The heart of pregnancy

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Pregnancy leads to a significant physiological change in the cardiovascular system. Women with cardiovascular compromise due to cardiac disease may not be able to meet these physiological demands, requiring multidisciplinary specialist input and careful management during pregnancy, labour and post-delivery.

Physiological changes in pregnancy

The 'normal' physiological changes occurring during pregnancy ask additional demands of the already compromised cardiovascular system in pre-existing cardiac disease:

- Increase in blood volume by the fifth week, with plasma volume increasing more than blood cell mass, leads to physiological anaemia of pregnancy.
- There is peripheral vasodilatation leading to a decrease in systemic vascular resistance.
- Cardiac output increases by 20 per cent in the first trimester and by up to 40 to 50 per cent at 20 to 28 weeks. (This is achieved mainly by an increase in stroke volume and less by increase in heart rate.)

'Established labour' adds even further strain. This is compounded by pain and anxiety, leading to a sympathetic response, elevating blood pressure and heart rate:

- Cardiac output is increased by 15 per cent in the first stage and 50 per cent in the second stage due to the auto-transfusion of 300 to 500ml of blood back into the circulation with each uterine contraction.
- Cardiac output increases again immediately after delivery due to auto-transfusion of blood via uterine contraction and relief of aortocaval compression.
- This may increase cardiac output by as much as 60 to 80 per cent, followed by a rapid decline to pre-labour values within one hour.[1]

There is an associated physiological thrombophilic state:

- Increased concentrations of coagulation factors: factor I, V, VII, VIII, IX, X, XII.
- Reduced endogeneous anticoagulations: protein S.
- Fibrinolysis suppression due to increase in endothelial plasminogen activator inhibitor.

These factors combine to lead to an increased risk of thrombosis beginning in early pregnancy.

The impact of these physiological changes to a pregnancy with pre-existing cardiac disease will vary according to the type and severity of the disease. Women with heart disease are likely to decompensate antenatally or during labour and postnatally due to the physiological changes described above.
These changes combined with the reduction in serum colloid osmotic pressure make women with cardiovascular compromise particularly susceptible to pulmonary oedema at the time of delivery and immediately postpartum. This risk is further increased if the woman has preeclampsia with the increase in pulmonary capillary permeability.

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Planning management in pre-existing cardiac disease

Preconception

Women of reproductive age with congenital or acquired heart disease are usually known to their medical practitioner and should be referred to an interested obstetrician and/or cardiologist for pre-conceptual counselling. This must include an assessment of pre-pregnancy cardiac function, the risks of pregnancy to maternal short and long-term health, the risk of inheritance of congenital heart disease and discussions around mode of delivery.

Antenatal

Following pregnancy, a multidisciplinary approach, involving obstetricians, cardiologists, anaesthetists and neonatologists is required.[2] Management involves optimisation of maternal health and fetal wellbeing with appropriate cardiac investigations required to monitor maternal status. Women with heart failure can be safely treated with diuretics, digoxin and hydralazine, nitrates, or both as vasodilators to reduce left ventricular load.

Labour and delivery

Vaginal delivery is safe in most women with heart disease. There will be a necessity to deliver early by caesarean section if there is deterioration in the cardiovascular status or other obstetric or fetal indications. Epidural analgesia should be considered early for pain relief, to stabilise any additional cardiovascular compromise due to anxiety and pain:[3]

- The first stage of labour can be managed as normal, avoiding prolonged labour, with judicious use of syntocinon.
- The second stage of labour can be shortened by ventouse or forceps delivery to prevent maternal exhaustion and auto-transfusion of blood with every contraction.
- The third stage of labour is best managed by syntocinon infusion rather than bolus. Ergometrine leads to vasoconstriction and is best avoided as is carboprost in view of the risk of myocardial ischemia and pulmonary oedema.

Postnatal

This group of pregnant women is at particular risk of decompensation following delivery and extra vigilance should be provided by healthcare professionals in caring for these women. A formal plan for the immediate postnatal period and long-term care should be devised, including contraception.

There is a particular risk of pulmonary oedema and cardiomyopathy in the pueperium.

Specific high-risk groups

Valvular heart disease

Congenital valvular lesions are now more common in the Western world with improved outcomes following cardiac surgical interventions, while acquired rheumatic valvular disease is more common in immigrant communities.

Women with congenital heart disease and pre-existing poor maternal cardiac function (NYHA Class II, III,IV; cyanosis; heart failure; left heart outflow obstruction; or arrhythmias) are associated with significantly poorer maternal outcome.

Atrial septal defects (ASDs) are common lesions. Uncorrected large lesions may be complicated by paradoxical embolus and arrhythmias frequently occur. Thromboprophylaxis is indicated if any other risk factors for thrombosis are present. Ventricular septal defects (VSDs) are usually well tolerated but require antibiotic prophylaxis for labour and delivery.

Acquired heart disease with mitral stenosis carries a significant risk in pregnancy because of the increased vascular volume and cardiac output during pregnancy and labour. Severe mitral stenosis associated with a large left atrium may be treated by beta-blockers and heparin in pregnancy to prevent atrial fibrillation.[4] However, associated pulmonary hypertension is an absolute contraindication to pregnancy.

The management of other valvular conditions in pregnancy depends on the severity of the condition. Mitral and aortic regurgitation are usually well-tolerated in pregnancy, provided there is no significant left ventricular dysfunction. The decrease in systemic vascular resistance in pregnancy is usually associated with a reduction in 'regurgitant' flow across the affected valve. The general principle during labour for women with severe aortic stenosis is to avoid sudden decreases in systemic vascular resistance, achieved via careful regional or general anaesthesia and labour, based on the principles described earlier.

Thromboprophylaxis in women with metallic prosthetic heart valves remains controversial. Warfarin provides the best protection against thrombosis, but increases the risk of fetal teratogenicity and miscarriage. Low molecular weight heparin is much safer for the fetus but carries a greater risk of valve thrombosis to the mother. The method of thromboprophylaxis is therefore usually determined after discussion with pregnant women.

Percutaneous catheter interventions are safe in the management of mitral and pulmonary stenosis during pregnancy. However, balloon dilatation for aortic valve disease should only be considered in high-risk cases as it carries a lower success rate and a higher risk.

Ischemic cardiac disease

Women with risk factors such as obesity, older age and high parity, smoking, diabetes, pre-existing hypertension, and a family history are at high risk for myocardial ischemia. A low threshold for diagnosis of myocardial infarction and acute coronary syndrome in women with risk factors is recommended and appropriate intervention in the form of coronary angiography, emergency coronary intervention and thrombolysis should not be withheld in the pregnant or puerperal woman.[5]

Literature review suggests management of delivery in women with coronary artery disease, ranging from spontaneous delivery with or without epidural analgesia to elective caesarean under combined spinal epidural anaesthesia[6] or general anaesthesia[7].

Pulmonary hypertension

Pulmonary arterial hypertension carries a very high risk during pregnancy (30 to 50 per cent mortality). A multidisciplinary risk assessment has to be carried out. Termination of pregnancy should be strongly considered as an alternative. Owing to the increased risk of mortality at delivery, elective caesarean section under a cardiac general anaesthetic is recommended with invasive monitoring.[8]

Marfan's syndrome and aortic dissection

Aortic dissection is a particular risk in women with Marfan's syndrome. However, it can also occur in previously apparently normal women. Preconceptual couselling and the consideration of corrective surgery for major aortic root dilatation prior to embarking on pregnancy is suggested. Beta-blockers can be safely used in pregnancy with fetal growth monitoring.[1] The differential diagnosis of aortic dissection includes pulmonary embolism, pneumonia, pneumothorax, myocardial ischaemia, pericarditis and musculoskeletal pain.

Peripartum cardiomyopathy

Peripartum cardiomyopathy is one of the commonest cardiac causes of maternal death in Australia and is defined as 'the onset of heart failure with no identifiable cause in the last month of pregnancy or within five months after delivery'. It is more common in older, obese, multiparous women with hypertension in pregnancy. All clinicians must have a high index of suspicion when tachycardia, tachypnoea, dysponea or pulmonary oedema develop in the context of these risk factors in the late third trimester or early postpartum period.[9]

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