm (see Fig. 1). A fetal cardiac rhythm disturbance may be noted by at least one of the following conditions: an irregular fetal heart rate, an abnormally slow fetal heart rate (< 100 bpm), or an abnormally fast fetal heart rate (> 200 bpm).[1] Regularity and rate can easily be evaluated by using M-mode sonography. The M-mode cursor should be positioned through the atrial and ventricular wall to allow simultaneous display of cardiac wall motion (see Fig. 2). If an abnormal rhythm is noted by M-mode, further analysis requires color Doppler assessment.

The rhythm of the heart is controlled by a built-in electrical system. The continuous, rhythmic repetition of the cardiac cycle depends on the transmission of electrical impulses through the myocardium. The myocardium also contains its own conduction system-specialized cells that enable it to generate and transmit pulses without stimulation from the nervous system. These cells are placed at certain sites in the myocardium, called nodes. The heart does not need neural impulses to maintain the cardiac cycle. Thus, the heart will beat in the absence of any nervous connection. The

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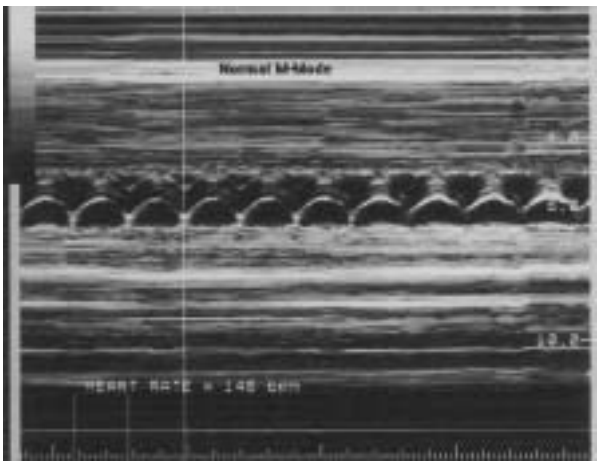

FIG. 1. Normal M-mode tracing of a fetal heart rate at 146 bpm.

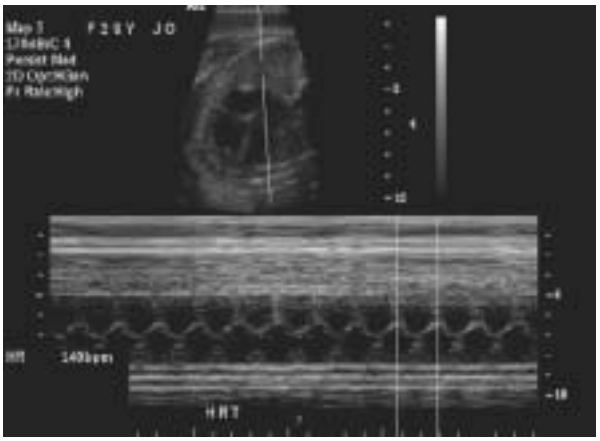

FIG. 2. Normal M-mode tracing showing placement of the cursor through the atrial and ventricular wall.

sympathetic and parasympathetic nerves affect the speed of the cardiac cycle (heart rate).

Normally, electrical impulses arise in the sinoatrial node (SA node), which is often called the pacemaker of the heart. The SA node is located at the junction of the right atrium and superior vena cava, just above the tricuspid valve. Each electrical potential travels rapidly from cell to cell and through special pathways in the atrial myocardium, causing both atria to contract. Meanwhile, ventricular contractions are delayed. An action is transmitted from the atria to the ventricular myocardium through fibers of the conduction system. It travels first to the atrioventricular node (AV node), then to the bundle of His (AV bundle), and finally through the bundle branches of the interventricular septum to Purkinje fibers in the heart wall. The AV node is located in the right atrial wall above the tricuspid valve and anterior to the coronary sinus. Conducting fibers from the AV node converge to form the bundle of His, a triangle within the posterior border of the interventricular septum. The Purkinje fibers extend from the ventricular apexes to the fibrous rings and penetrate the heart wall to the outer myocardium. It is within this electrical system that abnormal heart rhythms occur. Abnormal heart rhythms may signal that there is a problem in the natural electrical system that regulates the beating fetal heart. Depending on the nature of the problem, the fetal heart rate may be too slow (bradycardia), too fast (tachycardia), or irregular (premature atrial contractions or premature ventricular contractions).

PAC

Arrhythmias are present in approximately 10% to 20% of fetuses. Premature atrial contractions (PACs) are abnormal and account for nearly 75% of fetal arrhythmias.[2] Premature atrial contractions can occur at any time from 22 to 40 weeks gestation. Most frequently, an irregular fetal heart rate results from isolated premature atria contractions. These are characterized by an incomplete compensatory pause that is the interval between the ectopic and the next normal heart beat (see Fig. 3). The atrial pacemaker is "reset" so that the next normal atrial beat is also early when compared with normal preextrasystolic beats. Isolated premature atrial contractions are fairly common. They are benign and transient and do not require any treatment. When a fetus has been diagnosed with PACs, the mother is recommended to abstain from smoking and ingesting caffeine-containing products. The physician should follow this rhythm disturbance because supraventricular tachycardia may develop in up to 2.5% of fetuses with PACs.[3] The survival rate for premature atria contractions is 100%, with 85% of these arrhythmias detected by sonography.[3] These premature beats may originate in the atria, the atrioventricular node, or the ventricle. In most cases, premature atrial contractions are sporadic,

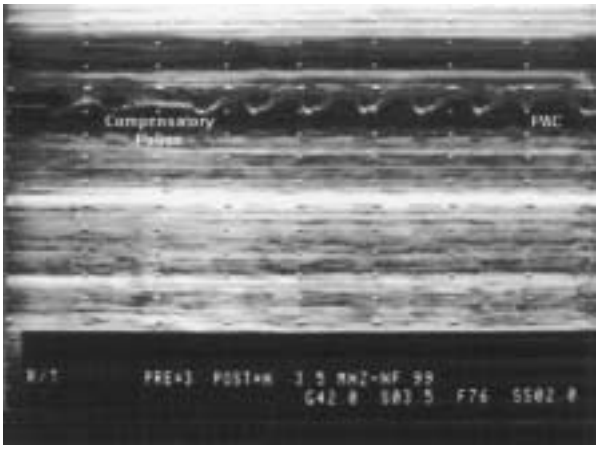

FIG. 3. M-mode tracing demonstrating a premature atrial contraction.

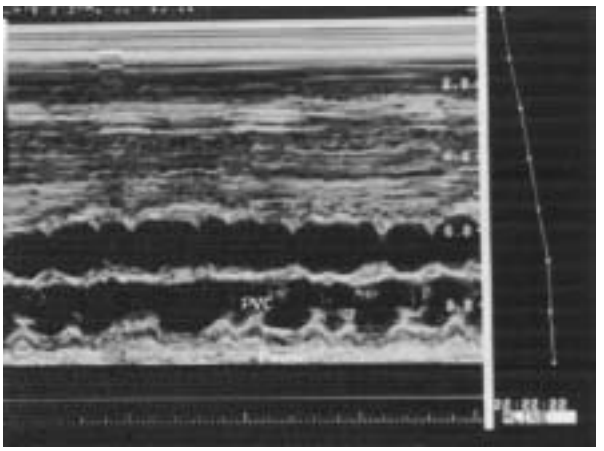

FIG. 4. M-mode tracing demonstrating a premature ventricular contraction.

but there is a small subgroup of patients with familial preexcitation syndrome. Survival and prognosis correlate with the development of tachycardia and hydrops.

PVC

A more rare form of heart arrhythmia is premature ventricular contractions (PVCs). PVCs account for 8% of fetal arrhythmias.[2] Premature ventricular contractions are also known as "extrasystoles" or extra heartbeats. They are also characterized by a complete compensatory pause. PVCs have an early ventricular contraction that is not preceded by an atrial contraction (see Fig. 4). PVCs interrupt the normal heart rhythm and cause an irregular beat, therefore allowing the preexisting rhythm to continue. They arise from the heart's lower pumping chambers, the ventricles. PVCs are often harmless, but when they occur very often or repetitively, they can lead to more serious rhythm disturbances. These are more commonly associated with structural heart disease, myocardial disease, and tumors, or they may result from extracardiac or even maternal conditions (cocaine ingestion).[4] Most PACs and PVCs disappear in utero or in the early neonatal period.

Tachycardia

The normal fetal heart rate usually ranges from 110 to 180 beats per minute. Tachycardia refers to

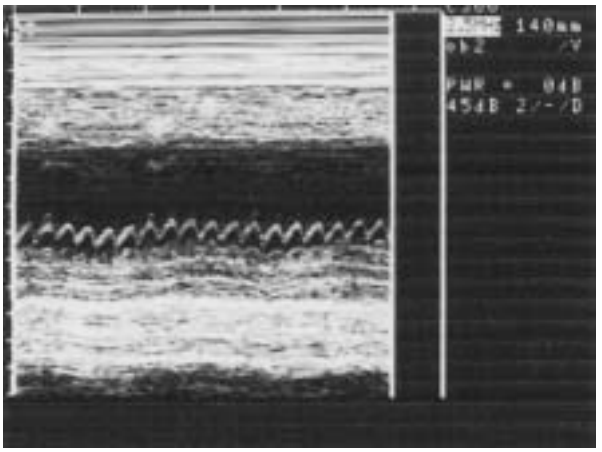

FIG. 5. M-mode tracing demonstrating tachycardia at 187 beats per minute.

a heart rate faster than 180 beats per minute (see Fig. 5). There are three classifications: supra-ventricular tachycardia, atrial flutter, and atrial fibrillation. Supraventricular tachycardia is more common and has an atrial rate of 180 to 300 bpm with a 1:1 conduction between the atria and the ventricle. Atrial flutter is a rate of 300 to 400 bpm with an AV block. Atrial fibrillation is a rate > 400 bpm, which produces an AV block. Accessory pathways between the atrium and the ventricle cause supraventricular tachycardia. PAC typically initiates supraventricular tachycardia, and the tachycardia has an abrupt onset and offset. Ventricular tachycardia is defined as a rapid heart rate as-

sociated with three or more consecutive premature ventricular systoles.

Many factors play into fetal tachycardia: type and duration of arrhythmia, presence of structural cardiac anomalies, gestational age, and response to intrauterine therapy.[5] Most fetal tachycardias have a good prognosis and can be treated in utero with various pharmacological agents. Administration of amiodarone and adenosine are safe and effective treatments for fetal tachycardia. Amiodarone is given for life-threatening recurrent ventricular fibrillation and recurrent ventricular tachycardia. Digitalis is the drug of choice because it rapidly crosses the placenta. Factors that affect fetal outcome with any form of tachycardia are the formation of hydrops, gestational age at onset, and prematurity. In the presence of hydrops, immediate and aggressive therapy is warranted.

Bradycardia

Another form of fetal arrhythmia is bradycardia. Bradycardia is defined as a ventricular rate less than 100 beats per minute (see Fig. 6). The incidence of fetal bradycardia is not clear. There are multiple causes of fetal bradycardia, with complete heart block accounting for 80% of cases.[6] Structural heart disease is also a cause, with the most common being heterotaxy syndrome or corrected transposition. Transient bradycardia can be related to increased pressure within the uterus due to transducer pressure. Below 80 beats per minute, sinus bradycardia may be associated with fetal asphyxia. Heart rates lower than 100 beats per minute during the first trimester have an increased risk of fetal demise.[2] A fetus with sustained bradycardia should have careful follow-up and monitoring for signs of cardiac failure.

Fetal cardiac rhythm disturbances are diagnosed by using M-mode. M-mode is useful in identifying and characterizing PACs, PVCs, tachycardia, and bradycardia by observing the atrial and ventricular rates. The M-mode tracing is obtained through the heart to allow independent assessment of the atria and ventricle wall motion. Further evaluation can be done by Doppler. Doppler evaluation has revealed a decrease in mean flow velocities and cardiac output. Of fundamental importance in

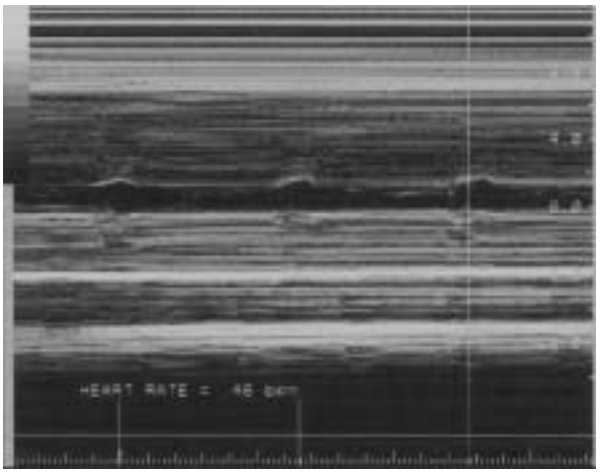

FIG. 6. M-mode tracing demonstrating bradycardia at 46 beats per minute.

determining fetal arrhythmia is identification of atrial and ventricle contraction and the relationship between them. These can be evaluated in four different ways:

- Simultaneous M-mode of the artial wall and a ventricular event. This method of evaluation is most accurate.
- Spectral pulse Doppler can be performed within the left ventricular outflow tract so that mitral inflow and aortic outflow are recorded. This is not more sensitive than M-mode in the interpretation of arrhythmia, but Doppler tracings to analyze rate and rhythm may be easier to obtain.
- Similar to the pulsed Doppler technique, color Doppler M-mode across the left ventricular outflow tract and mitral valve inflow can be performed. It may be quicker to obtain color Doppler M-mode images.
- Simultaneous pulsed Doppler velocimetry of the fetal abdominal aorta and inferior vena cava to access timing.[6]

With the use of sonography to evaluate fetal arrhythmias, other parameters can be evaluated. Fetal arrhythmias can be diagnosed transabdominally by 16 to 18 weeks. With transvaginal ultrasound, arrhythmias can be seen in the first trimester. Sonography should also be used to monitor for changes of hydrops, including

pericardial and pleural effusions, ascites, skin thickening, and placentomegaly. The sonographer should look for structural cardiac anomalies and check for intrauterine growth retardation. Tachycardia occurs with sepsis and fetal anemia.

An arrhythmia is a disturbance in the normal rhythm of the heart. An abnormal arrhythmia can include PAC, PVC, tachycardia, and bradycardia. All of these arrhythmias can occur as an individual arrhythmia or together. The significance of diagnosing a fetal arrhythmia is related to the affects of the arrhythmia on the fetus. Hydrops with prolonged affects of a fetal heart with arrhythmia can result in fetal death. With sonography, the diagnosis can be made early, and therapeutic treatment can be administered as early as possible.

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