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Pregnancy and Cardiovascular Disease (N Scott, Section Editor)

Cardiac Arrhythmias and Pregnancy

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This article is part of the Topical Collection on _Pregnancy and Cardiovascular Disease_

Abstract

Purpose of reviews In this review, the challenges of managing cardiac arrhythmias and syncope in the setting of pregnancy will be discussed.
Recent findings Arrhythmias in pregnancy are increasing, as diagnostic and therapeutic options have advanced and women are older at the time of gestation. Atrial fibrillation has become the most common arrhythmia in pregnancy. Inherited arrhythmia has become a more common entity, with advances in treatments and genetic testing, and require specialized treatments in pregnancy.
Summary The majority of arrhythmias in pregnancy are benign. The potential risk of increased cardiac morbidity and mortality exists for mother and fetus, especially in women with structural heart disease, which is becoming increasingly common. Early evaluation, diagnosis, and appropriate treatment are necessary to achieve optimal outcomes for both mother and fetus.

Case presentation

The following highlights an arrhythmia during pregnancy. A 34-year-old G5P1 at the gestational age of 31 weeks presented with a history of recurrent rapid palpitations accompanied by presyncope. Documentation of the palpitations was obtained on a 12-lead electrocardiogram (ECG) which demonstrated runs of non-sustained ventricular tachycardia (NSVT) (Fig. 1).

There was no prior history of structural cardiac disease. The ECG demonstrated polymorphic ventricular tachycardia initiated by a ventricular premature beat (VPB) originating from the right ventricular outflow tract, consistent with malignant right ventricular outflow tract ventricular tachycardia (RVOT VT). This entity has been described in the literature and is thought to be due

to triggered activity resulting in polymorphic ventricular tachyardia, as shown in Fig. 1. Similar triggered activity arising from the right or left Purkinje system has been described [1, 2]. Monomorphic VPBs led to short-coupled polymorphic VT (297 ± 41 ms), just as was observed in this case.

Introduction

Complex cardiovascular alterations occur during pregnancy over the 40-week gestational period [3]. These changes provide the increased metabolic requirements of the mother during the gestational period and the needs of the developing fetus [4]. The multi system events begin early, just after conception [5].

The physiologic changes can be seen both in function and structure of the cardiovascular system. The initial change is characterized by a rise in blood volume by 35–40%, combined with subsequent increases in heart rate (by as much as 15 beats per minute by the 28th–32nd week) and stroke volume, resulting in an increase in cardiac output by 30 to 50% (Fig. 2) [6–8]. The greatest rate of rise of the cardiac output is usually seen in the first 16 weeks of gestation. It is thought to be a direct compensation for the fall in systemic vascular resistance mediated by endothelial, hormonal, and prostaglandin factors. Specifically, progesterone, estrogen, and relaxin have been implicated in the increased vascular smooth muscle relaxation during pregnancy [7]. The main prostaglandin noted to play a role is prostacyclin, in concert with nitric oxide, exerts local effects on endothelial cells resulting in vascular smooth muscle relaxation [9].
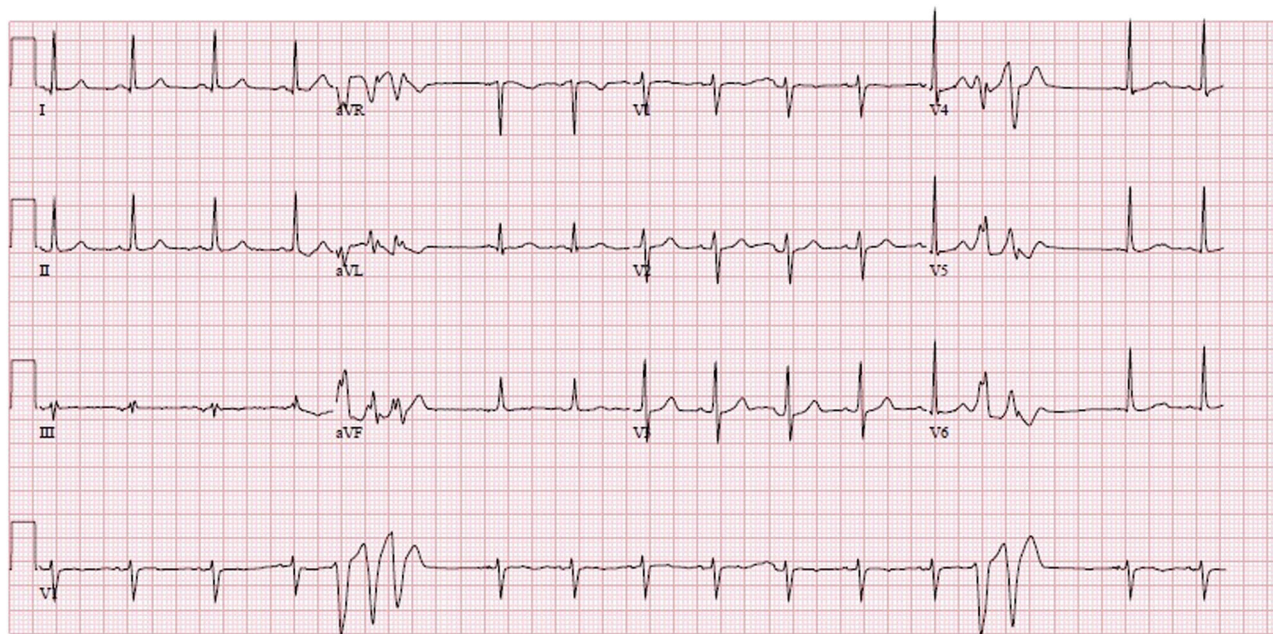
Fig. 1. Electrocardiogram demonstrating nonsustained polymorphic ventricular tachycardia, with the initiating ventricular beat originating from the right ventricular outflow tract.
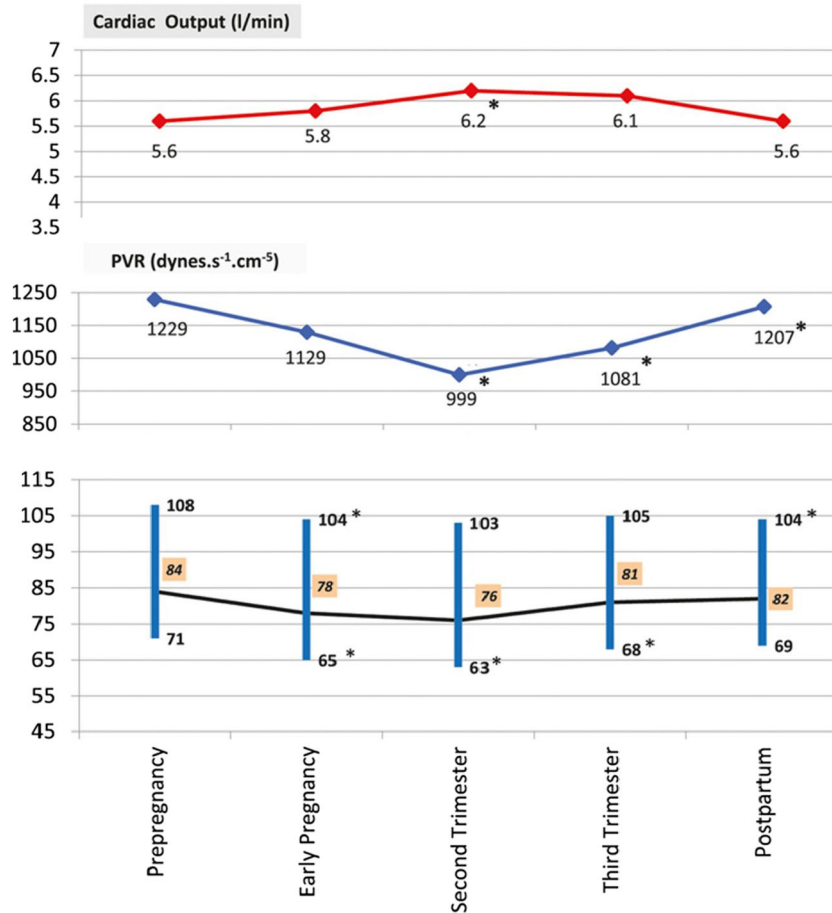
Fig. 2. Detailed hemodynamics were longitudinally studied in 54 women with normal pregnancies preconception and then at 6, 23, and 33 weeks during pregnancy and 16 weeks postpartum. Radial artery waveforms were obtained with a high-fidelity micromanometer; a central waveform was generated with a validated central transfer function; and mean arterial pressure (MAP) was determined with integrated software. Cardiac output (CO) was assessed with a noninvasive, validated inert gas rebreathing technique, and peripheral vascular resistance (PVR) was calculated from the formula PVR = MAP (mm Hg) × 80/CO (L/min). The reciprocal relationship of CO and PVR in pregnancy is demonstrated. CO increases from preconception to the second trimester and then falls to the preconception level 16 weeks postpartum. The PVR fell significantly by the second trimester (a 19% fall), followed by an increase in the third trimester and a return to preconception levels by 16 weeks postpartum. Blood pressures were measured in the nondominant arm with a validated automated measuring device. Blood pressure reached a nadir in the second trimester, although the majority of the reduction occurred early in pregnancy, followed by an increase in the third trimester and postpartum period. Systolic, diastolic, and mean arterial blood pressures are displayed. Figure created from the data of Mahendru et al. Adapted with permission from Sanghavi et al. [7, 8].

There are also structural changes involving remodeling of the cardiac chambers, with a temporary increase in size which reverts to pre-pregnancy levels post-partum [6]. These structural changes are adaptive due to increased preload from increased venous return [10]. Echocardiographic and cardiac MRI studies show significant remodeling of the left ventricle (LV) with increased LV end

diastolic and systolic volumes, LV wall thickness, and LV wall mass [9, 10]. Right ventricular (RV) mass is also increased. A summary of the normal structural cardiac changes associated with pregnancy is listed in Table 1.

During labor, a combination of anxiety, pain, and uterine contractions among other factors contributes to an increased heart rate and stroke volume, resulting in an even greater cardiac output. Increased blood volume in the immediate post-partum period arises from the relief of vena caval compression, in addition to uterine auto transfusion [11]. The second stage of labor is marked by an increase in heart rate by 52%, but a decrease in cardiac output by 32%, a decrease in stroke volume by 44% and an increase in systemic vascular resistance and systolic arterial pressure by 88 and 36%, respectively [10].

Arrhythmias in pregnancy

Most healthy women have relatively uncomplicated pregnancies; however, gestation can be described as a 'short term stress test [3]. The significant hemodynamic changes can unmask undiagnosed cardiac problems. Not uncommonly, they could worsen pre-existing disorders, including arrhythmias [12].

Arrhythmias are among the most common cardiac conditions in pregnancy [6]. The frequency of cardiac arrhythmias in pregnancy is on the rise [13] (Fig. 3).

Factors implicated in the creation of a pro-arrhythmic environment include the increased myocardial stretch induced by hypervolemia, the increased catecholamine activity in pregnancy, and hormonal influences on myocardium [14, 15]. In addition, greater myocardial irritability, changes to myocardial refractoriness, and greater cardiac excitability have been described as contributing factors [16]. 'Electrical remodeling' in pregnancy ('gestational re-modeling'), evidenced by ECG changes such as an increased heart rate and a lengthened QT interval that predispose to arrhythmic activity, has been described [17] (Table 2). A higher frequency of atrial and ventricular premature extra systoles, which could trigger arrhythmias, is seen in pregnancy. Importantly, structural heart disease secondary to congenital causes has become relatively more common in pregnancy, as greater numbers of women with congenital heart disease survive to adulthood [18].

Maternal mortality rates in the USA have increased dramatically in the past 20 years, from a rate of about 10 per 100,000 in the early 1990s to about 17 per 100,000 in 2013 [19]. Cardiovascular disease accounted for the highest percentage (16.5%) of pregnancy-related deaths. Cardiomyopathy was responsible for an additional 11% [20]. Cardiac evaluation is of greater importance in view of the higher incidence of cardiac disease in pregnancy, with the growing trends

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Table 1. Structural cardiac changes in a normal pregnancy

Increased LV wall thickness (more than 25% above pre-pregnancy levels)

Increased LV wall mass (more than 50% above pre-pregnancy levels)

Increase in RV mass

Dilatation of all 4 chambers to varying degrees

Transient trivial mitral regurgitation, physiologic tricuspid regurgitation, and pulmonic regurgitation
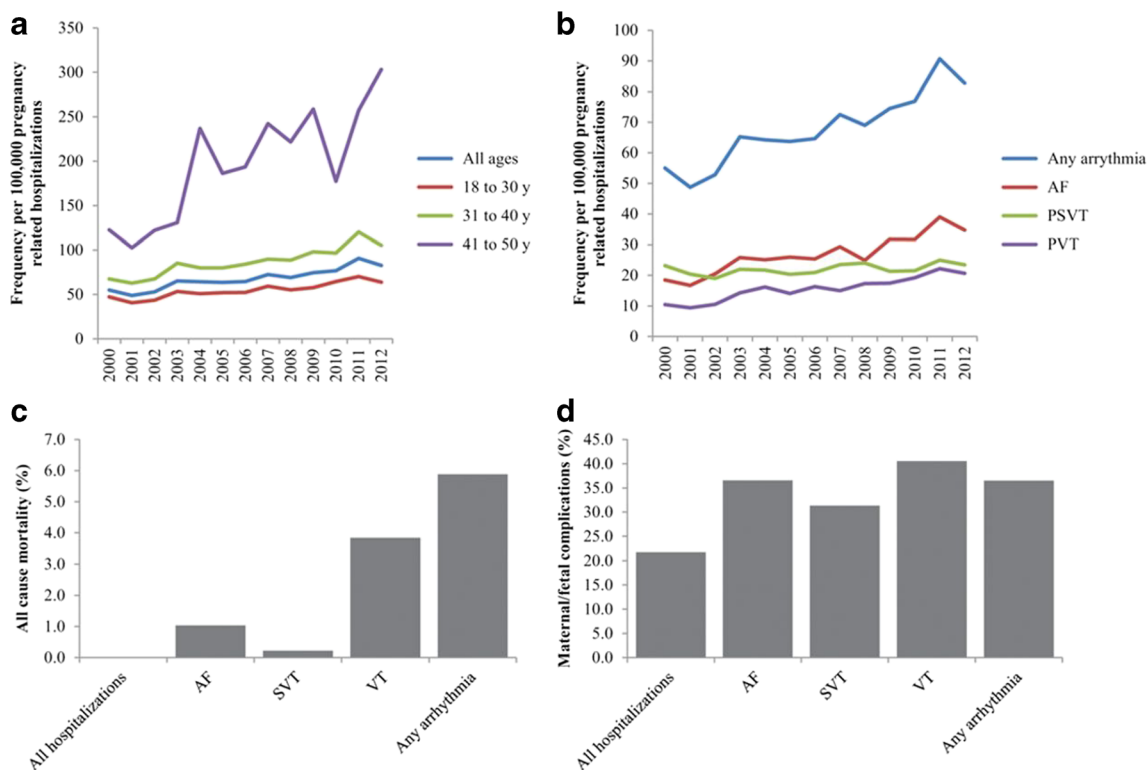
Fig. 3. Frequency of arrhythmia in pregnancy and associated mortality and complications. **a** Frequency of any arrhythmia per 100,000 pregnancy-related hospitalizations for the entire study period, stratified by age. **b** Frequency of arrhythmias per 100,000 pregnancy-related hospitalizations by arrhythmia type for the entire study period. **c** All-cause mortality in percentage for the entire study period. **d** Maternal/fetal complications (including preterm labor, ante- or postpartum hemorrhage, preeclampsia, eclamspia, gestational hypertension, transfusion, postpartum infection, and fluid and electrolyte imbalance) in percentage for the entire study period. Reproduced with permission from Vaidya et al. [13].

of maternal age as women delay childbirth, increasing cardiovascular risk factors among pregnant women, and more patients with congenital heart disease surviving to reproductive age.

Investigations for women with symptoms suggestive of arrhythmia may consist of 12 lead ECGs, trans thoracic echocardiogram (TTE), baseline blood tests (complete blood count, electrolytes including calcium, magnesium and phosphorus, and renal function), and thyroid function tests. Ambulatory

Table 2. Normal electrocardiographic changes in pregnancy [14]

Increased ventricular rate
Left axis deviation
Premature atrial and ventricular complexes
Non-pathologic Q waves lead III
Lengthening of QT interval
Inverted T waves lead III
ST segment depression and T wave inversion in inferior and lateral leads

Holter or trans telephonic monitoring may be indicated to obtain symptom-rhythm correlation and document the nature of the arrhythmia in order to guide therapy. An exercise stress test may assist in diagnosis if exertion is a known trigger. Cardiac MRI may be helpful in select situations such as detection of a cardiomyopathy, though gadolinium is avoided during pregnancy. Conventional electrophysiology (EP) studies may be needed for patients, but would generally be avoided during pregnancy due to the risk of fluoroscopy exposure.

Management of specific arrhythmias in pregnancy

The spectrum of arrhythmias in pregnancy mirrors that in the female population, albeit with greater complexity. They range from relatively benign rhythm disturbances such as atrial and ventricular extra systoles to life-threatening arrhythmias including ventricular tachycardias and asystole. Atrial premature beats are the most frequently observed arrhythmias in pregnancy and are generally benign [21]. Patients are asymptomatic in most cases, with no hemodynamic instability. A minority may experience palpitations [22]. Identification and avoidance of triggers may be helpful. No specific treatment is required unless symptoms become burdensome, in which case, cardio selective beta blockers may be considered [21]. The FDA classification and possible adverse effects of anti-arrhythmic medications are summarized in Table 3 (adapted and modified from Regitz-Zagrosek et al.) [23]. Further information on safety and potential adverse effects can be found at https://toxnet.nlm.nih.gov/newtoxnet/lactmed.htm [24].

*The United States Food and Drug Administration (FDA) classified drug pregnancy risks in five categories (A, B, C, D, X) (adapted and modified from Regitz-Zagrosek et al.) [23]. Category A—safe to use in pregnancy; category B—either animal reproduction studies have not demonstrated a fetal risk but there are no controlled studies in pregnant women, or animal reproduction studies have shown an adverse effect that was not confirmed in controlled studies in women; category C—either animal studies had shown adverse effect to the fetus or no animal studies had been conducted, but there are no adequate studies in pregnant women. Drugs should be given only if potential benefits justify the potential risk to the fetus; category D—there is evidence of human fetal risk, but the benefits from use in pregnant woman may be acceptable despite the risk (e.g., treatment of life-threatening conditions). It should be noted that the labeling of drugs will be changed to abide by the pregnancy and lactation labeling rule (PLLR), but this has not yet taken full effect [25].

Supraventricular tachycardia

Paroxysmal supraventricular tachycardias (PSVT) in pregnancy include AV nodal reentrant tachycardia (AVNRT), AV re-entrant tachycardia (AVRT), and atrial tachycardia. They are common in patients without structural heart disease [26]. They have been reported as the most common sustained tachyarrhythmias in pregnancy. Exacerbations of SVT have been found to occur in 20–44% of pregnant women with a documented history of SVT [27]. SVTs are typically a nuisance with a benign outcome in pregnancy. However, the well-being of the fetus could be jeopardized by hemodynamic changes triggered by the tachycardia.

If a patient with PSVT is found to be hemodynamically unstable, immediate direct current cardioversion (DCCV) is recommended. For stable patients, vagal

Table 3. Antiarrhythmic drugs: FDA classification and adverse effects in pregnancy and lactation

Drug	FDA category*	Placenta permeable	Transfer to breast milk	Adverse effects
Adenosine	C	No	No	No fetal adverse effects reported
Amiodarone	D	Yes	Yes	Thyroid insufficiency (9%), hyperthyroidism, goiter, bradycardia, growth retardation, premature birth.
Atenolol	D	Yes	Yes	Hypospadias (first trimester); birth defects, low birth weight, bradycardia and hypoglycaemia in fetus (second and third trimester).
Bisoprolol	C	Yes	Yes	Bradycardia and hypoglycaemia in fetus.
Digoxin	C	Yes	Yes	Serum levels unreliable, safe
Disopyramide	C	Yes	Yes	Uterine contraction
Diltiazem	C	No	yes	Possible teratogenic effects
Flecainide	C	Yes	Yes	Unknown
Labetolol	C	Yes	Yes	Intrauterine growth retardation (second and third trimester), neonatal bradycardia and hypotension (used near term)
Lidocaine	C	Yes	Yes	Fetal bradycardia, acidosis, central nervous system toxicity
Metoprolol	C	Yes	Yes	Bradycardia and hypoglycaemia in fetus
Mexilitine	C	Yes	Yes	Fetal bradycardia.
Procainamide	C	Yes	Yes	Unknown
Propafenone	C	Yes	Unknown	Unknown
Propranolol	C	Yes	Yes	Bradycardia and hypoglycaemia in fetus
Quinidine	C	Yes	Yes	Thrombopenia, premature birth, VIII th nerve toxicity.
Sotalol	B	Yes	Yes	Bradycardia and hypoglycaemia in fetus (limited experience).
Verapamil oral	C	Yes	Yes	Well tolerated (limited experience during pregnancy)
Verapamil IV	C	Yes	Yes	Intravenously use is may be associated with a greater risk of hypotension and subsequent fetal hypoperfusion.
Vernakalant	–	Unknown	Unknown	Unknown

maneuvers are first-line therapy to terminate SVTs. In pregnant women, aden-osine is safe and recommended if vagal maneuvers fail. Intravenous metoprolol is suggested as next-line therapy, followed by intravenous verapamil if the beta blockers fail or are not tolerated [28]. Adenosine has been reported to have successfully converted PSVTs to sinus rhythm in 90% of cases during pregnancy. Beyond the first trimester, sotalol or flecainide may be considered in highly symptomatic cases in the rare event that beta blockers do not succeed.

Interventional treatment (catheter ablation) is usually avoided due to the risk of fluoroscopic radiation exposure; however, the advent of electroanatomical mapping methods without the use of fluoroscopy may permit this to be performed safely, in situations where the arrhythmia is refractory to medical therapy, and remains symptomatic [29].

Focal atrial tachycardias occur less commonly. Adenosine may be useful to terminate a non-reentrant atrial tachycardia or to diagnose the underlying rhythm. DC cardioversion is recommended for unstable patients. The treatment strategy in stable patients is mainly rate control with drugs (such as oral digoxin, metoprolol, or verapamil). The ACC/AHA/HRS guidelines on the treatment of SVT in pregnancy are summarized in Table 4 [30]; refer to Table 3 for risks associated with the recommended drugs in pregnancy and lactation.

Atrial fibrillation (AF) and atrial flutter (AFl) were previously less common in pregnancy than PSVT, but has recently been found to have a rising prevalence [13, 31]. The reported prevalence is variable, with rates of 2 in 100,000 hospital admissions of pregnant women in one study [32] and between 59.3 per 100,000 pregnancies in a larger community-based population study [33]. Risk factors for AF/AFl in pregnancy include pre-pregnancy presence of AF/AFl, structural heart disease, increased maternal age of pregnancy, traditional cardiovascular risk factors (e.g., hypertension, hyperlipidemia, diabetes etc) [34]. AF risk may be highest in the third trimester, possibly related to expanded plasma volume, increased red cell mass, and cardiac load [31]. Unstable AF cases should be promptly treated by synchronized DC cardioversion. Guidelines recommend rate control as first-line therapy by digoxin, beta blocker, or non-dihyropyridine calcium channel blocker therapy for stable patients [35]. Rhythm control is not commonly employed in stable patients, but flecainide and sotalol were safely used for fetal arrhythmias in a non-randomized multi-center trial, and are thought to be acceptable options for treatment of symptomatic maternal AF [36]. Oral amiodarone carries a significant risk of fetal adverse effects and is suggested only for AF refractory to other methods of treatment, with ongoing unacceptable symtpoms [36].

With the recognized hypercoagulability that arises in pregnancy, and the stroke risk associated with AF, an additional consideration in pregnant women with AF is anticoagulation [31]. Heparin and low molecular weight heparins (LMWH) are generally preferred during pregnancy as they do not cross the placenta. Low-dose aspirin, LMWH, and unfractionated heparin are considered safe in pregnancy. Warfarin has known teratogenic effects in the first trimester and may be associated with fetal hemorrhage close to the end of pregnancy; hence, the suggestion is that it should be used from the second trimester up to 1 month before delivery. The newer oral anticoagulants (e.g., dabigatran, rivaroxaban, apixaban, edoxaban) are not recommended, as there is as yet insufficient data on their safety in pregnancy [23].

Ventricular arrhythmias

Ventricular arrhythmias include benign arrhythmias such as ventricular premature beats as well as potentially fatal ventricular tachycardias and ventricular fibrillation. Ventricular premature beats (VPBs), like atrial premature beats (APBs), are mostly asymptomatic, non-life threatening, and do not require treatment [37]. Sustained ventricular tachyarrhythmias (VT) or ventricular

Table 4. ACC/AHA/HRS guidelines on acute and chronic treatment of SVT in pregnancy [30]

Class of recommendation[a]	Recommendation
Acute treatment of SVT in pregnancy	
I	Vagal maneuvers are recommended for acute treatment in pregnant patients with SVT.
I	Adenosine is recommended for acute treatment in pregnant patients with SVT.
I	Synchronized cardioversion is recommended for acute treatment in pregnant patients with hemodynamically unstable SVT when pharmacological therapy is ineffective or contraindicated.
IIa	Intravenous metoprolol or propranolol is reasonable for acute treatment in pregnant patients with SVT when adenosine is ineffective or contraindicated.
IIb	Intravenous verapamil may be reasonable for acute treatment in pregnant patients with SVT when adenosine and beta blockers are ineffective or contraindicated.
IIb	Intravenous procainamide may be reasonable for acute treatment in pregnant patients with SVT.
IIb	Intravenous amiodarone may be considered for acute treatment in pregnant patients with potentially life-threatening SVT when other therapies are ineffective or contraindicated.
Chronic Treatment of SVT in pregnancy	
IIb	The following drugs, alone or in combination, can be effective for ongoing management in pregnant patients with highly symptomatic SVT: a. Digoxin b. Flecainide c. Metoprolol d. Propafenone e. Propranolol f. Sotalol g. Verapamil
IIb	Catheter ablation may be reasonable in pregnant patients with highly symptomatic, recurrent, drug-refractory SVT with efforts toward minimizing radiation exposure.
IIb	Oral amiodarone may be considered for ongoing management in pregnant patients when treatment of highly symptomatic, recurrent SVT is required and other therapies are ineffective or contraindicated.

[a]Class I: strong recommendation; class IIa: moderate recommendation (reasonable to perform); class IIb: weak recommendation; class III: no benefit

fibrillation (VF) are rare in pregnancy. Ertekin et al. evaluated the incidence of VT/VF in a registry of pregnancy and cardiac disease (ROPAC) that included 2966 pregnant women over 5 years [38]. They found an incidence of VT/VF of 1.4%, with highest rates among cardiomyopathy patients. VT/VF occurred more often in patients with structural heart disease or previous history of arrhythmia.

Monomorphic VT could present for the first time during pregnancy in women with structurally normal hearts [6]. Referred to as idiopathic VT, these arise mostly from the right ventricular outflow tract (RVOT), and less commonly the left ventricular outflow tract (LVOT). Prognosis is generally benign.

Cardio selective beta blockers are recommended for treatment of idiopathic RVOT VT, with sotalol reserved for cases non-responsive to this initial treatment. Verapamil is recommended for the less common idiopathic fascicular LVOT VT [37]. In a few cases which are refractory or poorly tolerated, catheter ablation may be an option.

Elevated catecholamine levels have been linked to idiopathic VT in the literature [37].

Inherited cardiac conditions

Inherited channelopathies and cardiomyopathies may pose specific challenges when present in pregnancy. The management of these conditions during pregnancy is discussed in this section.

Long QT syndrome is the commonest inherited channelopathy. It has been reported that VT risk is highest, not during the pregnancy itself, but post partum—up to 9 months after delivery, where the risk of experiencing a cardiac event is 2.7-fold [39]. This is particularly the case in LQT2, where the risk in the post-partum period is dramatically higher than in LQT1 or LQT3. During pregnancy, in particular the third trimester, increased heart rate and resultant shortened QT interval are partially protective; post partum, however, the increased adrenergic state, disrupted sleep pattern, and psychological stress may contribute to the increased event rate [40]. Guidelines recommend continued beta blocker therapy throughout pregnancy and the post partum period (even with breastfeeding). Implantable cardioverter defibrillators (ICDs) are a safe option in pregnancy, if an indication emerges [37].

Brugada syndrome is another rare but potentially life-threatening inherited disorder with an increased risk of sudden cardiac death. Studies have reported that event rates are not increased in pregnancy or the peripartum period [37]. ICDs are indicated only in high-risk patients. Quinidine has been successful in terminating unstable VT in one case report of a pregnant woman with this syndrome [41].

Catecholaminergic polymorphic ventricular tachycardia (CPVT) is an even rarer inherited disorder. Beta blockers without intrinsic sympathomimetic activity, e.g., nadolol, are first line for therapy/prophylaxis of CPVT, and exercise restriction is advisable. Flecainide is used when beta blockers are not successful in terminating the arrhythmia [42].

The prevalence of hypertrophic cardiomyopathy (HCM) in the general population is approximately 1 per 500 [43]. Women with HCM can typically progress through pregnancy and delivery with minimal risk. The maternal mortality rate has been estimated at 0.5% and is predominantly seen in patients with advanced disease [44]. In the asymptomatic patient population on no medical therapy, HCM is not a contraindication to pregnancy. In patients with mild to moderate left ventricular outflow tract (LVOT) obstruction with symptom control on medical therapy (beta blockers, verapamil, or disopyramide), pregnancy is reasonable with maternal fetal medicine and cardiology involvement. For these patients, medical therapy should be continued with no interruption with increased monitoring for fetal bradycardia. The maternal mortality rate is very low in this patient group [43]. In patients with advanced disease, including women with resting or provocable LVOT obstruction of 50 mmHg or higher, or symptoms not adequately controlled by medical therapy alone,

pregnancy is associated with increased risk [45]. In general, special precautions at the time of delivery and cesarean section are unnecessary. In the 24 h after delivery, however, careful monitoring is advised as significant fluid shifts increase the risk of pulmonary edema in these patients with non-compliant and hypertrophied left ventricles [45].

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is characterized by fibro-fatty replacement of the right ventricular myocardium predisposing patients to ventricular arrhythmia, heart failure, and an increased risk of sudden cardiac death [46, 47]. According to registry data, while most pregnancies in ARVC were tolerated well, 13% of pregnancies were complicated by ventricular arrhythmia and 5% were complicated by heart failure. That said, the likelihood of experiencing a first sustained ventricular arrhythmia or developing heart failure was not significantly increased during pregnancy when outcomes were compared to non-pregnant women with ARVC. Following recognition of pregnancy, medications with low fetal risk such as beta blockers should be continued. In most cases, vaginal delivery with cardiac monitoring is reasonable [48].

Syncope in pregnancy

Syncope has been reported in approximately 4.6% of pregnant women [49]. Higher rates in late pregnancy (up to 10% of women) have been linked mainly to vasovagal syncope due to abrupt hemodynamic fluctuations with position changes. It is believed to be caused by inferior vena caval occlusion by the enlarged uterus leading to significant hypotension and neuronally mediated severe bradyarrhythmia, with resultant cerebral hypoperfusion and syncope. It can be avoided or relieved if a left lateral position is adopted when supine. Small studies have demonstrated no increased risk of pregnancy-related complications in patients with vasovagal syncope, as compared to healthy controls [50]. The majority of syncopal events in pregnancy are due to a neurocardiogenic event; however, the history and subsequent investigations should focus on distinguishing vasovagal syncope from other causes such as a brady or tachyarrhythmia.

Conclusion

The majority of arrhythmias in pregnancy are benign. The potential risk of increased cardiac morbidity and mortality exists for mother and fetus, especially in women with structural heart disease, which is becoming increasingly common. Early evaluation, diagnosis, and appropriate treatment are necessary to achieve optimal outcomes for both mother and fetus.

Compliance with Ethical Standards

Conflict of Interest
Ciorsti MacIntyre, Chinyere Iwuala, and Ratika Parkash each declare no potential conflicts of interest.

Human and Animal Rights and Informed Consent
This article does not contain any studies with human or animal subjects performed by any of the authors.

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