

Arrhythmias and Pregnancy

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The incidence of certain arrhythmias, as well as some electrophysiological properties, differ between the male and female sex.¹ Women seem to have higher intrinsic heart rates and shorter sinus node recovery times than do men. In women the corrected QT interval is longer and the long QT syndrome more frequent. The incidence of drug-induced torsade de pointes is clearly greater in the female sex, whereas women suffer fewer episodes of sudden cardiac death. While men more often have accessory pathways, women are twice as likely to exhibit atrioventricular nodal re-entrant tachycardia. The precise mechanism behind all these sex-related differences is not fully understood, but sex hormones are very likely to play a part.²

Pregnancy, by dramatically altering the hormonal and haemodynamic state of the organism, can be shown to be proarrhythmic, triggering the appearance of new arrhythmias or exacerbating existing ones. The elevated levels of oestrogen and b-chorial gonadotropine appear from experimental models to affect the expression of cardiac ion channels. The haemodynamic changes, the result of a doubling of the circulating blood and the cardiac output, contribute to arrhythmogenesis by increasing the end-diastolic volume and myocardial stretch. In addition, the increased catecholamine concentrations during pregnancy form a suitable environment for the development of arrhythmias in a pregnant woman.³

By functioning in a proarrhythmic way, pregnancy gives rise to significant problems

concerning the diagnosis and treatment of certain arrhythmias, especially when drugs and/or non pharmaceutical therapeutic methods are required. It is in these cases that the risk-benefit ratio must be given serious consideration so as to avoid undesirable consequences for the foetus from the use of these methods.

Palpitations are the most common cause of concern for the pregnant woman and the most usual reason for her to consult a cardiologist. In the majority of cases there are no objective findings and it is the heart rate that is responsible, sometimes increasing by as much as 20% during pregnancy.⁴ The ECG may show rotation of the electrical axis to the left, as a result of displacement of the heart as the womb expands.⁵ Atrial and ventricular premature beats are very common and are usually benign when not related to organic heart disease. Following a non-invasive diagnostic approach it is sufficient for the specialist to inform and reassure the patient, who should be advised to avoid stimulants such as caffeine. If the diagnostic examinations include a stress test this should be carried out at low exercise levels, with simultaneous monitoring of the foetal heart rate.⁶

Since supraventricular re-entrant tachycardias have a greater incidence in the female population, it is logical that they should be the most frequently encountered persistent arrhythmia in pregnant women. The therapeutic approach to such an episode is the same as in any other adult. Vagotonic manoeuvres should be tried initially, and if

these are unsuccessful they should be followed by intravenous adenosine, which is the drug of choice and is quite safe provided that the foetal heart rate is monitored in order to avoid significant bradycardia.⁷ In cases where adenosine fails intravenous beta-blockers should be preferred to verapamil, because the latter is likely to cause prolonged hypotension. Of course, the failure of pharmaceutical agents or haemodynamic instability are indications for electrical cardioversion.⁸ It should be noted that according to some studies, either the incidence of paroxysmal supraventricular tachycardia increases during pregnancy or the episodes become more symptomatic.^{9,10} Indeed, it seems that pregnancy functions more proarrhythmically in patients with tachycardias from accessory pathways than in those with tachycardias due to atrioventricular nodal re-entry.¹¹ This observation, in combination with the potentially damaging consequences of chronic antiarrhythmic treatment during pregnancy, makes it advisable to provide definitive treatment of an arrhythmia with non-pharmacological means (ablation) before a planned pregnancy.

Atrial fibrillation and flutter are rarely encountered during pregnancy unless organic heart disease or endocrine disorders are present. Episodes of such arrhythmias appearing for the first time during pregnancy require further evaluation for possible congenital heart disease, rheumatic valvular disease or hyperthyroidism. The therapeutic strategy aims initially to control the ventricular response with beta-blockers and digitalis. Cardioversion should be performed within the first 48 hours in order to avoid the need for anticoagulant medication, since coumarinic anticoagulants are known to have a teratogenic action, especially during the first trimester. If cardioversion does not occur spontaneously it may be achieved by electrical means, avoiding the use of antiarrhythmics.^{6,12}

A fortunately rare, but intractable problem is that of women with hereditary long QT syndrome who wish to bear a child. A retrospective study revealed that in such cases events such as death, aborted cardiac arrest or syncope are seen mainly after birth rather than during pregnancy.¹³ This indirectly suggests that the increased heart rate during pregnancy exerts a protective effect that disappears during the laying in period. Treatment with beta-blockers should anyway be continued throughout, since this seems to be an independent factor in the reduction of cardiac events in this population, compensating for the likely risk of complications to the foetus.

Ventricular tachycardia is a rare arrhythmia during pregnancy. If organic heart disease and long QT syndrome have been ruled out by electrocardiography

and echocardiography, it is most likely to be an idiopathic tachycardia arising from the right ventricular outflow tract.¹⁴ This is viewed as a benign arrhythmia, but if it appears repeatedly and causes a haemodynamic burden the treatment of choice is considered to be beta-blocker administration.¹⁵ In pregnant women where an organic substrate is detected the risk of sudden cardiac death is increased and the possibility of antiarrhythmic medication or the implantation of a cardioverter-defibrillator should be considered. For the immediate treatment of ventricular tachycardia the drug of choice is lidocaine, but electrical cardioversion is recommended in cases with haemodynamic instability.^{8,12} Patients with a long QT are mainly at risk of torsade de pointes and should take a beta-blocker throughout pregnancy and laying in, as mentioned above.

As implantable cardioverter-defibrillator devices become more widespread the question arises whether their implantation amounts to a contraindication for pregnancy. A multicentre study by Natale et al¹⁶ found that pregnancy does not increase the risk of complications from use of the device nor the probability of inappropriate discharges. Therefore, women who have a defibrillator may become pregnant unless the underlying heart disease itself makes pregnancy dangerous. The defibrillator should remain activated during natural birth so that any arrhythmia may be treated directly. However, during a Caesarean section where electrical diathermy is used the device should be deactivated.

Of course, it is preferable that the treatment of an arrhythmiological problem with an implantable defibrillator device or intracardiac ablation should precede the pregnancy. Exposure of the developing foetus to radiation may cause significant complications. During the first half of pregnancy congenital malformations and mental retardation may result, while exposure during the second half increases the risk of neoplasia during childhood.¹⁷ Nowadays, some centres have the ability to implant catheters or electrodes under non-fluoroscopic guidance in cases where electrophysiological intervention is absolutely necessary.¹⁸

A major concern in the treatment of arrhythmias during pregnancy is the safety of the foetus when antiarrhythmic medication is used. No drug is considered to be absolutely safe, because there is a lack of controlled studies and hence uncertainty regarding their effect on the foetus.¹⁹ For most antiarrhythmic drugs the risk cannot be ruled out, while amiodarone is clearly contraindicated because of its known teratogenic effects.²⁰

For supraventricular tachycardias, preventive antiarrhythmic treatment, according to the guidelines of the American College of Cardiology, American Heart Association and European Society of Cardiology, includes digitalis or beta-blockers as first choice.⁸ Of the beta-blockers, which are better avoided during the first trimester, propranolol or metoprolol should be preferred whereas atenolol should be avoided.²¹ A second choice is sotalol, which is viewed as a drug with no proven risk.

For ventricular arrhythmias, preventive antiarrhythmic treatment in patients with underlying heart disease, according to the same guidelines, includes sotalol, if beta-blockers prove insufficient.⁸

Whichever antiarrhythmic drug is chosen, it should be given in the minimum effective dosage, as late as possible (after the eighth week), and its blood levels should be measured frequently, since drug concentrations depend on renal blood flow, liver metabolism and absorption by the gastrointestinal system, parameters that are sure to undergo significant changes during pregnancy.²² Since side effects are dose-dependent, a combination of drugs in low dosages is preferable to high doses of a single drug.^{23,24}

Summing up, not only during diagnostic evaluation, but also and much more importantly in the therapeutic approach to arrhythmias during pregnancy, the risk-benefit ratio for the mother and the foetus must constantly be borne in mind.²⁵ An arrhythmia attack is not treated differently than in any other individual, but chronic treatment should be given special thought. Benign arrhythmias, even when symptomatic, may be left untreated. Administration of antiarrhythmic treatment is only justified when the patient's life is at risk or the arrhythmias are very frequent and cause haemodynamic instability. Invasive treatment should be postponed until after birth, or even better, should be applied before a planned pregnancy.

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