Cardiac disease and pregnancy

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Abstract
Cardiac disease in pregnancy is an important cause of maternal mortality and morbidity in the UK. An understanding of the physiological cardiovascular changes in pregnancy and their impact on pre-existing cardiac disease is important. The increased incidence of congenital heart disease reflects advances in corrective and palliative surgery for severe defects. Rheumatic heart disease may present for the first time in pregnancy. Appropriate referral and multidisciplinary planning is very important in women with known cardiac disease. It is also important to be vigilant and monitor women for the development of new-onset cardiac disease in pregnancy or the puerperium.

Keywords cardiac disease; cardiomyopathy; maternal mortality; peripartum; pregnancy

Cardiac disease in pregnancy is an important cause of maternal mortality and morbidity in the UK. In the latest report of the Confidential Enquiries into Maternal and Child Health[1] cardiac disease was the second commonest cause of maternal mortality.

Currently there is emphasis on appropriate referral and multidisciplinary planning for women with known cardiac disease. The overall reported mortality due to cardiac disease has approximately doubled during 30 years. However, mortality due to known cardiac disease has changed little. Thus, a significant and increasing number of deaths occur in women who do not have a known disease. This subgroup of women needs better management, and all centres should be vigilant and monitor women for the development of cardiac disease throughout pregnancy and the puerperium.

The incidence of congenital heart disease in pregnancy is increasing, reflecting advances in corrective surgery for severe defects. Rheumatic heart disease (most commonly mitral stenosis) may present for the first time in pregnancy, especially in immigrants.

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Physiological cardiovascular changes in pregnancy

The cardiac output increases by about 40% during pregnancy (Table 1). The increase starts in early pregnancy and peaks by mid second trimester. The cardiac output increases as both stroke volume and heart rate increase. Blood pressure tends to fall in the first and second trimester because of peripheral vasodilatation. The serum colloid osmotic pressure decreases by 10–15%. This decrease makes pregnant women more susceptible to pulmonary oedema.

In late pregnancy, the pressure of a gravid uterus on the inferior vena cava causes a decrease in venous return and a reduction in cardiac output. Cardiac output can decrease by 25% when the mother changes position from lateral to supine. This fall in output can compromise the fetus by decreasing placental perfusion.

Labour causes an increase in cardiac output. The rise is about 15% in the first stage and 50% in the second stage. This rise is due to uterine autotransfusion secondary to uterine contractions, and raised blood pressure and heart rate as a response to pain and anxiety.

After birth the cardiac output increases by 60–80% because of the relief of the obstruction of the inferior vena cava and autotransfusion from the placental bed. The cardiac output declines rapidly to prelabour values about 1 hour after delivery. The highest risk of pulmonary oedema occurs in the third stage of labour and in the immediate postpartum period.

Normal cardiac findings during pregnancy

Because of the cardiovascular adaptive process, some common findings in pregnancy include a bounding pulse, third-heart sound, relative sinus tachycardia and dependent peripheral oedema. An ejection systolic murmur can be heard in 90% of pregnant women and is usually audible all over the praecordium. ECG changes are partly related to changes in the position of the heart. These changes include atrial and ventricular ectopic beats, inverted T in lead III, ST depression and T wave inversion in inferior and lateral leads.

Cardiovascular system physiological changes during pregnancy

Cardiac output	Increases by 40%
Stroke volume	Increases
Heart rate	Increases by 10–20 beats per minute
Blood pressure	Decreases in first and second trimester
Central venous pressure	Unchanged
PCWP	Unchanged
SVR and PVR	Decreases
Serum colloid osmotic pressure	Decreases by 10–15%

PCWP, pulmonary capillary wedge pressure; PVR, pulmonary vascular resistance; SVR, systemic vascular resistance

Table 1

General considerations during pregnancy

Whatever the underlying cardiac problem, the ability to tolerate pregnancy and delivery is related to the presence of cyanosis and pulmonary hypertension, the haemodynamic significance of any lesion and the New York Heart Association (NYHA) functional classification (Table 2). Cyanosis alone may not be as important in predicting poor outcome as the association of cyanosis with Eisenmenger's syndrome, poor functional NYHA class or both. Cyanosis increases the risk of thrombosis, intrauterine growth restriction, and fetal death secondary to reactive polycythaemia.

Cardiac events such as stroke, arrhythmia, pulmonary oedema and death are also predicted by a previous cardiac event, NYHA classification of IV, cyanosis, left ventricular ejection fraction less than 40% and left-heart obstruction.

General management

Antepartum: ideally, all women with cardiac disease should be seen and assessed before pregnancy. Counselling regarding fetal and maternal risks is vital to an informed decision-making process. Multidisciplinary care involving an obstetrician, cardiologist, obstetric anaesthetist and obstetric physician (if available) is essential. A clearly documented plan for the antenatal period and delivery should be made.

For women with congenital heart disease the risk of their fetus having congenital heart disease is double that (2–5%) of women without the condition. These women should be referred for detailed fetal cardiac ultrasound.

Some women might need elective admission for bed rest to maximize their oxygen saturation. These women also require serial fetal growth scans because maternal cyanosis and hypoxia may cause growth restriction, miscarriage and iatrogenic prematurity.

If anti-arrhythmics are required, digoxin and β-blockers can be used during early pregnancy. Verapamil, adenosine and direct-current cardioversion are also safe to use. Flecainide is safe in the second and third trimester.

Intrapartum: antibiotic prophylaxis for delivery is advocated for women with structural heart defects. Exceptions are those women with repaired patent ductus arteriosus, isolated ostium secondum, atrial septal defects, or mitral valve prolapse without regurgitation. The current recommended antibiotics include amoxicillin and gentamicin at the onset of labour or ruptured membranes, followed by amoxicillin 4 hours later. For women allergic to penicillin, vancomycin or teicoplanin may be used (Table 3).

Women should be treated in the left or right lateral position. The supine position should be avoided to minimize reduction in cardiac output and pulmonary oedema.

New York Heart Association (NYHA) functional classification

Class I	No breathlessness
Class II	Breathlessness on severe exertion
Class III	Breathlessness on mild exertion
Class IV	Breathlessness at rest

Table 2

Classification of cardiac conditions according to risk of bacterial endocarditis

High risk: endocarditis prophylaxis recommended
- Prosthetic valves
- Previous bacterial endocarditis
- Complex cyanotic heart disease (tetralogy of Fallot)
- Surgical shunts

Moderate risk: endocarditis prophylaxis recommended
- Acquired valvular disease
- Hypertrophic cardiomyopathy
- Mitral valve prolapse with regurgitation

Low risk: endocarditis prophylaxis not recommended
- Isolated secondum atrial septal defect
- Mitral valve prolapse without regurgitation
- Physiological heart murmurs
- Cardiac pacemakers

Table 3

Continuous ECG and oxygen saturation monitoring is recommended, and full resustation facilities should be available. Epidural anaesthesia and analgesia, when administered slowly in incremental doses with judicious preloading, are well tolerated in most conditions. Caution is needed when left ventricular outflow obstructions occur, such as aortic stenosis and hypertrophic cardiomyopathy. If a pudendal block is needed epinephrine should not be used with lidocaine.

Postpartum: depending on the underlying condition, syntocinon should be used without ergometrine for the third stage of labour if hypertension needs to be avoided. Slow syntocinon infusion or misoprostol should be used if vasodilatation needs to be avoided (e.g. in hypertrophic cardiomyopathy or pulmonary hypertension). Women should be sitting upright as soon as possible after delivery. They should be monitored on a high-dependency unit or an ICU, depending on clinical risk. Breast feeding is not contraindicated if the mother is well. Before discharge contraception and the safety of future pregnancies must be discussed.

Specific conditions

Pulmonary hypertension may be due to lung disease such as cystic fibrosis, primary pulmonary hypertension, connective tissue disease such as scleroderma or Eisenmenger's syndrome (atrial and ventricular septal defects with pulmonary hypertension and reversal of shunt, i.e. right to left).

For women with pulmonary hypertension the decrease in systemic vascular resistance in pregnancy combined with fixed pulmonary vascular resistance (which normally decreases in pregnancy) means that there is an increase in right-to-left shunting and therefore these women cannot increase pulmonary blood flow to match the increased cardiac output.

Pulmonary hypertension is defined as a non-pregnant elevation of the mean pulmonary artery pressure equal to or greater than 25 mm Hg at rest or 30 mm Hg on exercise in the absence of a left-to-right shunt. If there is pulmonary hypertension in the