Maternal cardiac arrhythmias during pregnancy and lactation

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Summary: Arrhythmias occurring during pregnancy can cause significant symptoms and even death in mother and fetus. The management of these arrhythmias is complicated by the need to avoid harm to the fetus and neonate. It is useful to classify patients with arrhythmias into those with and without structural heart disease. Those with a primary electrical problem, but an otherwise normal heart, often tolerate rapid heart rates without compromise whereas patients with problems such as rheumatic heart disease, congenital heart disease or cardiomyopathy may quickly decompensate during an arrhythmia.

Keywords: high-risk pregnancy, arrhythmia, cardiology, drugs (medical)

CARDIOVASCULAR PHYSIOLOGY AND ARRHYTHMOGENESIS DURING PREGNANCY AND LABOUR

Blood volume increases by an average of 50% during pregnancy. Blood pressure usually falls, reaching a nadir in mid-pregnancy. The systolic pressure usually returns to near pre-gestational levels by term.¹ Cardiac output rises by about 50% with a steep increase from the latter half of the first trimester. This is largely due to an expanding stroke volume that plateaus at around 24 weeks gestation. The increase in blood volume can cause atrial stretch that may be significant for arrhythmogenesis. Later in pregnancy a rise in heart rate, on average 20 beats per minute, accounts for further enhancement in cardiac output.² The rise in heart rate can be marked, especially in the multigravida pregnancy. The normal adjustment in sex hormones that occurs during pregnancy may exert a proarrhythmic effect on myocardial tissue.³ The increased sympathetic outflow that occurs in pregnancy probably also contributes to the proarrhythmic milieu.⁴

During labour, cardiac output rises substantially due to both a rise in heart rate and stroke volume. Anxiety, pain and uterine contractions all contribute. Immediately after delivery, relief of caval compression as well as autotransfusion from the uterus can cause a significant increase in blood volume despite the fluid losses in labour. Adequate analgesia can substantially attenuate the haemodynamic changes.¹

NORMAL ELECTROCARDIOGRAM DURING PREGNANCY

While the resting electrocardiogram (ECG) is usually unaltered in pregnancy apart from a physiological increase in heart rate, changes may occur. The electrical axis can be displaced slightly

due to cardiac rotation resulting from the gravid uterus, but tends to remain within the normal range.^{5,6} The increase in heart rate can be associated with shortened PR, QRS and QT intervals. Other more obvious changes should be regarded as abnormal and investigated.

DOES THE INCIDENCE OF CARDIAC ARRHYTHMIAS INCREASE DURING PREGNANCY?

Palpitations are frequently reported during pregnancy; however these symptoms often do not correspond to episodes of arrhythmia on 24-hour ECG Holter monitoring.⁷ For many women, pregnancy is a time of heightened awareness and it is likely that increased heart rate and stroke volume contribute to the sensation of a 'pounding heart' that at times is perceived as palpitations.

Benign atrial and ventricular ectopic beats are more frequent during pregnancy.⁷ Although sometimes annoying, in general they are benign in structurally normal hearts and usually do not require treatment. Frequent ventricular ectopics can sometimes indicate underlying heart disease and further investigation may be warranted. One large retrospective series found that only 0.17% of hospital admissions during pregnancy were for cardiac arrhythmia and for every 100,000 pregnant women, 24 will be admitted for management of supraventricular tachycardia (SVT) (Figure 1), two with atrial fibrillation (AF), two with ventricular tachycardia (VT) (Figure 2) or fibrillation and 1.5 with atrioventricular (AV) block.⁸ Arrhythmias of haemodynamic significance during labour are surprisingly rare.^{9,10}

Australian statistics indicate that between 2003 and 2005, 36 pregnant or peripartum women died from causes not directly related to their pregnancy. Only one of those deaths was caused by cardiac arrhythmia. That equates to 1.3 deaths per million women who give birth.¹¹

The literature is contradictory as to whether new onset SVT is more frequent during pregnancy, however, exacerbations of a pre-existing arrhythmia are more common.^{12,13} It seems that

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Figure 1 Suggested management of SVT in pregnancy. SVT = supraventricular tachycardia; DC = direct current

in Wolff–Parkinson–White (WPW) syndrome, pregnancy increases the propensity for SVT even in the previously asymptomatic patient.^{14,15}

RECOMMENDATIONS FOR CLINICAL ASSESSMENT

Many women experience the sensation of palpitations during pregnancy. A thorough history and examination are essential. History of structural heart disease, exacerbating situations, exercise capacity, a description of arrhythmia onset and termination as well as any associated symptoms, especially syncope, should be sought. A family history of sudden death or structural heart disease is important. Tachyarrhythmias can be caused or

exacerbated by hyperthyroidism. Contributing metabolic disorders should be excluded.

A baseline ECG must be obtained and the patient should be considered for a 24-hour ECG Holter monitor and echocardiogram. Unless the symptoms are occurring daily, the Holter recording is often inconclusive. If symptoms are exacerbated by exercise an exercise stress test can be useful. Diagnostic electrophysiological and tilt-table testing are rarely indicated in pregnancy.

Pregnant women with documented sustained or frequent arrhythmia, recurrent palpitations or syncope should be assessed by a cardiologist.

TACHYARRHYTHMIAS

Paroxysmal supraventricular tachycardia

The three common types of SVT are as follows:

- (1) *AV nodal reentrant tachycardia*: This is the most common type and involves a reentrant circuit utilizing the AV node and perinodal atrial tissue (Figure 3). This arrhythmia can be treated with antiarrhythmic drugs or AV nodal blocking drugs (such as adenosine, beta-blockers, calcium channel blockers or digoxin);
- (2) *Tachycardias using overt or concealed accessory pathways including WPW syndrome*: In most cases the reentrant impulse travels from the atria via the normal conduction system to the ventricles and then returns to the atria via an accessory pathway. Rarely the impulse travels in the reverse direction. This arrhythmia can be treated with antiarrhythmic drugs or AV nodal blocking drugs. In WPW syndrome the accessory pathway can conduct anterogradely and retrogradely. The abnormal accessory pathway may conduct at dangerously fast rates occasionally inducing ventricular fibrillation and sudden death (see below);
- (3) *Atrial tachycardia*: This tachycardia is caused by reentry or an automatic focus entirely within the atria. The AV node is not part of the reentrant circuit so drugs that delay AV conduction may slow the ventricular rate but do not terminate the tachycardia. Termination of the arrhythmia requires the use of an antiarrhythmic drug or electrical cardioversion.^{16,17}

Treatment of the acute episode of SVT

It is uncommon for brief episodes of supraventricular arrhythmia to cause significant compromise to mother or fetus in the absence of structural cardiac disease. Vagal manoeuvres should be tried initially. If vagal manoeuvres are ineffective, adenosine is the agent of choice. Given in sufficient dosage (6–24 mg), adenosine will always terminate SVT with the exception of atrial tachycardia (see above). Adenosine will not terminate most atrial tachycardias but will temporarily increase the degree of AV block. If the tachycardia resumes soon after termination, a longer acting agent such as intravenous verapamil or beta-blocker may be used with caution to avoid hypotension. If the patient is haemodynamically unstable, she should be electrically cardioverted. Persistent atrial tachycardia should be electrically cardioverted. Fetal monitoring is essential during cardioversion or in the setting of maternal haemodynamic instability and is also advisable during adenosine administration (discussed below).

Figure 2 Suggested management of VT in pregnancy. AA = antiarrhythmic; VT = ventricular tachycardia; DC = direct current

Figure 3 AV nodal reentrant tachycardia. A 24-year-old woman presented with frequent palpitations and sustained SVT during the 32nd week of pregnancy. The acute attack was terminated with intravenous adenosine (18 mg). Symptoms improved but were not eliminated with metoprolol. She underwent successful radiofrequency ablation 8 weeks postpartum

Prevention of recurrent SVT

Recurrent, brief episodes of SVT can be managed with simple reassurance. More troublesome SVT is managed with medication. AV nodal-blocking drugs—beta-blockers, verapamil or digoxin—are reasonable choices but beta-blockers have the best safety record (with the exception of atenolol as discussed below); however, these drugs will not prevent atrial tachycardia (see above). If the patient has atrial tachycardia or SVT resistant to AV nodal-blocking drugs, flecainide can be considered. If a woman is poorly responsive to medical therapy or thought to be at a high risk of malignant arrhythmia associated with WPW syndrome, radiofrequency ablation should be considered during pregnancy.

Atrial flutter and fibrillation

AF and flutter (AFL) are relatively rare in pregnancy in the absence of structural heart disease.^{8,18} These arrhythmias can be problematic for two main reasons. Firstly, the increased heart rate and loss of effective atrial contraction may cause cardiac failure. The second important consideration is the increased risk of thromboembolism. The loss of effective atrial contraction and the physiological prothrombotic state of pregnancy combine to increase the risk of thrombus formation in the left atrial appendage.

If required, beta-blockers, digoxin or verapamil are preferred for heart rate control during AF or AFL. In the event of new onset AF/AFL or haemodynamic instability, cardioversion (either electrical or pharmacological) should be undertaken to restore normal rhythm. Sotalol is probably the antiarrhythmic of choice but amiodarone and flecainide are second-line options. In the setting of underlying cardiac disease the issue of anticoagulation can be complex and needs to be decided on an individual basis. With respect to the choice of anticoagulant, in brief, warfarin can be used in the second and early third trimester and heparin (unfractionated or low molecular weight) is recommended at other times.¹⁹ If the heart is structurally

normal and there is no history of thromboembolic events or thrombophilia, anticoagulation is not usually indicated.

Ventricular tachycardia

The majority of women who present with new onset VT in pregnancy have a structurally normal heart (idiopathic VT). The most common type of idiopathic VT arises from the right ventricular outflow tract and has a distinctive ECG appearance (left bundle branch block morphology with inferior axis; see Figure 4). It is often precipitated by sympathetic stimulation and may respond to beta-blockade.²⁰ Data from non-pregnant patients with idiopathic VT suggest that the long-term prognosis is excellent;^{21,22} however, prolonged episodes could potentially result in hypotension and fetal hypoxia so adequate treatment is essential. If beta-blockade is not successful, class IC or III drugs may be used or radiofrequency ablation may be necessary. The second most common type of idiopathic VT arises in the left ventricle and is termed fascicular VT or idiopathic left ventricular VT. This VT also has a distinctive ECG appearance (right bundle branch block morphology with superior axis; see Figure 5). This VT is often mistaken for SVT because the QRS duration during tachycardia may be relatively narrow (0.12–0.14 s). The tachycardia may respond to verapamil or beta-blockade.

Treatment of the acute episode of VT

Patients with haemodynamic compromise should be electrically cardioverted immediately. In uncompromised patients intravenous lignocaine or sotalol can be used.²³ There is little evidence that intravenous amiodarone is useful in terminating an attack of VT.

Prevention of the recurrent VT

VT associated with structural cardiac disease is potentially malignant. Most patients with VT in this setting should

Figure 4 Idiopathic right ventricular VT. A 31-year-old woman presented with new onset intermittent, frequent episodes of broad complex tachycardia during the 24th week of pregnancy. The tachycardia had the ECG appearance of VT arising in the right ventricular outflow tract. The arrhythmia settled with oral metoprolol. She remained haemodynamically stable throughout. She had only occasional brief episodes after the commencement of regular metoprolol. Successful radiofrequency ablation was undertaken 8 weeks post delivery

undergo defibrillator implantation. Antiarrhythmic drug therapy can be used in patients with idiopathic VT in the absence of structural heart disease. Beta-blockers are the first-line therapy. If beta-blockers are ineffective or not tolerated calcium channel blockers (for idiopathic left ventricular VT), sotalol or flecainide can be considered but the severity and frequency of episodes must be weighed against adverse drug effects. Idiopathic VT can be cured with catheter ablation with a very high success rate (>90%). The choice of pharmacologic therapy in the setting of a structurally abnormal heart is more complex as some antiarrhythmic medication may worsen the situation.

Long QT syndrome

Hereditary long QT syndrome (LQTS) is caused by an abnormality of genes coding for cardiac ion channels. Patients with this

condition show a prolonged corrected QT interval on the ECG during sinus rhythm. LQTS may cause torsades de pointes, a potentially fatal form of polymorphic VT. The mainstay of treatment is beta-blockade but patients with recurrent arrhythmia despite beta-blockade should be considered for defibrillator implantation. It is possible that the increased heart rate and subsequent shortening of the QT interval that occur during pregnancy may be protective as several series have demonstrated that dangerous cardiac arrhythmias and sudden death occur rarely in the LQTS group until after delivery. The postpartum period is a time of increased risk.^{24–26} Treatment with beta-blockers has been associated with decreased chance of postpartum arrhythmia, especially in those with the LQT2 genetic mutation, and hence should be continued throughout pregnancy and postpartum in women already taking that medication.^{25,26} Data have also suggested that babies from mothers with LQTS requiring urgent caesarean section for fetal distress are extremely likely to be carriers of the genetic condition.²⁷

Figure 5 Idiopathic left ventricular VT. A 27-year-old woman presented with frequent, distressing palpitations during the 28th week of pregnancy. She remained haemodynamically stable throughout. The acute attack was initially thought to be SVT but was unresponsive to adenosine. She was reviewed by a cardiologist who diagnosed idiopathic left ventricular VT. The arrhythmia was terminated with intravenous lignocaine. Further episodes were prevented with oral verapamil. She underwent successful radiofrequency ablation 12 weeks postpartum

LQTS has also been linked to sudden infant death syndrome;²⁸ therefore, routine screening of the progeny of mothers with this condition is imperative. Drugs that potentially prolong the QT interval should be avoided. During an episode of torsades de pointes intravenous magnesium and temporary overdrive pacing may stabilize the patient.

BRADYARRHYTHMIAS

New onset symptomatic bradycardia is rare in young women and hence also during pregnancy. Significant sinus node dysfunction is uncommon. Isolated first-degree AV block is benign. Wenckebach-type second-degree AV block in a structurally normal heart is not a major concern.²⁹ Symptomatic second-degree AV block and complete AV block can be problematic. Congenital complete heart block may present in women of child-bearing age and conduction disease related to previous surgery in women with repaired congenital heart disease is becoming increasingly frequent. In general, if a woman with a structurally normal heart is asymptomatic from bradycardia without evidence of a broadened QRS, pregnancy and delivery is tolerated well without the need for temporary or permanent pacing.^{30,31} However, if required, a pacemaker can be inserted with echocardiographic^{32,33} or cautious fluoroscopic guidance, ideally after the first trimester. Even in normal women, delivery

and spinal anaesthesia may precipitate bradycardia. In women with a clinically significant bradyarrhythmia, a delivery plan may include cardiac monitoring, appropriately trained staff and a management plan for worsening bradycardia. Moreover, it may be wise to avoid spinal anaesthesia during labour.^{9,34}

GENERAL PRINCIPLES OF TREATMENT

Arrhythmias that cause prolonged maternal hypotension and threaten placental perfusion require urgent treatment. However, treatment may also be required to prevent symptoms such as syncope, dyspnoea or distressing palpitations. The decision to use a particular therapy must take into account the risks of that therapy to mother and child. Although randomized data in the setting of maternal arrhythmias do not exist, experience suggests that many antiarrhythmic drugs appear to be relatively safe in pregnancy (Table 1). The lowest therapeutic dose should be used. If possible, the introduction of drugs during the first trimester should be avoided and, where relevant, drug levels and medication-related ECG changes must be monitored closely.

If a woman is planning pregnancy and suffers from recurrent bouts of tachyarrhythmia, radiofrequency ablation prior to conception should be considered.

Table 1 Safety of antiarrhythmic drugs during pregnancy and lactation

Drug	Vaughan Williams class	ADEC category [†]	Effect on fetus	Safe in lactation?
Adenosine		B2	None	Unclear
Digoxin		A	?IUGR	Insufficient data
Disopyramide	IA	B2	May induce uterine contractions	Yes
Lignocaine	IB	A	Neonatal neurotoxicity	Unclear*
Mexiletine	IB	B1	?Minimal	Insufficient data
			Data limited	Yes*
Flecainide	IC	B3	Possible fetal cardiotoxicity	Probably
Beta blockers	II	C	IUGR	Yes*
			?Fetal bradycardia	Monitor infant
			?Neonatal hypoglycaemia	Yes*
Sotalol	III	C	Bradycardia	Less transfer with metoprolol
			?Teratogenic	
Amiodarone	III	C	Thyroid disease	
			Bradycardia	No*
			IUGR	
Diltiazem	IV	C	Teratogenic	
			Limited data	Yes*
			?IUGR	
Verapamil	IV	C	Avoid maternal hypotension	
			?IUGR	Yes*
			?Fetal bradycardia	

*Transferred to breast milk

[†]ADEC category: Australian Drug Evaluation Committee categories for safety in pregnancy (abbreviated)

A: Drugs taken by a large number of pregnant women without any proven increase in the frequency of malformations or other direct or indirect harmful effects on the fetus having been observed

B1: Drugs which have been taken by only a limited number of pregnant women without an increase in the frequency of malformation or other direct or indirect harmful effects on the human fetus having been observed. Studies in animals have not shown harm

B2: Drugs which have been taken by only a limited number of pregnant women without an increase in the frequency of malformation or other direct or indirect harmful effects on the human fetus having been observed. Studies in animals are inadequate but available data show no harm

B3: Drugs which have been taken by only a limited number of pregnant women without an increase in the frequency of malformation or other direct or indirect harmful effects on the human fetus having been observed. Studies in animals have shown evidence of an increased occurrence of fetal damage

C: Drugs which have caused or may be suspected of causing, harmful effects on the human fetus or neonate. These effects may be reversible

D: Drugs which have caused, are suspected to have caused or may be expected to cause, an increased incidence of human fetal malformations or irreversible damage

DRUG THERAPY

Pharmacokinetics during pregnancy

There are multiple reasons why maintaining therapeutic drug levels in pregnant women can be challenging. The volume of distribution rises and that can increase the necessary loading dose to reach adequate serum concentrations. There is reduced protein binding due to a reduction in plasma proteins. These changes in combination are important because even though the serum concentration on testing can appear low, the active free fraction of the drug is unchanged or elevated. Gastrointestinal absorption of drugs may be affected by alterations in gastric acidity and motility. The glomerular filtration rate increases, augmenting the excretion of renally cleared drugs, and liver enzymes are also variably induced that can affect hepatically excreted substances.

Adenosine

Adenosine is first-line therapy for the termination of SVT. It is an endogenous purine nucleoside and is highly effective at modulating conduction through the AV node. It can cause flushing and dyspnoea but these side-effects are short-lived. It should be avoided in women with severe asthma as it may cause bronchospasm. The extremely short half-life is advantageous with respect to side-effects and unlike calcium channel blockers and beta-blockers it does not cause hypotension, an important factor in the acute management of SVT. It is generally agreed that adenosine does not cross the placenta in significant amounts to cause fetal bradycardia, although there is one report in the literature of a transient drop in heart rate without any significant consequences.³⁵ Adenosine is not known to be teratogenic. It is advisable to monitor fetal heart rate during administration.

Digoxin

Digoxin is a cardiac glycoside that exerts its antiarrhythmic activity via inhibition of the sodium/potassium ATPase. The AV nodal tissue is particularly sensitive and responds with slowed conduction. A vagotonic effect may also be important. It has been used to control the rate of supraventricular and atrial tachyarrhythmias in the mother and fetus for decades. Maternal toxicity can cause fetal death.³⁶ Digoxin crosses the placenta and maternal levels to some extent reflect those of the fetus.³⁷ The various physiological changes of pregnancy alter the pharmacokinetics of digoxin variably³⁸ and regular monitoring of serum levels is essential; however, in the third trimester they can appear spuriously high due to digoxin-like substances that interfere with the radioassay.³⁹ Although digoxin is excreted in breast milk, the dose ingested by infants is small and unlikely to be of clinical significance.⁴⁰

Class IA antiarrhythmic agents: quinidine, procainamide and disopyramide

These drugs predominantly block sodium channels and also potassium channels. They have been used to treat broad and narrow complex tachycardia in pregnancy but are not commonly used in Australia. These agents prolong the action potential duration, refractory period and QT interval, and consequently can

predispose to torsades de pointes so monitoring of the QT interval is crucial. They do not appear to be teratogenic. Quinidine has been used safely in pregnancy for many years to treat fetal and maternal tachyarrhythmias although other agents have largely superseded it. It is excreted in breast milk, but is probably safe in breastfeeding. Disopyramide has a limited role in pregnancy as it may precipitate premature labour.⁴¹ Prolonged use of procainamide can cause a lupus-like syndrome and this drug is no longer available in Australia.

Class IB lignocaine and mexiletine

Class IB drugs shorten the action potential duration and effective refractory period particularly in ventricular muscle. Lignocaine is only available in intravenous form and can be used in the acute management of VT. It does cross the placenta, but is not thought to be teratogenic at clinically relevant doses.^{42,43} Neonatal toxicity is theoretically possible although at least one report was probably the result of inadvertent scalp injection during anaesthetic administration for episiotomy.⁴⁴ Acidosis resulting from fetal distress increases the potential for toxicity.⁴⁵ Lignocaine levels should be monitored carefully. This agent is secreted in small amounts in the breast milk, but the dose is insufficient to be harmful. Mexiletine is an oral agent structurally similar to lignocaine. Experience of use in pregnancy is limited; however, there are no reports of teratogenicity or long-term adverse effects to the fetus. Although it is excreted in breast milk and breast-feeding is discouraged, levels are unlikely to be harmful to the infant.^{46,47}

Class IC flecainide

Class IC drugs are potent sodium-channel blockers and cause marked slowing of conduction. Flecainide is the only drug of this class in use in Australia. Unfortunately it can be pro-arrhythmic in the setting of structural heart disease. It crosses the placenta and is sometimes used to treat fetal tachycardia. Rarely fetal cardiotoxicity can occur⁴⁸ and levels should be monitored carefully. It does not appear to be teratogenic. It is excreted in breast milk but the effect on the infant is not clear. It is not infrequently used in patients who are not pregnant with lone AF as an antiarrhythmic; however, given the lack of experience with this drug in pregnancy other agents may be more suitable in gravid women. Flecainide may be useful as chronic therapy in women with WPW syndrome.

Class II agents: beta-blockers

Beta-blockers are useful for rate control in atrial arrhythmias as well as suppressing some supraventricular and VTs via their anti-sympathetic actions. Beta-blockers, particularly atenolol, taken at the time of conception or during the first trimester, may be associated with intrauterine growth restriction (IUGR). Other questionable effects on the fetus include bradycardia and neonatal hypoglycaemia.^{49,50} These agents are safe and highly effective treatment for many tachyarrhythmias and the benefits often far outweigh the risks. Our agent of choice is metoprolol, largely because it is less protein bound and therefore less likely to be transferred to breast milk. Data are lacking to support a particular beta-blocker in the treatment of arrhythmias during pregnancy.

Class III agents: sotalol and amiodarone

Class III agents block potassium channels and prolong myocardial repolarization. This manifests as QT prolongation on the ECG. These drugs may cause an acquired LQTS and dangerous ventricular arrhythmias.

Sotalol has been used in the treatment of fetal and maternal tachyarrhythmia because it readily crosses the placenta. It can also cause fetal bradycardia⁵¹. Sotalol may be teratogenic, although data are inconclusive.^{52,53} Given these ambiguities it is not a first choice antiarrhythmic; however in some difficult cases its use is warranted and complications are rare. Close fetal monitoring is essential. Sotalol is readily excreted in breast milk and some data have suggested that the infant may ingest up to 20% of the maternal dose,⁵⁴ although breastfeeding is considered safe if the child is regularly assessed for the signs of toxicity.

Amiodarone does not cross the placenta readily but fetal serum levels may still be up to a quarter of the mother's.⁵⁵ It has a long half-life and contains significant amounts of iodine that contribute to a long list of well-known side-effects. It can cause maternal and fetal hyper- or hypothyroidism, bradycardia, QT prolongation and IUGR. There is a possible increased risk of learning difficulties and teratogenicity with the drug but evidence is scant.^{56–58} If possible amiodarone should be avoided in pregnancy, especially during the first trimester. Unfortunately in some patients amiodarone succeeds where other therapy has not and in those potentially life-threatening situations it may be necessary to use this agent. Amiodarone is secreted in breast milk in amounts so large that the child may effectively be on a low adult maintenance dose and breast feeding should be discouraged.⁵⁹

Class IV agents: calcium channel blockers

Verapamil and diltiazem are the commonly used calcium channel blockers with cardiac activity. Cardiac effects include negative inotropy and slowed conduction through the AV node. Data from animal studies suggest that diltiazem may be teratogenic,⁶⁰ but this has not been confirmed in humans. A recent prospective cohort study of 299 pregnant women did not show an increase in birth defects or fetal loss. Babies exposed to calcium channel blockers had lower birth weights but this may have been due to maternal factors.⁶¹ Verapamil and diltiazem are excreted in breast milk although no major adverse events have been reported. Verapamil when used intravenously can cause maternal hypotension and should be used cautiously. It does cross the placenta to some degree and can potentially cause fetal bradycardia although reports are rare. We could find no data to support the widely held view that diltiazem does not cross the placenta. Reassuringly, reports of fetal bradycardia do not exist but experience is limited.

IMPLANTABLE CARDIAC DEFIBRILLATORS

Women with implantable cardiac defibrillators (ICD) should not be discouraged from falling pregnant unless they have underlying structural heart disease that would be considered a contraindication. Data have shown that pregnancy does not increase the risk of major ICD-related complications, nor does it appear to increase the rate of discharges from the device. ICD discharge does not appear to affect fetal outcome.⁶² After a shock has been administered the fetus should be checked

largely because of the possibility of fetal hypotension associated with the arrhythmia. ICDs can be implanted during pregnancy (see comments above regarding implantation of pacemakers).

DIRECT CURRENT CARIOVERSION

Direct current cardioversion is well tolerated in all stages of pregnancy and should be performed if clinically indicated. There is a theoretical risk of inducing ventricular fibrillation in the fetus; however, there are no data to support this. There is one report in the literature of fetal distress following maternal cardioversion, albeit in the setting of maternal hypotension from tachyarrhythmia, requiring urgent caesarean section.⁶³ If possible, obstetric and paediatric staff should be close at hand and fetal heart rate should be monitored.

RADIOFREQUENCY CATHETER ABLATION

If an arrhythmia is poorly controlled and the patient is planning pregnancy, it is wise to consider radiofrequency catheter ablation prior to conception. *Elective* electrophysiological testing and radiofrequency ablation procedures are contraindicated in pregnancy because of potential radiation exposure to the developing fetus. Ablation may be warranted in cases where the tachyarrhythmia is not well controlled on medication placing the mother and fetus at risk of prolonged episodes of hypotension or even more sinister consequences. Where possible the procedure should be performed after the first trimester.

Studies in patients who were not pregnant have shown that the potential radiation dose to a developing embryo or fetus during an ablation procedure would be small, resulting in a minimal increase in risk for childhood cancers, genetic abnormalities or serious birth defects.^{64,65} Although there are many case reports in the literature regarding radiofrequency ablation in pregnancy without any negative outcome, the largest series reports only three cases.⁶⁶ Fluoroscopy time should be kept to a minimum. Using venous access routes other than the femoral vessels and ensuring the maternal bladder is empty are other ways to minimize the radiation dose. Lead aprons do not seem to be useful in reducing exposure significantly as scatter from the thorax of the mother is the major radiation source to the fetus.^{64,65} The use of electroanatomical mapping systems (Carto™ Biosense Webster, Diamond Bar, California, USA or NavX™, St Jude Medical, St Paul, Minnesota, USA) may decrease the risk by reducing the need for fluoroscopy during the procedure.

CARDIAC ARREST

In the event of cardiac arrest in pregnancy general standard guidelines for cardiopulmonary resuscitation and advanced life support should be followed with a few alterations. The gravid uterus pressing on the inferior vena cava and abdominal aorta can compromise venous return and cardiac output, in turn impeding the effectiveness of chest compression. Tilting the patient partly toward the left lateral position by using a rolled towel or even the lap of a kneeling attending resuscitator can help alleviate this problem.⁶⁷ If this technique is employed it is imperative that the back and shoulders are stabilized to enable adequate forceful chest compressions. Alternatively the uterus can be pulled to the side. Cricoid pressure should be applied during positive pressure ventilation because of the increased

risk of regurgitation and aspiration in pregnant women and chest compressions should be a little higher than usual to allow for the elevation of the hemidiaphragm and abdominal contents. Paediatric and obstetric staff should be on hand as soon as possible and when available the fetal heart rate should be monitored.

Emergency caesarean section should be considered if the pregnancy has progressed to the stage that the fetus could be viable but the complexities of this issue are beyond the scope of this article. The American Heart Association's most recent cardiopulmonary resuscitation guidelines provide an excellent outline of the topic.⁶⁸

CONCLUSION

Although haemodynamically significant cardiac arrhythmias are uncommon in pregnancy, a grasp of the types of arrhythmias and principles of management are essential for anyone working in the area of Obstetric Medicine.

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