ANAESTHESIA FOR THE PREGNANT PATIENT WITH ACQUIRED VALVULAR HEART DISEASE

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Valvular heart disease in pregnancy poses additional risk to both mother and fetus.[1] Although there is an ever-decreasing prevalence of rheumatic heart disease in developed nations, it is still occasionally encountered. In the developing world it remains a significant problem.[2]

Management in pregnancy should be multi-disciplinary. Obstetric, cardiology and anaesthetic opinions should all be sought. The following need to be addressed: accurate diagnosis as to which valves are involved, assessment of the severity of the lesion, degree of impairment resulting from the lesion and evaluation of concomitant therapy. In addition, as well as optimising management during pregnancy and labour, it is important that care be carried on into the puerperium. The majority of reported deaths in cases of valvular heart disease in pregnancy occur in the post-partum period.[3] Intensive monitoring should be continued for at least 72 hours after delivery, preferably in a high care or intensive care environment.[4]

There are few evidence-based recommendations in the literature regarding the management of acquired valvular heart disease in pregnancy. Most recommendations are derived from case reports and observational studies. This article reviews current data and discusses important aspects of the anaesthetic management of valvular heart disease in pregnancy.

Cardiovascular physiology in pregnancy

Pregnancy stresses the cardiovascular system. Wide fluctuations in haemodynamic stress can be anticipated during labour and delivery. Patients with stenotic valvular lesions are particularly prone to complications at delivery, and the anaesthesiologist should be familiar with anticipated difficulties and their management. Patients may require invasive cardiac monitoring during labour, particularly where an operative delivery is anticipated.

Although patients may present with previously diagnosed valvular disease, cardiac compromise frequently only becomes apparent during pregnancy. This is largely due to the fact that normal pregnancy is associated with a 30 to 50 percent increase in blood volume and corresponding increases in cardiac output. Stroke volume normally increases by 25 to 30 percent, with the remaining increase in cardiac output being accounted for by

changes in heart rate.[5] Not surprisingly, where valvular heat disease limits these changes, cardiac compromise with pulmonary oedema or bi-ventricular failure may present early in pregnancy.

Early signs of cardiac compromise may become apparent in the first trimester and peak at 20 to 24 weeks of pregnancy when cardiac output reaches a maximum. From 24 weeks onward cardiac output is maintained at high levels. Cardiac output only begins to decline in the post-partum period.

During labour, the sympathetic response to pain, as well as uterine contractions, induce profound fluctuations in the patient's haemodynamic status. Between 300 and 500 ml of blood is injected into the general circulation with each contraction. Stroke volume rises by an estimated additional 50 percent. At the same time, systemic vascular resistance is increased, exacerbating the additional stress placed on the cardiovascular system. At delivery a predicted blood loss of between 400 and 800 ml does little to maintain stability in an already compromised circulatory system.

Many normal women manifest subtle signs of cardiac failure during uncomplicated pregnancy and delivery. Dyspnoea and fatigue are common, together with a reduction in exercise capacity. A large proportion of pregnant patients have peripheral oedema together with distension of the central veins and many have audible flow murmurs and a third heart sound indicative of volume overload.

Where underlying valvular disease is present it is hardly surprising that symptoms and signs of cardiac failure may occur during pregnancy or at the onset of labour.

Following delivery the cardiovascular status of the patient will normalise at 6 to 8 weeks post delivery.[6]

Identifying cardiac disease in pregnancy

The anaesthesiologist should be able to identify cardiac disease in pregnancy and labour if appropriate management decisions are to be made. The presence of the following physical signs should always be regarded as abnormal in pregnancy and alert attending physicians to the potential presence of underlying cardiac disease:

- A loud fourth heart sound

- Any diastolic murmur

- A grade 3/6 or more systolic murmur

- Fixed splitting of the second heart sound

- An opening snap

The presence of one or more of these signs indicates the need for echocardiographic evaluation of the heart.[7] Echocardiography has the ability not only to diagnose specific cardiac disease, but also to quantify the severity of cardiac lesions observed. This information is invaluable in both planning anaesthesia and anticipating complications.

Assessing risk in pregnant patients with cardiac disease

The New York Heart Association functional class has been used to identify patients at high risk of complication in pregnancy. A

New York Heart Association functional class of III or IV has been estimated to carry a greater than 7 percent risk of mortality and a 30 percent risk of morbidity. Although women in these functional classes should be counselled against childbearing, it is not infrequent that they are encountered in the prenatal clinic (or even on the labour ward, or at the theatre door!).

New York Heart Association (NYHA) Classification
A functional classification of physical activity for acrdiac patients

- *Class I*: patients with no limitations of activities; they suffer no symptoms from ordinary activities

- *Class II*: patients with slight, mild limitation of activity; they are comfortable with rest or mild exertion

- *Class III*: patients with marked limitation of activity; they are comfortable only at rest

- *Class IV*: patients who should be at complete rest, confined to bed or chair; any physcial activity brings on discomfort and symptoms occur at rest

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Following a study of 252 completed pregnancies in patients with cardiac disease, five risk factors were identified as being predictive of poor maternal and or neonatal outcome.[8] These were:

1. Prior cardiac events (heart failure, transient ischaemic attack or stroke)

2. Prior arrhythmias (symptomatic brady- or tachy-arrhythmia requiring therapy)

3. New York functional > class II or the presence of cyanosis.

4. Valvular or outflow tract obstruction (aortic valve area of less than 1.5 cm^2 or mitral valve area of less than 2 cm^2. Left ventricular outflow tract pressure gradient of more than 30mmHg)

5. Myocardial dysfunction (left ventricular ejection fraction of less than 40 percent, or a restrictive or hypertrophic cardiomyopathy)

A subsequently revised risk index identified four factors as being predictive of poor maternal and fetal outcome: [9]

1. Prior cardiac event

2. Poor NYHA functional class or cyanosis

3. Left heart obstruction

4. Systemic ventricular dysfunction

Valvular heart lesions and risk during pregnancy

During pregnancy, valvular heart lesions may carry risk for both mother and fetus. Complications ascribed to valvular heart disease include: increased incidence of maternal cardiac failure and mortality, increased risk of premature delivery, lower APGAR scores and lower birth weight. In addition there is a higher incidence of interventional and assisted deliveries.[10] The American Heart Association has classified cardiac lesions according to their associated risk. This is shown in the table

Classification of valvular heart lesions according to maternal, fetal and neonatal risk			
Low maternal and fetal risk	**High maternal and fetal risk**	**High maternal risk**	**High neonatal risk**
Asymptomatic aortic stenosis low mean outflow gradient (<50 mmHg) with normal left ventricular function	Severe aortic stenosis with or without symptoms	Reduced left ventricular systolic function (LVEF<40%)	Maternal age <20yr or >35 yr
Aortic regurgitation of NYHA class I or II with normal left ventricular systolic function	Aortic regurgitation with NYHA class III or IV symptoms	Previous heart failure	Use of anticoagulant therapy throughout pregnancy
Mitral regurgitation of NYHA class I or II with normal left ventricular systolic function	Mitral regurgitation with NYHA class III or IV symptoms	Previous stroke or transient ischaemic attack	Smoking during pregnancy
Mild to moderate mitral stenosis (valve area > 1.5 cm^2, gradient < 5mmHg) without severe pulmonary hypertension	Mitral stenosis with NYHA class II, III, or IV symptoms		Multiple gestations
Mitral valve prolapse with no mitral regurgitation or with mild to moderate mitral regurgitation and with normal left ventricular systolic function	Aortic valve disease, mitral valve disease, or both, resulting in severe pulmonary hypertension (pulmonary pressure > 75% of systemic pressures)		
Mild to moderate pulmonary valve stenosis	Aortic valve disease, mitral valve disease, or both, with left ventricular systolic dysfunction (EF < 40%) Maternal cyanosis Reduced functional class status (NYHA class III or IV)		

Table 1: Modified from Reimold and Rutherford[11]

below. [11] It is important for the anaesthesiologist to be aware of the attendant risk that a patient suffers as a result of valvular heart disease. Those patients carrying the highest risk warrant additional care, invasive haemodynamic monitoring and appropriate modification of anaesthetic technique.

In the absence of echocardiography, where more than one valvular lesion co-exists, the anaesthesiologist must attempt to identify the most clinically significant problem.

Mitral stenosis

Mitral stenosis is a commonly encountered lesion. It is associated with a maternal mortality of 10 percent. This increases to more than 50 percent in patients in NYHA functional class III and IV. Should concomitant atrial fibrillation be present, the risk of maternal mortality rises by between 5 and 10 percent.

The increasing physiologic demands of pregnancy are poorly tolerated; a progressively larger pressure gradient between left atrium and left ventricle develops. Pulmonary oedema, pulmonary hypertension and consequent right ventricular failure may all occur. At delivery tachycardia, pain, anxiety and anaemia dramatically increase the risk of acute pulmonary oedema and cardiovascular compromise.

In a small percentage of patients with mitral stenosis, pulmonary vascular resistance is greatly elevated, resulting in severe pulmonary hypertension, right heart failure, and low cardiac output. These patients are at high risk, and termination of pregnancy for the health and survival of the mother may require consideration.

Pain-mediated tachycardia increases flow across the mitral valve and may precipitate acute pulmonary oedema. The careful

provision of epidural anaesthesia by a skilled operator is therefore desirable for vaginal delivery (unless contraindicated for obstetric reasons). Epidural anaesthesia should be performed using small increments of local anaesthetic to achieve an adequate level (T8-T10). These patients are very sensitive to changes in preload and afterload; fluid and vasopressor therapy must be very carefully titrated against blood pressure. In severe cases (NYHA class III and IV), this is particularly important. In many cases, elective caesarean section under general anaesthesia may be the best option. In patients with advanced disease, invasive arterial monitoring is advisable. Small boluses of phenylephrine (50 mcg) are effective in avoiding precipitous hypotension. Small-dose, single shot spinal anaesthesia (e.g.1mL 0.25% bupivacaine with 10-20mcg fentanyl or 0.1-0.2mg morphine) may be used as an alternative to epidural anaesthesia in less experienced hands. This provides 2-4 hours of analgesia for labour. Combined spinal-epidural anaesthesia may have a role in the hands of experienced anaesthesiologists.

Pulmonary artery catheterisation has been advocated in patients with severe mitral stenosis, or mild-to-moderate stenosis with severe symptoms.[12] Fluid restriction, the use of diuretics and supplemental oxygen may all be of benefit.

The literature reports a case in which variation of the Trendelenburg position was used to maintain a capillary wedge pressure of 25mmHg during operative delivery. A successful outcome for both mother and foetus was reported.[13]

Atrial fibrillation must be treated promptly should it occur. Cardioversion, beta-blockers and digoxin have all been used to treat recent-onset (<24 hours) atrial fibrillation. Rate control is an important objective in attempting to normalise haemodynamics by allowing adequate diastolic filling of the ventricle.[14]

Caesarean delivery requires a block to a level of T4 (for light touch). Spinal anaesthesia is thus best avoided. Careful epidural anaesthesia in experienced hands may be considered in class 1 and 2 patients, but NYHA class 3 and 4 patients are often better managed under general anaesthesia. Specific pharmacotherapy must be employed to obtund the intubation response. Bolus oxytocin is contraindicated in view of the risk of precipitous systemic hypotension and pulmonary hypertension. A brief period of postoperative ventilation may be required in some cases.

In areas where the incidence of pre-eclampsia is high and valvular heart disease is prevalent, any patient developing pulmonary oedema should have mitral valve disease excluded as a contributing cause (as this is a particularly dangerous combination).

Mitral regurgitation

Mitral regurgitation is usually tolerated well during pregnancy. The marked decrease in systemic vascular resistance that occurs during pregnancy alleviates the abnormal physiologic stress imposed by this lesion. Rarely, reactive pulmonary hypertension and severe right heart failure may ensue.

There are no specific recommendations for the management of mitral regurgitation during labour and delivery. Prior to labour symptoms may be managed with diuretics and vasodilators. During labour, regional anaesthesia is usually well tolerated. However, in complicated NYHA class 3-4 cases, general anaesthesia may be required.

Aortic stenosis

In general the symptoms of aortic stenosis are masked by progressive left ventricular hypertrophy and are thus easily missed. Overall, patients who were asymptomatic prior to pregnancy usually tolerate pregnancy relatively uneventfully.

Echocardiographic determination of valve area is the best guide to severity of aortic stenosis. The hyperdynamic circulation of pregnancy frequently leads to overestimation of the degree of stenosis.

These patients tolerate tachycardia, hypovolaemia and systemic vasodilatation poorly, since coronary perfusion is critically dependent upon maintaining aortic diastolic pressure. General anaesthesia and caesarean section, with the aid of invasive haemodynamic monitoring, appears to be the safest means of successful delivery. Aggressive maintenance of systemic blood pressure with vasopressors (e.g. phenylephrine), is paramount to the avoidance of severe hypotension, acute left ventricular failure and cardiac arrest.

Spinal anaesthesia is generally contraindicated in these patients. There are reports of the successful management of vaginal delivery under carefully introduced and limited epidural analgesia, but this should be restricted to very experienced hands.

Aortic regurgitation

Aortic regurgitation reduces both cardiac output and coronary blood flow. The principles of management are: a reduction in afterload (to improve forward flow) and maintenance of a relatively high heart rate (to reduce the regurgitant fraction). In patients with aortic regurgitation there is reduced coronary flow in diastole; coronary flow has been documented to reverse in patients with severe aortic regurgitation. It is thus important to maintain systolic blood pressure within 15% of baseline levels in these patients. Many patients with aortic regurgitation improve symptomatically during pregnancy. During labour, epidural analgesia improves forward flow, and is therefore the anaesthetic of choice in patient's requiring an operative delivery.

Pulmonary stenosis

Pulmonary stenosis increases right ventricular work and can dramatically impair left ventricular output (due to reductions in forward flow). It is important to maintain preload and optimise ventricular contractility, whilst bearing in mind that excess fluid may precipitate acute right heart failure. Atrial fibrillation is also a potential complication of fluid overload.

The goals of haemodynamic management include maintenance of right ventricular preload, left ventricular afterload and right ventricular contractility. In general hypothermia, hypercarbia, acidosis, hypoxia and high ventilatory pressures should be avoided. Aorto-caval compression may result in profound hypotension as a result of acute reductions in right ventricular

preload. Most reports recommend vaginal delivery under epidural anaesthesia, as operative delivery is associated with increased maternal mortality.[8]

Spinal anaesthesia may be associated with an uncontrolled reduction in right ventricular preload and should therefore be avoided in severe cases.

Managing valvular heart disease in pregnancy

Although most pregnant patients with valvular heart disease may be managed medically during pregnancy, it is occasionally necessary to consider valve replacement. This may become a necessity in the patient with severe valvular disease (particularly stenosis), where termination of pregnancy is not considered to be an option. Severe symptomatic disease, threatening maternal or fetal well-being is an accepted indication for either balloon-valvuloplasty or valve replacement.[15]

When needed, valve replacement is best undertaken during the second trimester. Cardiopulmonary bypass and hypothermia carry substantial risk for the fetus. Fetal bradycardia and death are not uncommon.[15] Meticulous care should be given to the maintenance of blood pressure during bypass and fetal well-being should be monitored continuously with a cardiotocograph.

Bacterial endocarditis prophylaxis

Pregnancy carries no additional risk for bacterial endocarditis. The Committee on Rheumatic Fever, Endocarditis and Kawasaki Disease (American Heart Association), do not recommend routine antibiotic prophylaxis in patients with valvular heart disease undergoing uncomplicated vaginal delivery or caesarean section (unless infection is suspected). Antibiotic prophylaxis as practiced for the prevention of wound sepsis is more than adequate.

Antibiotics are optional for high-risk patients with prosthetic heart valves, a previous history of endocarditis, complex congenital heart disease or a surgically constructed systemic-pulmonary conduit.[16]

Pregnant patients with valvular heart disease should receive prophylactic antimicrobial therapy for invasive urinary tract or gastrointestinal procedures.[17] Intravenous ampicillin and gentamicin, or oral amoxycillin should be used in the non-penicillin allergic patient. Vancomycin and gentamicin are used in the patient with penicillin allergy.[16]

Anticoagulant therapy in patients with prosthetic valves

Although it is undeniably important to provide ongoing anticoagulation to the pregnant patient with a prosthetic valve, there is debate regarding the optimal agent. Unfortunately there are no randomised trials providing guidance in this area.[18] The use of warfarin and other coumadin derivatives carries a well-established risk of embryopathy, whilst the use of subcutaneous unfractionated heparin has been reported to be ineffective. Low molecular weight heparins have been considered as an alternative but data is limited to trials and reports of only 25 patients, with a treatment failure rate of 20%.[19] Data suggests that the low molecular weight heparins are neither safe, nor effective, in preventing thromboembolic complications in patients with prosthetic heart valves (whether pregnant or not).[20]

Traditional teaching is that patients should be anti-coagulated with heparin in the first trimester of pregnancy and then converted to warfarin for the remainder of the pregnancy. Warfarin should then be stopped just prior to delivery.

Fetal, but not maternal outcome has been reported to be better where bioprosthetic valves are used instead of mechanical prostheses (in both aortic and mitral positions).[21]

Conclusion

As a rule, regurgitant valvular lesions are far better tolerated in pregnancy than are stenotic lesions. Patients who are asymptomatic, or only have minimal symptoms before falling pregnant, tend to tolerate pregnancy well. Patients with severe symptomatic valvular heart disease should ideally be counselled against pregnancy. In the event of pregnancy, early consultation between obstetrician and anaesthesiologist allows for planning with regards to both the timing of delivery and optimal analgesia/anaesthesia.

The main principles of management with regard to valvular heart disease in pregnancy are as follows:

- Early identification of the disease, and the assessment of the severity of the lesion(s).

- The appreciation of the severity of risk incurred by both mother and fetus.

- High-risk lesions, for either mother or fetus, should be managed in a high care environment where invasive monitoring is possible, both pre- and post delivery.

- Regional anaesthesia techniques in labour are an attractive option, and may be employed with good outcomes in many patients.

- Carefully titrated epidural anaesthesia for labour is associated with less sympathetic blockade than spinal or epidural anaesthesia for caesarean delivery. Thus, the effective use of regional anaesthesia for labour does not necessarily predict that this method will be safe for caesarean delivery in severe cases.

- Severe mitral or aortic stenosis, or any valvular heart condition associated with pulmonary oedema or heart failure, are contraindications to regional anaesthesia, (except in rare circumstances).

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