

# Arrhythmias and Pregnancy

## Management of Preexisting and New-Onset Maternal Arrhythmias



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### KEYWORDS

- Pregnancy • Maternal arrhythmias • Arrhythmias • Atrial fibrillation • Supraventricular tachycardia • Ventricular tachycardia • Structural heart disease

### KEY POINTS

- Women with preexisting arrhythmias are at high risk for recurrent arrhythmias and/or exacerbation arrhythmias with pregnancy.
- Arrhythmias may present at any time during pregnancy. Higher risk periods include the latter part of the second trimester, third trimester, and peripartum period.
- New-onset atrial fibrillation and ventricular arrhythmias should prompt evaluation for structural heart disease.

### BACKGROUND

Arrhythmias are the most common cardiovascular complication of pregnancy. Hospitalizations due to arrhythmias in pregnancy have increased by 58% from 2000 to 2012, mainly due to a rise in atrial fibrillation.<sup>1</sup> This rise is likely due to the increase in pregnancy in women with structural heart disease. Arrhythmias may present for the first time in pregnancy, and in women with a history of arrhythmias, pregnancy may lead to an exacerbation of a previously controlled arrhythmia. Identification and appropriate management of arrhythmias are of utmost importance in order to optimize maternal and fetal health.

### PATHOPHYSIOLOGY

Cardiac output increases by 30% to 50% in pregnancy, heart rate increases by 10 to 15 beats per

minute, and peripheral vascular resistance declines. These changes are amplified in multiple gestation pregnancy, with cardiac output increasing by 60% to 70%.<sup>2,3</sup> Physiologic changes peak in the second trimester, and again in labor and delivery where cardiac output increases due to “auto transfusion” with uterine contractions. Sympathomimetic tone is also increased due multiple factors including neurohormonal changes during pregnancy, and pain and anxiety during labor and delivery.<sup>3,4</sup>

Cardiac myocytes have estrogen and progesterone receptors. The downstream effects of estrogen and progesterone on cardiac myocytes is not well understood, but studies have shown these hormones play a role in repolarization.<sup>4</sup> Temporary cardiac remodeling during pregnancy may contribute to the development of arrhythmias. Atrial enlargement and stretch may create a substrate for atrial arrhythmias.<sup>5,6</sup>

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## PREMATURE BEATS

Premature atrial and ventricular beats are common in pregnancy, occurring in ~59% of pregnancies in one study.<sup>7</sup> Premature beats are often benign and patient reassurance can be provided. However, in some patients, premature beats can be associated with structural heart disease and further workup and evaluation are prudent.

Premature ventricular contractions (PVC) may be an initial presentation of a cardiomyopathy or lead to the development of a cardiomyopathy. PVC burden has been shown to correlate with left ventricular function. Most cases of PVC-induced cardiomyopathy occur in patients with a PVC burden of greater than 10% in 24 hours.<sup>8</sup> Tong and colleagues<sup>9</sup> performed a prospective case control study of 53 pregnancies in 43 women with a PVC burden of greater than 1%, mean PVC burden of 13.9%, and no structural heart disease. PVCs presented more commonly in the first trimester. In 25 of 53 pregnancies, beta-blocker therapy was initiated due to symptoms and/or a high burden. Adverse cardiovascular events occurred in 11% of pregnancies and included heart failure, and sustained and nonsustained ventricular tachycardia. Pregnancies with adverse cardiovascular events all had a PVC burden of greater than 5%. Adverse fetal events occurred in 13% of pregnancies and included small for gestational age and preterm birth.<sup>9</sup>

Patients with significant symptoms and preserved systolic function should be reassured.<sup>8</sup> Medical therapy for PVCs is indicated for symptoms or in the setting of a reduced left ventricular ejection fraction. First-line therapy with non-dihydropyridine calcium channel blockers or beta-blockers, excluding atenolol, is recommended.<sup>8</sup>

Premature atrial contractions (PACs) have primarily been studied in the nonpregnant population. Frequent PACs (>100 beats in 24 hours) have been shown to increase the risk of new-onset atrial fibrillation, supraventricular tachycardia, and cardiovascular morbidity and mortality in healthy patients and patients with multiple comorbidities, including structural heart disease.<sup>10,11</sup>

## SUPRAVENTRICULAR TACHYCARDIA

Supraventricular tachycardia (SVT) is the second most common arrhythmia in pregnancy, occurring in 22 per 100,000 pregnancy hospitalizations.<sup>1</sup> SVT may present at any stage of pregnancy, but commonly presents in the second trimester. SVT presents with sudden onset of palpitations, which may be associated with dyspnea, chest discomfort, or presyncope.

Atrioventricular nodal reentrant tachycardia (AVNRT) and atrioventricular reentrant tachycardia (AVRT) are the most common subtypes of SVT. AVNRT is characterized by dual AV node physiology allowing anterograde and retrograde conduction. In AVRT conduction may occur through the AV node or the accessory pathway. In antidromic AVRT, the tachycardia conducts anterograde down the accessory pathway and retrograde conduction through the AV node, creating a regular wide complex tachycardia. Antidromic AVRT accounts of 5% to 10% of AVRT.<sup>12</sup>

In patients with SVT, electrocardiograms in sinus rhythm are assessed for preexcitation, which may be asymptomatic and intermittently present on electrocardiogram. Findings of preexcitation include a short PR interval less than 120 ms, slurred upstroke of the QRS, and QRS prolongation greater than 110 ms. Concern arises in patients with preexcited atrial fibrillation that may degenerate into ventricular fibrillation. Preexcitation should be considered in patients with SVT who present with syncope or sudden cardiac death.

## ATRIAL FIBRILLATION

Atrial fibrillation (AF) is the most common arrhythmia in pregnancy, accounting for 27 per 100,000 pregnancy hospitalizations for arrhythmias.<sup>1,13</sup> In a meta-analysis of 7 studies totaling 301,638 pregnancies, AF incidence was significantly higher in women with structural heart disease compared with women without structural heart disease (0.3% vs 2.2%).<sup>14</sup>

Risk factors for AF in pregnancy are similar to risk factors in the nonpregnant state. Obesity and age older than 40 significantly increase risk of AF.<sup>15</sup> Additional risk factors for AF identified in the Registry of Pregnancy and Cardiac Disease (ROPAC) include congenital heart disease, preexisting history of AF, beta-blocker use before pregnancy, and valvular heart disease.<sup>16</sup>

AF in pregnancy is associated with adverse maternal and fetal outcomes. Adverse fetal outcomes include intrauterine growth restriction, respiratory distress syndrome, intraventricular hemorrhage, and higher rates of neonatal intensive care unit admissions. In addition, agents used for rate control may lead to maternal hypotension and decreased placental perfusion, increasing the risk for preterm labor. Adverse maternal outcomes include heart failure and thromboembolic events.<sup>15-17</sup>

Management of AF is similar to the nonpregnant state. In the nonpregnant population, trials have not shown a difference in cardiovascular

outcomes and overall mortality between rate and rhythm control strategies.<sup>18,19</sup> There are no data available comparing maternal and fetal outcomes in a rate control versus rhythm control approach. According to the 2018 European Society of Cardiology Guidelines, a rhythm control strategy is preferred for management of AF in pregnancy.<sup>20</sup> Rhythm control allows for lower doses of rate controlling medications, such as beta-blockers, which can be associated with hypotension, intrauterine growth restriction, and infant hypoglycemia. Rhythm control can be accomplished with cardioversion and/or antiarrhythmic therapy. Cardioversion is safe in pregnancy and should be considered if AF does not terminate within 24 hours of onset.<sup>21,22</sup> Cardioversion within 48 hours of AF onset does not negate the need for therapeutic anticoagulation.<sup>23</sup> Cardioversion results in atrial stunning and activation prothrombotic factors.<sup>24</sup> Thromboembolic events are highest the first month following cardioversion; thus, anticoagulation should be continued for a minimum of 4 weeks following cardioversion. Extended or long-term anticoagulation should be based on the patients' risk factors for thromboembolic events.<sup>23,25,26</sup> If the onset of AF cannot be determined with accuracy, transesophageal echocardiogram should be performed before cardioversion.

In nonpregnant women, the CHADS2 VASC Score (congestive heart failure, hypertension, age  $\geq$  75 years, diabetes mellitus, stroke or transient ischemic attack, vascular disease, age 65 to 74 years, sex category) guides anticoagulation management in AF. Therapeutic anticoagulation is recommended in patients with a nonsex CHADS2VASC score greater than 1.<sup>23</sup> The CHADS2VASC score is often used in AF in pregnancy; however, it has not been validated in pregnant women. There are case reports of left atrial appendage thrombus in pregnancy with persistent AF and structurally normal hearts.<sup>27</sup> Aspirin, therapeutic anticoagulation, and prophylactic enoxaparin have been reported in the literature. Antithrombotic therapy for AF in pregnancies varies. Use of aspirin, therapeutic anticoagulation, prophylactic enoxaparin and no therapy have all been reported.<sup>27</sup> If aspirin is prescribed for AF in pregnancy, the dose should not exceed 162 mg. Full-dose aspirin increases the risk of premature closure of the ductus arteriosus.

### INAPPROPRIATE SINUS TACHYCARDIA

Inappropriate sinus tachycardia (IST) may present during pregnancy and can be difficult to distinguish from postural orthostatic tachycardia syndrome as well as the physiologic increase in

heart rate with pregnancy. During pregnancy, the heart rate increases by 10 to 20 beats per minute but the resting heart rate rarely exceeds greater than 95 beats per minute.<sup>28</sup> IST is characterized by an elevated resting heart rate greater than 100 beats per minute or an average heart rate of greater than 90 beats per minute over 24 hours in the absence of secondary causes. Symptoms of IST include palpitations, fatigue, chest discomfort, dizziness, and poor exercise tolerance due to exaggerated rise in heart rate. In published case reports, IST appears to be well tolerated without adverse of maternal or fetal outcomes.<sup>29,30</sup>

### VENTRICULAR TACHYCARDIA

Ventricular arrhythmias (VA) pose a significant risk to maternal and fetal morbidity and mortality. VAs most commonly occur in the setting of structural heart disease, ischemia, inherited arrhythmia syndromes, or QT prolongation due to drugs or electrolyte abnormalities. In an ROPAC study of 2966 pregnancies (56% congenital heart disease, 32% valvular heart disease), VAs occurred in 1.4%. Predictors of VAs included New York Heart Association Class greater than 1 before pregnancy and moderate/severe left ventricular dysfunction. There was a trend toward higher mortality in women with VAs (2.4% vs 0.3%,  $P = .15$ ).<sup>31</sup> VAs are more likely to occur in women with a prior history of VAs.

### *Arrhythmogenic Right Ventricular Cardiomyopathy*

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is characterized by fibrofatty displacement and thinning of the myocardium leading to ventricular enlargement and dysfunction. ARVC predominantly affects the right ventricle but left ventricular dysfunction may also occur. The degree of ventricular dysfunction correlates with outcome. ARVC may be symptomatic or present with PVCs, ventricular tachycardia or sudden cardiac death. VAs often present before ventricular dysfunction. In patients with ARVC, VAs are often triggered by increased adrenergic activity, such as exercise.<sup>32</sup> Adverse cardiovascular events are not uncommon during pregnancy in women with ARVC. Wu and colleagues<sup>33</sup> reviewed 224 pregnancies in 120 women with ARVC. Ninety-one (76%) women had pregnancies before the diagnosis of ARVC. Adverse events occurred in 12 pregnancies and included VAs and heart failure. Women at highest risk of adverse outcomes had earlier onset of symptoms and left ventricular dysfunction (50% vs 60%,  $P = .004$ ). In the women who became pregnant after being diagnosed with ARVC, there

was no significant change in ventricular remodeling or function 1 year postpartum.<sup>33</sup> In a study by Hodes and colleagues<sup>34</sup> of 26 women with ARVC and 39 pregnancies, 5% developed heart failure and 13% developed VAs.

### ***Hypertrophic Cardiomyopathy***

Hypertrophic cardiomyopathy (HCM) is due to mutations in genes encoding sarcomere proteins, leading to increased left ventricular wall thickness and mass in the absence of secondary causes. It is inherited in an autosomal dominant manner and may occur with or without LVOT obstruction. Heart failure, arrhythmias, stroke, and sudden cardiac death account for most cardiovascular morbidity and mortality in HCM. In the ROPAC registry, VAs occurred in 22% of women with HCM with implantable cardioverter-defibrillators. There was no significant increase in VAs in women with HCM and no implantable cardioverter defibrillator.<sup>31</sup> In a pooled cohort of 9 studies with 207 women with HCM and 408 pregnancies, maternal mortality was less than 1% and 30% of pregnancies were associated with worsening symptoms or arrhythmias. Adverse fetal outcomes included spontaneous abortion, stillbirth, and premature birth. Maternal deaths were due to sudden cardiac death.<sup>35</sup> Validated risk factors of sudden cardiac death in HCM should be considered when counseling women on adverse cardiovascular outcomes with pregnancy.

### ***Inherited Arrhythmia Syndromes***

Inherited arrhythmia syndromes (IAS) include congenital long QT syndrome (LQTS), catecholaminergic polymorphic ventricular tachycardia (CPVT), Brugada syndrome, short QT syndrome, idiopathic ventricular fibrillation, and early repolarization syndrome. Data on IAS and pregnancy outcomes is limited as few studies specify types of IAS. IAS is not an absolute contraindication to pregnancy. LQTS is the most common channelopathy, occurring in 1 in 2000. QTc greater than 500 ms and severe genotype (LQT2 or LQT3) is associated with high risk of torsades de pointes.<sup>36,37</sup> In a study of 136 pregnancies in 76 women with LQTS and mean QTc 515 ms, 10.3% of pregnancies were associated with VAs. The increased risk of VAs persisted 9 months postpartum. Beta-blocker therapy was protective of VAs during pregnancy and postpartum.<sup>38</sup> Beta-blockers are a Class I indication in LQTS and should be continued in pregnancy and postpartum.<sup>39</sup>

### ***Catecholaminergic Polymorphic Ventricular Tachycardia***

CPVT is a rare inherited arrhythmia syndrome that often presents as syncope, VAs, or sudden death in the setting of exercise or an emotional stressor in the absence of structural heart disease or prolonged QT interval. Polymorphic VT, bidirectional VT and ventricular fibrillation are characteristic of CPVT. CPVT mimics LQT1 and is not uncommonly misdiagnosed as LQT1 despite a normal QTc interval. Medical therapy for CPVT includes nonselective beta-blockers and flecainide. Nadolol is the preferred beta-blocker in CPVT.<sup>39</sup>

### **BRADYARRHYTHMIAS**

Bradycardias are uncommon in pregnancy. If present, they are often due to chronotropic incompetence or high-degree atrioventricular block, which is often present before pregnancy. Women with repaired congenital heart disease or prior cardiac surgery are at an increased risk for bradyarrhythmias. In a study of 25 pregnancies in 18 women, those with untreated atrioventricular block were more likely to have progression in conduction disease with pregnancy. Women with new-onset atrioventricular block were more likely to require intervention compared with women with stable atrioventricular block before pregnancy.<sup>40</sup>

### **DIAGNOSIS AND MANAGEMENT**

Clinical evaluation should be performed in a stepwise approach starting with a detailed clinical history, obstetric history, family history and physical examination. Red flags include exertional syncope, syncope triggered by emotional stress and/or auditory stimuli, palpitations associated with anginal chest pain or syncope, and a family history of sudden cardiac death.

An electrocardiogram should be obtained in all patients with specific attention to signs of preexcitation, pathologic q waves, ventricular hypertrophy, and conduction delays and intervals. Mobile cardiac telemetry or Holter monitoring should be considered based on the frequency of symptoms. Use of implantable loop recorders in pregnancy have been reported. In a study of 40 pregnant women, implantable loop recorders increased detection of arrhythmias and led to changes in management.<sup>41</sup> Identification of arrhythmias or frequent premature ventricular should prompt assessment of structural heart disease. Identification of structural heart disease affects risk of cardiovascular complications with pregnancy and medical therapy. Transthoracic echocardiogram

is readily available and can be performed with contrast enhancement in pregnancy. Exercise stress testing or advanced imaging should be considered based on the clinical scenario (Table 1).

## PHARMACOTHERAPY

There are no randomized trials regarding use of cardiovascular disease medication in pregnancy. Most drugs are Food and Drug Administration (FDA) class C or D. Class C drugs have limited data in human pregnancy but have been studied in animal reproduction and shown to have adverse fetal effects. Class D drugs have shown adverse fetal effects when given in pregnancy in humans. Given limited data on pharmacotherapy in pregnancy, risk versus benefit must be considered. Triggers to arrhythmias should be considered before implementation of long-term medical therapy. Triggers include severe electrolytes abnormalities, illicit drug use, supplements, and certain obstetric medications such as terbutaline and magnesium sulfate. Severe hypermagnesemia may lead to cardiac and respiratory arrest. The PR interval and QRS duration increase with plasma levels of 5 mg/dL to 10 mg/dL. Conduction defect and cardiac arrest may occur with plasma levels greater than 10 mg/dL. It is important to remember that to improve fetal and maternal outcomes, maternal health must be prioritized.

### ***Beta-Blockers (Food and Drug Administration Class C)***

Beta-blockers increase the risk of intrauterine growth restriction (IUGR), preterm birth, and neonatal hypoglycemia, bradycardia and hypotension. In a cohort study of 18,477 women with hypertension in pregnancy, beta-blocker use in the first trimester was not independently associated with an increased risk of overall malformations or cardiac malformations.<sup>42</sup> Variation in risk of congenital malformation with beta-blocker dose in the first trimester has not been studied.

$\beta$ 1 selective beta-blockers are preferred in pregnancy due to lower rates of IUGR and decreased effects on uterine activity and peripheral vasodilation. Nonselective beta-blockers are associated with higher rates of IUGR. Atenolol is the only beta-blocker listed as FDA Class D due to increased risk of congenital malformations. Use of atenolol is not recommended in pregnancy.<sup>43</sup>

A recent study by Grewal and colleagues analyzed the determinants of birth weight to discern the relative impact of beta-blockers.<sup>44</sup> Of 1757 pregnancies, 404 women were treated with beta-blockers, most commonly metoprolol

(72%). Beta-blockers significantly reduced birth weight less than 200 g; however, this is unlikely to be clinically consequential. Metoprolol was associated with the smallest reduction in birth weight by 119 g. Atenolol was associated with the largest reduction in birthweight by 466 g and is not recommended for use in pregnancy.<sup>44</sup>

### ***Calcium Channel Blockers (Food and Drug Administration Class C)***

Calcium channel blockers (CCBs) have not been associated with increased risk of congenital malformation. Due to the mechanism of action, CCBs may cause hypotension and tocolysis. Prior studies suggested an increased risk of neonatal seizures with CCB use in the third trimester; however, this was not shown in recent large cohort study with 22,908 pregnancies.<sup>45</sup> Diltiazem has been associated with teratogenicity in animals but this has not been studied in pregnancy. Verapamil is considered safe in pregnancy and breastfeeding.<sup>20</sup>

### ***Digoxin (Food and Drug Administration Class C)***

Digoxin predominantly affects the resting heart rate and is often used as an adjunct for rate control in patients treated with beta-blockers or CCBs. Digoxin may also be used in heart failure with reduced ejection fraction. Serum levels of digoxin are not reliable in pregnancy due to an increase in unbound digoxin and an increase in renal clearance.<sup>46</sup> Clinical signs and symptoms should be used in addition with serum levels to assess for digoxin toxicity.

### ***Adenosine (Food and Drug Administration Class C)***

Adenosine is safe for use in pregnancy and has not been shown to have adverse fetal effects. Adenosine has a very short half-life, which prevents delivery to the fetus. It is recommended as first-line therapy for acute termination of supraventricular tachycardia in pregnancy if vagal maneuvers fail.<sup>46</sup> An intravenous line should be placed in the antecubital fossa or more proximal, given the short half-life. It is given as a bolus of 6 mg followed by rapid saline flush. Two subsequent doses of 12 mg can be given.<sup>12</sup>

### ***Flecainide (Food and Drug Administration Class C)***

Flecainide is a sodium channel blocker used in the treatment of supraventricular tachycardia, atrial arrhythmias and CPVT. Flecainide crosses the

<b>Arrhythmia</b>	<b>Diagnostic Testing</b>	<b>Treatment</b>	<b>Clinical Pearls</b>
Supraventricular tachycardia	Assess for structural heart disease with echocardiography before antiarrhythmic therapy	Vagal maneuvers β-blockers Non-dihydropyridine calcium channel blockers Flecainide Sotalol Adenosine DCCV Catheter ablation	Consider antidromic SVT in differential for a regular wide complex tachycardia
Atrial fibrillation and flutter	Assess for structural heart disease with echocardiography and/or advanced cardiac imaging	β-blockers Non-dihydropyridine calcium channel blockers Digoxin Flecainide Sotalol Dofetilide Amiodarone <sup>a</sup> DCCV Catheter ablation	Consider DCCV for hemodynamically stable AF>24 h Aspirin <162 mg daily Therapeutic anticoagulation in high risk patients (coumadin, LMWH) Preexcited AF: Procainamide, catheter ablation
Sinus tachycardia	Distinguish physiologic tachycardia from inappropriate sinus tachycardia and postural orthostatic tachycardia syndrome	Encourage adequate hydration Compression stockings β-blockers Non-dihydropyridine calcium channel blockers	Avoidance of precipitating factors (eg, alcohol, caffeine)
Premature ventricular contractions	Quantitate PVC burden with Holter monitor Consider assessment for structural heart disease with echocardiography and/or advanced cardiac imaging	Correct electrolyte abnormalities β-blockers Non-dihydropyridine calcium channel blockers Sotalol Dofetilide Amiodarone <sup>a</sup> Catheter ablation	Risk of PVC-induced cardiomyopathy with higher PVC burden
Ventricular tachycardia	Assess for structural heart disease with echocardiography and/or advanced cardiac imaging Consider inherited channelopathies and ischemia	Cardioversion Correct electrolyte abnormalities β-blockers Non-dihydropyridine calcium channel blockers Sotalol Dofetilide Amiodarone <sup>a</sup> Catheter ablation	Avoid QT-prolonging agents Consider antidromic SVT with a regular wide complex tachycardia Consider preexcited AF with an irregular wide complex tachycardia ICD implantation (transvenous or subcutaneous) during pregnancy in qualifying patients

**Abbreviations:** AF, atrial fibrillation; DCCV, direct current cardioversion; ICD, implantable cardioverter defibrillator; LMWH, low molecular weight heparin; PVC, premature ventricular contractions; SVC, supraventricular tachycardia

<sup>a</sup> Avoid amiodarone use, Food and Drug Administration Class D.

placenta and is present in breast milk. It is used in the treatment of both maternal and fetal arrhythmias. Coadministration of atrioventricular (AV) nodal blockers are recommended in patients with AF and flutter treated with flecainide as there is potential for one-to-one atrioventricular conduction. Flecainide should not be used in patients with coronary artery disease or structural heart disease.<sup>47</sup>

### ***Sotalol (Food and Drug Administration Class B)***

Sotalol is a potassium channel blocker with beta-blocker properties. Due to its QT-prolonging effects, there is risk of torsade de pointes. Sotalol exhibits reverse-use dependence on the action potential. As a result, QT-prolonging effects are highest at reduced heart rates. Drug efficacy is reduced at higher heart rates.

### ***Dofetilide (Food and Drug Administration Class C)***

Dofetilide is a potassium channel blocker with reverse-use dependence. The QT prolonging of effects are greater when compared with sotalol. Dofetilide must be initiated in an inpatient setting with close monitoring of the electrocardiogram.<sup>47</sup> Providers should pay close attention to drug interactions and avoid coadministration of QT-prolonging agents.

### ***Amiodarone (Food and Drug Administration Class D)***

Amiodarone is reserved for refractory and/or life-threatening arrhythmias due to its adverse fetal effects which are independent of dose and duration. Adverse fetal effects include congenital goiter, hypothyroidism, neurodevelopmental abnormalities, and preterm birth. Neonatal hypothyroidism is often transient and has been reported in 23% of neonates exposed to amiodarone.<sup>48</sup> Use of amiodarone is not recommended in women breastfeeding.

## **DIRECT CURRENT CARIOVERSION**

Cardioversion is safe and effective in pregnancy and should be performed immediately in patients with hemodynamic instability.<sup>21,22</sup> Continuous fetal monitoring and coordination of care with maternal fetal medicine and pediatric is recommended in viable pregnancies. If anticoagulation is indicated, consideration of gestational age and potential need for emergency delivery should play a role in choosing the appropriate agent.

## **ELECTROPHYSIOLOGY PROCEDURES**

Catheter ablation has been performed safely in pregnancy. Catheter ablation is considered in patients with refractory and/or life-threatening arrhythmias that cannot be managed with medical therapy.<sup>20,49</sup> If possible, catheter ablations should be performed in the second trimester with use of echocardiographic guidance to minimize or eliminate radiation exposure. Placement of implantable cardiac-defibrillators (ICD) and pacemakers are safe in pregnancy. ICD shocks have not been associated with adverse fetal effects.<sup>50</sup>

## **SUMMARY**

Arrhythmias, new-onset or exacerbation of preexisting arrhythmias, are the most common cardiovascular complication in pregnancy. A detailed evaluation should be performed in patients with arrhythmias and management should be in place outlining antepartum, intrapartum, and postpartum care. A multidisciplinary approach with cardiology, maternal fetal medicine, pediatrics, and anesthesia is of utmost importance to optimize maternal and fetal outcomes.

## **CLINICAL CARE POINTS**

- Arrhythmias are the most common cardiovascular complication of pregnancy, occurring in 68 per 100,000 pregnancies.
- Women with a prior history of arrhythmias are at high risk of recurrence (30%–50%) with pregnancy.
- There are no validated risk models to assess risk of thromboembolic events in nonvalvular AF in pregnancy. The CHA<sub>2</sub>DS<sub>2</sub>-VASc score has not been validated in pregnancy. High-dose aspirin increases the risk of premature closure of the ductus arteriosus.
- Ventricular tachycardia should prompt evaluation for structural heart disease, ischemia (eg, pregnancy associated myocardial infarction, coronary spasm), use of QT-prolonging agents, and inherited channelopathies.
- Amiodarone is associated with adverse fetal effects and should be reserved for refractory and life-threatening arrhythmias.
- Cardioversion, catheter ablation, and implantation of cardioverter-defibrillators are safe in pregnancy.

## **DISCLOSURE**

The authors have nothing to disclose.

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