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Treatment of Arrhythmias During Pregnancy

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Abstract: Cardiac disease is the most common cause of maternal mortality in developed nations. Cardiac arrhythmias are frequent among patients with structural heart disease and may require immediate treatment to prevent hemodynamic instability leading to acute maternal and fetal decompensation. Antiarrhythmic therapy during pregnancy should follow the same principles recommended for nonpregnant individuals. Although multidisciplinary management is recommended, obstetricians, and maternal-fetal medicine specialists may sometimes need to emergently recognize and treat rhythm anomalies before support services become available.

Key words: pregnancy, tachycardia, bradycardia, cardioversion

Introduction

Although cardiac arrhythmias are overall uncommon during pregnancy, their incidence is expected to increase secondary to the rise in cardiac disease among reproductive-age women. In fact, in developed nations, cardiovascular disease is currently the leading cause of maternal mortality.[1] Contributing factors include advanced maternal age, obesity, chronic hypertension, pregestational diabetes, and especially better surgical techniques of congenital heart defects resulting in more women with corrected congenital complex cardiac anomalies becoming pregnant.[1]

Pregnancy by itself is arrhythmogenic as a result of physiological changes including increased resting heart rate, volume expansion with cardiac chamber dilation, and increased levels of placental-originated arrhythmogenic hormones. Increased effective blood volume results in atrial and ventricular stretching with activation of stretch-sensitive ion channels leading to membrane depolarization and conduction slowing.[2,3]

Pregnancy-induced heart chamber enlargement also increases the length of

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The authors declare that they have nothing to disclose.

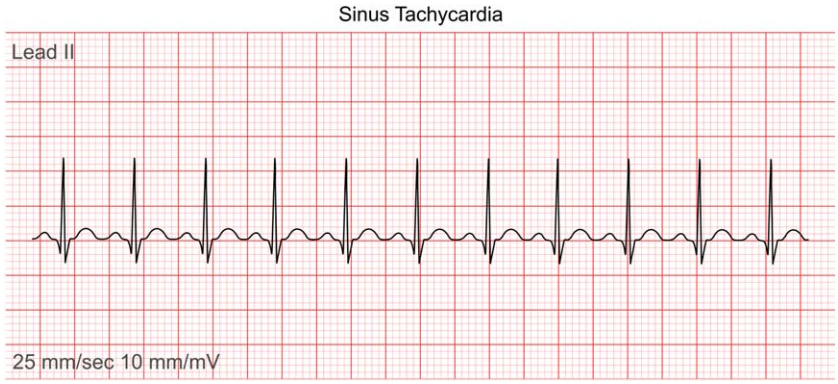

FIGURE 1. Sinus tachycardia. Regular R-R interval and each QRS complex are preceded by a P-wave. full color online

reentrant pathways potentially triggering reentrant tachyarrhythmias.[2]

At the same time, increased estradiol, progesterone, and free cortisol increase the risk of cardiac arrhythmias by augmenting the responsiveness of adrenergic receptors within the myocardium.[4]

In general, management of cardiac arrhythmias during pregnancy should follow the same principles as in nonpregnant individuals including early synchronized direct current cardioversion for unstable tachyarrhythmias and use of chronotropic agents and/or temporary intravenous pacing for symptomatic bradycardias. In cases of nonreassuring fetal status, delivery is rarely indicated as immediate interventions (electrical cardioversion or pharmacological agents) will improve peripheral perfusion by restoring a normal cardiac rhythm. In this article, we will provide a simplified practical approach to the initial basic management of the most common maternal cardiac arrhythmias during pregnancy. Although some rhythm disturbances will require immediate management, consultation with specialists such as cardiology, where available, is highly recommended.

TACHYARRHYTHMIAS

Tachyarrhythmias are usually divided into supraventricular and ventricular. Supraventricular tachycardias originate in the atrium, above the atrioventricular node, and have a narrow QRS complex (<0.12 s). Ventricular tachycardias originate in the ventricular tissue and have a wide QRS (>0.12 s). Although ventricular tachycardias are less common, they

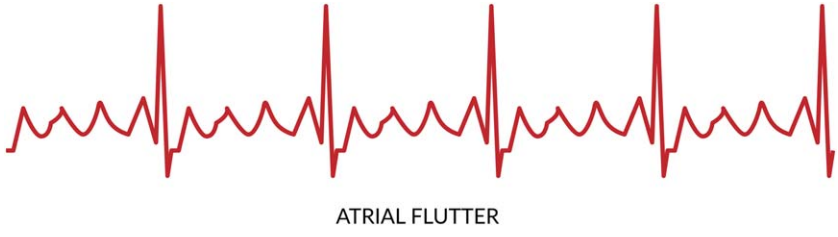

FIGURE 2. Atrial flutter with 4:1 AV block. Note regular R-R interval with sawtooth appearance of P-waves. full color online

carry the highest risk of complications such as sudden death and require immediate treatment.

Regardless of initial classification, any pregnant patient with new-onset tachyarrhythmia leading to hemodynamic instability (eg, systemic hypotension, chest pain, dyspnea, and altered mental status) will require immediate synchronized direct current cardioversion. There is no evidence that cardioversion is harmful during pregnancy.[2]

Supraventricular Tachyarrhythmias

As previously discussed, these groups of tachyarrhythmias have a narrow QRS complex and may be classified as regular or irregular based on the R-R interval. The most common forms of regular supraventricular tachycardia include sinus tachycardia, atrial flutter with a fixed atrioventricular conduction ratio, atrioventricular nodal reentrant tachycardia (AVNRT), and atrial tachycardias. Irregular narrow complex supraventricular tachycardias include atrial fibrillation (AF) and atrial flutter with variable atrioventricular conduction ratios. A brief description of each rhythm and initial management strategies are discussed next.

Sinus tachycardia manifests as sinus rhythm with a rate above 100 beats/min (bpm) and may be as high as 220 minus the age of the patient. Importantly, the onset of sinus tachycardia is usually *gradual* and reflects an underlying process such as pain, anxiety, fever, congestive heart failure, pulmonary embolism, hypovolemia, anemia, or thyrotoxicosis, among others. The diagnosis is made through a detailed physical exam and history together with classic electrocardiographic (EKG) findings. Each QRS complex is preceded by a P-wave, and the R-R interval is regular (Fig. 1). In most cases, treatment of the underlying cause will result in the cessation of sinus tachycardia.

In atrial flutter with fixed AV block, the R-R interval is regular resulting in a narrow complex regular tachycardia. For example, if AV conduction is 2 to 1 and the heart rate is 150 bpm, this means that the atria is contracting at a rate of 300 bpm and the ventricle at half this rate as the AV conduction is fixed at 2:1. Unlike sinus tachycardia, atrial flutter usually has a sudden onset.[5] As most flutters have a 2:1 conduction and the usual atrial rate is 280 to 300 bpm, a sudden onset of narrow complex tachycardia with a rate of 140 to 150 bpm should raise the suspicion of atrial flutter.[5] In these cases, the atrial

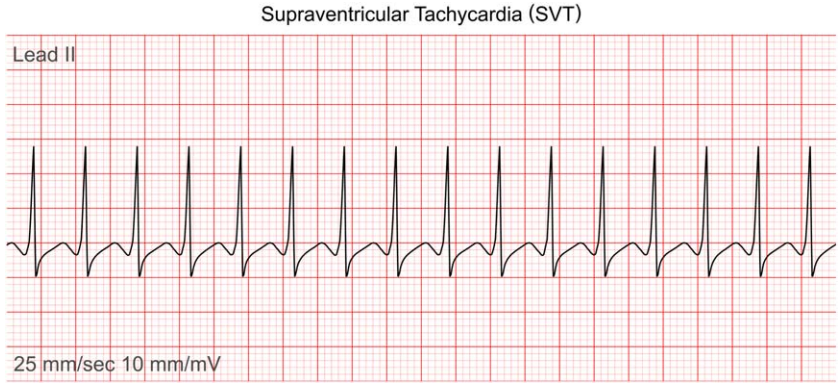

FIGURE 3. Atrioventricular nodal reentrant tachycardia. Note the regular R-R interval with unrecognizable P-waves. full color online

flutter waves may not be visible as they will be superimposed on the QRS complex and/or the T-wave. The use of adenosine as a diagnostic maneuver may unmask the flutter waves as they will become apparent after the transient suppression of the sinus node induced by adenosine. In patients with slower conduction rates (eg, 3:1 or 4:1), the diagnosis is easily made by identifying the flutter (sawtooth) waves (Fig. 2).

If a patient presents with atrial flutter and is hemodynamic unstable, immediate synchronized electrical cardioversion is indicated. A starting energy as low as 50 joules (J) is often effective.[6] In hemodynamically stable patients, 2 treatment strategies are available. Rhythm control consists of cardioverting the patient back to sinus rhythm, either electrically or pharmacologically with agents such as amiodarone, ibutilide, or flecainide. In contrast, a rate control strategy consists of slowing the atrioventricular node conduction aiming for a heart rate <110 bpm providing sufficient diastolic ventricular filling time leading to a preserved ejection fraction.[6] Usually, rate control may be achieved with beta-blockers (esmolol, metoprolol), calcium channel blockers (diltiazem), or digoxin. We favor a rate control strategy during pregnancy as outcomes are overall comparable to a rhythm control approach and will result in less fetal exposure to antiarrhythmics. Although adenosine is mainly indicated for AVNRT, it may be used when the differential between an AVNRT and atrial flutter with fixed 2:1 ratio is unclear (as discussed previously). Adenosine transiently blocks the sinus and atrioventricular nodes; following its administration a short period of asystole is seen in cases of AVNRT. If the arrythmia was in fact a 2:1 flutter, instead of a flat line, the flutter waves will become evident as the QRS will disappear from the EKG transiently.[5]

AVNRT usually presents as a sudden onset narrow QRS regular tachycardia with rates between 150 and 250 bpm. P-waves are not visible as they are "hidden" behind the terminal portion of the QRS. AVNRT is due to the presence of a short circuit pathway within the atrioventricular node. Depolarization moves normally from the atria to the ventricles, but once in the ventricles, the electrical signal travels retrogradely to the atrial tissue through the abnormal pathway resulting in a new depolarization of the atria.[6] A circular motion follows with repetitive depolarization of the atria and ventricles. Therapy is aimed to slowing

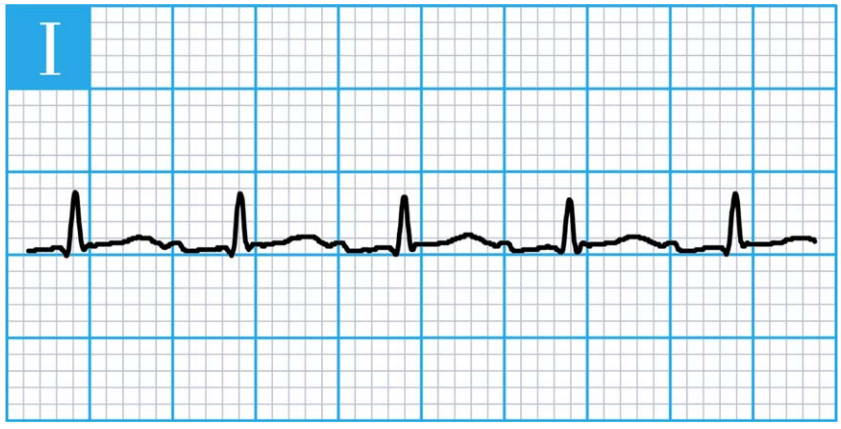

FIGURE 4. Atrial tachycardia. Note regular R to R interval, narrow QRS, and abnormal P-wave morphology indicating an ectopic focus. full color online

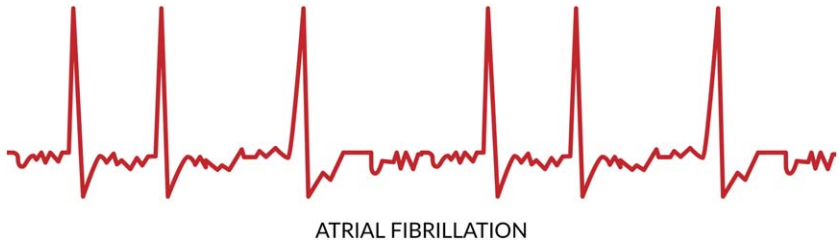

FIGURE 5. Atrial fibrillation. Note irregular R-R interval with no identifiable P-wave. full color online

conduction in the atrioventricular node. If the patient is hemodynamically unstable, direct current synchronized electrical cardioversion starting with 50 J is indicated. If stable, vagal maneuvers (Valsalva maneuver, carotid sinus massage, and immersion of the face in ice water) may be attempted. If unsuccessful, the first line of therapy is adenosine (which will terminate nearly all AVNRT).[6] Adenosine can cause bronchospasm, so it should be avoided in patients with severe reactive airway disease. In cardiac transplant patients, lower doses of adenosine are required due to a higher risk of prolonged atrioventricular block in denervated hearts; a starting dose of 1.5 mg is recommended.[7] As previously discussed, when adenosine is administered the cardiac monitors usually show a short period of asystole (as transient inhibition of the sinus node occurs) before the resumption of electrical activity. Most patients usually experience transient chest pain, flushing, and dyspnea. Immediate defibrillation equipment should be readily available anytime adenosine is utilized.

If adenosine is unsuccessful, slowing of the atrioventricular node may be achieved with beta-blockers, calcium channel blockers, or digoxin. Figure 3 depicts an AVNRT.

Atrial tachycardia is secondary to an automated focus in the atrial tissue firing

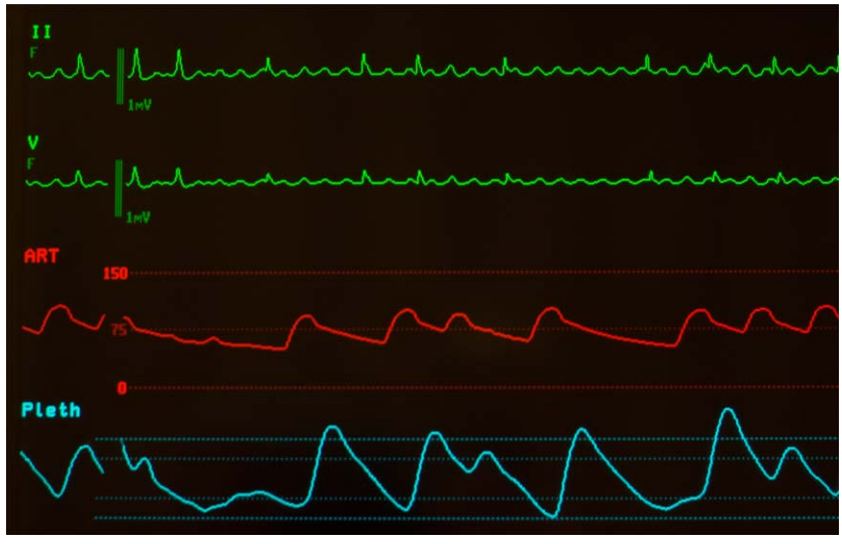

FIGURE 6. Atrial flutter with variable AV block. Notice the baseline sawtooth appearance of flutter waves. full color online

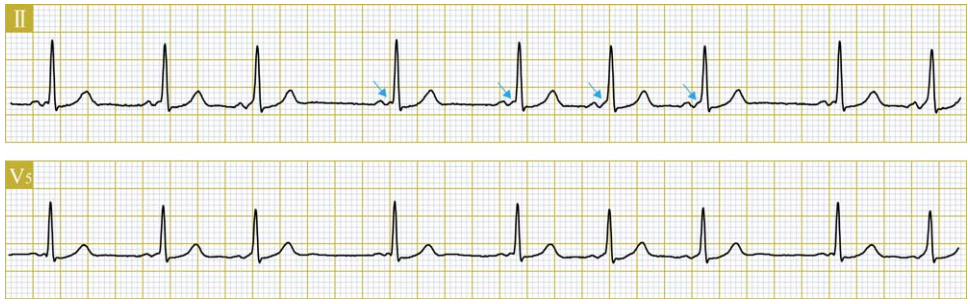

FIGURE 7. Wolf Parkinson White. Note the short PR interval and a slurred upstroke of the QRS, also known as a Delta wave. full color online

faster than the sinus node. It is usually of rapid onset, regular, narrow QRS, and rates between 150 and 250 bpm.[5] P-waves, when visible, are of different morphology compared with P-waves seen, although in sinus rhythm. Similarly, the atrioventricular interval may be abnormally short. Treatment is directed at slowing atrioventricular conduction with vagal maneuvers, beta- blockers, and calcium channel blockers. Adenosine may also be used; however, its efficacy is lower than for AVNRT. Of note, atrial tachycardia is highly resistant to electrical cardioversion as it is secondary to increased automaticity. An example of atrial tachycardia is shown in Figure 4.

Irregular supraventricular tachycardias, on the other hand, have an irregular R-R interval with a narrow QRS and include mainly AF and atrial flutter with a variable atrioventricular conduction ratio.

AF accounts for approximately one-third of hospitalizations for cardiac arrhythmias worldwide. It is the most common sustained cardiac arrhythmia; however, it occurs infrequently during pregnancy. When identified, clinicians should rule out secondary causes such as valvular heart disease, myocardial ischemia, thyroid disease, congenital heart defects, or pulmonary disease. Regarding treatment, if the patient is hemodynamically unstable, synchronized electrical cardioversion is indicated with a starting energy of 100 to 200 J.[6] If hemodynamically stable, either a rate control or rhythm control strategy may be pursued as previously described for atrial flutter. Ventricular rate control can be achieved using beta-blockers, nondihydropyridine calcium channel blockers (diltiazem or verapamil), or digoxin. The goal of this strategy is to slow AV conduction allowing adequate ventricular filling time. A rate below 110/bpm is considered appropriate.[8] Rhythm control consists of restoring sinus rhythm through either electrical or pharmacological cardioversion with the use of agents such as amiodarone, procainamide, ibutilide, flecainide, and sotalol. Overall, outcomes are similar with either strategy with less side effects from antiarrhythmic use in

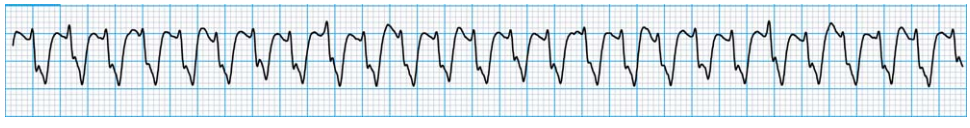

FIGURE 8. Monomorphic ventricular tachycardia. Note regular R-R interval with the wide QRS complex. full color online

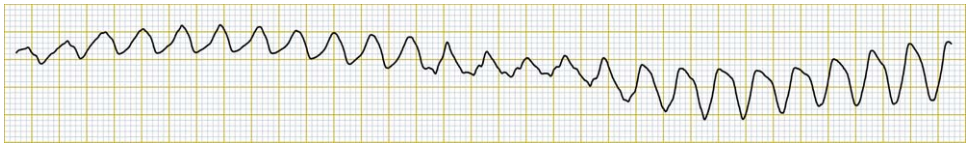

FIGURE 9. Polymorphic ventricular tachycardia. Note different QRS morphologies with irregular R-R intervals. full color online

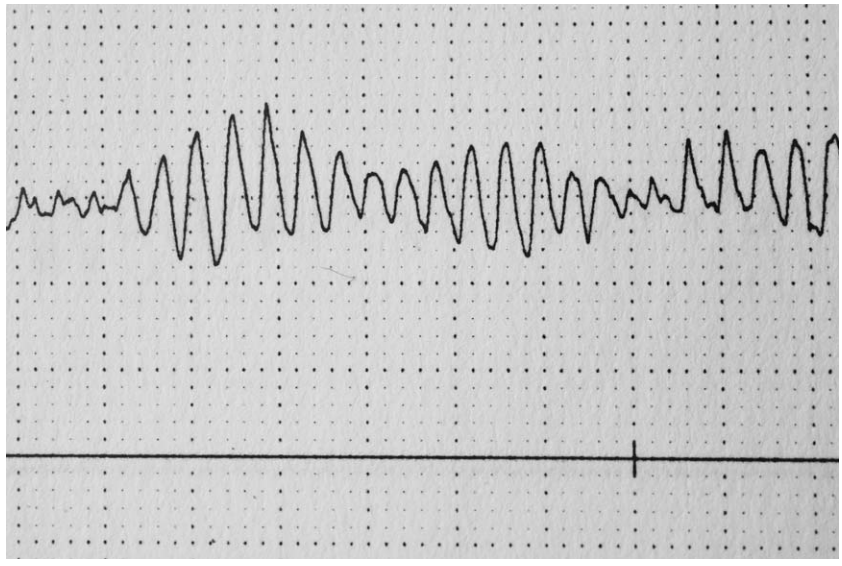

FIGURE 10. Torsades de Pointes. Note irregular R-R intervals and polymorphic QRS which appear to be twisting around the EKG baseline.

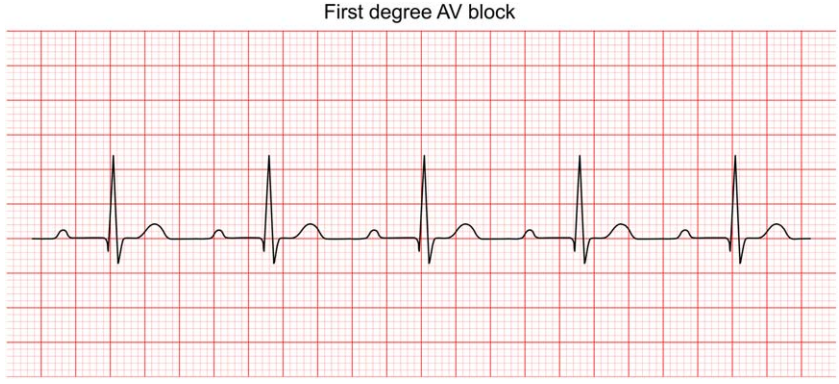

FIGURE 11. First-degree AV block. Notice bradycardia with a prolonged PR interval. full color online

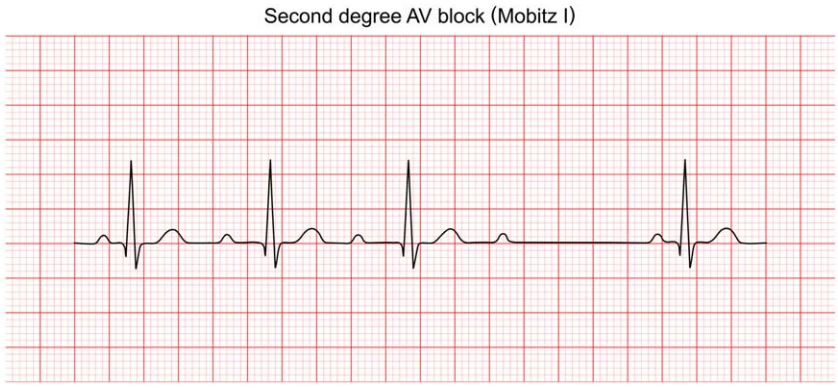

FIGURE 12. Second-degree AV block Mobitz I. Note progressive prolongation of the PR interval until 1 P-wave is not conducted. After the non-conducted P-wave, the PR interval shortens again. full color online

rate-controlled patients.[9] Recently, lower mortality and stroke rates have been described with early rhythm control in patients with AF of <12 months as long as the rhythm control strategy includes AF ablation.[10] For the acute management of AF in pregnancy, we recommend an initial rate control strategy unless the patient is hemodynamically unstable. Figure 5 depicts AF.

Atrial flutter with variable AV block also presents with a narrow QRS irregular rhythm. The diagnosis is suspected by the presence of sawtooth flutter atrial waves. Importantly, the management of atrial flutter and AF is identical as patients commonly fluctuate between periods of AF and flutter[6] (Fig. 6).

The Wolf Parkinson White (WPW) syndrome occurs in people with an accessory pathway capable of electrical conduction between atria and ventricles. In most cases, the electrical impulse originating in the sinus node will travel from the atria to the ventricles through the AV node. However, electricity will travel again to the atria through the accessory pathway resulting in a new atrial depolarization (orthodromic

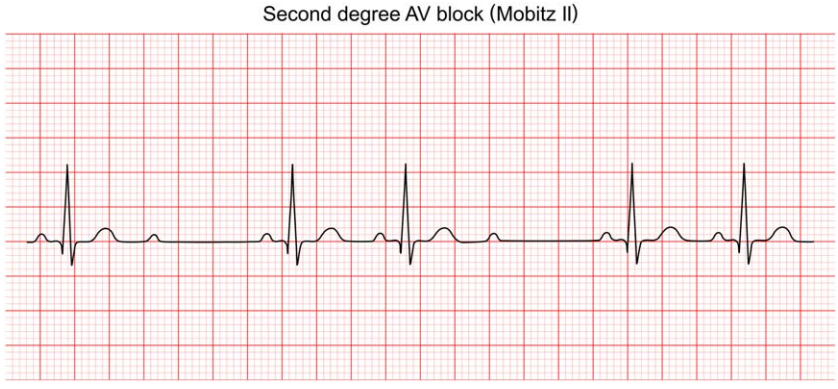

FIGURE 13. Second-degree AV block Mobitz II. Note some P-waves are conducted with a constant PR interval whereas other P-waves are not conducted at all. full color online

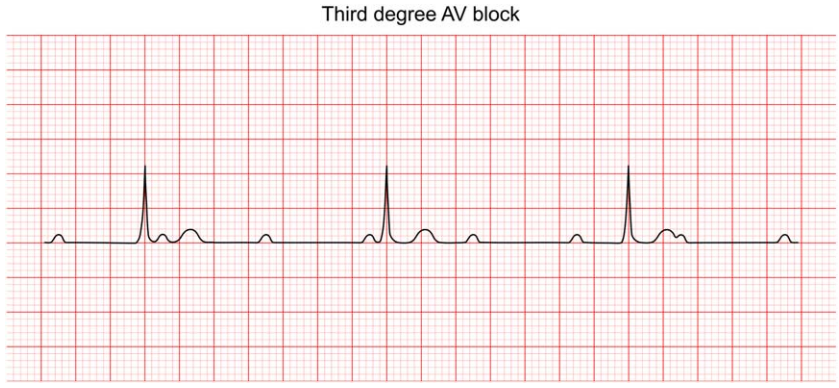

FIGURE 14. Complete heart block. Note complete dissociation between P-waves and QRS complexes. The interval between P-waves is constant as is the interval between each QRS complex (R-R interval). full color online

conduction) that will be conducted again to the ventricle through the AV node leading to a narrow complex regular tachycardia. Classic EKG findings include a short PR interval (P-wave is very close to the QRS) and a delta wave in the QRS (slurred upstroke in the QRS). Treatment of WPW with a narrow complex regular tachycardia is like other supraventricular tachycardias aiming at slowing the AV node conduction with vagal maneuvers, beta-blockers, adenosine, or nondihydropyridine calcium channel blockers.[11] If hemodynamically unstable, synchronized electrical cardioversion is indicated. Less commonly, the accessory pathway may be capable of conducting directly from the atria to the ventricles resulting in a wide QRS complex tachycardia (antidromic conduction).[11] These cases should be treated with intravenous procainamide.[11] Importantly, patients with WPW who develop AF should not receive AV node slowing medications (adenosine, beta-blockers, calcium channel blockers, digoxin, and amiodarone) as slowing the AV node could increase conduction down the accessory pathway leading to potentially lethal ventricular arrythmias. If unstable, synchronized electrical cardioversion is indicated. If stable, medications that slow both the AV node and the accessory pathway, such as procainamide, are preferred.[11] Figure 7 shows a classic WPW EKG tracing.

Ventricular Tachyarrhythmias

Ventricular tachycardia (VT) is diagnosed based on the presence of tachycardia with a wide QRS complex and it may further be subdivided into monomorphic and polymorphic. Monomorphic VT is treated with immediate synchronized electrical cardioversion (100 J) if the patient is hemodynamically unstable.[6] In hemodynamically stable patients, pharmacological treatment includes lidocaine, amiodarone, procainamide, or sotalol.[6] In patients with a history of a right or left bundle block (both of which will have a baseline prolonged QRS), a supraventricular tachycardia will appear as a regular wide complex tachycardia and should be treated following usual management of such arrythmias. However, when in doubt about the origin of a wide complex tachycardia, it is recommended to treat as VT until proven otherwise. Importantly, VT may be associated with myocardial ischemia and structural anomalies; both should be investigated as part of the initial management.[12] Figure 8 depicts monomorphic VT.

TABLE 1. Common First Line Antiarrhythmic Agents Used During Pregnancy

Medication	Dose	Comments
Metoprolol (beta 1 selective blocker)	2.5-5 mg intravenously every 5 min, maximum 15 mg	Caution in severe reactive airway disease. Safe in pregnancy[15]
Esmolol (beta 1 selective blocker)	Initial bolus 0.5 mg/kg, infusion 25-300 μg/kg/min	Short acting (half-life 3-8 min) allowing rapid titration and short-lived effect. Caution in severe reactive airway disease. May use if indicated during pregnancy
Diltiazem (nondihydropyridine calcium channel blocker)	Bolus 0.25 mg/kg followed by infusion of 5-15 mg/h	May decrease cardiac output and lead to hypotension. Limited data during pregnancy[15]
Digoxin (cardiac glycoside)	0.5 mg intravenously, may repeat 0.25 mg intravenously every 1-2 h to a maximum of 1.5 mg in 24 h	Avoid in severe renal dysfunction. A good option in patients with low ejection fraction. Safe in pregnancy.[15] Correct concomitant hypokalemia
Amiodarone (type 3 antiarrhythmic agent, potassium channel blocker)	150 mg intravenous bolus given over 10 min followed by a continuous infusion at 1 mg/min for 6 h and then 0.5 mg/min for 18 h	Effective in both supraventricular and ventricular tachyarrhythmias. Minimal effect on cardiac output. Effective for chemical cardioversion in atrial fibrillation. *Not the first line in pregnancy due to concerns of fetal toxicity*; however, may use in cases where it is the only optimal alternative to achieve arrythmia control[15]
Lidocaine (class 1 antiarrhythmic)	1-1.5 mg/kg bolus may repeat 0.5-0.75 mg/kg as needed (to maximum of 3 mg/kg). Maintenance infusion: 1-4 mg/min	Alternative to amiodarone in monomorphic ventricular tachycardia. Safe in pregnancy[15]
Atropine (antimuscarinic)	0.5-1 mg intravenously. May repeat as needed every 3-5 min. Maximum 3 mg	Avoid in advanced heart blocks (Mobitz 2- and third-degree blocks). Safe in pregnancy
Procainamide (class 1 antiarrhythmic)	100 mg intravenously every 5-10 min until arrhythmia is controlled, QRS widens by more than 50%, hypotension, or to a maximum total dose of 1 g. Maintenance infusion: 1-6 mg/min	The first line in atrial fibrillation with Wolf Parkinson White. Alternative in monomorphic ventricular tachycardia. Safe in pregnancy[16]
Sotalol (class 3 antiarrhythmic)	80 mg orally every 12 h	Alternative for monomorphic ventricular tachycardia. Safe in pregnancy[17]
Epinephrine (alpha, beta agonist)	2-10 μg/min	Use in bradycardia nonresponsive to atropine. May use in pregnancy indicated
Dopamine (alpha, beta, dopamine receptor agonist)	2.5-20 μg/kg/min	Use in bradycardia nonresponsive to atropine. May use in pregnancy if indicated[15]

Polymorphic ventricular tachycardia is less common than monomorphic VT. Polymorphic VT has an irregular R-R interval and variable QRS morphology. It is commonly associated with myocardial ischemia/infarction. If polymorphic VT presents in the context of hemodynamic instability, unsynchronized high-energy cardioversion is indicated (defibrillation). In these cases, synchronized cardioversion is not possible as the monitor will be unable to identify a constant R wave due to the chaotic rhythm. Torsades de Pointes is a form of polymorphic VT frequently associated with electrolyte disturbances and QT prolongation. Medical management of Torsades includes administration of magnesium sulfate (2 gm over 5 min), discontinuation of any medications that prolong the QT interval, and correction of any electrolyte anomaly including hypokalemia, hypocalcemia, and hypomagnesemia.[6,13] Examples are depicted in Figures 9 and 10.

BRADYARRHYTHMIAS

Although many bradycardias may be asymptomatic and require no intervention, others can be life threatening requiring rapid pharmacological and electrical interventions (eg, temporary ventricular pacing).

The most common form of bradycardia is sinus bradycardia. Heart rate is lower than 60 bpm and every QRS is preceded by a P-wave with a normal PR interval (<0.2 s). If symptomatic (hypotension, angina, pulmonary edema, and confusion), it should be treated with intravenous atropine.

First-degree heart block is characterized by prolongation of the PR interval (>0.2 s). This finding is commonly asymptomatic and rarely requires treatment. If symptomatic, treatment with atropine is indicated (Figure 11).

Second-degree block is divided into 2 subcategories: Mobitz type I and type II. In Mobitz type I, there is a gradual

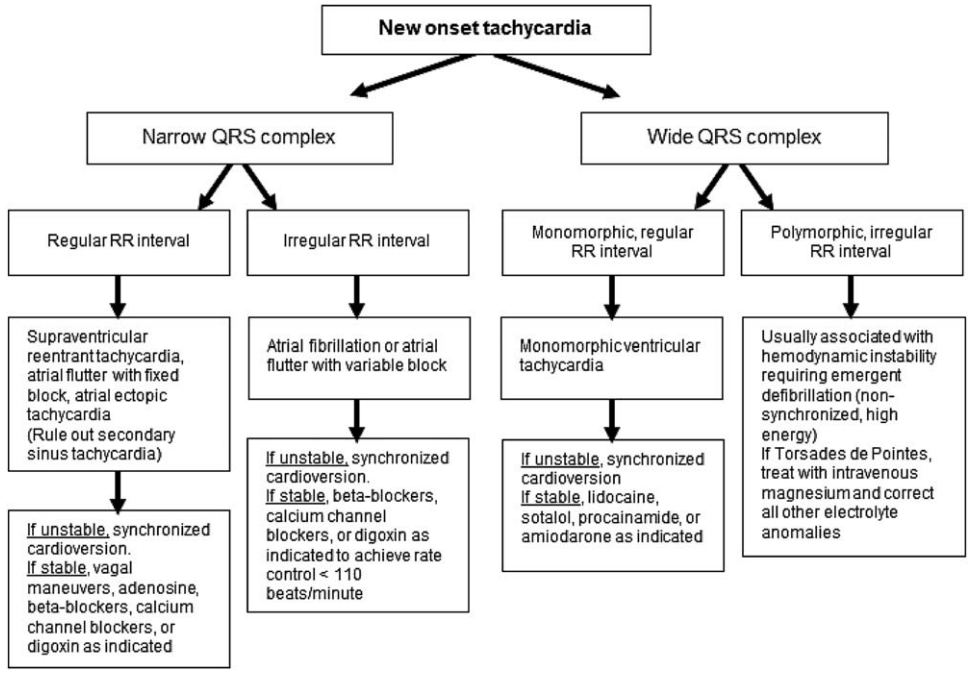

FIGURE 15. Initial management of a pregnant patient with new-onset tachyarrhythmia.

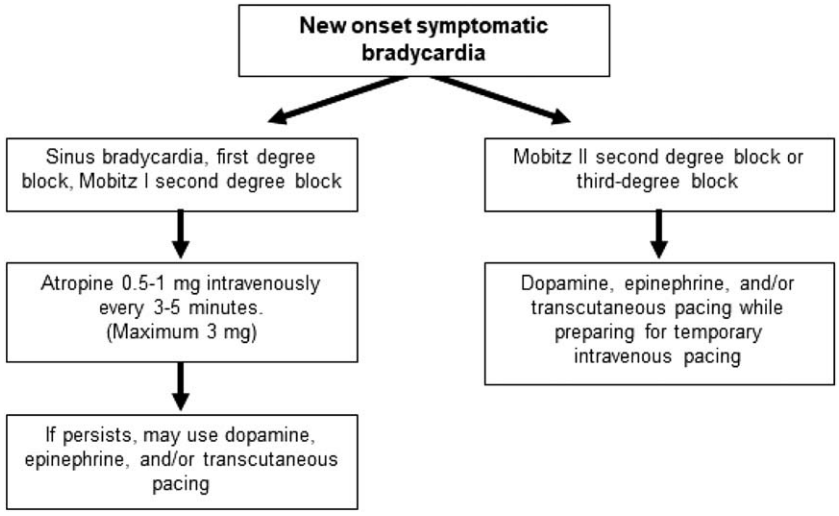

FIGURE 16. Initial management of a pregnant patient with new-onset bradyarrhythmia.

prolongation of the PR interval until there will be an unconducted atrial beat (P-wave).[14] Every conducted impulse will have a narrow QRS complex. Mobitz type I, also known as Wenckebach, is usually asymptomatic and does not require treatment unless symptomatic (Fig. 12). In Mobitz type II, some P-waves are conducted and followed by a narrow complex QRS whereas some P-waves are not conducted. This block may progress to a complete heart block and requires close follow up; if symptomatic it should be treated with catecholamines to increase chronotropism (epinephrine or dopamine) and/or temporary pacing. Atropine is usually not recommended for advanced heart blocks.[14] Figure 13 depicts a Mobitz type 2 block.

Lastly, in third-degree AV block (complete heart block), there is a complete dissociation between the electrical activity of the atria and the ventricles (there is no correlation between P-wave and QRS complexes). New-onset complete heart block is a medical emergency and usually requires chronotropic catecholamines like dopamine and epinephrine and/or transcutaneous pacing while preparation for temporary intravenous pacing is undergoing.[14] As mentioned earlier, atropine is not effective in advanced heart blocks. Figure 14 depicts a complete heart block.

Table 1 summarizes the different antiarrhythmic agents discussed in this focused article. Figures 15 and 16 provide a practical algorithm for the acute management of pregnant women with new-onset arrythmias.

Conclusion

Heart disease is the most common cause of maternal mortality in the United States. Obstetricians and maternal-fetal medicine specialists should prepare to play a fundamental role in the care of these patients and contribute to their care actively as part of a multidisciplinary team. Initial management of tachy and bradyarrhythmias in pregnancy should follow the same principles as in nonpregnant individuals including early cardioversion for unstable patients. Although some antiarrhythmic agents may pose some teratogenic potential, if indicated, they should not be withheld in cases where they are the optimal choice for maternal care.

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