PREGNANCY AND HEART DISEASE

Pregnancy and native heart valve disease

Sara Thorne

Correspondence to
Dr Sara Thorne, Department of Cardiology, University Hospital Birmingham NHS Trust, Queen Elizabeth Hospital, Edgbaston, Birmingham B15 2TH, UK; Sara.thorne@uhb.nhs.uk

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Learning objectives

- To understand the haemodynamic changes of pregnancy and the effects of pregnancy on women with heart valve disease.
- To understand how to assess and manage women with native valve disease who present before or during pregnancy.
- To understand that regurgitant valve lesions are better tolerated in pregnancy than stenotic lesions.
- To appreciate the differences in presentation, management and pregnancy outcome between aortic and mitral stenosis.

INTRODUCTION

Heart disease is the biggest killer of pregnant women in the developed world.[1] Likewise, heart disease emerges as the main indirect (non-obstetric) cause of maternal death in low/middle-income countries once basic maternity services and sufficient infrastructure are in place. However, while deaths from complications of ischaemic heart disease, heart failure and sudden arrhythmic death syndrome are the leading causes of cardiac maternal death in the UK,[2] rheumatic mitral stenosis is the single most common cause of cardiac maternal mortality in the developing world.[3 4]

Valvular heart disease is not uncommon in women of childbearing age, but there is a paucity of population-based data. Nonetheless, rheumatic valve disease predominates in the developing world, accounting for 50%–90% of maternal cardiovascular complications. In contrast, in the developed world, valve disease is responsible for around 15% of pregnancy-related complications, most commonly as bicuspid aortic valve and mitral valve prolapse.[5]

This article will review the haemodynamic changes of pregnancy and consider how native valve disease interacts with the physiological demands of pregnancy and delivery. Valve diseases of the normally connected heart are addressed in detail; valve lesions that form part of complex congenital heart disease are beyond the scope of the article.

Cardiovascular demands of pregnancy and delivery

Cardiac output increases during pregnancy by 50% above the non-pregnant state, with most of the rise occurring before the middle of the second trimester and being maintained until the end of pregnancy. The rise in cardiac output is achieved by a modest (10%) and gradual rise in heart rate and by a 30% rise in stroke volume by the end of the second trimester. Total vascular resistance falls as a result of systemic vasodilation and the low resistance uteroplacental circulation, accommodating the 50% increase in blood volume (figure 1).

During labour, cardiac output rises during uterine contractions by a further 80%. Sympathetic tone increases due to pain and anxiety resulting in an increase in heart rate and blood pressure, and blood volume increases due to autotransfusion from the contracting uterus. Unless there is significant haemorrhage, there is a further increase in blood volume post-delivery from the placenta and from relief of inferior caval vein obstruction.[6] The volume changes are more rapid when delivery is by caesarean section. Thus, the mode of delivery associated with the least cardiovascular stress is a spontaneous vaginal delivery with adequate analgesia and a low threshold for instrumental assistance.

An understanding of the cardiovascular physiology of pregnancy allows the prediction of which valve lesions will be well tolerated or fare badly during pregnancy: conditions with a fixed cardiac output such as severe mitral or aortic stenosis are at greatest risk of complication and decompensation. In comparison, regurgitant lesions are likely to be well tolerated, with the fall in vascular resistance compensating for the volume loading effects of pregnancy.

Pregnancy risk in valvular heart disease

Valvular conditions can be classified according to a validated modified WHO scale,[7] shown to be the best risk model currently available (box 1).[8]

Thus, in general, the maternal risk of pregnancy for women with isolated valve disease and normal ventricular size and function may be classified as shown in box 1.[5 7] However, risks are additive; so the risks will be higher if the valve lesion coexists with more complex heart disease, ventricular dysfunction, aortopathy or if it is as a result of underlying heart disease. In addition, risks are higher if the woman has non-cardiac risk factors including hypertension, obesity, diabetes or renal disease (table 1).

There are little data on the risks of pregnancy in women with mixed valve disease; a reasonable approach is to add the risks of each lesion.

Preconception counselling

Preconception assessment and counselling is important in reducing maternal risk. It allows the woman's condition to be optimised, gives her the

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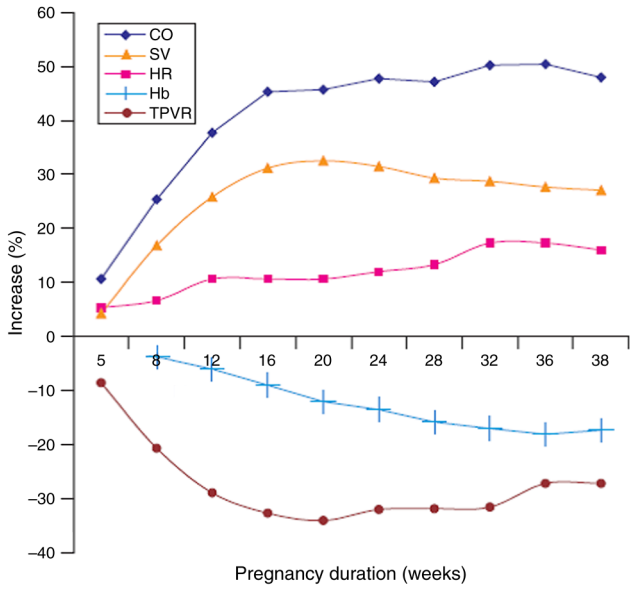

Figure 1 Plot showing the changes in cardiac output (CO), stroke volume (SV), total peripheral vascular resistance (TPVR), heart rate (HR) and haemoglobin concentration (Hb) during pregnancy. Reproduced from: Karamermer and Roos-Hesselink.[25]

opportunity to understand the risk of pregnancy and provides her antenatal care and delivery to take place in the most appropriate setting—for example, at a high-risk unit rather the nearest local unit for a woman with severe disease. Clearly, only those with known heart disease can have access to preconception care. This is usually case for women with bicuspid aortic stenosis, which tends to be picked up in childhood with an easily heard murmur. In contrast, those with undiagnosed rheumatic mitral stenosis usually only present for the first time in pregnancy when the haemodynamic changes cause them to decompensate. These women are dependent on maternity or emergency department staff, realising that their symptoms are due to heart disease rather than respiratory disease or simply pregnancy and on being referred early for expert cardiac assessment. It is salutary that the majority of women who die in pregnancy in the UK were not known to have heart disease before they became pregnant: delayed diagnosis is a risk factor for maternal death.[9]

Box 1 Modified WHO pregnancy risk scale

- WHO-1—pregnancy carries the same risk as the general population.
- WHO-2—pregnancy carries a small increased risk of morbidity and mortality.
- WHO-3—pregnancy carries a significantly increased risk; expert obstetric and cardiology care are needed.
- WHO-4—pregnancy carries a very high risk of maternal morbidity and mortality and is therefore contraindicated. Termination should be discussed if pregnancy occurs and if pregnancy continues, care should be as for WHO-3.

Pre-pregnancy assessment should include a thorough assessment of symptoms, cardiac status, usually with echocardiography and functional status with cardiopulmonary exercise testing±exercise echocardiography. This information aids risk stratification,[7 10 11] and counselling.

Maternal drug therapy should also be reviewed and the balance of risk to the mother from stopping or changing each medication during pregnancy should be weighed against the risk to the fetus of continuing.

Antenatal care and delivery

Pregnant women with WHO-3 or WHO-4 risk need expert and individualised antenatal care. The signs and symptoms of heart disease in pregnancy are distinguishable from that of normal pregnancy with careful history taking and examination (table 2).

Delivery planning and anaesthetic review are important, as is a readiness to change plans as pregnancy progresses according to maternal, fetal and obstetric needs. In general, the mode of delivery that carries the least cardiovascular stress is term, spontaneous and vaginal, with adequate regional analgesia and a low threshold for instrumental assistance. However, women with severe valvular disease may need early delivery if there is evidence

Table 1 Pregnancy risk for women with isolated valve lesions, normal ventricular size and function and no other risk factors

WHO risk	Mitral	Aortic	Pulmonary	Tricuspid	Presence of other lesions or risk factors
WHO 1	Trivial MR	Trivial AR	Mild PS, PR	Mild TR	RISK INCREASES
WHO 2	Mild MS, MR	Mild AS, AR	Moderate PS, PR	Mild TS, moderate TR (both rare in isolation)	
WHO 2–3	Moderate MR	Moderate AR	Severe PR		
WHO 3	Severe MR, moderate MS	Severe AR, moderate to severe AS	Severe PS	Severe TR and TS (but very rare in isolation—likely to be part of complex underlying disease)	
WHO 4	Severe MS	Severe and critical AS			

AR, aortic regurgitation; AS, aortic stenosis; MR, mitral regurgitation; MS, mitral stenosis; PA, pulmonary artery; PR, pulmonary regurgitation; PS, pulmonary stenosis; TR, tricuspid regurgitation; TS, tricuspid stenosis.

Table 2 Signs and symptoms in normal pregnancy and pregnancy with cardiac compromise

Signs and symptoms that may occur in normal pregnancy	Signs and symptoms suggesting cardiac decompensation during pregnancy
Breathlessness on exertion	Extreme breathlessness eg minor exertion, talking and eating
Difficulty sleeping due to discomfort	Orthopnoea and paroxysmal nocturnal dyspnoea
Sinus tachycardia <100 bpm (10–20 bpm higher than prepregnancy)	Sinus tachycardia persistently >100 bpm
Chest discomfort due to reflux	Exertional, tearing or pleuritic chest pain
Vasovagal syncope, postural hypotension	Exertional or palpitation related syncope
Palpitation due to atrial and ventricular ectopics	Sustained tachyarrhythmia
Jugular venous pulse visible+2 cm	Jugular venous pressure raised >2 cm
3rd heart sound	4th heart sound
Mild peripheral oedema	Marked peripheral oedema

of maternal cardiac decompensation, in which case a caesarean section is likely to be necessary. Care to avoid the haemodynamic stress of peripartum haemorrhage is also important—in most cases, routine syntocinon and ergometrine should be given during the third stage of labour. The decision to use syntocinon and ergometrine and the manner in which it is given should be individualised. Ergometrine is a vasoconstrictor and may cause hypertension; in general, it should be avoided in women with severe valvar stenosis, ventricular dysfunction or hypertension. Syntocinon is a vasodilator and may cause transient hypotension and tachycardia. These effects are ameliorated by the use of a continuous infusion rather than a bolus dose; nonetheless, caution is required for women with severe valvar stenosis. It should be remembered however that postpartum haemorrhage is an important cause of maternal mortality and is particularly poorly tolerated by women with significant heart disease. Care by obstetricians, cardiologists and anaesthetists with expertise in the management of women with heart disease is needed; the balance of risk is usually in favour of careful use of uterotonics in the active management of the third stage of labour.

Valvular regurgitation

Isolated mitral or aortic regurgitation

Mitral and aortic regurgitation are generally well tolerated in pregnancy. However, it should be remembered that risks are additive, so ventricular dysfunction or the presence of additional valve lesions will increase the risk. Broadly speaking, as long as ventricular size and function are normal, trivial mitral or aortic regurgitation can be classified as WHO-1, mild regurgitation as WHO-2, moderate as WHO-2–3 and severe regurgitation as WHO-3.

Care should be taken to exclude coexisting conditions that would add to the risk of pregnancy or require pre-pregnancy treatment. In those with aortic valve disease (usually bicuspid), an aortopathy or aortic coarctation should be excluded.

The decision as to whether to repair or replace a severely regurgitant mitral or aortic valve prior to embarking on a pregnancy is complex. If the ventricle is dilating, or the function has begun to deteriorate, then valve surgery is indicated. Similarly, if a mitral valve is considered likely to be repairable, ventricular function is good and the criteria for intervention[12] have been reached, pre-pregnancy surgery should be considered: a subsequent pregnancy with a competent native valve and good ventricular function is likely to be low risk (WHO-2).

However, if the valve needs to be replaced and the patient is asymptomatic with severe mitral or aortic regurgitation and good ventricular function, the decision is more complex and needs to be individualised. The pregnancy and lifetime risk of three possible paths need to be considered (box 2):

If the ventricle is severely impaired and the patient is symptomatic such that she is considered too high risk for mitral or aortic valve surgery, then pregnancy is also likely to be high risk, WHO-3 or WHO-4.

Antenatal review in pregnancy should focus on symptoms of breathlessness or palpitation and signs of decompensation such as tachycardia, atrial

Box 2 Timing of pregnancy versus valve replacement: pregnancy and lifetime risk

(i) Pregnancy without intervention with severe mitral or aortic regurgitation. Will pregnancy be well tolerated? Will pregnancy have a long-term deleterious effect on ventricular function?

(ii) Pregnancy after tissue mitral or aortic valve replacement. Pregnancy with a competent tissue valve will be low risk, but the risks of pre-pregnancy cardiac surgery and that of inevitable further cardiac surgery within 5–10 years need to be taken into account.

(iii) Pregnancy after mechanical valve replacement. Pregnancy will still be high risk (WHO-3) because of anticoagulation. The risk of pre-pregnancy surgery and lifelong anticoagulation needs to be considered, but the chance of needing further redo mitral or aortic valve surgery is low.

fibrillation, elevated venous pressure, a third heart sound and basal crepitations. Echocardiography should be performed in each trimester in women with moderate or severe regurgitation.

Diuretics may be helpful if the woman is breathless. The onset of other signs and symptoms suggest significant decompensation and although medical therapy (including anticoagulation with low-molecular-weight heparin if arrhythmia occurs) may alleviate symptoms, intervention such as early delivery may be necessary. Emergency cardiac surgery for isolated mitral or aortic regurgitation should only very rarely be needed during pregnancy: most women should be able to deliver—even if prematurely—before surgery.

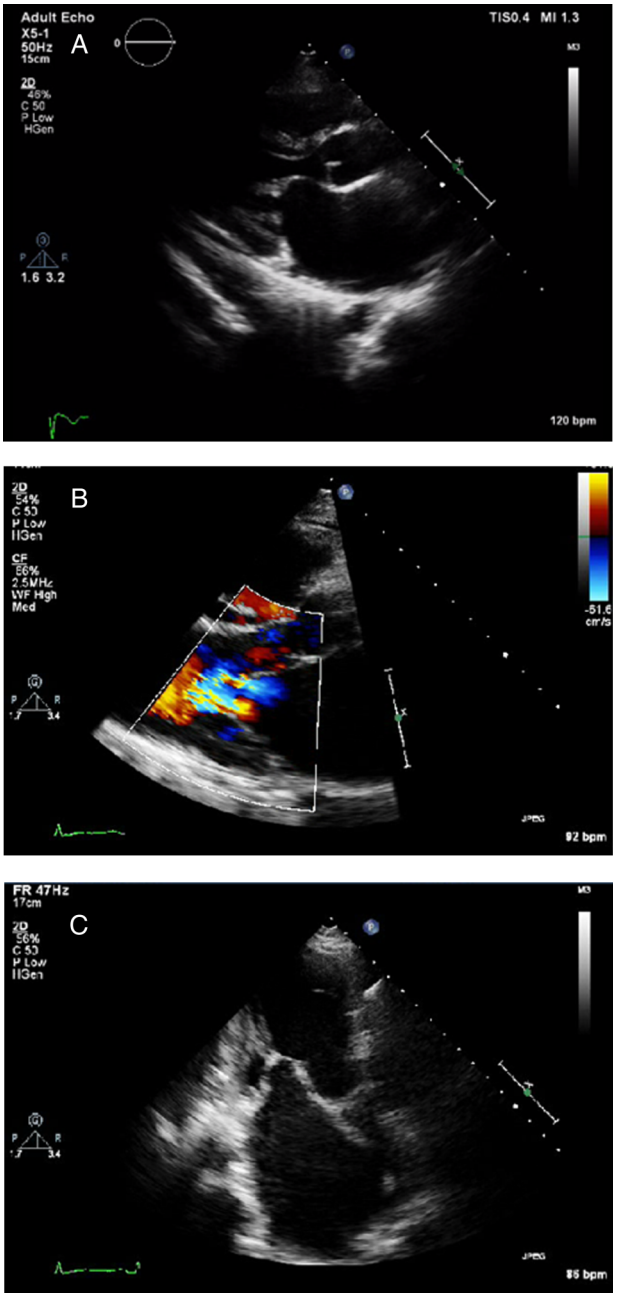

Figure 2 Two-dimensional echocardiographic images showing parachute mitral valve. Parasternal long axis (A and B) and apical two chamber (C) views are shown. There is a single papillary muscle. The leaflet tips are thickened with no retraction. The stenotic valve orifice is at the tips of the leaflets as they connect to the single papillary muscle; thus, the orifice is displaced into the body of the left ventricle.

Severe pulmonary regurgitation

This is usually long-standing post-pulmonary valvotomy or repair of tetralogy of Fallot in childhood. Some reports suggest a high complication rate[13] but this is not general experience.[14] Asymptomatic women with good right ventricular function and isolated severe pulmonary regurgitation are likely to tolerate pregnancy well (WHO-2). The question as to whether the extra volume load of pregnancy on the already volume-loaded right ventricle might have a long-term detrimental effect on its function remains unanswered. In general, the pulmonary valve should only be replaced pre-pregnancy if valve replacement criteria are met.[15]

Women should be followed during pregnancy; diuretics and rest may be helpful if there is breathlessness, peripheral oedema and rise in jugular venous pressure. Echocardiography should be performed in each trimester: if the right ventricle dilates or function worsens, early (usually 36–37/40) delivery may need to be considered.

Severe tricuspid regurgitation

If tricuspid regurgitation is due to primary valve pathology (eg, dysplastic valve or Ebstein's anomaly), then pregnancy is likely to be well tolerated provided the woman is asymptomatic and right ventricular function is maintained (WHO-2). Management is similar for women with pulmonary regurgitation.

However, if tricuspid regurgitation is secondary to dilation of the annulus, there may be important underlying disease affecting the right ventricle and pregnancy may be high risk (WHO-3 or WHO-4). Careful pre-pregnancy assessment and management of the underlying condition are needed.

Valvular stenosis

Mitral stenosis

The pathology of mitral stenosis is usually rheumatic, even in the developed world. The increased stroke volume and tachycardia of pregnancy combine with impaired diastolic flow through the stenotic valve to increase left atrial and pulmonary venous pressure and reduce cardiac output. If atrial fibrillation develops, the loss of atrial transport and the increase in heart rate can precipitate pulmonary oedema and cardiovascular collapse. Right ventricular failure may develop as a result of pulmonary venous hypertension. Delivery and the immediate postpartum period is particularly dangerous, with the rapid fluid shifts and tachycardia precipitating pulmonary oedema and a low output state.

Severe mitral stenosis with a valve area of $<1\ cm^2$ is predictably poorly tolerated (WHO-4)—women should be expected to decompensate and intervention should be planned as soon as the diagnosis is made.

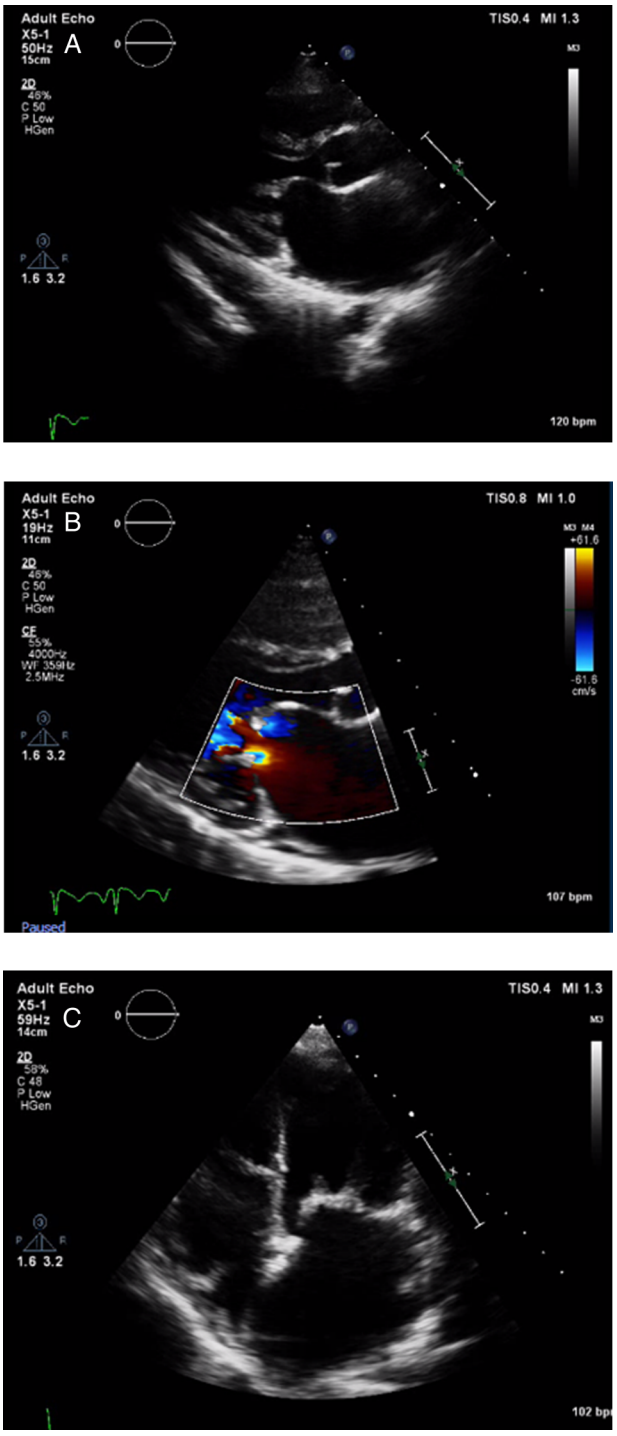

Figure 3 Two-dimensional echocardiographic images showing rheumatic mitral valve. Parasternal long axis (A and B) and apical four chamber (C) views are shown. There are two papillary muscles. The leaflet tips are thickened and the posterior leaflet is retracted giving a typical rheumatic appearance. The stenotic valve orifice is at the tips of the leaflets and in contrast to the parachute valve, it is not displaced into the left ventricular cavity.

Most women present in the second trimester as cardiac output nears its peak, either with exertional breathlessness, orthopnoea and paroxysmal nocturnal dyspnoea or with frank pulmonary oedema. The mainstay of medical treatment is oxygen and diuretics (furosemide) in the acute phase and β-blockade to reduce heart rate and allow time for ventricular filling once pulmonary oedema is cleared. The rheumatic mitral stenotic valve in a woman of childbearing age is usually suitable for balloon dilatation. If severe mitral stenosis is confirmed, plans should be made to proceed with percutaneous valvuloplasty before inevitable decompensation despite medical therapy, to allow a safe delivery.

The timing of intervention depends on the mother's clinical state and the likelihood of later decompensation, but gestational stage should also be taken into consideration. At 19/40, the fetus is beyond the most radiation-sensitive first trimester and the uterus is still below the level of the umbilicus so radiation exposure is less than later in pregnancy. If the procedure is done after 20 weeks gestation, an obstetrician must be present to empty the uterus in the unlikely event of cardiac arrest, in order to allow effective maternal resuscitation. If mitral valvuloplasty is done after 24 weeks gestation, a full obstetric and neonatal team must be present, since maternal cardiac decompensation or arrest would necessitate the emergency caesarean section delivery of a viable neonate.

The procedure must be done by an operator experienced in percutaneous mitral valvuloplasty—if necessary, referring the woman to another centre, since, in the UK, few cardiologists have extensive experience. Radiation dose should be kept to a minimum and a pelvic wedge placed under the right hip to shift the gravid uterus off the inferior caval vein and maintain cardiac output. The operator should be cautious—residual moderate stenosis is likely to be better tolerated for the remainder of pregnancy and delivery than acute severe mitral regurgitation.

Although even in the developed world, rheumatic disease is the most common cause of mitral stenosis presenting in pregnancy, rare congenital anomalies such as parachute mitral valve also cause mitral stenosis and may present in pregnancy. It is important to determine the underlying cause of mitral stenosis, since parachute mitral valve is not amenable to balloon valvuloplasty (figures 2 and 3). In addition, it often exists as part of Shone's syndrome: serial left-sided obstruction (supramitral membrane, subaortic ridge, bicuspid valve, aortic coarctation or interruption) and carries a high recurrence rate (10%) with hypoplastic left heart syndrome at the extreme end of its spectrum.

Surgical intervention during pregnancy, or after a termination, may be necessary for severe mitral stenosis that is not amenable to balloon mitral valvuloplasty, as is the case for parachute valve and mixed mitral stenosis and regurgitation. Cardiac surgery during pregnancy is discussed below.

Aortic stenosis

In contrast to mitral stenosis, valvular aortic stenosis in women of childbearing age is usually due to previously diagnosed bicuspid aortic valve disease, so the opportunity for pre-pregnancy counselling should be taken. If rheumatic aortic stenosis is

present, there is also likely to be significant mitral stenosis.

Unlike mitral stenosis, severe aortic stenosis in a previously asymptomatic patient is often well tolerated. However, the pressure gradient across the stenotic aortic valve rises during pregnancy due to the increased circulating volume and stroke volume of pregnancy that result in an increase in left ventricular pressure. There may therefore be a failure of coronary blood flow to increase with consequent compromise to myocardial perfusion. As a result, there is a risk that pregnant women with severe aortic stenosis may develop symptoms for the first time: breathlessness, angina, syncope, pulmonary oedema, left ventricular failure and sudden death. In the past, the risk of maternal death in severe aortic stenosis has been considered as high as 17%,[16] but more recent studies have shown low or no maternal mortality.[17] Nonetheless, women who are mildly symptomatic with severe aortic stenosis before pregnancy are likely to need intervention afterwards.[18]

The majority of adults with severe bicuspid aortic stenosis will eventually require valve replacement—balloon valvotomy or surgical repair is rarely satisfactory. Thus, a careful assessment is important to distinguish those who need valve replacement before pregnancy from those who are able to complete a pregnancy safely without intervention.

The patient with moderate-to-severe aortic stenosis is likely to tolerate pregnancy well, with specialist care (WHO-3) if (box 3).

Exercise echocardiography is particularly helpful in refining risk stratification in truly asymptomatic women with severe aortic stenosis: those who meet criteria for surgery[19] should delay pregnancy until after surgery Pregnancy is contraindicated (WHO 4) and pre-pregnancy surgery is indicated, for women who have[5] (box 4).

Women with significant aortic stenosis need close expert antenatal follow-up, with particular attention to the development of symptoms such as exertional chest pain, light-headedness or syncope that indicate decompensation. Echocardiography and ECG should be performed every 4–6 weeks to ensure that left ventricular function is maintained, that pulmonary arterial hypertension is not developing and that there are no new ischaemic changes. The velocity and Doppler-derived gradient across the stenotic valve will increase as pregnancy progresses, due to the increased stroke volume—the increase is not a sign of worsening aortic stenosis. Indeed, a failure of the aortic valve velocity to increase, or a fall in velocity, indicate that cardiac output is unable to rise and suggests heart failure and imminent decompensation.

Should the patient with severe aortic stenosis deteriorate during pregnancy, medical management includes bedrest with oxygen therapy, diuretics and, if there is no left ventricular failure or pulmonary

Box 3 Pregnancy risk in severe aortic stenosis

- The patient is asymptomatic pre-pregnancy
- ECG shows no left ventricular hypertrophy strain pattern
- Pre-pregnancy echocardiographic findings show
  - normal left ventricular function
  - aortic valve area ≥1 cm^2
  - Maximum velocity <4.5 m/s with mean <50 mm Hg and peak <80 mm Hg Doppler-derived gradients
- Exercise stress shows
  - normal exercise capacity with no symptoms
  - normal blood pressure response to exercise (surrogate for cardiac output)
  - no ST segment ECG changes
- Exercise echo shows
  - increase in mean aortic valve gradient of <18 mm Hg
  - increase in left ventricular ejection fraction (indicating contractile reserve)
  - no exercise-induced systolic pulmonary artery (PA) hypertension (PA pressure <60 mm Hg)

Box 4 Exercise echo for risk stratification in severe aortic stenosis to determine the need for pre-pregnancy surgery

- Symptoms at rest or on exercise
- Left ventricular dysfunction (eg, ejection fraction <50%) at rest
- No improvement in left ventricular ejection fraction during exercise
- ECG changes at rest or during exercise
- A failure to increase, or a fall in blood pressure during exercise
- An exercise induced rise in mean aortic valve gradient of ≥20 mm Hg
- Exercise induced systolic pulmonary artery hypertension ≥60 mm Hg
- Critical aortic stenosis (valve area <0.75 cm^2), regardless of symptoms or other parameters, since decompensation during pregnancy is almost inevitable.

Box 5 Treatment options when decompensation due to severe aortic stenosis occurs during pregnancy

- Ending the pregnancy: either as termination or delivery, depending on gestation
- Palliative balloon valvotomy if the valve is amenable (not likely in an adult)
- Surgical aortic valve replacement

oedema, the cautious use of β-blockade. However, medical therapy alone is unlikely to be sufficient and if intervention is necessary the options are as provided in box 5.

In comparison with mitral stenosis, there are little data on balloon aortic valvotomy in pregnancy.[20] If the valve has clear commissures, is not thickened or calcified and is not regurgitant, cautious palliative balloon valvuloplasty may provide a small increase in valve area, sufficient to allow the pregnancy to progress to a gestation at which the woman can be delivered more safely. The most experienced interventionist (usually a paediatric cardiologist for this procedure) and cardiac pregnancy teams should be involved, with cardiac surgical standby. If moderate or severe aortic regurgitation develops, emergency aortic valve replacement is likely to be needed.

Cardiopulmonary bypass carries increased maternal risks during pregnancy, with a mortality of up to 15%. The hormonal effects of pregnancy increasing tissue elasticity contribute to the risks of maternal morbidity. There is a risk of fetal demise of approximately 30%,[21] with hypothermic non-pulsatile flow during bypass and heparin-related placental bleeding being significant factors. Some data suggest that near-normothermia and some pulsatility may be beneficial.[22] The risks to the mother and fetus of cardiac surgery before delivery, or delivery before cardiac surgery need to be carefully considered and individualised, and if aortic valve surgery is performed before delivery, the most experienced surgical team should be involved.

Delivery in women with moderate or severe aortic stenosis may need to be by caesarean section, because of maternal or fetal decompensation. However, if the mother has tolerated pregnancy well, the lowest risk mode of delivery is a term vaginal delivery in a high-risk centre with slow, incremental epidural analgesia and invasive monitoring and observation on labour ward high-dependency unit for 48 h postpartum.[23 24]

Pulmonary and tricuspid stenosis

Provided right ventricular function is good, pregnancy in an asymptomatic woman with isolated severe pulmonary stenosis may be well tolerated (WHO-3), but close monitoring is needed. If there is severe stenosis, any symptoms or concern about right ventricular function, and the valve is thin and doming and amenable to balloon valvotomy, consideration should be given to performing the procedure in the second trimester. If however, the valve is unsuitable for a percutaneous approach, the patient should be closely monitored and management individualised.

Isolated tricuspid stenosis is very rare in pregnancy and more likely to form part of more complex underlying disease. Management should be individualised in a specialist high-risk pregnancy centre.

SUMMARY AND CONCLUSION

Pregnancy in a woman with a regurgitant valve lesion usually carries a lower risk than one with a stenotic valve. Although severe aortic stenosis is often better tolerated than severe mitral stenosis, the aortic valve is less likely to be amenable to percutaneous intervention, so decompensation during pregnancy is more difficult to manage, since surgical intervention may be required.

Native valve disease is common in women of childbearing age and is a major cause of preventable maternal morbidity and mortality worldwide. Rheumatic mitral stenosis accounts for the majority of cardiac maternal deaths in the developing world where poor socioeconomic conditions are associated with continued prevalence of rheumatic fever.

Expert pre-conception counselling reduces risk for women already known to have heart disease. Similarly, expert, individualised cardiac and obstetric antenatal and peripartum care reduces the risk of avoidable major morbidity and mortality. For those with undiagnosed disease, vigilance is needed

Key messages

- Valvar stenosis is less well tolerated in pregnancy than regurgitation.
- Cardiac output rises by 50% during pregnancy, with the majority of the increase before the end of the second trimester. Women with cardiac lesions who are unable to meet this demand are at high risk during pregnancy.
- Rheumatic mitral stenosis is the leading cardiac cause of maternal death in the developing world. The initial presentation of many women with rheumatic mitral stenosis occurs when pregnancy precipitates symptoms for the first time.
- Diuretics and β-blockers are useful in the medical management of mitral stenosis, but balloon mitral valvuloplasty has a high success rate and is the mainstay of treatment for severe mitral stenosis in pregnancy.
- Preconception assessment is important to determine whether a woman with moderate or severe aortic stenosis is likely to tolerate pregnancy well, or whether she should undergo pre-pregnancy aortic valve replacement.
- The severely stenotic bicuspid aortic valve in adulthood is often unsuitable for balloon valvotomy, so surgical aortic valve replacement may be necessary if a woman with severe aortic stenosis decompensates during pregnancy.

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to recognise the cardiac nature of their symptoms as they start to decompensate during pregnancy and to act appropriately.

Competing interests None declared.

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