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Treatment of Cardiac Arrhythmias During Pregnancy

Safety Considerations

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Contents

Abstract

Maternal and fetal arrhythmias occurring during pregnancy may jeopardise the life of the mother and the fetus. When arrhythmias are well tolerated and patients are minimally symptomatic, conservative therapy, such as observation and rest or vagal manoeuvres, should be employed. When arrhythmias cause debilitating symptoms or haemodynamic compromise, antiarrhythmic drug therapy is indicated. Although no antiarrhythmic drug is completely safe during pregnancy, most are well tolerated and can be given with relatively low risk.

Physiological changes that occur during pregnancy mandate caution when administering antiarrhythmic drugs, with close monitoring of serum concentration and patient response. Drug therapy should be avoided during the first trimester of pregnancy if possible, and drugs with the longest record of safety should be used as first-line therapy.

Several therapeutic options exist for most arrhythmias in the mother and fetus. Of the class IA agents, quinidine has the longest record of safety during pregnancy, and is generally well tolerated. Procainamide is also well tolerated, and should be a first line option for acute treatment of undiagnosed wide complex tachycardia. All IA agents should be administered in the hospital under cardiac monitoring due to the potential risk of ventricular arrhythmias (torsade de pointes).

The IB agent, lidocaine (lignocaine), has local anaesthetic role but is also generally well tolerated as an antiarrhythmic agents. Phenytoin should be avoided

due to the high risk of congenital malformations and limited role as an antiarrhythmic drug. Of the IC agents, flecainide has been shown to be very effective in treating fetal supraventricular tachycardia complicated by hydrops. β-Blockers are generally well tolerated and can be used with relative safety in pregnancy, although recent data suggest that they may cause intrauterine growth retardation if they are administered during the first trimester.

Amiodarone, a class II agents with characteristics of the other antiarrhythmic drug classes, has been reported to cause congenital abnormalities; it should be avoided during the first trimester and used only to treat life-threatening arrhythmias that fail to respond to other therapies. Adenosine is generally safe to use in pregnancy, and is the drug of choice for acute termination of maternal supraventricular tachycardia. Digoxin has a long track record of treating both maternal and fetal arrhythmias, and is one of the safest antiarrhythmics to use during pregnancy.

Direct current cardioversion to terminate maternal arrhythmias is well tolerated and effective, and should not be delayed if indicated. The use of an implantable cardioverter-defibrillator should be considered for women of childbearing potential with life-threatening ventricular arrhythmias.

When arrhythmias strike during pregnancy, they jeopardise the health of both the mother and the fetus by causing haemodynamic instability, decreased uterine blood flow and even fetal hydrops.[1] Any treatment is complicated by the potential for the therapeutic intervention to aggravate the risk to the mother and fetus/neonate. The problem is made even more complex by the maternal changes in cardiovascular physiology that occur during pregnancy, which affect antiarrhythmic drug dosage requirements.

An increased incidence of maternal cardiac arrhythmias is observed during pregnancy.[2,3] This is explained in part by the metabolic, hormonal and haemodynamic changes that occur during pregnancy which result in increased myocardial irritability, altered myocardial refractoriness, increased cardiac excitability, and exacerbation of pre-existing organic heart disease.[1,2]

In patients with structurally normal hearts and asymptomatic or minimally symptomatic arrhythmias (such as premature ventricular or atrial beats), no therapy other than reassurance is necessary. Antiarrhythmic drug therapy should be reserved for maternal or fetal arrhythmias that result in debilitating symptoms or are life threatening to the mother or fetus. When drug therapy is necessary during pregnancy, it should be administered without hesitation and with understanding of which drugs are appropriate.

1. Physiological Changes During Pregnancy

The normal physiological changes that occur in the maternal-fetal circulatory system during pregnancy can affect the dosage requirements of antiarrhythmic agents.[1,4] Increases in intravascular volume (by 40 to 50%) can raise the loading dose necessary to achieve therapeutic drug concentrations. Reduction in total protein level, which occurs as a result of increased intravascular volume, can lead to reduced drug protein binding and lower total drug concentrations, despite adequate free drug concentrations. Drug absorption in the gastrointestinal tract might be altered by changes in gastric secretions and gut motility, leading to changes in drug bioavailability. Increases in cardiac output by 30 to 50% cause increased renal flow and may cause increased clearance of renally excreted drugs. Finally, progesterone-induced hepatic stimulation may increase the hepatic clearance of some drugs.

As a result of these factors, more frequent measurement of drug concentrations and adjustment in

antiarrhythmic drug dosages are required during pregnancy. It is also important to closely monitor the clinical and electrophysiological response of the patient to the particular agent, rather than relying solely on serum drug concentration measurements, since these might be less accurate because of altered protein binding.

2. Effects of Drugs on the Fetus and Newborn

A major concern when administering drugs during pregnancy is the potential risk to the fetus. Congenital malformations may occur as a result of exposure of the fetus to teratogenic drugs during the first trimester; these abnormalities also depend on the nature of the drug, duration of the exposure, and genetic predisposition.[5] After the first trimester, organogenesis is essentially complete and the risk to the fetus is substantially reduced. The main concern with drug therapy during the second and third trimesters is interference with the fetal growth and development.[5] Another source of concern is the possibility of proarrhythmic effects on the fetal heart, which may occur when the drug is administered to treat fetal or maternal arrhythmias.

Table I. Antiarrhythmic drugs in pregnancy

Drug	Vaughan Williams class	FDA class[a]	Placental transfer	Adverse effects	Teratogenic	Transfer to breast milk	Risk of causing injury to the fetus
Quinidine	IA	C	Yes	Thrombocytopenia, rarely oxytocic	No	Yes[b]	Minor
Procainamide	IA	C	Yes	None	No	Yes[b]	Minor
Disopyramide	IA	C	Yes	Uterine contraction	No	Yes[b]	Minor[c]
Lidocaine (lignocaine)	IB	B	Yes	Bradycardia, CNS adverse effects	No	Yes[b]	Minor
Mexiletine	IB	C	Yes	Bradycardia; low birthweight, low APGAR, low blood sugar	No	Yes[b]	Minor[c]
Tocainide	IB	C	Unknown	Unknown	Unknown	Unknown	Minor[c]
Phenytoin	IB	D	Yes	Mental and growth retardation, fetal hydantoin syndrome	Yes	Yes[b]	Significant
Flecainide	IC	C	Yes	None	No	Yes[b]	Minor[c]
Propafenone	IC	C	Yes	None[c]	No	Unknown	Minor[c]
Moricizine	IB	B	Unknown	Unknown	No	Yes	Minor[c]
Propranolol	II	C	Yes	Growth retardation, bradycardia, apnoea hypoglycaemia	No	Yes[b]	Minor
Atenolol	II	C	Yes	Low birthweight	No	Yes	Minor
Sotalol	III	B	Yes	β-Blocker effects, torsade de pointes	No	Yes[b]	Minor[c]
Amiodarone	III	D	Yes	Hypothyroidism, growth retardation, premature birth, large fontanelle	Yes	Yes	Significant
Bretylium	III	C	Unknown	Unknown	Unknown	Unknown	Unknown
Ibutilide	III	C	Unknown	Unknown	Unknown	Unknown	Unknown
Verapamil	IV	C	Yes	Bradycardia, heart block, hypotension	No	Yes[b]	Moderate
Diltiazem	IV	C	No	Unknown	Unknown	Yes[b]	Moderate[c]
Digoxin	NA	C	Yes	Low birthweight	No	Yes[b]	Minor
Adenosine	NA	C	No[c]	None	No	Unknown	Minor[c]

a US Food and Drug Administration (FDA) class (see table II).[8]

b American Academy of Pediatrics considers the drug to be 'usually compatible with breast feeding'.[7]

c Very limited experience.

Potential exposure of the neonate to drugs excreted into the milk is another important consideration. Fortunately, excretion of antiarrhythmic drugs into human milk is minimal in most cases (1 to 2% of the maternal dose), so most agents are can be taken by nursing mothers (table I).[6,7]

3. General Considerations in Selecting Therapy

Experience with the use of antiarrhythmic agents during pregnancy varies among the different classes of drugs. In general, the medications that have been available for longer periods of time have the most clinical data regarding safety. Most antiarrhythmic drugs appear to be relatively safe to use in pregnancy, although there are important exceptions. The majority of antiarrythmic drugs are classified as US Food and Drug Administration (FDA) category C (table II), which means that there are either animal studies suggesting risks but no confirmatory human studies, or no controlled studies in both humans or animals.[8]

Table II. Definitions of US Food and Drug Administration (FDA) classifications (use in pregnancy ratings)

FDA classification	Definition
Category A	Controlled studies show no risk. Adequate, well-controlled studies in pregnant women have failed to demonstrate risk to the fetus
Category B	No evidence of risk in humans. Either animal studies show risk, but human studies do not; or, if no adequate human studies have been done, animal findings are negative
Category C	Risk cannot be ruled out. Human studies are lacking, and animal studies are either positive for fetal risk, or are lacking as well. However, potential benefits may justify the potential risk
Category D	Positive evidence of risk. Investigational or post-marketing data show risk to the fetus. Nevertheless, potential benefits of the drug may be acceptable when they outweigh the potential risk
Category X	Contraindicated in pregnancy. Studies in animals or humans, or investigational or post-marketing reports have shown fetal risk which clearly outweighs any possible benefits to the patients

All antiarrhythmic medications have potential adverse effects in both the mother and the fetus, so the smallest recommended dose should be used initially, accompanied by regular monitoring of serum drug concentrations and clinical response. The continued need for the medication should also be reassessed on a regular basis.

The most worrisome adverse effect for the mother is proarrhythmia, which can occur with almost all class I and III antiarrhythmic agents; these medications should be initiated during continuous cardiac monitoring.

In contrast to the treatment of maternal arrhythmias, in some situations antiarrhythmic medications are administered to the mother in order to treat fetal arrhythmias (i.e. transplacental therapy). When intractable fetal arrhythmias are seen, the mother is simply the conduit for transplacental administration of the drug. Obviously this situation calls for dosage adjustments such that fetal serum concentration reaches a therapeutic concentration, all the while guarding against maternal drug toxicity or proarrhythmia. Although this review primarily concentrates on treatment of the maternal arrhythmia, much of the information provided here applies to the situation of fetal arrhythmias as well.

It is important to emphasise that, if it is determined that a patient would benefit from a specific drug and no other drug would be as beneficial, the drug should be given to the patient regardless of the fact that she is pregnant and close monitoring should be provided. In other words, no woman should receive suboptimal medical care solely because she is pregnant.

4. Safety Profile of Specific Antiarrhythmic Agents

4.1 Class I Drugs: Sodium Channel Blocking Agents

In the Vaughan Williams classification of antiarrhythmic drugs, the class I agents are subdivided into class IA (drugs that prolong the action potential duration), IB (drugs that cause shortening of

the action potential duration), and IC (drugs that cause profound slowing of conduction).[9]

4.1.1 Class IA

Of the IA agents, quinidine has the longest record of safety during pregnancy and there exists over 60 years of experience with this agent in the management of a variety of maternal and fetal arrhythmias.[10-12] Although isolated cases of adverse effects have been reported, such as fetal thrombocytopenia and eighth nerve toxicity, the drug is considered to be relatively well tolerated,[4,5] since only rarely have significant adverse effects been reported when the drug was administered in therapeutic doses. The drug should be avoided during lactation because of potential accumulation of the drug in the immature liver of the neonate.[11]

Procainamide, like quinidine, has proven to be well tolerated and useful in the management of maternal and fetal arrhythmias and has the advantage of intravenous administration.[4,13] It is an appropriate choice for the short term treatment of undiagnosed wide complex tachycardia,[14] and is one of the best therapeutic options for the treatment of arrhythmias in pregnancy. The adverse effect profile of procainamide, when the drug is administered over a limited period of time (such as during gestation), has proven to be quite favourable based on general experience. Dose adjustments during the different stages of pregnancy may be necessary, and some caution is warranted due to the possibility of a lupus-like syndrome that can occur with long term therapy.[5]

Disopyramide is also a category IA agent and has similar antiarrhythmic properties to quinidine and procainamide. However, lack of experience with this agent during pregnancy makes therapy with disopyramide less attractive than the other class IA drugs.

Care must be taken when administering class IA drugs, due to the risk of potentially fatal ventricular arrhythmias (torsade de pointes) that are associated with prolongation of the QT interval on ECG. All class IA medications must be administered in hospital during continuous cardiac monitoring and serum drug concentrations should be adequately monitored. Quinidine, procainamide, and disopyramide are considered FDA category C agents (table II).

4.1.2 Class IB

Class IB agents include lidocaine (lignocaine), mexiletine, tocainide, moricizine and phenytoin. Lidocaine has been widely used during pregnancy mainly as an anaesthetic agent during labour and delivery. Experience with lidocaine as an antiarrhythmic agent during pregnancy is more limited, but animal and human registry data has shown lidocaine to be well tolerated by both the mother and the fetus,[4,15,16] although occasional cases of toxic effects of lidocaine on the fetus have been reported. The drug is best avoided with prolonged labour or fetal distress; when lidocaine is administered in the presence of fetal acidosis, fetal cardiac and CNS toxicity could occur due to higher free drug concentrations.[17] Since the metabolism of lidocaine occurs in the liver, mothers with impaired liver function or heart failure should receive reduced loading and maintenance dosages. Lidocaine is considered to be an FDA category B agent (table II).

Mexiletine is an oral antiarrhythmic agent with structure similar to lidocaine. The experience with mexiletine during pregnancy is much more limited, but it also appears to be well tolerated, especially during breast feeding since the amount of drug that is excreted in breast milk is very small.[18-20] Despite the reports of safe administration, caution is still advised due to the very limited amount of data available.

Phenytoin is an anticonvulsant agent that has been used in the past primarily for treating arrhythmias resulting from digitalis toxicity.[21] In recent years, it has become of limited value as an antiarrhythmic agent due to the availability of other therapeutic alternatives (including β-blockers, digoxin-specific antibodies and lidocaine), and the routine practice of monitoring serum digoxin concentrations. This drug should be avoided during pregnancy (it is in FDA category D) due to the high risk of congenital malformations and adverse ef-

fects to the fetus, including the 'fetal hydantoin syndrome', which may occur with drug exposure during the first trimester.[22] Nevertheless, it may be safe when used for short periods of time, especially after the first trimester, and could still be an option for short term management of digitalis-induced arrhythmias that do not respond other therapies.[5]

Experience during pregnancy with the agents, tocainide and moricizine, is very limited. Since they offer no unique advantage, it is best to avoid these drugs in favour of agents with better established utility and safety in pregnancy.

4.1.3 Class IC

The class IC agents include propafenone and flecainide, both of which are useful in the management of ventricular and supraventricular tachycardias and appear to be relatively well tolerated during pregnancy.

Flecainide is contraindicated in patients with previous myocardial infarctions in light of the results of the Cardiac Arrhythmias Suppression Trial (CAST), which showed an increased mortality in post-infarct patients who were treated for suppression of high grade ventricular ectopy.[23] Otherwise, it appears to be relatively well tolerated and effective in the treatment of maternal arrhythmias.[14] The question of whether it is safe to use flecainide during the early stages of fetal development remains unanswered.

For the management of fetal arrhythmias, maternally administered flecainide has shown to be very effective. Allan et al.[24] reported successful termination of supraventricular arrhythmias in 12 of 14 hydropic fetuses within 48 hours of therapy initiation. A recent retrospective analysis showed improved survival when fetuses were treated with flecainide, as compared with digoxin.[25] No adverse effects from flecainide were observed in this study. Sporadic cases of adverse effects to the fetus have been reported, but generally patients did well when flecainide was administered for fetal arrhythmias.[26] The drug is excreted in human breast milk, but there are scant data regarding pharmacokinetics of flecainide in nursing infants. In light of the previously mentioned studies, flecainide should be considered as a first-line option in the management of fetal supraventricular tachycardia complicated by fetal hydrops.

Experience with propafenone is more limited, although no adverse effects to the mother or the fetus have been reported when the drug is administered during the third trimester.[27] The concerns regarding patients with prior myocardial infarction may be similar to those with flecainide, since these agents share similar cellular properties. The are no reports regarding administration of propafenone during the first trimester. Both flecainide and propafenone are considered to be FDA category C drugs (table II).

4.2 Class II Drugs: β-Adrenergic Blocking Agents

The β-adrenergic blocking agents are useful in the management of a variety of maternal and fetal supraventricular and ventricular arrhythmias, and for control of the ventricular response in atrial fibrillation and flutter. They have been used extensively during pregnancy for the management of a number of other clinical conditions, including hypertension, hyperthyroidism and hypertrophic cardiomyopathy.[4,28] The cardiac effects are mediated by β_1-receptors, while β_2-receptors act predominantly in bronchi, blood vessels, and are involved in uterine relaxation.[1]

While there have been reports of β-blockers having adverse effects on the fetus such as bradycardia, apnoea, hypoglycaemia, metabolic abnormalities and induction of premature labour, the incidence of these complications is low.[4] Prospective randomised trials failed to show statistically significant higher incidence of these complications with β-blockers as compared with placebo, raising the possibility that these reaction occurred as the result of fetal distress in high risk pregnancies.[14,28]

The main adverse fetal effect that has been reported with propranolol is intrauterine growth retardation,[29] although other studies have failed to show similar results. Placebo controlled studies of

atenolol and metoprolol have likewise demonstrated no evidence of growth retardation.[30,31] A more recent study reported growth retardation in babies receiving atenolol in the first trimester.[32] The authors attribute their findings to the administration of the β-blocker during the first trimester (and not the second or third trimesters, as with the other studies). Although it is difficult to discern between growth retardation due to maternal disease versus drug, in view of these results, β-blockers should be avoided during the first trimester, if possible. Cardioselective β_1-antagonists, such as metoprolol and atenolol, or agents with intrinsic sympathomimetic activity, are the preferred agents since they may interfere less with β_2-mediated peripheral vasodilatation and uterine relaxation.[14,29]

Almost all β-blockers have been shown to be secreted in breast milk but generally do not cause adverse effects to the nursing infant with normal hepatic and renal function;[33,34] however fetal bradycardia has been reported.[35] Thus, the use of β-blockers is not contraindicated in lactating women.[7]

4.3 Class III Drugs: Potassium Channel Blocking Agents

The class III agents primarily block potassium channels, causing delay of repolarisation and prolonging the QT interval. This class includes amiodarone, bretylium, sotalol, and ibutilide.

Amiodarone is a potent antiarrhythmic agent with properties similar to class I, II, III and IV agents. It is used for the management of ventricular and, less often, supraventricular arrhythmias. It contains a high concentration of iodide, which contributes to some of its adverse effects, and it has a long half-life of 26 to 107 days.

Multiple adverse effects to the fetus have been reported with the use of amiodarone, including fetal hypothyroidism, low birthweight, prematurity, bradycardia and QT prolongation.[36-38] Concomitant use of amiodarone with β-blockers should be avoided.[38] Congenital abnormalities have been reported in neonates who were exposed to amiodarone during the embryonic period.[38,39] Therefore, amiodarone should not be administered during the first trimester and it should be strongly avoided throughout gestation. The adverse effect profile of amiodarone has lead to this agent being reserved for life-threatening conditions where other agents have failed, in spite of the substantial efficacy of the drug. It is an FDA category D agent (table II).

Relatively high concentrations of amiodarone can be found in breast milk,[40,41] so it is best avoided during lactation.

Sotalol is a class III agent with noncardioselective β-blocker properties. The most worrisome adverse effect related to this agent is the occurrence of torsade de pointes.[42] Although experience with the use of sotalol during pregnancy is limited, it appears to be well tolerated,[43,44] and is an FDA category B agent (table II). No adverse events have been reported with nursing, but since a significant amount of drug is found in human milk, caution is advised during breast feeding.[4]

Bretylium is an intravenous class III agent used to treat ventricular tachycardia and fibrillation. The experience with bretylium during pregnancy is rather limited, so it can not be recommended for routine use except for cases of life-threatening arrhythmias in which other options have failed. It is an FDA category C agent (table II).

Ibutilide is a new class III intravenous agent that is used for acute termination of atrial fibrillation or flutter. The main adverse effect of this drug is the occasional (1.7%) induction of sustained torsade de pointes requiring direct current cardioversion.[45] There are no data for the use of ibutilide during pregnancy, so although it is labelled as an FDA category C agent (table II), it should be avoided during pregnancy.

4.4 Class IV Drugs: Calcium Channel Blocking Agents

This class includes verapamil and diltiazem, which exert their electrophysiological effect by blocking the calcium channel, thus slowing or blocking conduction in the atrioventricular node.[9] These agents are mainly used in the management

of supraventricular tachycardias, for control of the ventricular response in patients with atrial fibrillation or flutter, and in some cases for the treatment of idiopathic ventricular tachycardia. Although favourable results with verapamil in the treatment of supraventricular tachycardia in pregnant women have been reported,[46] adverse effects to the fetus such as bradycardia, heart block, depression of contractility, hypotension, and death have occurred when verapamil was used in the management of fetal supraventricular arrhythmias.[25,47] For these reasons these drugs should be avoided during pregnancy when safer choices are available. The experience with diltiazem is more limited, but the same concerns may be justified. Both drugs are considered to be FDA category C agents (table II) and are compatible with breast feeding.

4.5 Adenosine

Adenosine and digoxin (section 4.6) do not fit into the Vaughn Williams classification of antiarrhythmic drugs. Adenosine is an endogenous nucleoside with a short half-life (less than 2 seconds), that has proven to be very effective in acute termination of supraventricular tachycardia by causing transient conduction block in the atrioventricular node.[48] The drug has been used safely for the termination of supraventricular tachycardia in pregnant women, with no significant adverse effects to the mother or fetus.[48] The usual doses of 6mg intravenously, followed by 12 or even 18mg, are indicated, and safe administration of up to 24mg has been reported.[14] Minor maternal adverse effects, such as bradycardia and dyspnoea, are usually transient and of no consequence.

Although adenosine deaminase is reduced in pregnancy by about 25%,[49] the potency of the drug is not usually increased because of changes in intravascular volume,[50] and even higher than usual doses may be required.[51] Experimental data suggest that significant amounts of adenosine do not cross the placenta, probably due to the short half-life.[52] Because most of the reports of adenosine administration were in the second and third trimester, caution is advised when administering this drug in the first trimester. Nevertheless, it is the drug of choice for acute termination of supraventricular tachycardia during pregnancy.[53] Adenosine should not be administered to patients with asthma, since it can induce bronchospasm.[48] Adenosine is considered an FDA category C agent (table II).

4.6 Digoxin

Digoxin is a glycoside that exerts its antiarrhythmic action mainly through vagally-mediated atrioventricular conduction slowing and block. Digoxin freely crosses the placenta,[54,55] and for many years has been use for the treatment of a variety of maternal and fetal supraventricular arrhythmias.[56-58] Direct fetal intramuscular administration of digoxin for the management of fetal supraventricular tachycardia has been reported, with good results and no adverse effects.[59] There is extensive experience with digoxin and it is probably one of the safest antiarrhythmic drugs to use during pregnancy.[14,60]

As in nonpregnant patients, the digoxin dose needs to be adjusted for renal impairment or if there is concomitant administration of drugs that increase the digoxin concentration.[5] Serum drug concentrations need to be monitored, although in the third trimester the serum concentration measurement may be spuriously high, due to a circulating digoxin-like substance that interferes with the radioimmune assay for the drug.[61] Despite a history of safe use, digoxin is considered an FDA category C agent (table II). Digoxin is considered acceptable to use by lactating mothers.[7,55]

5. Nonpharmacological Therapy

The degree of aggressiveness in the acute treatment of cardiac arrhythmias should depend mainly on the haemodynamic effects of the arrhythmia on the mother and the fetus.[14] Conservative management, such as observation and rest, or vagal manoeuvres, such as carotid massage or valsalva, are appropriate in selected patients where the arrhythmia is well tolerated.

Direct current cardioversion has been used to acutely terminate both supraventricular and ventricular arrhythmias, with no significant adverse effects to the mother or fetus.[62-64] Fetal arrhythmias have been reported,[62] so fetal monitoring is advised; however, effects on the fetus are uncommon, perhaps related to the high mammalian fetal fibrillation threshold,[65] or the fact that the uterus is outside the shocking vector and receives relatively little electrical current.

Implantable cardioverter defibrillator therapy, which is a highly effective therapy for the management of life-threatening ventricular tachycardia and fibrillation, has rarely been employed in women of childbearing potential. In 1 report, no adverse effect to the mother or fetus occurred when the device discharged during pregnancy.[66] The implantable cardioverter defibrillator represents an option for women with life-threatening arrhythmias who plan to conceive in the future, since exposure to potentially toxic drugs can be obviated.

If a pregnant woman experiences a cardiac arrest, and cardiopulmonary resuscitation is necessary, it should be performed as in the nonpregnant state; however, modification is necessary to take account of the fact that the gravid uterus may cause compression of the aorta and inferior vena cava. It is recommended that cardiopulmonary resuscitation be performed with the patient in a lateral decubitus position or with the uterus displaced manually cephalad and to the left.[67-70] If the fetus is older than 25 weeks, Caesarean section should be considered and undertaken promptly.[67]

6. Conclusions

A variety of maternal and fetal arrhythmias can occur during pregnancy, for which antiarrhythmic drug therapy may be indicated. Although no drug is completely safe, most are well tolerated and can be given with relatively low risk. Drug therapy should be avoided during the first trimester of pregnancy if possible and drugs with the longest record of safety should be used as first line therapy. Conservative therapies should be used when appropriate. Several therapeutic options exist for most arrhythmias in the mother and fetus. The use of an implantable cardioverter defibrillator is an option for women of childbearing potential with life-threatening ventricular arrhythmias.

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