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REVIEW

Principles of Arrhythmia Management During Pregnancy

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Abstract

This paper reviews current recommendations on the appropriate evaluation and management of cardiac arrhythmias in the pregnant patient. Most arrhythmias during pregnancy are benign and require no intervention. When required, the decision to treat should be based on symptom severity and the associated risk to mother and fetus posed by potentially recurring arrhythmia episodes throughout the pregnancy. Any treatment strategy in this patient population has inherent risk to both mother and unborn child. Before the initiation of any intervention, documentation of a clinical arrhythmia and correlation with clinical symptoms should be obtained. There is no role for empiric therapy.

Keywords: arrhythmias; pregnancy; pregnancy and heart disease

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Introduction

Isolated rhythm disturbances are infrequent but may occasionally occur during an otherwise uncomplicated pregnancy. Fortunately for both the patient and the clinician, life-threatening arrhythmias during pregnancy are rare. It is not unusual for women during an otherwise normal pregnancy to experience symptoms of palpitations, dizziness, near syncope, and even occasional syncope. Arrhythmias, when present, most commonly present as symptomatic palpitations, which, in most cases, represent sinus arrhythmia or an increased level of background ventricular or atrial ectopic activity that largely resolves following delivery [1]. In 2008 Li et al. [2] published an analysis of 9 years of hospitalizations to a high-volume obstetric service with the admission diagnosis of "arrhythmia." Sixty percent involved a group of patients with sinus tachycardia, sinus bradycardia, and sinus arrhythmia. Pre-

mature atrial and ventricular ectopy accounted for 19%, supraventricular tachycardia (SVT) accounted for14%, atrial fibrillation (AF) and atrial flutter (AFL) accounted for 1%, ventricular tachycardia (VT)/ventricular fibrillation accounted for 1%, and atrioventricular (AV) block accounted for 1%.

Risk of Arrhythmias

Women with tachyarrhythmias predating their pregnancy have an increased risk of recurrence or worsening of their arrhythmia during pregnancy, and patients with no history may present for the first time with an arrhythmia [3, 4]. Women who become pregnant with preexisting structural heart disease have the greatest risk of hemodynamically significant arrhythmias during their pregnancy. This is likely to become a growing clinical concern as many women are now delaying pregnancy until later in life, adding degenerative cardiovascular disease to the clinical picture. Additionally, many more young women with surgically repaired congenital heart disease now routinely survive into their reproductive years and choose to have children of their own. These women inherently have a higher risk of cardiovascular complications. including cardiac arrhythmias, throughout their pregnancy [5–7].

Evidence suggests that the state of pregnancy increases a woman's susceptibility to a variety of arrhythmias. Although no single mechanism has been definitively cited, increased mechanical stress, intravascular volume shifts, elevated hormonal levels, and emotional changes occurring during pregnancy likely account for this increase. During pregnancy, total body water increases by approximately 5–8 L and plasma volume increases to 150% of pregestational levels. leading to increased cardiac end-diastolic volume, wall tension, and stroke volume. Heart rate increases by 10–20 beats per minute at term. The net result is a rise in cardiac output by 30–50% above the baseline [8]. Additionally, peripheral vascular resistance falls in proportion to the rise in total body water in early pregnancy. Estrogen and progesterone levels rise, as does adrenergic receptor sensitivity. These changes all place significant mechanical demands on the maternal heart, alter the electrophysiologic properties of the myocardium. and ultimately promote arrhythmogenesis [8, 9].

General Management of Arrhythmias

Most arrhythmias during pregnancy can be safely and adequately managed with conservative therapies without an adverse effect on either the mother or the child. In general, the approach to evaluating and treating arrhythmias in these women is similar to that taken with any other patient. The decision to treat should be based on symptom severity and the potential risk to both the mother and the fetus should the arrhythmia recur throughout pregnancy. If treatment is initiated, several factors unique to pregnancy must be considered. These include the direct effect of planned therapy on the developing fetus, characteristic hemodynamic changes seen in pregnant women that may alter the anticipated therapeutic effect, and the effect of antiarrhythmic therapy on labor, delivery, and lactation.

Initial Evaluation of Symptoms

The main rule to live by is that there is no room for empirical treatment in pregnant patients. Careful documentation of the arrhythmia, symptom severity, and correlation of symptoms with the arrhythmia, whatever it may be, is vitally important. Patients complaining of palpitations with no correlating arrhythmia on ambulatory monitoring likely require no further evaluation. Arrhythmias causing hemodynamic compromise to the mother compromise the fetus by reducing placental blood flow and are of primary concern.

- Take a detailed history: onset, duration, and frequency of symptoms, known structural heart disease, family history of sudden death, history of arrhythmias, prior unexplained syncope.
- Attention should be given to symptoms of hemodynamic compromise: dizziness, syncope, near syncope.
- Baseline laboratory work should screen for electrolyte imbalances, renal failure, thyrotoxicosis, and gestational diabetes.
- Noninvasive diagnostic testing to document the arrhythmia and screen for underlying structural heart disease can include 12-lead ECG, ambulatory monitor, echocardiogram, and exercise treadmill testing.

- Invasive studies such as cardiac catheterization and electrophysiology testing (in most laboratories) require fluoroscopy. Ionizing radiation acts both directly on the cell's biochemical structures (protein, DNA, etc.) and indirectly by the formation of tissue-damaging free radicals. The embryo and fetus are most susceptible to the consequences of radiation-induced cellular damage during the first 15 weeks of pregnancy, the period of organogenesis. The effects of exposure can be teratogenic, carcinogenic, and mutagenic. Fetal risk is directly related to the level of radiation exposure. Noncancer health effects of fetal radiation exposure are generally not seen at doses of less than 5 rad (0.05 Gy). The long-term, stochastic effects of fetal radiation exposure are less defined and likely do not have a threshold dose. Any exposure to ionizing radiation during fetal development is potentially harmful from this standpoint [7, 10–12]. Because of the small, but finite, increased risk of childhood malignancy, congenital malformations, and mental retardation from fetal radiation exposure, routine use of fluoroscopy is not recommended. If X-ray exposure is deemed absolutely necessary, recommended shielding should be used and the as low as reasonably achievable (ALARA) dosing principle should be followed.

Management of Specific Arrhythmias

Bradyarrhythmias

Congenital complete heart block is usually diagnosed in childhood. Asymptomatic individuals may be discovered incidentally during pregnancy but, in general, no intervention in them is required. For symptomatic patients, pacemaker placement is likely unavoidable. This has been a dilemma in the past over concerns for fetal radiation exposure particularly during the first and second trimesters. This may soon not be a significant issue. Conventional pacing lead implantation requires fluoroscopic guidance. The development of nonfluoroscopic 3D catheter navigation systems, which allow catheter navigation through the vascular system and cardiac chambers, has made it feasible to substantially minimize or eliminate the use of fluoroscopy in certain cardiac procedures that require vascular access [13, 14]. If the patient is symptomatic, pacemaker implantation is recommended, with care being taken to reduce fetal radiation exposure as much as possible, preferably by means of either echocardiography-guided lead placement or an electroanatomical mapping system. A number of centers have now published reports on the use of electroanatomical mapping systems for device implantation [15, 16]. This technique will likely become more widely used over time in centers well versed in use of this technology. Congenital third-degree AV block with an average heart rate of less than 50 beats per minute, even if asymptomatic, is a class IIb indication for permanent pacing and a class IIa indication if abrupt pauses in ventricular rate are seen [17]. These may not be indications to proceed with pacemaker implantation during the pregnancy in an asymptomatic individual.

Vasodepressor syncope/neurocardiogenic syncope is one of the most common causes of symptomatic bradycardia in young women and accounts for as much as 20% of unexplained syncope in the general population [18]. Susceptible patients may demonstrate varying degrees of the cardioinhibitory and vasodepressor components of this syndrome, and most will have a mixed response of both a decrease in heart rate and a decrease in blood pressure. Neurocardiogenic syncope usually improves in susceptible patients during pregnancy, likely due to the increase in plasma volume while the patient is pregnant. This is largely a clinical diagnosis and symptoms will usually predate the pregnancy. Tilt-table testing is a useful tool in evaluating orthostatic symptoms, unexplained syncope, and positional palpitations associated with neurocardiogenic syncope, postural orthostatic tachycardia syndrome, and dysautonomia. Individuals can experience transient bradycardia, hypotension, and even asystole during the study and therefore it may not be appropriate during pregnancy. It remains a useful diagnostic tool in the setting of recurrent, otherwise unexplained syncope when conducted in a carefully controlled setting. Patients should be educated to pay attention to the warning signs of impending syncope so as to lie down before experiencing syncope. Adequate hydration, not skipping meals, and liberalization of salt intake if the patients is not hypersensitive, are usually adequate and effective measures. Pharmaceutical therapy has mixed results and is probably best avoided during pregnancy.

Supraventricular Tachycardia

AV Nodal Reentrant Tachycardia and AV Reentrant Tachycardia

Palpitations during pregnancy are common, usually representing sinus arrhythmia or increased ventricular, atrial, and junctional ectopy. They are generally considered benign in the setting of a structurally normal heart and largely dissipate following delivery [1]. If there is no history of SVT, first onset of paroxysmal SVT during pregnancy is rare (3.9%) [4]. However, when tachycardia is present before pregnancy, there is an increased risk of arrhythmia exacerbation generally distributed equally throughout the pregnancy. Silversides et al. [3] found a 50% recurrence rate of arrhythmia during pregnancy in patients with prior paroxysmal SVT. Adverse fetal and neonatal events occurred in 20% of these pregnancies, most commonly prematurity. The risk of adverse fetal complications increases if arrhythmia recurrence occurs during the antepartum period. Accessory pathway–mediated reentrant tachycardia, both the preexcited Wolff-Parkinson-White form and the non-preexcited form of AV reciprocating tachycardia, and AV nodal reentrant tachycardia are the two most common sustained arrhythmias seen during pregnancy [19, 20].

Therapeutic options for managing SVT during pregnancy can be roughly broken down into acute treatment and ongoing management. They include drug therapy, external cardioversion, and if warranted because of severity and drug-refractoriness, ablation. All medications have potential side effects on the mother and fetus at any stage of pregnancy (Table 1). The risk of teratogenicity is generally higher during the first trimester. After 8 weeks the risk to the fetus is substantially reduced. Most approved drugs used to treat cardiac arrhythmias are classified as category "C" for use in pregnancy by the US Food and Drug Administration (FDA) largely on the basis of outcomes from case reports and small series in the medical literature (Table 2). If possible, drugs should be avoided in the first trimester. The physiologic effects of pregnancy on drug therapy are significant owing to increases in renal blood flow and glomerular filtration rate, decreased protein binding, increased progesterone levels and hepatic metabolism, and increased gastric mobility, all of which impact on expected drug clearance and biologic activity [21]. If the decision to initiate drug therapy is made, as few drugs as possible at the lowest therapeutic dose should be used.

Table 1 US Food and Drug Administration Use-in-Pregnancy Ratings.

Category	Interpretation
A	Controlled studies show no risk
B	No evidence of risk in humans
C	Risk cannot be ruled out
D	Positive evidence of risk
X	Contraindicated in pregnancy

From Physicians' Desk Reference, 70th edition. Montvale, NJ: PDR, LLC; 2015: 211.

Acute Treatment

- Vagal maneuvers (class I indication in pregnancy): Rapid, regular, narrow complex tachycardia is often due to a reentrant mechanism and can be quickly terminated by vagal maneuvers, including Valsalva maneuver and carotid massage, performed with the patient supine.
- Adenosine (class I indication): When carotid maneuvers fail, adenosine is considered a first-line drug option in pregnancy for rapidly terminating reentrant SVT. It suppresses AV node conduction and sinus node automaticity with rapid onset and short half-life. The standard dose is 6 or 12 mg rapid intravenous push. A history of severe reactive airway disease is a contraindication [22].
- Synchronized cardioversion (class I indication): External cardioversion of refractory SVT may be necessary and is indicated in the setting of hemodynamic instability. Energy dosing is the same as in nonpregnant patients. Fetal monitoring during and immediately after cardioversion is recommended. There are case reports of direct current cardioversion leading to sustained uterine contraction [23], but overall cardioversion is safe and effective during pregnancy.
- Metoprolol or propranolol (class IIa indication): Intravenous β-blockers are considered second-line agents when adenosine fails or its use is contraindicated. β-Blockers are the best choice for initial therapy in the setting of narrow-complex SVT (SVT with the absence

Table 2 Antiarrhythmic Drugs during Pregnancy.

Antiarrhythmic drug	Vaughan Williams classification	FDA category	Safety during lactation
Disopyramide	IA	C	S
Procainamide*	IA	C	S
Quinidine	IA	C	S
Lidocaine	IB	B	S
Mexiletine	IB	C	S
Flecainide	IC	C	S
Moricizine	IC	B	Unknown
Propafenone	IC	C	Unknown
Propanolol	II	C	S
Metoprolol	II	C	S
Pindolol	II	B	S
Atenolol	II	D	S
Amiodarone	III	D	NS
Azimilide	III	Unknown	Unknown
Dofetilide	III	Unknown	Unknown
Ibutilide	III	C	Unknown
Sotalol	III	B	S
Verapamil	IV	C	S
Diltiazem	IV	C	S
Adenosine	–	C	Unknown
Digoxin	–	C	S

FDA, Food and Drug Administration; 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.
*Commercially available in intravenous formulation only.

of clear preexcitation). There is extensive literature backing their safe use in pregnancy, although they have been associated with intrauterine growth retardation. The exception is atenolol (FDA category D), which has been linked to fetal hypotonia, neonatal respiratory depression, low birth weight, and hypoglycemia especially in mothers who received the drug at an earlier gestational stage and for longer duration during the pregnancy [24].

- Verapamil (class IIb indication): Intravenously administered verapamil has a long history of safe use in the rapid termination of SVT in pregnancy. There is a greater risk of maternal hypotension with use of calcium channel blockers and they should not be used in the setting of preexcited SVT as they may enhance conduction over the accessory pathway. There is less clinical experience in the use of diltiazem in this clinical setting but it has also been safely used in pregnancy [25]; however, there is some evidence of possible congenital malformations associated with its use during the first trimester [25].
- Procainamide (class IIb indication): Intravenously administered procainamide is a Vaughan Williams class IA antiarrhythmic drug with a long history of safe use in the short-term treatment of both maternal and fetal SVT in pregnancy [26]. It is available commercially only in an intravenous formulation. Quinidine is another similar antiarrhythmic with an extensive history of safe use in pregnancy [27] but is now rarely used in clinical practice. Both drugs can be proarrhythmic and should be avoided in the setting of underlying structural heart disease.
- Amiodarone (class IIb indication): Intravenously administered amiodarone is highly effective in treating multiple cardiac arrhythmias but its safety profile in pregnancy is poor (FDA category D) given its adverse effects on the fetus, the most dangerous being hypothyroidism reported in 17% of cases [24, 28]. That said, amiodar-

one has been used safely transplacentally (oral ingestion by the mother) for the treatment of drug-refractory fetal tachycardia with excellent effect and low fetal mortality [29]. Short-term use of amiodarone would seem acceptable given that its toxic effects are cumulative.

Ongoing Management of Symptomatic SVT

The 2015 American College of Cardiology, American Heart Association, and Heart Rhythm Society SVT guidelines [22] list the following drugs, alone or in combination, for ongoing management in pregnant patients with highly symptomatic SVT (for long-term oral arrhythmia suppression, β-blockers–metoprolol and propranolol–and digoxin are considered the safest first-line agents given their longer clinical history):

- Digoxin (class IIa indication): Digoxin is used transplacentally for the treatment of fetal SVT either alone or in combination with another drug such as flecainide [26]. Like verapamil and diltiazem, it should not be used in the setting of preexcited SVT as it can enhance conduction over the accessory pathway and should not be used in the setting of AF as it can induce ventricular fibrillation.
- Flecainide and propafenone (class IIa indication): Both of these drugs are Vaughan Williams class IC antiarrhythmic agents and have been used to suppress atrial and ventricular arrhythmias during pregnancy. Both should not be used in the setting of ischemic/structural heart disease and in the setting of significant AV conduction disease. Both have been used in combination with a β-blocker, digoxin, or a rate-slowing calcium channel blocker to effectively manage recurrent atrial tachycardia, AF, AFL, and reentrant SVT during pregnancy.
- Sotalol (class IIa indication): This is a Vaughan Williams class III antiarrhythmic drug similar to the newer agent dofetilide. Sotalol is the antiarrhythmic drug of choice to suppress refractory SVT in the presence of underlying structural heart disease. It is the only class I or class III antiarrhythmic drug to be classified as FDA category B for use in pregnancy. It can cause significant QT prolongation and so its administration must be started in a monitored setting and it should not be used in the setting of significant renal impairment as it is 90% excreted unchanged in the urine. There is little current literature on the use of dofetilide for arrhythmia management during pregnancy.
- Amiodarone (class IIb indication): As noted earlier, orally administered amiodarone is highly effective in treating a variety of both maternal and fetal arrhythmias. If it must be used long term, fetal monitoring for the development of goiter and for signs of clinical hypothyroidism is recommended. Its use has also been linked to neurodevelopmental abnormalities [28].
- Catheter ablation (class IIb indication): There a growing body of literature documenting the successful ablation of SVT without fluoroscopy with use of nonfluoroscopic 3D catheter navigation systems [13, 15]. The first reports of its use for SVT ablation during pregnancy are now making their way into the medical literature [30]. If a standard ablation is performed, it should be avoided in the first trimester and the latest radiation-reduction protocols should be used [31]. As the long-term stochastic effects of fetal radiation exposure are unknown, it would seem that the goal of ablation during pregnancy should be no radiation exposure of the fetus at all. As such, we consider an attempt at catheter ablation during pregnancy to be a second-line therapy reserved for the management of highly symptomatic arrhythmias that have proven to be refractory to medical therapy.

AF/AFL/Focal Atrial Tachycardia

New-onset AF, AFL, and focal atrial tachycardia (FAT) are rare in pregnancy. FATs seen during pregnancy mainly occur in patients with underling structural heart disease. They can be a challenge to manage as they are often persistent and refractory to both cardioversion and medication. Fortunately, FATs are usually associated with a slower ventricular rate and are thus better tolerated than AF or AFL, which can manifest themselves as rapid ventricular response rates in the presence of a healthy AV node. The goal of therapy for FAT is heart rate control to prevent possible development of tachycardia-induced cardiomyopathy. Initial therapy can

include β-blockers, non-dihydropyridine calcium channel blockers, and digoxin alone or a combination as blood pressure allows. If FAT is refractory and symptomatic, antiarrhythmic therapy including flecainide, propafenone, or sotalol can be tried. Use of amiodarone is recommended only if the above-mentioned therapies fail. Catheter ablation may need to be considered if the FAT is incessant, poorly tolerated, and drug resistant [32, 33].

Silversides et al. [3] reported an AF/AFL recurrence rate of 52% during pregnancy in women with a pre-pregnancy history of AF/AFL. As with FAT, structural heart disease was present in 96% of these women. In the absence of structural heart disease, AF and AFL are rare during pregnancy, but are infrequent even in its presence. Salam et al. [6] prospectively studied 1321 pregnant women with known structural heart disease throughout their pregnancy. The incidence rate of AF/AFL was only 1.3%, with the highest occurrence rate at the end of the second trimester [6]. Pre-pregnancy predictors of occurrence were AF before pregnancy, mitral valve disease, β-blocker use, and left-sided lesions. AF/AFL was associated with higher maternal mortality (11.8% vs 0.9%) and low birth weight (35% vs 14%). Thus, if a women develops AF during pregnancy, workup for undiagnosed cardiomyopathy, valve disease, hyperthyroidism, electrolyte imbalance, and alcohol abuse should be undertaken. Management of AF/AFL should be no different from that in a nonpregnant women but requires faster intervention, even in patients with normal heart function because of the thrombogenic state of pregnancy and the deleterious effects it may be having on fetal blood supply [34].

Acute Treatment

- Electrical cardioversion should be performed early in the setting of hemodynamic instability with fetal monitoring if possible.
- In hemodynamically stable patients, intravenous administration of unfractionated heparin (UFH) or weight-adjusted low molecular weight heparin (LMWH) should be started. If the AF/AFL duration is less than 48 h, the patient's CHA_2DS_2-VASc score is less than 2, or in the setting of "lone AF," postcardioversion oral anticoagulation may not be necessary. Initial treatment should focus on rate control. β-Blockers are the preferred first-line agent for rate control. Digoxin or non-dihydropyridine calcium channel blockers are considered second-line agents. Neither should be used in the setting of obvious preexcitation. If spontaneous return to sinus rhythm does not occur, external cardioversion can be safely performed during all stages of pregnancy.
- If direct current cardioversion is not available or sedation not desirable, intravenously administered ibutilide has been used safely in pregnancy to terminate AF/AFL [35, 36]. Ibutilide is a Vaughan Williams class III, FDA category C antiarrhythmic drug. It is particularly useful in terminating AF in the setting of preexcitation because of its negative effects on accessory pathway conduction. As it has significant QT-prolonging effects, there is risk of polymorphic VT (torsade de pointes) with its use. Pretreatment with intravenously administered magnesium sulfate is therefore recommended and the patient should be continuously monitored during drug administration and for at least 4 h after with a crash cart in the room [37].
- The risk for a cardioembolic event with cardioversion of AF/AFL of more than 48 h duration is significant. In a pregnant women, anticoagulation with UFH or LMWH for at least 3 weeks is considered mandatory before an elective cardioversion unless a transesophageal echocardiogram is performed before cardioversion to rule out left atrial appendage thrombus. The continuation of anticoagulation for at least 4 weeks following cardioversion is then recommended because of possible left atrial appendage stunning [38].

Ongoing Management

- Anticoagulation should be initiated and maintained throughout pregnancy in the presence of AF/AFL in patients with known embolic stroke risk: CHA_2DS_2VASc score of 2 or greater or in the setting of valvular AF.
- Warfarin (FDA category X) should be avoided in pregnancy as it is linked to spontaneous abortion, fetal hemorrhage, mental retardation, and birth malformations particularly when used in the first

trimester. Its use is limited to those patients with mechanical heart valves from week 13 through the middle of the third trimester [39].

- The preferred agents for anticoagulation in pregnancy are heparin compounds. UFH (FDA category C) and LMWH (FDA category B) do not cross the placenta and are considered safe in pregnancy. Their use should be discontinued 12 h before planned induction of labor [34].
- None of the new novel oral anticoagulants are currently recommended for use in pregnancy.
- Rhythm control is preferred in cases of new-onset AF/AFL. Antiarrhythmic drugs to maintain rhythm control, including quinidine, flecainide, propafenone, and sotalol, have been used safely in this setting [21]. Flecainide and propafenone should be used in combination with an AV nodal slowing agent and their use should be avoided in patients with known structural heart disease. Amiodarone should be used only if absolutely needed.
- Rate control using an AV nodal slowing agents is a reasonable strategy in refractory cases. Unfortunately, we are not aware of any literature defining what is considered adequate rate control in pregnancy.

Ventricular Tachycardia

Nonsustained ventricular arrhythmias have been reported to occur in approximately 50% of pregnancies [40]. The vast majority of these women have structurally normal hearts with no history of VT, and as such there is a very low associated risk of morbidity and death [41]. Silversides et al. [3] reported a 27% recurrence rate of VT during pregnancy in women with a history of VT. Of these women, 27% had congenital long QT (LQT) syndrome and 50% had underlying structural heart disease. Sustained VT during pregnancy is due to idiopathic VT (structurally normal heart) in most cases. These VTs tend to be catecholamine sensitive and rarely degenerate into an unstable rhythm. Right ventricular outflow tachycardia is the most common form of idiopathic VT seen during pregnancy. Fascicular VT is a distant second and generally arises from the left posterior fascicle. Idiopathic, catecholamine-sensitive VT generally responds to cardioselective β-blockers. Fascicular VT often responds to calcium channel blockers (verapamil) [42]. Recognized causes of hemodynamically unstable VT during pregnancy include arrhythmogenic right ventricular dysplasia, hypertrophic cardiomyopathy, LQT syndrome, coronary artery disease, and peripartum cardiomyopathy.

Arrhythmogenic right ventricular dysplasia is a rare genetic disease which results in progressive right (and rarely left) ventricular failure. The affected individuals have been reported to have an increased incidence of ventricular arrhythmias particularly during the third trimester of pregnancy and following delivery [43].

LQT syndrome is a genetic disorder resulting in excessive prolongation of the QT interval with increased risk of torsade de pointes. Affected women (particularly those with LQT2) have been reported to have a significantly increased risk of torsade de pointes/cardiac arrest in the immediate 9 months postpartum. β-Blockers are recommended during pregnancy and in the postpartum period [44].

Peripartum cardiomyopathy should always be suspected in women presenting with new-onset VT during the last 6 weeks of pregnancy or in the early postpartum period.

Few women of childbearing age have coronary artery disease and an even smaller number have scar-related reentrant VT. This may become less of a rarity as women delay having children until later in life.

Management During Pregnancy

- External cardioversion should be performed early in the setting of sustained VT with hemodynamic compromise. Fetal monitoring is recommended if possible.
- β-blockers, if tolerated, are the first drug of choice in the treatment of most forms of VT and symptomatic ventricular ectopy and remain so in pregnancy.
- Lidocaine (FDA category B drug during pregnancy) was previously the first drug of choice in the management of stable or shock-resistant VT. It has been supplanted by amiodarone in the present advanced cardiovascular life support (ACLS) guidelines but remains an option [25] for immediate management with non-LQT-related sustained VT during pregnancy.

- Long-term antiarrhythmic therapy may be unavoidable in the setting of refractory VT. Quinidine, procainamide, and flecainide have all been used in pregnancy with no adverse fetal outcome [45]. Flecainide is useful for the suppression of frequent ventricular ectopy. It should not be used for VT in the setting of structural heart disease as it can promote sustained VT in this setting. Sotalol can be used safely in patients with structural heart disease so long as renal function is preserved. Long-term amiodarone use should be avoided in all but the most refractory cases of VT during pregnancy for the reasons noted previously.
- Implantable cardioverter-defibrillators (ICDs) are indicated in patients at risk of sudden cardiac death due to nonreversible causes. Pregnancy does not increase the risk of major ICD-related complications or result in a higher number of ICD discharges. Retrospective studies have not demonstrated adverse fetal outcome as a result of ICD discharges delivered during pregnancy. It is recommended that ICD therapy be left "on" during vaginal deliveries and "off" during cesarean deliveries given the risk of inappropriate shock with the use of cautery [46].
- The LifeVest external defibrillator (Zoll LifeCor, Pittsburgh, PA, United States) is a wearable external defibrillator which has been used successfully to treat VT due to congenital LQT in pregnancy [7]. This technology would seem a logical alternative in patients for whom ICD implantation is not safe or feasible. Concern for fetal radiation exposure makes standard ICD implantation during a pregnancy problematic. As noted previously, the emergence of 3D electroanatomical mapping systems has allowed safe and accurate pacemaker lead placement without the need for fluoroscopy in centers proficient in this technology. It should be remembered, however, that these "fluoro-less" procedures are still being performed in standard fluoroscopy laboratories in case things do not go as planned.
- Catheter-based ablation of VT remains an option of last resort during pregnancy. This procedure is conventionally guided by fluoroscopy-based catheter visualization. Because of the complexity of this procedure, patients are frequently exposed to high levels of radiation. However, Lamberti et al. [47] recently reported successful ablation in 19 patients with idiopathic VT using only intracardiac echocardiography and an electoanatomical mapping system for catheter guidance. No fluoroscopy was used during any of the procedures, with 100% short-term success rates and no reported complications. In addition, newer fluoroscopy-integrated 3D mapping systems using electoanatomical localization of diagnostic and ablation catheters in prerecorded X-ray images or short X-ray loops have allowed dramatic reductions in radiation exposure without prolonging procedure times or compromising patient safety [48].

Management of Cardiac Arrest Associated with Pregnancy

Cardiac arrest during pregnancy is fortunately rare, occurring in only 1 in 30,000 pregnancies as a result of complications during the maternity, labor, and delivery, or in the immediate postpartum period [49]. The survival rate for these mothers is unfortunately lower than that reported for the traditional cardiac arrest patient despite these women generally being of younger age and having fewer comorbidities [50]. Common causes in this patient population include amniotic fluid embolism, pulmonary embolism, hemorrhage, and eclampsia [51]. Cardiopulmonary resuscitation (CPR) in these patients is complicated by several unique physiologic factors, particularly in late-term pregnancy. After the 25th week of gestation, fetal growth significantly hinders the effective delivery of CPR to the mother. The increasing abdominal mass results in progressive aortocaval obstruction with the mother in the supine position. At term, the vena cava is completely compressed in 90% of supine pregnant patients [52]. This effectively reduces venous return to the heart and forward movement of arterial blood with each chest compression.

Performing CPR on the Pregnant Patient

Positioning

Before the 25th week of gestation, CPR should be performed as in the nonpregnant patient with the patient supine on her back.

After the 25th week of gestation or in an individual who is obviously pregnant, before initiating CPR, the rescuer should place the patient in the left lateral position at 27° to 30° to displace the uterus and decrease aortocaval compression. Left lateral tilt in non-cardiac arrest patients has been reported to improve maternal hemodynamics and improve fetal parameters of oxygen, nonstress test, and fetal heart rate [53].

Chest Compressions

Unfortunately, chest compressions with the patient in the left lateral position result in less forceful chest compressions than can be achieved with the patient in the supine position. With two-person CPR, the same effect can be achieved with the patient supine by having one rescuer on the patient's right side "push" the uterus to the left with one hand or "pull" the uterus to the left using two hands from the patient's left side. This technique is reported to be at least as effective if not better than left lateral tilt in relieving aortocaval compression and allows optimal delivery of chest compressions during CPR. In one-person CPR, a rolled towel or wedge can be placed under the patient's right hip to tilt the abdomen/uterus at least 15° but no more than 30° to the left [54, 55]. Chest compression should be delivered higher on the sternum, just above the center of the sternum, with increased force.

Breathing

Airway management during pregnancy is more difficult. In late pregnancy the diaphragm is displaced upward, resulting in a 20% reduction in lung functional residual capacity, and resting oxygen demand increases by 20%. Thus, patients in late-term pregnancy can quickly become hypoxic [56]. Rescuers should monitor oxygen saturation closely and be ready to quickly support oxygenation and ventilation with 100% oxygen. Early intubation should be strongly considered and has the added benefit of reducing the risk of aspiration of gastric contents, a complication that is significantly likelier in these patients [57].

Circulation

One should follow ACLS guidelines for resuscitation. There is no evidence that current ACLS guideline medications or their dosage should be altered during the management of cardiac arrest in pregnancy [55]. Although there is a small risk of fetal complications with defibrillation, external cardioversion and defibrillation is considered safe at all stages of pregnancy [58, 59]. Defibrillation should be performed at the recommended ACLS defibrillation doses [55, 60].

Emergent Caesarean Delivery

At 25 weeks of gestation, the best survival rate for the fetus occurs when the infant is delivered within 5 min of the mother's cardiac arrest. This may also facilitate the successful resuscitation of the mother as well. Neonatal and obstetric personnel should be involved early in the resuscitation effort, and emergency cesarean delivery may be considered within 4 min of the onset of maternal cardiac arrest if there is no return of spontaneous circulation [55, 61–63].

Labor and Delivery and the Provocation of Cardiac Arrhythmias

The stress of labor and delivery can provoke cardiac arrhythmias. Patients with underlying heart disease should have continuous ECG monitoring even if no previous arrhythmia has been documented [64]. Arrhythmias in the peripartum period can be managed as previously prescribed. If the arrhythmia proves to be refractory to therapy or fetal compromise is suspected, cesarean delivery may be required.

Management of the Patient Postpartum

These patients are likely to experience improvement in their arrhythmias and symptoms following delivery. It is therefore essential to stress to these individuals that the substrate and potential for future arrhythmia problems remain unchanged and will likely complicate any future pregnancy. Patients with arrhythmias that are treatable by catheter ablation should be encouraged to have these arrhythmias treated before their next pregnancy. The patient who experienced management issues from congenital complete heart block should be evaluated for pacemaker implantation. In the setting of symptomatic

VT associated with either structural or genetic abnormalities (particularly LQT2), ICD implantation for primary or secondary prevention should be undertaken as per current guidelines. The temporary use of a LifeVest in the immediate postpartum period for patients with significant peripartum cardiomyopathy would seem reasonable to allow time for recovery of ventricular function before consideration of permanent ICD implantation.

Summary and Take-Home Messages

- Serious arrhythmias that threaten the lives of both the mother and the unborn child during pregnancy are rare.
- Most arrhythmias during pregnancy are benign and require no intervention.
- Documentation of the arrhythmias and correlation of findings with symptoms is imperative before initiation of any therapy.
- Women with previously diagnosed arrhythmias will frequently experience worsening of their arrhythmia during pregnancy.
- Advances in fluoro-less mapping technologies are opening the door to the possibility of performing advanced, invasive arrhythmia therapies during any stage of pregnancy without subjecting the mother and fetus to high doses of radiation. Women with known structural heart disease and preexisting arrhythmia disorders should be counseled on the advantages of preemptive treatment by means such as catheter ablation or device implantation before their next planned pregnancy.

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