Opinion statement

This article reviews the appropriate evaluation and management of cardiac arrhythmias in the pregnant patient. Any treatment strategy in this patient population has the inherent potential to adversely affect the health of the unborn child. As such, there is no room for empiric therapy in these patients. Adequate arrhythmia documentation is paramount, preferably by noninvasive means. The decision to treat should be based on symptom severity and the risk to both mother and fetus posed by potentially recurring arrhythmia episodes throughout the pregnancy. Minimal symptoms in the setting of a structurally normal heart call for a conservative approach. Less is better. If pharmacologic therapy is justified, drugs with historically demonstrated safety profiles in pregnancy should be tried first. The safety profiles of virtually all drugs used to treat cardiac arrhythmias during human pregnancy are based solely on an accumulation of past clinical experience. Newer antiarrhythmics therefore carry a largely unknown risk. Most inherent rhythm disorders manifest long before a woman reaches childbearing age. Women with previously diagnosed arrhythmias frequently experience a recurrence or worsening of their arrhythmia during the pregnancy. Counseling of these individuals and perhaps preemptive treatment by means such as arrhythmia ablation prior to a planned pregnancy would seem optimal.

Introduction

Cardiac arrhythmias during pregnancy are infrequent but not uncommon [1•]. As such, most cardiologists have minimal training or clinical experience in the appropriate management of abnormal heart rhythms in this unique patient population. Fortunately, life-threatening tachyarrhythmias in women of childbearing age are rare, as are symptomatic bradyarrhythmias. However, clinicians may expect to see a rise in arrhythmia occurrence rates in this group of individuals, as many young women now commonly delay pregnancy until later in life, adding degenerative cardiovascular disease to the clinical picture. Additionally, young women with repaired congenital heart disease now routinely survive into their reproductive years. These individuals are inherently at risk for cardiac arrhythmia disorders. A new arrhythmia during pregnancy also may be the first clinical manifestation of underlying heart disease [2, 3, 4].
In general, the approach to evaluating arrhythmias in a pregnant patient is similar to that taken in any other patient. The decision to treat, however, must be tempered by several factors unique to the state of pregnancy. These factors include the presence of the developing fetus; the characteristic hemodynamic changes seen in pregnant women; the effect of antiarrhythmic therapy on labor, delivery, and lactation; and the direct effects of these therapies on the fetus.

During pregnancy, significant hemodynamic changes take place to meet the physiologic needs of both the mother and the developing fetus. These changes must be accommodated by the maternal cardiovascular system. Total body water increases by approximately 5 to 8 L in normal pregnancy, even more so in the presence of clinical edema. The resulting rise in blood volume increases cardiac output by approximately 40%, placing significant mechanical demands on the maternal heart [5, 6].

The physiologic effects of pregnancy on drug therapy are significant. Renal blood flow increases as much as 80% by midpregnancy, resulting in a glomerular filtration rate 50% higher. Increased metabolism of renally cleared drugs is thus seen; similarly, the metabolism of hepatically cleared drugs is increased because of increased progesterone levels during pregnancy. Other factors, such as increased volume of distribution, decreased protein binding, and changes in gastric motility, all affect drug bioavailability and potential effect. The combined result of these physiologic factors makes careful assessment of clinical drug effect essential [7] and measured drug levels difficult to interpret.

Initial evaluation
A detailed history should be taken, documenting onset, duration, and frequency of symptoms; known structural heart disease; family history of sudden death; history of arrhythmias; or prior unexplained syncope. Attention should be given to symptoms suggestive of hemodynamic compromise: dizziness, syncope, or near syncope. Baseline laboratory work should screen for electrolyte imbalances, renal failure, thyrotoxicosis, and gestational diabetes. Noninvasive diagnostic testing to document the arrhythmia disorder and evaluate for underlying heart disease may include any or all of the following modalities: 12-lead electrocardiography, ambulatory monitoring, echocardiography, exercise treadmill testing, and tilt table testing. Invasive studies such as cardiac catheterization or electrophysiology testing require fluoroscopy and should be considered in only the most urgent situations. Fetal radiation exposure has been linked to congenital malformations, mental retardation, and childhood malignancies [8, 9, 10].

Management of specific arrhythmias

### Congenital complete heart block

- Although usually diagnosed in childhood, asymptomatic individuals may be discovered incidentally during pregnancy.
- In general, in the asymptomatic patient, no acute intervention is required.
- For symptomatic patients in the first or second trimesters, there are no clear guidelines. If the mother is at risk for hemodynamic compromise, permanent pacemaker implantation will likely be unavoidable. Permanent pacemaker implantation is recommended, if possible using echocardiographic guidance to avoid fetal radiation exposure, which has been linked to congenital malformations, mental retardation, and increased risk of childhood malignancies. The risk appears to be greatest with exposure during the first and third trimesters of pregnancy [8, 9, 10]. Shielding of the mother’s abdomen with a lead apron is advisable when the need for fluoroscopy is unavoidable and has been reported in clinical literature without adverse fetal outcome [11].
Congenital third-degree atrioventricular (AV) block with an average heart rate less than 50 bpm, even if asymptomatic, is considered a class IIa indication for permanent pacemaker implantation [12]. These individuals should ideally be referred to a cardiologist prior to pregnancy to address this issue.

Vasodepressor syncope

- Neurocardiogenic syncope is one of the most common causes of symptomatic bradycardia in young women and is believed to account for as much as 20% of unexplained syncope in the general population [13].
- Even when present prior to pregnancy, neurocardiogenic syncope usually improves in susceptible patients during pregnancy, likely because of the marked increase in intravascular volume associated with this state.
- Tilt table testing can be performed if there is no prior diagnosis to evaluate for positional causes of syncope, including orthostatic hypotension, autonomic dysfunction, and neurocardiogenic syncope. It is generally safe to perform during pregnancy and may be of diagnostic value if the appropriate management of positional syncope or recurrent, unexplained syncope is the issue at hand.

Diet and lifestyle treatment

- Management of symptoms usually can be achieved by educating the individual to recognize the warning signs of impending syncope so she can lie down prior to experiencing syncope.
- Adequate hydration, not skipping meals, and liberalization of salt intake if not hypertensive are usually effective measures.
- Pharmaceutical therapy has mixed results and is probably best avoided during pregnancy.

Supraventricular tachycardia

- Palpitations during pregnancy are common, usually representing increased atrial, ventricular, or junctional ectopy due to the hemodynamic, hormonal, and emotional changes associated with pregnancy. They are generally considered benign in the structurally normal heart.
- There is a recognized increased incidence of paroxysmal supraventricular tachycardia (SVT) during pregnancy with the risk generally equally distributed throughout the pregnancy [3, 14].
- Contributing factors are thought to include hemodynamic, autonomic, and hormonal changes, intravascular volume shifts, and increased cardiac mechanical stress [4].
• Sinus tachycardia is a normal response to pregnancy.
• Accessory pathway–mediated reentrant SVT—both the preexcited, Wolff-Parkinson White (WPW) and non-preexcited forms of AV reciprocating tachycardia (AVRT)—and AV nodal reentrant tachycardia (AVNRT) are the two most common sustained arrhythmias seen during pregnancy [15, 16].
• Atrial tachycardia, atrial flutter, and atrial fibrillation are infrequent arrhythmias in women of childbearing age, except in those with underlying heart disease or repaired congenital heart disease.

### Pharmacologic treatment

• Most approved drugs used to treat cardiac arrhythmias are classified by the US Food and Drug Administration (FDA) as category “C” for use in pregnancy. This classification means that risk cannot be ruled out with their use, as there are either animal studies suggesting risk, but no human studies, or no controlled studies in either humans or animals. Please see Table 1 for the complete list of FDA use-in-pregnancy drug categories and definitions. There are no clear guidelines for fetal monitoring during the period of drug administration either long or short term. Fetal heart rate monitoring, if possible, would seem optimal during the period of initial drug loading or bolus administration of antiarrhythmic drugs for the acute termination of cardiac arrhythmias.

#### Adenosine

- Rapid, regular, narrow complex tachycardia is often the result of a re-entrant mechanism. AVNRT and AVRT can be acutely terminated by adenosine bolus infusion if initial vagal maneuvers fail. Adenosine is a purine nucleoside that characteristically depresses AV nodal conduction and sinus node automaticity. Thus, it is highly effective in rapidly terminating reentrant SVT, using the AV node as part of the circuit [17, 18].

<table>
<thead>
<tr>
<th>Standard dosage</th>
<th>6- or 12-mg rapid intravenous (IV) push (class I).</th>
</tr>
</thead>
<tbody>
<tr>
<td>Contraindications</td>
<td>History of severe reactive airways disease.</td>
</tr>
<tr>
<td>Special points</td>
<td>Very rapid onset and short duration of action.</td>
</tr>
<tr>
<td>FDA category</td>
<td>Category C drug in pregnancy. It appears to have no direct effect on the fetus when fetal monitoring is performed during bolus IV administration [19].</td>
</tr>
</tbody>
</table>

#### Calcium channel blockers/digoxin

Verapamil and digoxin have a long history of use in the management of SVT in pregnancy (Table 2) [20, 21, class IIa]. IV verapamil, digoxin, and diltiazem may be used for the acute termination of reentrant SVT as well as for rate control in other forms of sustained SVT, such as atrial fibrillation, atrial flutter, and atrial tachycardia. Chronic management of these arrhythmias is similar to that in the nonpregnant patient using oral dosing. Diltiazem has less clinical history of use during pregnancy for arrhythmias but has been used safely to treat premature labor [22].
Standard dosage
Verapamil initial dose: 2.5 to 5 mg IV bolus over 2 minutes. Diltiazem initial dose: 15 to 20 mg IV bolus over 2 min.

Contraindications
Calcium channel blockers, as well as digoxin, should not be used to treat SVT in the presence of overt or suspected preexcitation. These drugs may enhance conduction over an accessory pathway, potentially causing induction of ventricular fibrillation by allowing rapid conduction of underlying atrial fibrillation to the ventricles across the accessory pathway.

Special points
Verapamil and diltiazem are recommended as second-line therapy for the acute termination of SVT \[23\]. Safe use may be limited if the patient is hemodynamically unstable and likely unable to bear the hypotensive effects of these drugs.

FDA category
All three drugs are category C in pregnancy.

**β-Blockers**

β-Blockers have been used extensively in pregnancy for both the acute termination of SVT and for rate control of sustained SVTs (Table 2). β-

### Table 2. Calcium channel blocker, digoxin, and β-blocker therapy during pregnancy

<table>
<thead>
<tr>
<th>Drug</th>
<th>FDA category</th>
<th>Safety during lactation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diltiazem</td>
<td>C</td>
<td>S</td>
</tr>
<tr>
<td>Diltiazem IV</td>
<td>C</td>
<td>NS</td>
</tr>
<tr>
<td>Verapamil</td>
<td>C</td>
<td>S</td>
</tr>
<tr>
<td>Digoxin</td>
<td>C</td>
<td>S</td>
</tr>
<tr>
<td>Acebutolol</td>
<td>B</td>
<td>S</td>
</tr>
<tr>
<td>Atenolol</td>
<td>D</td>
<td>S</td>
</tr>
<tr>
<td>Bisoprolol</td>
<td>C</td>
<td>Unknown</td>
</tr>
<tr>
<td>Esmolol</td>
<td>C</td>
<td>Unknown</td>
</tr>
<tr>
<td>Inderal</td>
<td>C</td>
<td>S</td>
</tr>
<tr>
<td>Labetalol</td>
<td>C</td>
<td>S</td>
</tr>
<tr>
<td>Lopressor</td>
<td>C</td>
<td>S</td>
</tr>
<tr>
<td>Metoprolol</td>
<td>C</td>
<td>S</td>
</tr>
<tr>
<td>Nadolol</td>
<td>C</td>
<td>S</td>
</tr>
<tr>
<td>Pindolol</td>
<td>B</td>
<td>Unknown</td>
</tr>
<tr>
<td>Propranolol</td>
<td>C</td>
<td>S</td>
</tr>
<tr>
<td>Timolol</td>
<td>C</td>
<td>S</td>
</tr>
</tbody>
</table>

*IV intravenous; NS generally regarded as unsafe and contraindicated or requires cessation of breast-feeding; S generally regarded as safe, maternal medication usually compatible with breast-feeding.*
Blockers are the best choice for initial therapy in the presence of SVT and preexcitation, often in combination with an antiarrhythmic drug. No studies or case reports implicate any β-blocker in fetal malformation. Adverse outcomes with their use have been reported, predominantly consisting of fetal hypotonia, neonatal respiratory depression, low birth weight, and hypoglycemia [6, 24, 25••, class IIa].

**Standard dosage**
Metoprolol initial dose: 5 mg IV bolus over 5 min. Esmolol IV loading dose: 0.5 mg/kg over 1 min followed by a 4-minute infusion of 0.05 mg/kg/min.

**Contraindications**
Atenolol is the only β-blocker currently contraindicated for use during pregnancy, primarily because of an increased incidence of reduced birth weight when compared with other β-blockers [25••].

**Special points**
Safe use may be limited if the patient is hemodynamically unstable. If use of β-blockers is necessary, the clinician should look for low birth weight and neonatal hypoglycemia in the infant following delivery. Infant blood glucose should be monitored for 24 to 48 h post delivery [6].

**FDA category**
Acebutolol and pindolol are category B drugs in pregnancy. All other β-blockers are C, with the exception of atenolol, which is D.

**Antiarrhythmic drugs**

The use of membrane-sensitive antiarrhythmic drugs should be reserved for patients with clearly defined, symptomatic arrhythmias (Table 3). Flecainide, quinidine, procainamide, and sotalol are the most commonly used drugs in this category.

**Class IA antiarrhythmic drugs**

Quinidine and procainamide are the two drugs in this class with the longest history of safe use in pregnancy. Quinidine readily crosses the

<table>
<thead>
<tr>
<th>Antiarrhythmic drug</th>
<th>Vaughan Williams classification</th>
<th>FDA category</th>
<th>Safety during lactation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disopyramide</td>
<td>IA</td>
<td>C</td>
<td>S</td>
</tr>
<tr>
<td>Procaainamide</td>
<td>IA</td>
<td>C</td>
<td>S</td>
</tr>
<tr>
<td>Quinidine</td>
<td>A</td>
<td>C</td>
<td>S</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>IB</td>
<td>B</td>
<td>S</td>
</tr>
<tr>
<td>Mexiletine</td>
<td>IB</td>
<td>C</td>
<td>S</td>
</tr>
<tr>
<td>Flecainide</td>
<td>IC</td>
<td>C</td>
<td>S</td>
</tr>
<tr>
<td>Moricizine</td>
<td>IC</td>
<td>B</td>
<td>Unknown</td>
</tr>
<tr>
<td>Propafenone</td>
<td>IC</td>
<td>C</td>
<td>Unknown</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>III</td>
<td>D</td>
<td>NS</td>
</tr>
<tr>
<td>Azimilide</td>
<td>III</td>
<td>Unknown</td>
<td>Unknown</td>
</tr>
<tr>
<td>Dofetilide</td>
<td>III</td>
<td>Unknown</td>
<td>Unknown</td>
</tr>
<tr>
<td>Ibutilide</td>
<td>III</td>
<td>C</td>
<td>Unknown</td>
</tr>
<tr>
<td>Sotalol</td>
<td>III</td>
<td>B</td>
<td>S</td>
</tr>
<tr>
<td>Adenosine</td>
<td>—</td>
<td>C</td>
<td>Unknown</td>
</tr>
</tbody>
</table>

*Commercially available in intravenous formulation only.

NS generally regarded as unsafe and contraindicated or requires cessation of breast-feeding; S generally regarded as safe, and maternal medication usually compatible with breast-feeding.
placental barrier, a property that has been harnessed historically to terminate fetal tachyarrhythmias [26]. Procainamide also crosses the placental barrier readily and has also been used to treat fetal arrhythmias. It has been used effectively to terminate and manage maternal SVTs, particularly accessory pathway-mediated SVT, because of its slowing effects on pathway conduction [27, class IIa]. Both have a history of safe use in the treatment of atrial fibrillation and flutter in pregnancy.

**Standard dosage**
Quinidine: variable in both immediate- and extended-release formulations; consult the pharmacy. Procainamide: IV formulation only.

**Contraindications**
Both drugs may be proarrhythmic in structurally abnormal hearts and therefore are contraindicated for use in such patients. They should also be avoided in the presence of an underlying AV conduction abnormality or significant hypotension.

**Special points**
Of the class IA antiarrhythmics, quinidine has the longest safety record of use during pregnancy. It is considered relatively safe in pregnancy [26]. There are no reports of adverse fetal outcomes with use of procainamide. Oral procainamide is no longer available, limiting its use to acute arrhythmia management only as an IV agent.

**FDA category**
Quinidine and procainamide are category C drugs in pregnancy.

*Class IC antiarrhythmic drugs*

Flecainide and propafenone both easily cross the placental barrier. Neither of these drugs has been reported to be teratogenic [27]. There are many reports of the safe use of flecainide during pregnancy [28]. In the management of recurrent AVNRT or preexcited AVRT, flecainide may be used in combination with a β-blocker. These drugs may be used to effectively manage recurrent atrial fibrillation, atrial flutter, and atrial tachycardias with regular dosing in combination with a β-blocker or rate-slowing calcium channel blocker [29].

**Standard dosage**
Flecainide: start 50 mg twice daily orally; maximum, 300 mg/d. Propafenone, immediate release: start 150 mg every 8 h; maximum dose, 900 mg/d. Propafenone, slow release: start 225 mg twice daily; maximum dose, 425 mg twice daily.

**Contraindications**
Both drugs may be proarrhythmic in structurally abnormal hearts and therefore are contraindicated for use in such patients. They also should be avoided in the presence of an underlying AV conduction abnormality.

**Special points**
These drugs should always be administered in combination with an AV nodal slowing agent to avoid possible 1:1 conduction to the ventricle of a slowed atrial arrhythmia due to drug effect.

**FDA category**
Flecainide and propafenone are category C drugs in pregnancy.

*Class III antiarrhythmic drugs (sotalol and amiodarone)*

Sotalol is the drug of choice in the presence of underlying structural heart disease when an antiarrhythmic is required to suppress refractory SVT [30]. Amiodarone is a highly effective drug in treating multiple cardiac arrhythmias. However, its safety profile in pregnancy is poor. It has multiple serious adverse effects on the fetus, the most dangerous being hypothyroidism. Congenital abnormalities also have been reported
Recent literature, however, reports the use of amiodarone transplacentally (orally) for the treatment of drug-refractory fetal tachycardia, with excellent efficacy and low associated fetal mortality [33••].

**Standard dosage**
- Sotalol: 80 mg twice daily orally initially; maximum, 640 mg/d. Amiodarone: IV load per Advanced Cardiac Life Support (ACLS) protocol; oral loading is variable.

**Contraindications**
- Sotalol is 90% excreted unchanged in the urine; thus, it should be avoided in patients with renal insufficiency. It should not be used in the presence of baseline QT prolongation.

**Special points**
- Sotalol causes significant QT prolongation in susceptible patients. This must be closely watched for in a monitored setting while starting or titrating the dose, to avoid torsades de pointes. Pulmonary, thyroid, hepatic, corneal, and optic nerve effects are well documented and must be watched for as long as the patient remains on amiodarone.

**FDA category**
- Sotalol is the only class I or III agent to be classified as category B in pregnancy. Amiodarone is a category D drug in pregnancy and should be avoided if at all possible.

**Class III antiarrhythmic drugs (ibutilide)**

Ibutilide is a newer class III agent approved for IV termination of atrial fibrillation and flutter. It is particularly useful in terminating atrial fibrillation in the presence of WPW syndrome because of its negative effects on pathway conduction as well as atrial and ventricular myocardium [34, class IIb]. There are now published case reports describing its safe and successful use in termination of both atrial fibrillation and flutter during pregnancy [35,36]. It has significant QT-prolonging effects.

**Standard dosage**
- 0.01 mg/kg up to 1 mg IV over 10 min; may repeat if no response after 10 additional minutes. Consider pretreatment with 1 mg of magnesium sulfate.

**Contraindications**
- Baseline QT prolongation, because of the risk of inducing torsades de pointes.

**Special points**
- The patient should be monitored for at least 4 h after administration to watch for torsades de pointes. Ibutilide does not cause hypotension and has a rapid onset of action, making it useful in an already hypotensive patient.

**FDA category**
- Ibutilide is a category C drug in pregnancy

**External cardioversion**

- Synchronized external cardioversion of refractory SVT may be necessary and is indicated in the setting of hemodynamic instability [37].

**Standard procedure**
- In the setting of SVT, the shock must be delivered synchronously with ventricular activation to avoid induction of ventricular fibrillation. Cardioversion with up to 300 J has been demonstrated to be safe during pregnancy [38]. If cardioversion is not being performed emergently, the fetus should be monitored for signs of fetal distress during and immediately after administration.

**Complications**
- There are case reports of direct-current cardioversion leading to sustained uterine contraction [39].
Contraindications

The risk of an embolic event with cardioversion of atrial fibrillation or flutter of more than 48 h’ duration off anticoagulation is at least the same, if not slightly higher, during pregnancy. Early cardioversion should be considered to avoid the need for anticoagulation.

Symptomatic palpitations

- If symptoms are severe enough to warrant therapy, a β-blocker is the initial drug of choice once they are documented.

Ventricular tachycardia

- Wide-complex tachycardia, until proven otherwise, should be treated as ventricular tachycardia (VT).
- Nonsustained ventricular arrhythmias reportedly occur in approximately 50% of pregnant women [40]. Most occur in the presence of a structurally normal maternal heart and thus are associated with a low risk of subsequent morbidity and mortality [41].
- Recognized causes of paroxysmal VT during pregnancy include arrhythmogenic right ventricular dysplasia, long QT syndrome (LQTS) [42•], hypertrophic cardiomyopathy, peripartum cardiomyopathy, and coronary artery disease (CAD).
- Sustained VT during pregnancy is the result of idiopathic VT in most cases. These women have normal hearts with catecholamine-sensitive VT [41]. Right ventricular outflow tachycardia and fascicular VT are two such forms of idiopathic VT.
- Few women of childbearing age have CAD, and even fewer have scar-related reentrant VT.
- Peripartum cardiomyopathy should always be considered with new-onset VT within 6 months of delivery.

Pharmacologic treatment

Class IB antiarrhythmic drugs

Lidocaine was previously the drug of choice in the initial management of sustained VT and cardiac arrest [43,44], class indeterminate. In the most recent ACLS guidelines, lidocaine has been replaced with amiodarone as the first drug of choice in the management of VT in a patient who is stable or has shock-resistant VT [23].

<table>
<thead>
<tr>
<th>Standard dosage</th>
<th>Initial dose per ACLS guidelines: 1 to 1.5 mg/kg IV.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Special points</td>
<td>There is conflicting evidence as to its effects on newborn neurobehavioral responses when administered during delivery [45].</td>
</tr>
<tr>
<td>FDA category</td>
<td>Lidocaine is a category B drug during pregnancy.</td>
</tr>
</tbody>
</table>

β-Blockers and calcium channel blockers

β-Blockers are the first drug of choice in the treatment of essentially all forms of VT and frequent, symptomatic ventricular premature complexes. Idiopathic catecholamine-sensitive VT often responds to
cardioselective β-blockers. Fascicular VT often responds to calcium channel blockers.

**Antiarhythmics**

Antiarhythmic therapy may be unavoidable in the face of refractory VT. Quinidine, procainamide, and flecainide all have been used in pregnancy for this purpose, with no adverse fetal outcome [46]. These class I drugs should be used in combination with a β-blocker. They should not be used in the presence of CAD or a structurally abnormal heart because of their proarrhythmic effects in this setting. Sotalol remains a good alternative in an abnormal heart, so long as renal function is not significantly impaired. Amiodarone should be avoided in all but the most refractory cases of VT.

**External cardioversion**

- External cardioversion should be performed early in the setting of hemodynamic compromise.
- Defibrillation shocks transfer no significant current to the fetus.

**Implantable cardioverter–defibrillators**

- Implantable cardioverter–defibrillators (ICDs) are now commonly used to treat patients with life-threatening ventricular arrhythmias. Having an ICD is not a contraindication to becoming pregnant. This “safety net” may actually simplify patient management by allowing the safe discontinuation of suppressive drug therapies until proven necessary.
- In rare cases, it may be desirable to implant an ICD during pregnancy. Fetal radiation exposure remains a concern in early pregnancy, as described earlier. Echocardiographic guidance for lead placement would be optimal. The need for defibrillation testing at the time of implant should be decided on a case-by-case basis. Retrospective studies report ICD shocks delivered during pregnancy have not been associated with adverse fetal outcome as a result of the discharges [47].
- Wearable external defibrillator harnesses are now available but do not have FDA approval for use in pregnant women. These would seem a logical alternative in cases in which ICD implantation is not feasible. So far, there has been only unpublished work on the use of the LifeVest (ZOLL LifeCor; Pittsburgh, PA) in peripartum cardiomyopathy and unpublished documentation of its successful use for VT due to LQTS in a pregnant patient (ZOLL Corporation, personal communication).
- Pregnancy does not increase the risk of major ICD-related complications or result in a higher number of ICD discharges. It is recommended that ICD therapy be left “on” during vaginal deliveries and “off” during cesarian section because of the likely use of cautery [47].
Cardiac arrest

- Cardiac arrest during pregnancy is rare, occurring in one in 30,000 pregnancies as a result of complications during pregnancy, labor, and delivery, and in the immediate postpartum period [48].
- Common causes in this patient population include amniotic fluid embolism, pulmonary embolism, hemorrhage, and eclampsia [49].
- Cardiopulmonary resuscitation (CPR) has unique challenges in these patients.

Performing CPR on the pregnant patient

- Prior to the 25th week of gestation, CPR should be performed as in the nonpregnant patient.
- After the 25th week of gestation, fetal growth results in significant hindrance to the effective delivery of CPR to the mother. The increasing abdominal mass results in aortocaval obstruction. At term, the vena cava is completely occluded in 90% of supine pregnant patients [50]. The result is reduced venous blood return to the heart and forward movement of arterial blood with each chest compression.

Chest compressions

When a patient is noticeably pregnant, it is extremely important to manually displace the uterus to decrease aortocaval compression [51, 52••]. A wedge or rolled towel should be placed under the right hip to tilt the abdomen/uterus at least 15° but no more than 30° to the left. With two-person CPR, one rescuer should position him- or herself to the left of the patient and use both arms to manually pull the uterus toward the left while the second rescuer performs chest compressions. Chest compressions should be administered higher on the sternum, just above the center of the sternum, with increased force [52••].

Breathing

In late pregnancy, the diaphragm is displaced upward, resulting in a 20% decrease in lung functional residual capacity. Additionally, resting oxygen demand increases 20% during pregnancy. As a result, pregnant patients become hypoxic, quickly making adequate oxygen delivery with standard CPR difficult. Early intubation should be strongly considered to improve oxygenation and reduce the chance of aspiration of gastric contents [53].

Circulation

Follow ACLS guidelines for resuscitation medications. Amiodarone has replaced lidocaine as the first drug of choice for refractory ventricular fibrillation [52••]. In pregnancy, it may be reasonable to try lidocaine first if the initial defibrillation attempt is unsuccessful, followed by amiodarone in rapid succession if cardioversion fails. Al-
though vasopressor agents will decrease blood flow to the uterus, the chances of fetal resuscitation depend on successful resuscitation of the mother. Defibrillation should be performed per ACLS guidelines without modification, but any fetal or uterine monitors should be removed before shock delivery.

**Emergent caesarean section**

Neonatal and obstetric personnel should be involved early in the resuscitation effort. After 25 weeks, caesarean section to save the fetus should be considered within 5 min of arrest if spontaneous circulation cannot be restored [14]. It may facilitate the successful resuscitation of the mother as well.

**Anticoagulation therapy in pregnancy**

Anticoagulation should be initiated and maintained throughout pregnancy in the presence of atrial fibrillation in patients with a known embolic stroke risk: hypertension, congestive heart failure, prior stroke or transient ischemic attack, or rheumatic heart disease.

**Warfarin**

Warfarin should be avoided in pregnancy. It passes the placental barrier and is known to cause spontaneous abortion, fetal hemorrhage, mental retardation, and birth malformations, particularly when used during the first trimester.

**Standard dosage**

Initially, 2 to 5 mg/d orally; the dosage is adjusted based on monitored international normalized ratio.

**Special points**

Prevention of maternal mechanical valve thrombosis presents a dilemma. Only IV heparin and warfarin are adequate to prevent valve thrombosis. In pregnant patients with mechanical heart valves, the use of warfarin is limited to the period between week 13 and the middle of the third trimester [54]. Warfarin is safe to use during breast-feeding.

**FDA category**

Warfarin is a category X drug in pregnancy.

**Enoxaparin**

Low molecular weight heparin is not adequate for prophylaxis against mechanical heart valve thrombosis. Recent consensus papers suggest there are enough data to support its safe and effective use in patients without mechanical valves to prevent and treat thromboembolism in high-risk pregnancies [55].

**Standard dosage**

1 mg/kg subcutaneously every 12 h. Dose adjustment is required in the presence of renal insufficiency.

**Special points**

Enoxaparin carries a lower incidence of heparin-induced thrombocytopenia, and there is no evidence of teratogenicity of this agent. There are now published protocols for its use in pregnancy [56].

**FDA category**

Enoxaparin is a category B drug in pregnancy.

**Heparin**

Unfractionated heparin in high doses has been used subcutaneously
as a substitute for warfarin in the first trimester. IV heparin infusion should be discontinued at the onset or 24 h before the induction of labor.

**Standard dosage** Continuous infusion with or without an initial bolus based on weight. The goal is a partial thromboplastin time of 1.5 to 2.5 times baseline.

**Special points** Heparin does not cross the placental barrier. It is safe during breast-feeding.

**FDA category** Unfractionated heparin is a category C drug in pregnancy.

**Labor and delivery**

- The stress of labor may provoke arrhythmia onset.
- Patients with underlying heart disease should have continuous cardiac monitoring during labor and delivery, even if no previous arrhythmia has been documented [57].
- SVTs in the peripartum period can be managed as previously described.
- If the arrhythmia proves to be difficult to manage or fetal compromise is a concern, cesarean section may need to be considered.

**Disclosure**

No potential conflicts of interest relevant to this article were reported.

**References and Recommended Reading**

Papers of particular interest, published recently, have been highlighted as:

- Of importance
- • Of major importance


This article covers 9 years of hospitalizations in a high-volume obstetric service; 226 of 136,422 admissions were for the diagnosis of "arrhythmia." This study is important because of the large number of cases reviewed in terms of better understanding the epidemiology of cardiac arrhythmias seen during pregnancy in the United States.


This article provides a detailed review of the physiologic changes that occur during pregnancy, the effects of pregnancy on drug kinetics, and the use of the most common antiarrhythmic drugs in pregnancy and lactation.

This article is of significant importance because it reports on the safe and successful use of oral amiodarone to treat refractory fetal tachyarrhythmias in a series of 26 patients less than 36 weeks of gestational age. This suggests amiodarone may be able to play a larger role in treating maternal arrhythmias than previously believed.
39. Barnes RJ, Eben F, Patterson D: Direct current cardioversion during pregnancy should be performed with facilities available for fetal moni-
This article addresses the increased risk for torsades de pointes/cardiac arrest in women with congenital LQTS, particularly LQT2, in the immediate 9 months postpartum.
These are the latest published ACLS guidelines. Part 10.8 specifically addresses performing ACLS on the pregnant patient.