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# Cardiac arrhythmias in pregnancy

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ABSTRACT

As more women with repaired congenital heart disease survive to their reproductive years and many other women are delaying pregnancy until later in life, a rising concern is the risk of cardiac arrhythmias during pregnancy. Naturally occurring cardiovascular changes during pregnancy increase the likelihood that a recurrence of a previously experienced cardiac arrhythmia or a de novo arrhythmia will occur. Arrhythmias should be thoroughly investigated to determine if there is a reversible etiology, and risks/benefits of treatment options should be fully explored. We discuss the approach to working up and treating various arrhythmias during pregnancy with attention to fetal and maternal risks as well as treatment of fetal arrhythmias. Acute management in stable patients includes close monitoring and intravenous pharmacologic therapy, while DC cardioversion should be used to terminate arrhythmias in hemodynamically unstable patients. Long-term management may require continued oral antiarrhythmic therapy, with particular attention to fetal safety, to prevent complications associated with arrhythmias.



## Maternal arrhythmias

### *Introduction*

As more women with repaired congenital heart disease survive to their reproductive years, a rising concern is the risk of pregnancy and the associated incidence of cardiac arrhythmias. At the same time, a growing number of women are delaying pregnancy until later in life, increasing their risk of developing arrhythmias as the incidence of heart disease increases with age.[1,2] During pregnancy, as the cardiac output increases and the plasma volume expands, increased heart size causes a rise in wall tension, thereby stimulating the stretch-activated ion channels.[3] These changes may predispose the mother to new-onset arrhythmias but more likely raise the likelihood of recurrence in women with previously documented ones during pregnancy. The increase in heart rate may lead to a shortening of the PR, QRS, and QT intervals. The rotation of the heart on its long axis in a left-upward direction, due to an enlarging uterus and elevation of the diaphragm, can shift the electrical axis to the left. A small Q wave and/or an inverted T wave may be seen in lead III.[4,5] Any other change from baseline should be considered abnormal and further evaluated. Ectopic beats during pregnancy are common, usually benign, and require no specific treatment.[6] Sustained arrhythmias in patients without any underlying structural heart disease are likely to be pathway-related supraventricular tachycardia (SVT), such as atrioventricular (AV) nodal reentry[1,2] or idiopathic ventricular tachycardia (VT). By contrast, in patients who have cardiomyopathy, rheumatic, or other valve disease, or who have undergone corrective cardiac surgery for congenital heart disease, the mechanism is likely to be atrial or ventricular tachyarrhythmia related to the pathological substrate.[7] A prior history of arrhythmias or structural heart disease or a family history of sudden death increases the risk of tachyarrhythmias during pregnancy.[3]

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### Epidemiology

Cardiac arrhythmias constituted an event rate of 166/100,000 in unselected pregnancy-related admissions in a large series.[8] SVTs had an event rate of 24/100,000, atrial fibrillation/flutter (AF/AFL) an event rate of 2/100,000, VT an event rate of 2/100,000, and high-grade AV block an event rate of 1.5/100,000. The 8 cases of cardiac arrest (6/100,000) included 5 patients with asystole and 3 with ventricular fibrillation (VF).[8] The incidence of cardiac arrhythmias during pregnancy is much higher in women with previously known cardiac arrhythmia. In a retrospective study conducted during 87 pregnancies in a selected group of 73 patients who had a history of SVT, paroxysmal AF/AFL, persistent AF/AFL, or VT, 44% of the patients with normal sinus rhythm at the beginning of their pregnancy developed a recurrence of their known tachyarrhythmia during or within 1 month after their pregnancy.[9] The recurrence rate of SVT, paroxysmal AF/AFL, and VT in women with a history of these arrhythmias was 50%, 52%, and 27%, respectively.

### Specific arrhythmias in pregnant women with no organic heart disease

Pregnant women who develop palpitations, near syncope, or syncope should be evaluated to determine the etiology. Serum electrolyte abnormalities and hyperthyroidism should be ruled out. A resting ECG, ambulatory ECG recording, and echocardiography should be performed. New-onset SVT is rare but the recurrence rate of previously documented SVT reaches 50% during pregnancy. Atrioventricular nodal reentrant tachycardia is the most common form unless the patient has Wolff–Parkinson–White (WPW) syndrome, where the type of SVT would be AV reentrant tachycardia (AVRT), whereas focal ectopic atrial tachycardia (EAT) is generally rare and is usually associated with structural heart disease.[4,7] AF and AFL during pregnancy are also rare in patients who do not have structural heart disease or hyperthyroidism[1]and, if observed, should raise the suspicion of previously undiagnosed cardiomyopathy or valve disease and further investigated with an echocardiogram.

Wide-complex tachycardias may be SVT with aberration, or pre-excitation, or VT. Without structural heart disease VT is idiopathic, usually originating from the right ventricular (RV) outflow tract with its typical ECG configuration of an inferior frontal axis and left bundle branch block QRS mimicry[10] and less commonly idiopathic left VT, which has a superiorly directed frontal axis and a right bundle branch QRS morphology. A recent study found 73% of the complex ventricular ectopy originating in the RV outflow tract during pregnancy.[11] A specific type of polymorphic VT, torsade de pointes, occurs in patients with long QT syndrome. A retrospective study of women affected with congenital long QT syndrome, who had one or more pregnancies, showed no increase in cardiac events during pregnancy but a definite rise in the ventricular arrhythmias during the postpartum period, especially in LQT2 genotype.[12]

### Specific arrhythmias in pregnant women with organic heart disease

Focal EAT, intra-atrial reentry, atrial flutter, atrial fibrillation, and ventricular tachycardia in young and middle-age women are generally associated with structural heart disease, including surgically corrected congenital heart disease, cardiomyopathy, and valve disease. Focal EAT tends to be persistent and has a propensity to be drug-resistant.[4,7] Clinically significant arrhythmias occur in 4.6% of the women with an unrepaired atrial septal defect (ASD) and in 3.6% in those with repaired ASD.[13] Right atrial EAT and typical AFL are also common in patients with repaired tetralogy of Fallot (TOF) and pulmonic stenosis. In patients with an atrial switch operation for d-transposition of the great arteries (d-TGA) and in patients after a Fontan procedure, the atrial tachycardias and flutters are usually scar related and complex, and may have dire hemodynamic consequences. In women with atrial switch operation, maternal complications include 12.5% incidence of tachyarrhythmias in mid to late pregnancy.[14] In a recent report, 33 of the 71 pregnancies in 45 women with Fontan operation were complicated by an adverse maternal cardiac event, primarily atrial arrhythmias, resulting in heart failure.[15] Atrial fibrillation may occur in valve disease, and if associated with rheumatic mitral stenosis, pulmonary edema may develop rapidly, especially in late pregnancy, necessitating emergent ventricular rate control and even prompt DC cardioversion.[5]

Ventricular tachycardia may cause hypotension and even collapse if the rates are high enough, severely compromising both maternal and fetal circulation. A retrospective review of 40 pregnancies in 25 women with repaired TOF reported 17% cardiac events, but only a couple of these were VT related.[16] Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a rare genetic disease that causes RV dysfunction and VT. During the third trimester and after delivery there is an increased incidence of arrhythmias in women with ARVC.[17]

Symptomatic bradycardia due to sinoatrial disease may result from an atrial switch operation, a Fontan operation, or repair of partial anomalous venous return. The incidence of second- and third-degree heart block during pregnancy is rare unless there is congenital heart disease that has been repaired, such as TOF, ventricular septal defect, or endocardial cushion defect.[18]

### Treatment

#### *Acute management*

Without organic heart disease, narrow-complex tachycardias are usually well tolerated, but if there is hemodynamic instability, DC cardioversion should be used to terminate the tachycardia. DC cardioversion is safe in all stages of pregnancy.[5] Although the DC current exposure to the fetus is minimum, fetal monitoring during the procedure is recommended. Rare instances of DCCV precipitating prolonged uterine contractions have been reported.[19] In hemodynamically stable patients, the preferred method of termination is IV adenosine, which is extremely effective for AV node dependent SVT. Adenosine has a half-life shorter than 10 s and is not placenta permeable; no fetal adverse effects have