Anesthesia and Valvular Heart Disease

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The anesthetic management of patients undergoing noncardiac surgery or for valve replacement/repair requires an understanding of the natural history and pathophysiology of the valve disease. The patient's physiologic adaptation to their valve disease can be acutely disturbed by perioperative events and anesthetic drugs. This summary will focus on disease of the mitral and aortic valves in the adult. The reader is referred to a standard textbook of cardiology and recent anesthesiology textbooks for detailed reviews of pathophysiology and anesthetic considerations.[1,2,3]

The ability of the left ventricle to generate the stroke volume (SV) depends on adequate filling or "preload," the contractile state of the muscle, and the impedance to ejection ("afterload"). Valvular lesions impose additional requirements for compensation. Thus, stenotic lesions require the heart to force an adequate volume through a small orifice; regurgitant lesions require the to heart eject a large volume because part of the ejected volume returns backwards. Compensation involves both the myocardium and the peripheral vasculature. Preoperative assessment in all patients with valve disease should include a recent (ie at least within 6 months) evaluation such as echocardiography, and a detailed assessment of symptom progression.

Aortic Stenosis (AS)

Aortic stenosis is the commonest of the major valve lesions. While rheumatic disease was historically the most important cause, this has been displaced by degeneration of congenital bicuspid disease. This latter abnormality occurs in 1-2% of the population. Elderly patients may also have significant "senile" degeneration of a normal (tricuspid) aortic valve. Both rheumatic disease and congenital bicuspid disease become hemodynamically significant over a period of decades, with patients presenting with symptoms and the need for valve replacement usually during or after the 5th decade.[4]

The gradual process of narrowing of the aortic orifice leads to concentric left ventricular hypertrophy and a reduction in left ventricular compliance – the myocardium becomes thick, the end-diastolic pressure (LVEDP) rises, but there is no dilatation. Typically this occurs as the valve area decreases over years from the normal 2.5 – 3.5 cm^2 to about 1 cm^2. The left ventricle (LV) generates very high systolic pressures to overcome the stenosis, but aortic pressures are normal. Because of the decreased compliance, LV filling during diastole depends on adequate preload as well as atrial contraction. While the latter contributes less than 20% of filling in the normal heart, it may contribute twice this amount in AS. This phase of AS is termed "mild" stenosis with physiologic compensation.

As the aortic valve area diminishes below 1 cm^2 down to 0.5 cm^2, the stenosis is termed "moderate." Patients begin to develop symptoms as the heart struggles to maintain flow through the narrowing lesion. The increased work of the heart in association with decreased compliance and increased LVEDP results in angina in the majority of patients. This occurs in the absence of coronary artery disease (CAD), although up to 50% of patients may have significant CAD.[1] The left ventricle begins to dilate, the atrium may develop fibrillation, and the patient begins to experience symptoms of pulmonary congestion or even syncope with any type of excitement or exertion. "Critical" stenosis is present if the valve area is less than 0.5 cm^2. The onset of angina is associated with an average survival of 5 years; heart failure or syncope are associated with less than 3 years survival.[5] Valve replacement is recommended when the valve area is less than 0.8 cm^2 or if there is ventricular dysfunction, ventricular ectopy or an inadequate blood pressure response to exercise.[6] Percutaneous balloon valvuloplasty is possible in selected patients.

Anesthetic considerations

Maintenance of adequate coronary flow to the hypertrophied myocardium in the face of an elevated LVEDP requires an adequate aortic blood pressure. Thus, the anesthetic goals are to maintain adequate preload and to avoid atrial dysrhythmias, to maintain contractility, and to avoid systemic hypotension.

Preoperative assessment must include a review of the patient's history and medical records to be sure the degree of stenosis, both clinically and objectively, is appreciated. This review may result in a referral for valve replacement or valvuloplasty. Because tachycardia is undesirable as it will interfere with adequate filling and increases myocardial oxygen demand, cautious premedication of the anxious patient is indicated. Induction agents, muscle relaxants, and potent vapors must be administered in such a way that the hemodynamic compensations are not lost. Spinal and epidural anesthesia should be used very cautiously due to the risks of hypotension. Invasive monitoring with an arterial catheter is probably indicated for most procedures; the use of a pulmonary artery catheter (PAC) or transesophageal echo (TEE) for monitoring LV preload may be desirable for major procedures where large fluid shifts are anticipated. Hypotension and dysrhythmias must be treated early and aggressively. Conversely, hypertension should be treated very cautiously.

Two reports from the Mayo Clinic document non-cardiac surgery performed in patients with moderate or severe aortic stenosis.[7,8] While the patient population, degree of stenosis, anesthetic techniques and procedures were varied, these reports are remarkable in that only two mortalities occurred in 66 patients. In all cases the degree of stenosis was understood before the procedure, and the anesthetic was planned accordingly, in some cases including spinal or epidural techniques.

Hypertrophic Cardiomyopathy ("HOCM")

While not a true valvular lesion, HOCM is a myocardial disease that causes left ventricular outflow obstruction (similar to aortic stenosis) and associated mitral

regurgitation. It may be unrecognized, associated with few symptoms or may cause heart failure, dysrhythmias or sudden death.[1] Various terminologies have been used for the syndrome, however the key pathophysiology is a hypertrophied interventricular septum which can lead to "functional" outflow tract obstruction if the ventricle is empty or hypercontractile. An associated abnormality of the anterior leaflet of the mitral valve causes it bulge into the outflow tract, contributing to the obstruction and resulting in mitral regurgitation. This abnormality is called systolic anterior motion (of the anterior mitral leaflet) or "SAM." Haering et al reviewed 77 patients with HOCM who underwent noncardiac surgery, noting that 40% had one or more adverse cardiovascular events (but no mortality).[9] An important feature of this syndrome is that outflow tract obstruction and mitral regurgitation are exacerbated by inotropic therapy and hypovolemia. Thus, the usual treatment for acute heart failure – diuresis, nitrates and inotropic agents – make the patient worse. If the patient is monitored with a PAC, the cardiac output will decrease with standard therapy while pulmonary artery occlusion pressure increases. Echocardiography is the only tool which can lead to the correct diagnosis and management, which includes volume administration, slowing the heart, and increasing the afterload.

Aortic Regurgitation (AR)

The commonest causes of AR in the adult are rheumatic fever, bacterial endocarditits, trauma, and aortic dissection. Congenital diseases such as Marfan syndrome and cystic medialnecrosis may also cause incompetence at least partly through ascending aortic dilatation or dilatation of the aortic "root". Especially with rheumatic disease there may be combined stenosis and regurgitation. A gradual increase in regurgitation occurs over decades, with the clinical presentation of shortness of breath in the 5th decade. Unlike AS, patients with severe regurgitant disease may live many years; the amount of regurgitation alone is not indicative of the stage of disease. The appearance of symptoms follows a rise in LVEDP and progressive LV dysfunction, which may not be reversible. Once shortness of breath begins to occur, mortality is common within 2-4 years.

The pathophysiology of AR is volume overload of the left ventricle, with dilatation and eccentric hypertrophy rather than the concentric hypertrophy seen with AS. The left ventricle generates a very large stroke volume, aided by peripheral dilatation, to make up for the backwards flow through the aortic valve during diastole. The heart rate is usually elevated as this reduces the proportion of time spent in diastole (when regurgitation occurs). With severe, compensated AR the regurgitant flow may exceed 20 L/min, giving a total cardiac output of 25 L/min.[1]

Anesthetic management

The anesthetic management of patients with AR is focused on maintaining forward rather than regurgitant cardiac output. Based on the pathophysiology this requires maintaining an adequate preload to assure filling of the hypertrophied, dilated LV, a high-normal heart rate to reduce the proportion of time spent in diastole, and low-normal systemic blood pressure to encourage forward rather than regurgitant flow.

Premedication is often associated with venodilation and a reduction in heart rate which may not be an advantage. Similarly, anesthetic induction and maintenance must be designed to avoid these changes. While reductions in contractility are undesirable in almost all valvular disease conditions, for AR the maintenance of adequate preload and reduced afterload are most important. When using vasodilator drugs this must be kept in mind: nitroglycerin may be of little or no benefit, while nitroprusside or other arterial dilators significantly improve forward flow and reduce LVEDP. Maximizing forward cardiac output with the correct balance of afterload reduction and preload augmentation is difficult to gauge without a PAC. The period of emergence which is associated with pain and hypertension is of particular concern, as this may be associated with an acute rise in LVEDP and pulmonary edema. Unlike AS where the reduction in blood pressure may be life threatening, epidural or spinal anesthesia may be a good choice for appropriate patients and surgeries in patients with compensated AR.

Acute AR

Acute onset of AR is usually secondary to diseases such as endocarditis, aortic dissection, or valve trauma. Without time to develop compensatory LV dilatation, acute AR is a hemodynamic "emergency," requiring valve replacement. There is an acute rise in LVEDP during diastole, sometimes even causing premature closure of the mitral valve as the LVEDP exceeds left atrial pressure (LAP). The sympathetic response of tachycardia and peripheral vasoconstriction is a two-edged sword, the vasoconstriction in particular causing a reduction in forward flow. Maintenance of tachycardia and avoidance of myocardial depression are anesthetic goals in this condition.

Mitral Stenosis (MS)

Mitral stenosis is usually secondary to rheumatic disease, and usually becomes functionally significant within 2 decades. Most patients are female, and more have combined stenosis and regurgitation (40%) than isolated stenosis (25%).[10] The following discussion refers to isolated stenosis.

The normal mitral valve area is 4-6 cm^2; it is not until the valve area decreases to below 1.5 cm^2 that symptoms other than mild dyspnea with exertion develop. As the valve area decreases to below 1 cm^2 ("critical" stenosis) symptoms occur at rest. As the valve area narrows, the pressure gradient across the valve must increase to maintain stroke volume. For example, with an area of 1 cm^2 a pressure gradient of 20 mm Hg corresponding to a left atrial pressure of at least 25 mm Hg, is required to generate a normal cardiac output at rest.[1] With exercise and the requirement for an increased cardiac output this gradient must increase. This inevitably leads to pulmonary congestion and symptoms of shortness of breath. Unlike aortic valve lesions, MS is a disease proximal to the LV: the compensation to MS is left atrial hypertension, resulting in left atrial dilatation, atrial fibrillation, and pulmonary congestion. Some patients in the later stages develop a ventricular cardiomyopathy.

Mitral stenosis is likely to become clinically apparent when there is a relatively acute rise in cardiac output such as occurs with pregnancy, or when there is a tachydysrhythmia. In the latter case, the duration of diastole is reduced which means there is less time for ventricular filling requiring an increased trans-mitral pressure gradient – leading to pulmonary congestion. This is very relevant to perioperative events which can often lead to tachycardia. As the valve area decreases and left atrial pressure increases, pulmonary hypertension and eventually right ventricular dysfunction can develop. Mitral valve replacement should generally be performed when patients are symptomatic and the valve area is less than approximately 1.5 cm^2. Percutaneous balloon valvuloplasy may be an alternative to surgery, and may be particularly valuable in the high-risk patient who needs an urgent noncardiac procedure.

Anesthetic management

Based on the pathophysiology, the goals of anesthetic management are to maintain cardiac output through the tight mitral valve while avoiding pulmonary congestion. The conflicting goals of maintaining an adequately high preload (ie, high left atrial pressure) while avoiding acute pulmonary congestion, pulmonary hypertension and right ventricular dysfunction, are difficult to achieve without careful monitoring using a PAC. That being said, the LVEDP will be significantly less than the pulmonary artery occlusion pressure (PAOP or "wedge"). Of particular concern is the avoidance of tachycardia. Patients in sinus rhythm who develop atrial fibrillation in the perioperative period should be cardioverted. Patients already in atrial fibrillation should have the rate controlled aggressively. The pulmonary vasculature is sensitive to hypoxemia or hypercarbia so meticulous attention should be paid to these values. While epidural and spinal anesthesia can be safely performed in patients with MS, adequate preload must be given.

Mitral regurgitation (MR)

Mitral regurgitation from a number of causes is a relatively common valvular abnormality.[1] Probably the most common cause is mitral valve prolapse; other relatively common causes include rheumatic disease (often combined with stenosis); myxomatous degeneration of the valve leaflets, annular degeneration, annular dilatation secondary to LV failure, ischemic papillary dysfunction, or ischemic papillary muscle/chordal rupture. Not surprisingly, this range of causes is associated with a wide range in clinical course. The nature and severity of symptoms is related largely to LV function as well as the degree of regurgitation.

The pathophysiology of MR is volume overload of the LV, similar to AR. In both lesions the circulatory compensation is peripheral vasodilatation to promote forward rather than backward flow. The major difference is that in AR the regurgitation occurs during diastole from aorta into LV, while in MR the regurgitation occurs during systole, from LV back into the left atrium (LA). With chronic MR the LA dilates over decades, maintaining a low left atrial pressure until late in the disease and thereby avoiding pulmonary congestion.[11] As the regurgitant fraction exceeds 60% of the stroke volume,

forward cardiac output becomes compromised and symptoms of "forward" heart failure (fatigue, weakness) occur. Eventually LAP and pulmonary pressures become elevated. Because systolic ejection is partly into the low pressure LA, the ejection fraction is a poor indicator of LV function – an LVEF less than 50% means severe dysfunction. Onset of symptoms of forward heart failure is associated with a very high 5 year mortality.

Acute mitral regurgitation (from papillary muscle rupture, for example) occurs in a heart where the LA cannot rapidly dilate to accommodate the regurgitated blood. As a result there is a rapid increase in LA pressure and LVEDP. This can cause acute pulmonary edema, pulmonary hypertension, and RV dysfunction or failure. Similar to acute AS this may be a hemodynamic emergency requiring immediate surgery.

Anesthetic considerations

Similar to AR, forward cardiac output is best when the heart is full and reasonably fast, and the blood pressure is low-normal.[12] Determination of the optimal end-diastolic volume or pressure is difficult without a PAC or TEE, and may require observing the response to a fluid load. Bradycardia is associated with an increase in ventricular size leading to valvular dilatation and an increase in regurgitant fraction, so should be avoided. The need for a low "afterload" as determined by blood pressure is explained above. Because the true contractile state is difficult to determine (ie the LVEF is misleading), avoidance of myocardial depression within reason should be a goal. Patients with MR often require inotropic assistance to accomplish these hemodynamic goals in the face of general anesthesia. Epidural or spinal anesthesia are options where appropriate. Patients who undergo mitral valve replacement or repair for MR often require inotropic assistance after the procedure, as the loss of the low-resistance pathway (ie, regurgitant flow) poses an acute stress on the LV and may reveal underlying dysfunction.

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