Heart Diseases in Pregnancy

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ABSTRACT

Heart disease is the leading cause of maternal mortality in the UK. Cardiomyopathy, myocardial infarction and aneurysm or dissection of the aorta are the leading causes of death from acquired heart disease while pulmonary hypertension is the leading cause of death from congenital heart disease. Congenital heart disease is the predominant form of heart disease encountered in pregnancy as most women with congenital heart disease now survive to adulthood due to the successes of pediatric cardiology and cardiac surgery. Patients with heart disease should receive multi-disciplinary counseling and risk assessment to enable an informed decision regarding pregnancy. Appropriate contraceptive advice should also be given. Physiological changes in pregnancy should be understood as they may precipitate de compensation in patients with previously well tolerated lesions. This article briefly reviews congenital and acquired cardiac lesions that are important because they are common conditions or because pregnancy poses a particular risk. Early involvement of a cardiologist is recommended in any pregnant woman with chest pain and ECG changes. Ischaemic heart disease and cardiomyopathy (dilated, peripartum, hypertrophic) are discussed. The management of pregnant women with prosthetic heart valves can be complex and choice of anti-coagulant needs to be individualized with close monitoring by a specialist team. Pregnancy is in-advisable in pulmonary hypertension, severe un-operated left sided stenosis, severely impaired ventricular function and Marfan syndrome with a dilated aortic root.

INTRODUCTION

At present, 0.2–4% of all pregnancies in western industrialized countries are complicated by cardiovascular diseases (CVD),[1] and the number of the patients who develop cardiac problems during pregnancy is increasing. Heart disease is the leading cause of maternal mortality in the UK, after psychiatric illness (maternal mortality rate of 2.2/100,000 maternities). Causes of maternal death include acquired and congenital heart disease; incidence 0.1% and 0.8%, respectively.[2] Cardiomyopathy (predominantly peripartum), myocardial infarction and aneurysm or dissection of the thoracic aorta are the leading causes of death from acquired cardiac disease while pulmonary hypertension is the leading cause of death from congenital heart disease.[2] The spectrum of CVD in pregnancy is changing and differs between countries. In the western world, the risk of CVD in pregnancy has increased due to increasing age at first pregnancy and increasing prevalence of cardiovascular risk factors—

diabetes, hypertension, and obesity. Also the treatment of congenital heart disease has improved, resulting in an increased number of women with heart disease reaching childbearing age.[3] In western countries maternal heart disease is now the major cause of maternal death during pregnancy.[4] Hypertensive disorders are the most frequent cardiovascular events during pregnancy, occurring in 6–8% of all pregnancies.[5] In the western world, congenital heart disease is the most frequent cardiovascular disease present during pregnancy (75–82%), with shunt lesions predominating (20–65%).[6,7] Congenital heart disease represents just 9–19% outside Europe and North America. Rheumatic valvular disease dominates in non-western countries, comprising 56–89% of all cardiovascular diseases in pregnancy.[6,7] Cardiomyopathies are rare, but represent severe causes of cardiovascular complications in pregnancy. Peripartum cardiomyopathy (PPCM) is the most common cause of severe complications.[8]

PHYSIOLOGICAL CHANGES DURING PREGNANCY

An understanding of the basic haemodynamic changes that take place throughout pregnancy and delivery is essential. The total blood volume increases by up to 50% (up to 1500 ml), starting as early as the fifth week of pregnancy, with an increase in red cell mass (increased oxygen carrying capacity) and an even greater increase in total plasma volume resulting in a relative anaemia. To deal with the increased blood volume and the additional demand for oxygen, there is a 40% increase in cardiac output peaking at the 25th week. This is achieved via an increased stroke volume in addition to a 15–20% increase in heart rate. Failure to achieve this is marked by a resting tachycardia which may signal diminished cardiovascular reserve and may in turn be detrimental in conditions where left ventricular filling is slow. An increase in cardiac output across a stenosed valve will cause an increase in transvalvular gradient which often manifests with worsening symptoms in the second trimester. The increased cardiac output is balanced by peripheral vasodilatation (due to hormonal changes) with a subsequent reduction in systemic vascular resistance and reduction in after load. This may decrease the regurgitant fraction in regurgitant valve disease and explains why pregnancy is frequently well tolerated with valvular regurgitation.[9] Other physiological changes include hypercoagulability with reduced antithrombotic factors (decreased protein C and protein S) and increased prothrombotic factors (fibrinogen, platelet activating inhibitor, platelet adhesion and aggregation) thus increasing the risk of valve thrombosis and other

thrombotic complications. All of these physiological changes may precipitate de compensation in patients with previously well tolerated lesions.

CARDIAC PHYSICAL EXAMINATION DURING NORMAL PREGNANCY

The carotid upstroke is brisk and the jugular venous pressure is normal or slightly increased. The apex beat may be displaced and increased. The first heart sound is loud, the pulmonary component of the second may be prominent and an S3 may be heard. An ejection systolic murmur may be heard in 90% of pregnant women caused by an increase in pulmonary outflow. Benign venous hums and mammary murmurs can also be heard.

CARDIOVASCULAR EVALUATION

All patients with known or suspected cardiac disease should have a detailed clinical history and examination ideally prior to conception and then at regular intervals according to the underlying cardiac condition. ECGs and echocardiograms are very useful non-invasive investigations. Chest x-ray (CXR) and other radiological procedures are best avoided (particularly during the first trimester), however a CXR carries a negligible risk if the fetus is shielded and should be considered if the patient has chest pain.[2] Caution should be used when considering computed tomography (CT) scanning due to the high maternal radiation dose involved. MRI is generally considered to be safe. Coronary angiography is occasionally necessary but should be avoided until at least 7 weeks gestation. Shielding should be used with an upper limb approach. No investigation should be withheld from a patient just because she is pregnant. Pulmonary embolism, aortic dissection and myocardial infarction (MI) account for a number of maternal deaths each year, and failure to investigate correctly probably accounts for a significant proportion of these.

FETAL ASSESSMENT

First trimester ultrasound allows accurate measurement of gestational age and early detection of multiple pregnancy and of malformations. Diagnosis of congenital cardiac malformations can be made as early as 13 weeks, and, in families with heart disease, this timing is appropriate to start screening for congential heart disease. A review of the accuracy of first-trimester ultrasounds for detecting major congenital heart disease showed a sensitivity and specificity of 85% [95% confidence interval (CI) 78–90%] and 99% (95% CI 98–100%), respectively. Early examination in pregnancy allows parents to consider all options, including termination of pregnancy, if there are major malformations.[10] The optimum time for screening of normal pregnancies for congenital heart diseases[11] is 18–22 weeks of gestation when visualization of the heart and outflow tracts is optimal. It becomes more difficult after 30 weeks since the fetus is more crowded within the amniotic cavity. Second-trimester screening (18–22 weeks) for detection of fetal anomalies should be performed by experienced specialists, particularly in pregnancies with risk factors for congenital heart anomalies.[12]

INTERVENTIONS IN THE MOTHER DURING PREGNANCY

Percutaneous therapy: The effects of radiation on the fetus depend on the radiation dose and the gestational age at which exposure occurs. If possible, procedures should be delayed until at least the completion of the period of major organogenesis (<12 weeks after menses). If an intervention is absolutely necessary, the best time to intervene is considered to be after the fourth month in the second trimester. By this time organogenesis is complete, the fetal thyroid is still inactive, and the volume of the uterus is still small, so there is a greater distance between the fetus and the chest than in later months. Fluoroscopy and cineangiography times should be as brief as possible and the gravid uterus should be shielded from direct radiation. Heparin has to be given at 40–70 U/kg, targeting an activated clotting time of at least 200 s, but not exceeding 300 s.

Cardiac surgery with cardiopulmonary bypass: Maternal mortality during cardiopulmonary bypass is now similar to that in non-pregnant women who undergo comparable cardiac procedures.[1] However, there is significant morbidity including late neurological impairment in 3–6% of children, and fetal mortality remains high.[13] For this reason cardiac surgery is recommended only when medical therapy or interventional procedures fail and the mother's life is threatened. The best period for surgery is between the 13th and 28th week.[14,15]

TIMING AND MODE OF DELIVERY: RISK FOR MOTHER AND CHILD

High risk delivery

Induction, management of labour, delivery, and post-partum surveillance require specific expertise and collaborative management by skilled cardiologists, obstetricians, and anaesthesiologists, in experienced maternal–fetal medicine units.[16,17]

Timing of delivery

Spontaneous onset of labour is appropriate for women with normal cardiac function and is preferable to induced labour for the majority of women with heart disease. Timing is individualized, according to the gravida's cardiac status, Bishop score, fetal well-being, and lung maturity. In women with mild unrepaired congenital heart disease and in those who have undergone successful cardiac surgical repair with minimal residua, the management of labour and delivery is the same as for normal pregnant women.

Labour induction

Oxytocin and artificial rupture of the membranes are indicated when the Bishop score is favourable. A long induction time should be avoided if the cervix is unfavourable. While there is no absolute contraindication to misoprostol or dinoprostone, there is a theoretical risk of coronary vasospasm and a low risk of arrhythmias. Dinoprostone also has more profound effects on BP than prostaglandin E1 and is therefore contraindicated in active CVD. Mechanical methods such as a Foley catheter would be preferable to pharmacological agents, particularly

in the patient with cyanosis where a drop in systemic vascular resistance and/or BP would be detrimental.[18]

Vaginal or caesarean delivery: The preferred mode of delivery is vaginal, with an individualized delivery plan which informs the team of timing of delivery (spontaneous/ induced), method of induction, analgesia/ regional anaesthesia, and level of monitoring required. In high risk lesions, delivery should take place in a tertiary centre with specialist multidisciplinary team care. Vaginal delivery is associated with less blood loss and infection risk compared with caesarean delivery, which also increases the risk of venous thrombosis and thrombo-embolism.[19] In general, caesarean delivery is reserved for obstetric indications. Caesarean delivery should be considered for the patient on oral anticoagulants (OACs) in pre-term labour, patients with Marfan syndrome and an aortic diameter >45 mm, patients with acute or chronic aortic dissection, and those in acute intractable heart failure. Cesarean delivery may be considered in Marfan patients with an aortic diameter 40–45 mm.[20,21]

Haemodynamic monitoring
Systemic arterial pressure and maternal heart rate are monitored, because lumbar epidural anaesthesia may cause hypotension. Pulse oximetry and continuous ECG monitoring are utilized as required.

Anaesthesia/analgesia
Lumbar epidural analgesia is often recommendable because it reduces pain-related elevations of sympathetic activity, reduces the urge to push, and provides anaesthesia for surgery. Continuous lumbar epidural analgesia with local anaesthetics or opiates, or continuous opioid spinal anaesthesia can be safely administered. Regional anaesthesia can, however, cause systemic hypotension and must be used with caution in patients with obstructive valve lesions.

Labour
Once in labour, the woman should be placed in a lateral decubitus position to attenuate the haemodynamic impact of uterine contractions.[22] The uterine contractions should descend the fetal head to the perineum, without maternal pushing, to avoid the unwanted effects of the Valsalva manoeuvre.[23,24] Delivery may be assisted by low forceps or vacuum extraction. Routine antibiotic prophylaxis is not recommended. Continuous electronic fetal heart rate monitoring is recommended.

Delivery in anticoagulated women with prosthetic valves
OACs should be switched to LMWH or unfractionated heparin (UFH) from the 36th week. Women treated with LMWH should be switched to i.v. UFH, at least 36 h before the induction of labour or caesarean delivery. UFH should be discontinued 4–6 h before planned delivery, and restarted 4–6 h after delivery if there are no bleeding complicationsIf emergent delivery is necessary while the patient is still on UFH or LMWH, protamine should be considered. In the event of urgent delivery in a patient on therapeutic OACs, caesarean delivery is preferred to reduce the risk of intracranial haemorrhage in the fully anticoagulated fetus. If emergent delivery is necessary, fresh frozen plasma should be given prior to caesarean delivery to achieve a target international normalized ratio (INR) of ≤2.4 Oral vitamin K (0.5–1 mg) may also be given, but it takes 4–6 h to influence the INR. If the mother was on OACs at the time of delivery, the anticoagulated newborn may be given fresh frozen plasma and should receive vitamin K. The fetus may remain anticoagulated for 8–10 days after discontinuation of maternal OACs.

Post-partum care
A slow I.V. infusion of oxytocin (2 U/min), which avoids systemic hypotension, is administered after placental delivery to prevent maternal haemorrhage. Prostaglandin F analogues are useful to treat post-partum haemorrhage, unless an increase in pulmonary artery pressure (PAP) is undesirable. Methylergonovine is contraindicated because of the risk (.10%) of vasoconstriction and hypertension. [25, 26] Meticulous leg care, elastic support stockings, and early ambulation are important to reduce the risk of thrombo-embolism. Delivery is associated with important haemodynamic changes and fluid shifts, particularly in the first 12–24 h, which may precipitate heart failure in women with structural heart disease. Haemodynamic monitoring should therefore be continued for at least 24 h after delivery.[27]

Breastfeeding
Lactation is associated with a low risk of bacteraemia secondary to mastitis. In highly symptomatic/unwell patients, bottle-feeding should be considered.

INFECTIVE ENDOCARDITIS
Infective endocarditis during pregnancy is rare, with an estimated overall incidence of 0.006% (1 per 100 000 pregnancies)[28] and an incidence of 0.5% in patients with known valvular or congenital heart disease.[29] The incidence is higher in drug addicts. Patients with the highest risk for infective endocarditis are those with a prosthetic valve or prosthetic material used for cardiac valve repair, a history of previous infective endocarditis, and some special patients with congenital heart disease.

Prophylaxis
The same measures as in non-pregnant patients with recent modifications of guidelines apply.[30] Endocarditis prophylaxis is now only recommended for patients at highest risk of aquiring endocarditis during high risk procedures, e.g. dental procedures. During delivery the indication for prophylaxis has been controversial and, given the lack of convincing evidence that infective endocarditis is related to either vaginal or caesarean delivery, antibiotic prophylaxis is not recommended during vaginal or caesarean delivery.[30,31]

ISCHAEMIC HEART DISEASE (IHD)
An acute coronary event occurs in 1 in 10–35,000 pregnancies, predominantly in the third trimester, caused by coronary thrombosis, spasm or dissection and less commonly atherosclerosis. Risk factors include increased age, diabetes, hypertension and eclampsia with greatest

risk occurring before and during labour. Mortality is 21–37% increasing up to 50% in patients with diabetes. Fetal mortality ranges between 13–34%. Investigations include ECG and troponin I, 3 which are reliable biomarkers for myocardial injury during pregnancy (unlike creatinine kinase). Angina can be treated conventionally but an acute coronary syndrome (ACS) is a difficult problem with little data in pregnancy. Since the mortality of acute MI is so high, a 'wait and see' approach is not good practice. Thrombolysis carries a risk of maternal and fetal haemorrhage and will not be effective if the pathology is coronary dissection. The best option in a woman with an acute MI is urgent transfer to the catheter laboratory where angiography will reveal the pathology and allow effective intervention for dissection or ruptured plaque.

Early involvement of a cardiologist is recommended in any pregnant woman with chest pain and ECG changes.

DILATED CARDIOMYOPATHY

Pregnancy in patients with dilated cardiomyopathy is poorly tolerated and related to New York Heart Association (NYHA) class. Mortality in classes I–II is 1%, increasing to at least 7% in classes III–IV. Fetal mortality is strongly related to NYHA class (30% in class IV). Adverse prognostic features include left ventricular (LV) ejection fraction <20%, mitral regurgitation, right ventricle (RV) failure, atrial fibrillation and hypotension. Most patients should be advised against pregnancy but if they do become pregnant they should be advised to limit strenuous exercise, to obtain adequate rest, restrict salt intake and attend for regular specialist review. Anaemia should be avoided. Diuretics, β-blockers and vasodilators can be used. ACE inhibitors should be avoided unless poor maternal cardiac status means that the risk of a poor maternal and fetal outcome is considered to be greater than the associated ~17% risk of fetal renal agenesis.

PERIPARTUM CARDIOMYOPATHY

This rare condition is defined as cardiac failure in the last month of pregnancy or within 5 months of delivery with no identifiable cause of heart failure and no recognisable pre-existing heart disease. Mortality ranges between 6–50% with mortality risk in subsequent pregnancy estimated as:

- 0–2% if normal ejection fraction (EF) before subsequent pregnancy

- 8–17% if depressed EF.

Adverse risk factors include age, multiparity, twins and persisting left ventricle (LV) dilatation and dysfunction. Around 50–60% of patients show complete or near-complete recovery of clinical status and cardiac function six months post partum. Subsequent pregnancies should be discouraged if the LV does not recover.[32,33]

HYPERTROPHIC CARDIOMYOPATHY

Most women tolerate pregnancy well. In a study of 91 consecutive families with 199 live births, symptoms deteriorated in less than 10% of patients. Mortality was increased but confined to patients known to be at high risk (massive LV hyperthrophy, severe restrictive physiology). Maternal death was uncommon and risk of sudden death was not increased by the pregnancy.[34] Vaginal delivery is generally well tolerated with careful fluid management in labour to avoid hypovolaemia-induced hypotension due to decreased left ventricular filling or pulmonary oedema due to overzealous fluid replacement.

MITRAL STENOSIS

Regurgitant lesions are generally well tolerated in pregnancy whereas left-sided stenotic lesions are not. Impaired diastolic flow through a stenotic valve may cause tachycardia and increased stroke volume leading to increased left atrial (LA) pressure, pulmonary oedema and atrial fibrillation. Treatment includes bed rest, diuretics, rate control and heparin. Balloon valvotomy should be used if delivery is not advisable (e.g. fetal prematurity) or if the patient is haemodynamically compromised.

PROSTHETIC HEART VALVES

The high risk of thrombo-embolic complications means that choice of anticoagulant is extremely important. The interests of the mother and fetus are in conflict:

- warfarin is a more effective anticoagulant, but crosses the placenta and may cause embyropathy and fetal haemorrhage

- heparin does not cross the placenta, so has no fetal adverse effects, but is associated with a 12–24% risk of valve thrombosis or embolism.[35]

Women with mechanical valves need care from a specialized team which includes a cardiologist, obstetrician and haematologist. The choice of anticoagulation regime needs to be individualized taking into account factors such as valve site and type and required warfarin dose (very low risk of embryopathy if <5 mg).[36] The choice of regimens includes:

- warfarin throughout pregnancy with elective caesarean section at 38 weeks

- heparin and aspirin for the 1st trimester, warfarin in the 2nd trimester, converting back to heparin for delivery

- heparin and aspirin throughout.

There is no consensus about the type of heparin used and choice is controversial in the absence of good data. Unfractionated heparin is associated with osteoporosis and thrombocytopenia whereas low molecular weight (LMW) heparin is not. It is also possible to more effectively control LMW heparin with 2–4 weekly anti-Xa levels. LMW heparin should be given as a twice-daily regimen.[37,38]

CONGENITAL HEART DISEASE

This section is not an exhaustive review of the congenital cardiac lesions that may be encountered in pregnancy. It includes some of those that are important because they are common conditions or because pregnancy poses a particular risk.

BICUSPID AORTIC STENOSIS

Most young people with aortic stenosis are asymptomatic and pregnancy is well tolerated if the patient has a:

- normal resting ECG or voltage increase only

- normal exercise test

- good LV function

- pre-pregnancy echo gradient – peak <80, mean <50 mmHg.

Signs of decompensation include disproportionate dyspnoea, angina, pulmonary oedema, new ECG changes and an unexpected fall in peak Doppler gradient. Treatment includes bed rest and β-blockade while in severe cases aortic valvotomy and aortic valve replacement may need to be considered. Vaginal delivery is advisable with avoidance of vasodilators and hypovolaemia (as with all obstructive lesions).

AORTIC COARCTATION

Major complications are infrequent but are a cause for concern. Obstetric and neonatal outcomes are similar to the general population.[39] Each patient should have formal preconception haemodynamic assessment (exercise stress test, echocardiogram, MRI aorta) with very close monitoring of blood pressure during pregnancy.

MARFAN SYNDROME

The main maternal risk in this condition is type A aortic dissection – 1%. Patients with the following are at high risk:

- poor family history

- cardiac involvement

- aortic root >4 cm or rapidly expanding

Every patient should receive appropriate pre-conception counseling with regard to maternal as well as fetal risk. β-blockers are frequently used with elective Caesarean section if at higher

risk. Staff should remain vigilant about the possibility of dissection as a cause of chest or interscapular pain in pregnancy, particularly if the woman is hypertensive. Aortic dissection should always be considered in pregnant women with atypical chest pain and features of pulmonary embolism who do not get better with treatment. Other diagnostic features include pulse deficits and signs of aortic regurgitation.

REPAIRED TETRALOGY OF FALLOT

Pregnancy is well tolerated if ventricular function is good and there is no significant right ventricular (RV) outflow tract obstruction.[40] If the woman has significant pulmonary regurgitation, a successful pregnancy outcome may be anticipated, but she may become breathless earlier than expected in pregnancy and may need bed rest and diuretics.

CONGENITALLY CORRECTED TRANSPOSITION OF THE GREAT ARTERIES

In this condition the right ventricle supports the systemic circulation. Pregnancy is well tolerated but RV function may deteriorate during the course of pregnancy and systemic AV valve regurgitation may worsen. Treatment is as for any patient with heart failure. There is also a risk of complete heart block.

TRANSPOSITION OF THE GREAT ARTERIES POST MUSTARD OR SENNING

With atrial redirection the RV supports the systemic circulation and there is a risk of atrial arrhythmia as well as RV failure and systemic atrioventricular valve regurgitation. The patency of the pulmonary venous pathways must be checked (by echocardiography) since obstruction may mimic mitral stenosis. Pregnancy is well tolerated if patients are NYHA class I–II.

FONTAN PROCEDURE

This is a palliative procedure for patients with univentricular hearts e.g. tricuspid atresia, pulmonary atresia with intact ventricular septum. Both atrioventricular valves are connected to a single ventricular cavity (double-inlet ventricle) and the main ventricle is connected to a rudimentary chamber.

This changes a complex cyanosed patient into a complex patient who is now pink. With no pump to support the pulmonary circulation, it relies on phasic flow with limited ability to increase cardiac output. Atrial arrhythmias are poorly tolerated and the circulation is prothrombotic. If patients are in NYHA class I–II with good ventricular function, maternal risk is not excessively high. Strict care should be taken to maintain filling pressures, avoid dehydration or vasodilation. There is a high risk of fetal loss (30%). Most patients take warfarin and should be converted to LMW heparin for the duration of the pregnancy. Anti-arrhythmics are often needed raising the issue of arrhythmia risk vs teratogenicity. These patients need multidisciplinary expert care.[41,42]

PULMONARY HYPERTENSION

There is a high risk of maternal death and severe morbidity even if pulmonary artery pressures are half systemic. The risk of maternal death is 40–50% (unchanged in the last four decades) and therefore pregnancy is NOT advised. If a woman chooses to continue with pregnancy, she should receive multidisciplinary specialist antenatal and peripartum care. There is no evidence that intervention reduces mortality, but bed rest, heparin, oxygen, prostacyclin, phosphodiesterase inhibitors and endothelin antagonists may all be considered. The risk of death continues for at least two weeks postpartum. Appropriate contraceptive advice is extremely important. While sterilization can be considered, there are other more effective methods which avoid a general anaesthetic e.g. Implanon (sub dermal implant).

RISK ESTIMATION: CONTRAINDICATIONS FOR PREGNANCY

Maternal risk assessment is carried out according to

the modified World Health Organization (WHO) risk classification.[43] This risk classification integrates all known maternal cardiovascular risk factors including the underlying heart disease and any other co-morbidity. It includes contraindications for pregnancy. The general principles of this classification. A practical application. In women in WHO class I, risk is very low, and cardiology follow-up during pregnancy may be limited to one or two visits. Those in WHO II are at low or moderate risk, and follow-up every trimester is recommended. For women in WHO class III, there is a high risk of complications, and frequent (monthly or bimonthly) cardiology and obstetric review during pregnancy is recommended. Women in WHO class IV should be advised against pregnancy but, if they become pregnant and will not consider termination, monthly or bimonthly review is needed.

CONCLUSION

All patients should receive multi-disciplinary counselling and cardiac assessment prior to conception. They should all receive appropriate contraceptive advice. An estimate of maternal and fetal risk should be made to enable an informed decision regarding pregnancy. Pregnancy is inadvisable in:

- pulmonary hypertension
- severe unoperated left sided stenosis
- severely impaired ventricular function (EF<20%)
- Marfan syndrome with a dilated aortic root.

Treatment and management approaches in pregnant women are similar to those in non-pregnant patients with awareness of altered physiology and careful attention to fetal and maternal effects of pharmacological agents.

All pregnant and recently delivered women with congenital heart disease should be appropriately supervised, preferably by a consultant cardiologist with a special interest in congenital heart disease in close conjunction with a consultant obstetrician.

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