Valvular heart disease is becoming more common in our aging population. An estimate of the prevalence of moderate to severe disease in patients > 75 years old is 13.3%. Maintenance of hemodynamic stability in these patients can be quite challenging. This review will focus on anesthetic management of the classic lesions: aortic stenosis (AS), aortic regurgitation (AR), mitral stenosis (MS), and mitral regurgitation (MR).

General guidelines for hemodynamic management (heart rhythm, heart rate, preload, afterload, and contractility) will be presented for each valvular lesion. However, the anesthesiologist should bear in mind that “mixed” valvular lesions are more common than “pure” valvular lesions. Thus, the clinician will need to determine which is the most severe (hemodynamically significant) lesion and/or will need to “split the difference” between management goals for multiple valve lesions.

AORTIC STENOSIS
Aortic stenosis (AS) is a commonly encountered cardiac valve lesion in the U.S. Acquired AS is due to idiopathic senile degeneration with sclerosis and calcification of the valve. There is a clear association between clinical risk factors for atherosclerotic disease and the development of AS, including the process of chronic inflammation. An increased incidence with aging occurs due to greater mechanical stress over time and longer exposure to risk factors such as hypertension, smoking, diabetes, and hypercholesterolemia. Aortic stenosis is now seen in 2%-4% of adults greater than 65 years of age, and this prevalence is expected to increase.

Another common etiology of aortic stenosis is a congenital defect in the valve, because 1%-2% of the population are born with a bicuspid aortic valve. Inheritance has been found to play a role, with an autosomal dominant pattern and a variable penetrance. A bicuspid aortic valve that does not yet show any signs of damage nevertheless tends to open and close with abnormal folding and creasing, leading to scarring and calcification. Although patients with a bicuspid aortic valve are asymptomatic until late in the disease process, severe, symptomatic AS with or without aortic regurgitation (AR) may develop in mid-life. In undeveloped countries, rheumatic disease is a fairly common cause of AS, and it is usually associated with concomitant AR.

Aortic stenosis is an important clinical entity because of the potential for sudden death and because of the relative ineffectiveness of external cardiac massage during a cardiac arrest. The diagnosis of AS is made from a detailed history and physical examination, supplemented by echocardiography. Symptoms of AS can range from decreased exercise tolerance and exertional dyspnea to angina, CHF, and syncope. On physical examination, a systolic ejection murmur with radiation to the carotids is strongly suggestive of AS.
Echocardiography can be used to determine multiple aspects of the pathophysiology of the lesion including the severity of AS, any structural abnormalities of the valve causing left ventricular outflow tract (LVOT) obstruction, and any accompanying disease that can be found in the other heart valves. A commonly used parameter of severity is the aortic valve area (AVA), with normal AVA being 3-4 cm². In severe AS, AVA is ≤ 1 cm². Another parameter commonly used to determine the severity of AS is the gradient across the aortic valve, whereby AS is considered severe if the mean gradient is ≥ 40 mmHg.

ANESTHETIC MANAGEMENT

Operative risk depends upon the severity of the AS, whether the patient has concomitant coronary disease, and the risks of the surgical procedure. Also, it is important to realize that the presence of severe AS reduces the usefulness of CPR for maintaining a cardiac output sufficient to meet the patient’s physiologic needs. Anesthetic management of patients with AS revolves around avoiding fluctuations in the patient’s hemodynamics, while achieving adequate anesthetic depth.

Every effort should be made to ensure that the patient with AS stays in sinus rhythm. Due to diastolic dysfunction and impaired relaxation, the “atrial kick” may contribute as much as 40% of the total cardiac output. Anything interfering with atrial function (e.g., junctional rhythm or atrial fibrillation) can lead to severe hypotension. Therefore, in order to treat any possible arrhythmia, external cardioversion pads should be considered, preferably before induction of anesthesia.

It is also important to avoid either tachycardia or bradycardia. Bradycardia is undesirable because the stroke volume is already limited by the stenotic valve itself; therefore, cardiac output is unacceptably low in the presence of bradycardia. Tachycardia can usually be tolerated for short periods, but can further jeopardize an already compromised coronary supply/demand relationship in the presence of ventricular hypertrophy and concomitant coronary disease.

Preload should be maintained or increased in order to adequately fill the noncompliant left ventricle. Afterload should be maintained or increased. Systemic hypotension causes reduced coronary perfusion pressure and should be managed with the early use of α-adrenergic agonists. Contractility should be maintained.

Premedication may help to prevent perioperative tachycardia in patients with AS. Monitoring includes standard noninvasive modalities. Usually, an arterial line is placed in the preoperative period. Invasive monitoring of central venous pressure is considered, if the surgical procedure involves the potential for blood loss and volume shifts. TEE monitoring is nearly always desirable if there are no contraindications to its placement.

AORTIC REGURGITATION

Aortic regurgitation (AR) is the flow of blood from the aorta backwards into the left ventricle during the diastolic phase of the cardiac cycle. Chronic AR is more prevalent and carries a much better prognosis than acute AR. Causes of chronic AR include congenital lesions, connective tissue disorders, inflammatory diseases,
appetite suppressant medications, rheumatic disease, and annular dilation from aging and chronic hypertension. These processes cause malcoaptation of the AV leaflets by causing abnormalities in the leaflets themselves or dilation of the AV annulus, the aortic root, or both.12

Progressive volume loading in chronic AR increases end-diastolic wall tension. The left ventricle undergoes a process of remodeling due to series replication of sarcomeres and myofibril elongation, with development of eccentric ventricular hypertrophy and chamber enlargement.13

Figure 2: Pressure-volume loops in acute and chronic aortic insufficiency (regurgitation).

Figure 2 shows that the pressure-volume loop is shifted far to the right in patients with chronic AR. Notably, left ventricular end-diastolic pressure (LVEDP) remains relatively normal because left ventricular end-diastolic volume (LVEDV) increases slowly. Therefore, patients with chronic AR may remain asymptomatic for years or even decades. However, patients with chronic AR eventually present with symptoms of left heart failure, e.g., exercise intolerance, dyspnea, and paroxysmal nocturnal dyspnea or orthopnea. Even when these patients have normal coronary arteries, a few nevertheless present with angina due to poor coronary perfusion resulting from low diastolic aortic pressure.

In patients with AR, forward flow is improved by peripheral vasodilation. Typically, a normal ejection fraction is maintained by a large stroke volume. However, over time, increases in the left ventricular wall stress and afterload result.14 Eventually, as left ventricular dilation and hypertrophy progress, irreversible left ventricular dysfunction develops, and patients become symptomatic. Further impairment of systolic function occurs secondary to oxidative stress, collagen degradation, and matrix metalloproteinase activation.15 As a compensatory mechanism for poor cardiac output, sympathetic constriction of the peripheral vasculature occurs to maintain blood pressure, although this worsens regurgitation and cardiac output.

Acute AR is less common than chronic AR but carries a more ominous prognosis. Common causes of acute AR include trauma, bacterial endocarditis, and aortic dissection. The pathophysiology of acute AR centers around the fact that it causes an acute increase in the volume coming into the left ventricle. Because the left ventricle has not had time to undergo the process of eccentric hypertrophy, as it does in chronic AR, it is unprepared to accommodate this sudden increase in volume. As shown in the middle loop in Figure 2, this sudden increase in the LVEDP causes a rightward shift in the pressure-volume loop.13 A sympathetic response is activated ─ tachycardia and increased contractile state are the chief compensatory mechanisms for maintaining adequate cardiac output. Unless the AR is managed appropriately, these compensatory mechanisms rapidly fail, necessitating emergency cardiac surgery.

Echocardiography is the most important diagnostic tool. Regurgitant volume consisting of < 20% of the total left ventricular stroke volume is considered mild, 20%-39% is considered moderate, 40%-60% is considered moderately severe, and > 60% is considered severe.9 In actuality, the regurgitant volume depends in part upon the diastolic time interval and the diastolic pressure gradient across the valve, as well as the regurgitant orifice area.

ANESTHETIC MANAGEMENT

Maintaining a relatively fast heart rate (approximately 90 beats/min) will minimize the time spent in diastole and leads to a decreased regurgitant fraction. Subendocardial blood flow may actually improve with tachycardia due to a higher diastolic pressure and a lower LVEDP. Sinus rhythm is preferable, but rapid supraventricular tachyarrhythmias are better tolerated in patients with AR than in patients with AS.

Also, due to the increased left ventricular volume, preload should be augmented to maintain filling of the dilated left ventricle and maintain forward flow. Furthermore, reducing afterload (maintaining a relatively low systemic vascular resistance [SVR]) will minimize the pressure gradient back across the aortic valve during diastole, improving forward flow and decreasing LVEDP. Finally, left ventricular contractility should be maintained.
In considering the choice of drugs for general anesthesia in these patients, medications that cause bradycardia should be avoided. Pharmacologic interventions that produce venous dilation may significantly impair cardiac output by reducing preload. Increases in ventricular afterload should be avoided. In some patients with AR, inodilator agents such as phosphodiesterase inhibitors or other inotropic agents such as β-agonists may be needed to improve left ventricular function.

**MITRAL STENOSIS**

Rheumatic heart disease was once the primary cause of mitral stenosis (MS), although its prevalence in the U.S. is decreasing. In the US and other industrialized nations, mitral valve disease is usually caused by primary degenerative (i.e., age-associated), congenital mitral valvular abnormalities (e.g., mitral valve prolapse), or ischemic heart disease resulting in functional mitral incompetence, rather than by rheumatic heart disease. However, rheumatic heart disease remains a large-scale medical and public health problem for many countries and is still seen in the U.S. due to immigration.

The normal mitral valve orifice area is approximately 4-5 cm². Symptoms of MS, usually dyspnea, can occur with a valve area less than 2.5 cm² and can be precipitated by clinical events associated with increased cardiac output and consequent increased flow across the stenotic valve, e.g., stress, exercise, anemia, pregnancy, or febrile illness. MS is considered to be mild if the valve area is 1.5-2.5 cm², moderate if 1.1-2.5 cm², and severe if ≤ 1.0 cm². Currently, MS is primarily diagnosed and monitored with echocardiography, although MVA can be calculated during cardiac catheterization by using the Gorlin equation. Obstructed flow across the mitral valve is also associated with a pressure differential or “gradient” across the valve. The more severe the MS, the greater the gradient, as long as flow across the valve is held constant. Notably, severe MS may be present with a low measured or calculated gradient across the valve if the patient has low flow due to right heart failure and pulmonary hypertension.

The increased left atrial pressure in patients with MS gradually produces left atrial dilation. Such atrial enlargement can lead to the onset of atrial fibrillation and also to thromboembolic complications if a clot forms in the atrium or appendage due to low velocity blood flow. Treatment may include anticoagulation with IV heparin or oral coumadin, pharmacologic rate control, and pharmacologic or electrical cardioversion for hemodynamically significant or acute onset atrial fibrillation. In patients scheduled for cardioversion, TEE may be performed first to rule out the presence of LA thrombus.

Elevated pressure in the left atrium also leads to passive increases in pulmonary venous and arterial pressures. Many patients with MS have elevated pulmonary pressures secondary to reactive pulmonary vasoconstriction or histologic changes in the medial and intimal layers of pulmonary arteries and arterioles. Chronic elevation in pulmonary pressure caused by MS leads to compensatory right ventricular hypertrophy, similar to the pathophysiology of lesions that obstruct outflow of the left ventricle. However, the response in the right ventricle is less efficient than the left ventricle because of its shape, wall thickness, and smaller muscle mass of the right ventricle. Therefore, chronic pulmonary hypertension can lead to progressive right ventricular dilation and failure.

The effect of MS on the left ventricle is primarily due to obstruction of diastolic inflow. The narrowed mitral valve orifice leads to prolonged early diastolic mitral inflow and delayed left ventricular filling. Late diastolic filling, occurring during atrial systole, is further compromised in patients who have atrial fibrillation secondary to MS. In cases of MS, pressure-volume loops are shifted to the left, so that LVEDP and LVEDV are lower (Figure 3).

Stroke volume is diminished, especially in clinical situations that result in elevated heart rate and shortened diastolic filling intervals. Although left ventricular function or contractility was once thought to be normal in most patients with MS, it has been shown that left ventricular dysfunction is common in patients with MS. Proposed mechanisms include reduced filling of the left ventricle, muscle atrophy, inflammatory myocardial fibrosis leading to wall motion abnormalities, scarring of
the subvalvular apparatus, abnormal patterns of left ventricular contraction, reduced left ventricular compliance with diastolic dysfunction, increased afterload leading to left ventricular remodeling, right-to-left ventricular septal shift secondary to the effect of pulmonary hypertension on the right ventricle, and coexisting diseases such as systemic hypertension and coronary artery disease.20

ANESTHETIC MANAGEMENT

Primary concerns in patients with MS include management of heart rate, ventricular preload, potentially diminished right and left ventricular contractile function, and coexisting pulmonary hypertension. The most important hemodynamic goal is to avoid tachycardia (keep heart rate within its normal range). Tachycardia is poorly tolerated because of the decreased time for diastolic filling. Also, pressure gradients are somewhat flowdependent in MS. Elevated flow states, such as increased sympathetic activity from any source, can dramatically increase the pressure gradient across the valve. Echocardiographically, the concept of valve gradients is derived through the use of a modified form of Bernoulli’s equation, ΔP = 4v², where “v” is the measured velocity of blood flow through the valve. Thus, any increase in transvalvular flow rate caused by an increase in heart rate will have a significant impact on transvalvular flow dynamics and on left atrial pressure.

Also, if possible, sinus rhythm should be preserved. Atrial contributions to stroke volume may be elevated in MS patients who are in the early stages of the disease and who are not in atrial fibrillation. Once atrial fibrillation has occurred, the atrial kick is lost. In this case, however, the most important factor in the deterioration of the patient’s clinical condition is tachycardia itself, rather than loss of the atrial kick. In any event, digoxin should be continued perioperatively. Short-acting β-blockers can then be used for heart rate control.

Flow through a stenotic mitral valve requires a higher-than-normal pressure gradient between the left atrium and the left ventricle. Thus, reduction in preload, from the venodilatory effects of anesthesia or from blood loss, can markedly affect cardiac output. However, patients with MS already have elevated left atrial pressures, so that overly aggressive use of fluids can lead a patient in borderline CHF into florid pulmonary edema.9 In patients with MS, afterload reduction is usually not helpful in augmenting forward flow, because stroke volume is determined by the mitral valve orifice area and the diastolic filling interval.

Left ventricular contractility and SVR are usually preserved in MS. If anything, the left ventricle is chronically underloaded. Nevertheless, global systolic dysfunction develops in some MS patients.18

Right ventricular dysfunction probably poses a greater challenge in treating patients with MS than does left ventricular dysfunction. Every effort should be made to avoid increases in pulmonary arterial pressures (e.g., avoid hypoxia, hypercarbia, acidosis, lung hyperexpansion, and nitrous oxide).

In patients with MS, oversedation in the preoperative period should be avoided to prevent hypoventilation. Bleeding complications from chronic anticoagulation in patients with atrial fibrillation should be anticipated. Monitoring for these patients includes standard noninvasive modalities and, depending upon the type of surgery, may involve invasive monitoring of blood pressure, central venous pressure, and intraoperative echocardiography. Monitoring pulmonary artery (PA) pressure and monitoring cardiac output with a PA catheter is sometimes employed, but care and judgment must be exercised, given the propensity for PA rupture in patients with long-standing pulmonary hypertension. Management of right ventricular dysfunction includes optimizing acid-base balance and using hypocarbia, hyperoxia, and possibly vasodilators to decrease pulmonary vascular resistance. Inotropic support may be needed for patients with secondary right ventricular dysfunction or failure. Epinephrine and milrinone are good therapeutic options. Newer therapeutic options for treatment of refractory pulmonary hypertension include inhaled prostacyclin or nitric oxide.

MITRAL REGURGITATION

Mitrail regurgitation (MR) is a commonly encountered valve lesion. MR can either involve structural abnormalities in the valve or in its subvalvar components, or functional abnormalities due to annular or left ventricular dilatation causing malcoaptation of the mitral valve leaflets.21 Examples of structural abnormalities of the mitral valve include mitral valve prolapse, myxomatous degeneration of the mitral valve, rheumatic mitral insufficiency, cleft mitral valve associated with an atroventricular septal defect, and any infiltrative/fibrotic processes. Functional MR is present in 10%-20% of patients with chronic ischemia due to coronary artery disease. Unlike primary valvular causes of MR, the morphology of the mitral valve is normal in these patients. Nonetheless, the long-term morbidity and mortality associated with this type of MR are significant.21 Today, in developed countries, the most common causes of MR are either myxomatous degeneration of the mitral valve (resulting in annular dilation, chordal elongation and rupture, and redundant,
prolapsing, or flail mitral valve leaflets) or mitral insufficiency caused by ischemic heart disease.\(^3\)

The incompetent mitral valve allows retrograde passage of blood from the left ventricle into the left atrium during systole. The magnitude of the regurgitant volume is a function of the size of the regurgitant orifice, the pressure differential between the left atrium and the left ventricle, and the duration of the regurgitant cycle.\(^1\)

The severity of MR is assessed in the context of whether the MR is acute or chronic. In patients with chronic MR, symptoms range from nonspecific complaints such as easy fatigability and palpitations to severe CHF. Echocardiography is used to serially follow patients with chronic MR.\(^2\) Quantitative estimates of regurgitant fraction (the fraction of regurgitant volume in relation to total stroke volume) are made from the LV angiogram or measured echocardiographically with Doppler. If regurgitant volume is < 30% of total left ventricular stroke volume, the MR is considered mild, 30%-39% is considered moderate, 40%-60% is considered moderately severe, and > 60% is severe.\(^9\) Pulmonary venous systolic flow reversal is another indication that mitral regurgitation is severe.

The left atrium is exposed to both volume and pressure increases. However, in chronic MR, left atrial pressure increases are not dramatic because of compliance changes in the left atrium as a function of gradual chamber dilation. Progressive left atrial enlargement eventually leads to atrial fibrillation, which occurs in about 50% of patients who present for surgical correction of MR. When left atrial compliance thresholds are reached, left atrial pressure and pulmonary arterial pressure become elevated. Eventually, if chronically exposed to elevated PA pressure, the right ventricle progressively enlarges and right ventricular dysfunction develops.

The long-term sequelae of MR are related to chronic pressure and volume effects on the left atrium and left ventricle. The left ventricle is exposed to a chronic, isolated volume-overload state. Eccentric hypertrophy of the left ventricle develops, causing chamber enlargement without significant increases in wall thickness. Forward cardiac output is preserved because of eccentric hypertrophy and the low impedance of the left atrium—a physiologic equivalent of afterload reduction.\(^1\) The larger stroke volume ejected by the left ventricle is composed of normal venous return into the left atrium plus the regurgitant volume from the prior cardiac cycle. With time, however, compensatory eccentric hypertrophy fails to preserve left ventricular systolic function, and gradual systolic failure ensues, as noted on pressure-volume loops (Figure 4).

A reduction in left ventricular ejection fraction below 60% or an increase in end-systolic dimension exceeding 40 mmHg indicates the need for surgical repair or replacement of the mitral valve.\(^2\)

With acute onset of MR (e.g., due to myocardial infarction and rupture of papillary muscles), there has been no time for left atrial compensatory changes to occur. Therefore, there is a sudden increase in left atrial pressure and pulmonary capillary wedge pressure. Patients with acute severe MR are usually in cardiogenic shock and do not present for noncardiac surgery. Pharmacologic support of the left ventricle, often accompanied by mechanical support with intraaortic balloon pump (IABP) counterpulsation, may be necessary to prepare the patient for emergency cardiac surgery.

**ANESTHETIC MANAGEMENT**

The primary goal in patients with chronic MR is maintaining forward systemic flow.\(^3\) The heart rate should be maintained in the high-normal range, i.e., 80 to 100 beats/minute. Tachycardia decreases the regurgitant volume by shortening systole. Bradycardia has dual detrimental effects on MR: it increases the systolic period duration, thus prolonging regurgitation, and it increases the diastolic filling interval, which can lead to LV distention. A sinus rhythm is preferred, but there is less dependency on the atrial kick than in stenotic valvular heart disease.

As with most compensated forms of valvular heart disease, patients with hemodynamically significant MR are sensitive to ventricular loading conditions. It must be
remembered that anesthetic effects on afterload and preload can drastically alter the severity of MR from its baseline level as seen in preoperative echocardiographic or catheterization assessments. In general, afterload reduction in combination with mild preload augmentation will enhance forward cardiac output and blood pressure. Adequate anesthetic depth, systemic vasodilators, or inodilators may be clinical options, depending on the situation. However, higher systolic driving pressures, as in hypertension, can increase the regurgitant volume, while fluid overload with ventricular distension can lead to expansion of an already dilated mitral annulus and thus worsen MR.

In early compensated MR, left ventricular contractility may be preserved. However, in patients with moderate to severe MR, ejection fraction indices are poorly correlated with left ventricular systolic function so that underlying systolic dysfunction may be underestimated. Hypotension in patients with significant MR can often be managed by manipulating heart rate and volume, but persistent hemodynamic instability may be best treated with inotropic support. Direct-acting α1-agonists increase SVR and blood pressure, lower heart rate, and may worsen MR. Temporary use of small doses of ephedrine may be a better choice. Dobutamine, lowdose epinephrine, and milrinone are all acceptable inotropic choices for continuous infusion.

Pulmonary artery pressures and pulmonary vascular resistance may be elevated in patients with MR. Factors that may increase pulmonary vascular resistance and unfavorably load an already dysfunctional right ventricle, such as hypoxia, hypercarbia, and acidosis, should be avoided.

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