Chapter 15

Valvular Heart Disease: Replacement and Repair

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Key Points

1. Although various valvular lesions generate different physiologic changes, all valvular heart disease is characterized by abnormalities of ventricular loading.
2. The left ventricle normally compensates for increases in afterload by increases in preload. This increase in end-diastolic fiber stretch or radius further increases wall tension in accordance with Laplace's law, resulting in a reciprocal decline in myocardial fiber shortening. The stroke volume is maintained because the contractile force is augmented at the higher preload level.
3. Treatment modalities for hypertrophic obstructive cardiomyopathy, a relatively common genetic malformation of the heart, include β-adrenoceptor antagonists, calcium channel blockers, and myectomy of the septum. Newer approaches include dual-chamber pacing and septal reduction (ie, ablation) therapy with ethanol.
4. The severity and duration of symptoms of aortic regurgitation may correlate poorly with the degree of hemodynamic and contractile impairment, delaying surgical treatment while patients are undergoing progressive deterioration.
5. Mitral regurgitation causes left ventricular volume overload. Treatment depends on the underlying mechanism and includes early reperfusion therapy, angiotensin-converting enzyme inhibitors, and surgical repair or replacement of the mitral valve.
6. Rheumatic disease and congenital abnormalities of the mitral valve are the main causes of mitral stenosis, a slowly progressive disease. Surgical treatment options include closed and open commissurotomy and percutaneous mitral commissurotomy.
7. Most tricuspid surgery occurs in the context of significant aortic or mitral disease, and anesthesia management primarily is determined by the left-sided valve lesion.
8. Innovations in surgical valve repair include aortic valve repair and closed- and open-chamber procedures for mitral regurgitation.

Valve surgery is very different from coronary artery bypass grafting (CABG). Over the natural history of valvular heart disease (VHD), the physiology changes markedly. In the operating room, physiologic conditions and hemodynamics are quite dynamic and are readily influenced by anesthesia. For some types of valve lesions, it can be relatively difficult to predict before surgery how the heart will respond to the altered loading conditions associated with valve repair or replacement.

It is essential to understand the natural history of adult-acquired valve defects and how the pathophysiology evolves. Clinicians must also understand surgical decision making for valve repair or replacement. A valve operated on at the appropriate stage of its natural history has a good and more predictable outcome compared with a heart operated on at a later stage, for which the perioperative result can be poor. The dynamic physiology and natural history of each valve defect govern the anesthesia plan, which must include the requirements for preload, pacing rate, and rhythm; use of inotropes or negative inotropes; and use of vasodilators or vasoconstrictors to alter loading conditions.

Although valvular lesions impose different physiologic changes, a unifying concept is that all VHD is characterized by abnormalities of ventricular loading. The status of the ventricle changes over time because ventricular function and the valvular defect itself are influenced by the progression of volume or pressure overload. The clinical status of patients with VHD can be complex and dynamic. It is possible to have clinical decompensation in the context of normal ventricular contractility or have ventricular decompensation and performance with normal ejection indices. The altered loading conditions characteristic of VHD may result in a divergence between the function of the heart as a systolic pump and the intrinsic inotropic state of the myocardium. The divergence between cardiac performance and inotropy results from compensatory physiologic mechanisms that are specific to each of the ventricular loading abnormalities.

AORTIC STENOSIS

Clinical Features and Natural History

Aortic stenosis (AS) is the most common cardiac valve lesion in the United States. Approximately 1% to 2% of people are born with a bicuspid aortic valve, which is prone to stenosis with aging. Clinically significant aortic valve stenosis occurs in 2% of unselected individuals older than 65 years and in 5.5% of those older than 85 years.

Calcific AS has several features in common with coronary artery disease (CAD). Both conditions are more common in men, older people, and patients with hypercholesterolemia, and both result in part from an active inflammatory process. Clinical evidence indicates that an atherosclerotic process is the cellular mechanism of aortic valve stenosis. There is a clear association between clinical risk factors for atherosclerosis and the development of AS: increased lipoprotein levels, increased low-density lipoprotein (LDL) cholesterol, cigarette smoking, hypertension, diabetes mellitus, increased serum calcium and creatinine levels, and male sex. The early lesion of aortic valve sclerosis may be associated with CAD and vascular atherosclerosis. Aortic valve calcification is an inflammatory process promoted by atherosclerotic risk factors.

The average rate of progression is a decrease in aortic valve area (AVA) of 0.1 cm^2/year, and the peak instantaneous gradient increases by 10 mm Hg/year. The rate of progression of AS in men older than 60 is faster than in women, and it is faster in women older than 75 than in women 60 to 74 years old.

Angina, syncope, and congestive heart failure (CHF) are the classic symptoms of the disease, and their appearance is of serious prognostic significance because postmortem studies indicate that symptomatic AS is associated with a life expectancy of only 2 to 5 years.

There is evidence that patients with moderate AS (ie, valve areas of 0.7 to 1.2 cm^2) are also at increased risk for complications, with the appearance of symptoms further increasing their risk.

Angina is a frequent and classic symptom of the disease, occurring in approximately two-thirds of patients with critical AS, and about one-half of symptomatic patients have anatomically significant CAD.

It is probably never too late to operate on patients with symptomatic AS. Unlike patients with aortic regurgitation (AR), most symptomatic patients undergo valve replacement when left ventricular function is still normal. Even when impaired left ventricular function develops in AS, the relief of pressure overload almost always restores normal function or produces considerable improvement. Morbidity rates, mortality rates, and clinical results are favorable even for the oldest surgical candidates. Advances in operative techniques and perioperative management have contributed to excellent results after aortic valve replacement (AVR) in patients 80 years of age or older, with minimal incremental postoperative morbidity.

Preoperative assessment of AS with Doppler echocardiography includes measurement of the AVA and the transvalvular pressure gradient. The latter is calculated from the Doppler-quantified transvalvular velocity of blood flow, which is increased in the setting of AS. The maximal velocity (v) is then inserted into the modified Bernoulli equation to determine the pressure gradient (PG) between the left ventricle (LV) and the aorta:

$$PG = P(\text{left ventricle}) - P(\text{aorta}) = 4(v^2)$$

The *pressure gradient* is the maximal difference between the LV and aortic pressures that occurs during ventricular systole.

Pressure gradients determined invasively or by Doppler echocardiography correctly classify AS severity in less than 50% of cases compared with estimates of AVA. The preferred method of obtaining AVA requires only two Doppler-generated velocities: those proximal or distal to the stenotic valve. These values are inserted into the continuity equation, which relates the respective velocities and cross-sectional areas proximal and distal to a stenotic area:

$$V_{max} \times AVA = \text{area(LVOT)} \times V(\text{LVOT})$$

In the equation, AVA is the aortic valve area, V is the volume, and LVOT is the left ventricular outflow tract.

Pathophysiology

The normal AVA is 2.6 to 3.5 cm^2, with hemodynamically significant obstruction usually occurring at cross-sectional valve areas of 1 cm^2 or less. Accepted criteria for critical outflow obstruction include a systolic pressure gradient greater than 50 mm Hg, with a normal cardiac output, and an AVA of less than 0.4 cm^2. In view of the ominous natural history of severe AS (AVA <0.7 cm^2), symptomatic patients with this degree of AS are usually referred for immediate AVR. A simplification of the Gorlin equation to calculate the AVA is based on the cardiac output (CO) and the peak pressure gradient (PG) across the valve.

$$AVA = CO/\sqrt{(PG)}$$

An obvious corollary of the previously described relationship is that "minimal" pressure gradients may reflect critical degrees of outflow obstruction when the CO is significantly reduced (ie, generation of a pressure gradient requires some finite amount of flow). Clinicians have long recognized this phenomenon as a paradoxical decline in the intensity of the murmur (ie, minimal transvalvular flow) as the AS worsens.

Stenosis at the level of the aortic valve results in a pressure gradient from the LV to the aorta. The intracavitary systolic pressure generated to overcome this stenosis directly increases myocardial wall tension in accordance with Laplace's law:

$$\text{Wall tension} = P \times R/2h$$

In the equation, P is the intraventricular pressure, R is the inner radius, and h is the wall thickness.

The increase of wall tension is thought to be the direct stimulus for the further parallel replication of sarcomeres, which produces the concentrically hypertrophied ventricle characteristic of chronic pressure overload. The consequences of left ventricular hypertrophy (LVH) include alterations in diastolic compliance, potential imbalances in the myocardial oxygen supply and demand relationship, and possible deterioration of the intrinsic contractile performance of the myocardium.

Fig. 15.1 shows a typical pressure-volume loop for a patient with AS. Two differences from the normal curve are immediately apparent. First, the peak pressure generated during systole is much greater because of the high transvalvular pressure gradient. Second, the slope of the diastolic limb is steeper, reflecting the reduced left ventricular diastolic compliance that is associated with the increase in chamber thickness. Clinically, small changes in diastolic volume produce relatively large increases in ventricular filling pressure.

Increased chamber stiffness places a premium on the contribution of atrial systole to ventricular filling, which in patients with AS may account for up to 40% of the left ventricular end-diastolic volume (LVEDV) rather than the 15% to 20% characteristic of the normal LV. Echocardiographic and radionuclide studies have documented that diastolic filling and ventricular relaxation are abnormal in patients with hypertrophy

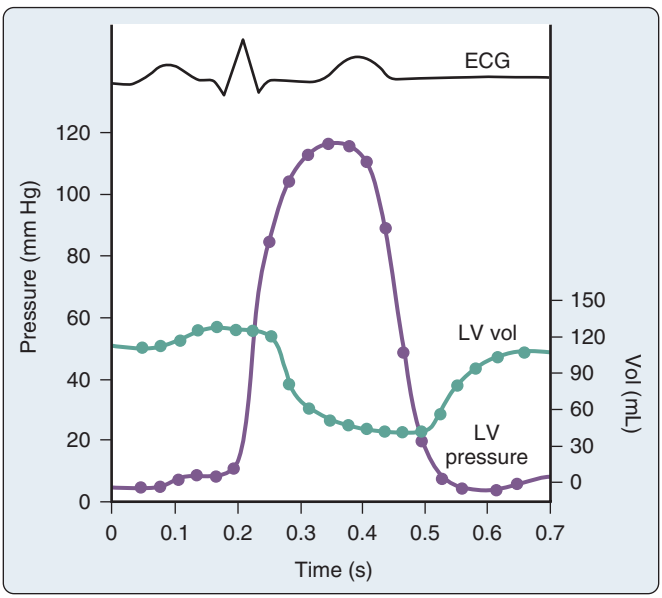


Fig. 15.1 Simultaneous left ventricular *(LV)* volume and pressure during one cardiac cycle. *ECG,* Electrocardiogram. (From Barash PG, Kopriva DJ. Cardiac pump function and how to monitor it. In: Thomas SJ, ed. *Manual of Cardiac Anesthesia.* New York: Churchill Livingstone; 1984:1.)

from a variety of causes, and significant prolongation of the isovolumic relaxation period is the most characteristic finding. This necessarily compromises the duration and amount of filling achieved during the early rapid diastolic filling phase and increases the relative contribution of atrial contraction to overall diastolic filling. A much greater mean left atrial pressure is necessary to distend the LV in the absence of the sinus mechanism. One treatment of junctional rhythm is volume infusion.

The systolic limb of the pressure-volume loop shows preservation of pump function, as evidenced by maintenance of the stroke volume (SV) and ejection fraction (EF). Use of preload reserve and adequate LVH are likely the principal compensatory mechanisms that maintain forward flow. Clinical studies have confirmed that ejection performance is preserved at the expense of myocardial hypertrophy, and the adequacy of the hypertrophic response has been related to the degree to which it achieves normalization of wall stress, in accordance with the Laplace relation. LVH can be viewed as a compensatory physiologic response; however, severe afterload stress and proportionately massive LVH could decrease subendocardial perfusion and superimpose a component of ischemic contractile dysfunction.

In AS, signs and symptoms of CHF usually develop when preload reserve is exhausted, not because contractility is intrinsically or permanently impaired. This contrasts with mitral regurgitation (MR) and AR, in which irreversible myocardial dysfunction may develop before the onset of significant symptoms.

The major threat to the hypertrophied ventricle is its exquisite sensitivity to ischemia. Ventricular hypertrophy directly increases basal myocardial oxygen demand ($M\dot{V}O_2$). The other major determinants of overall $M\dot{V}O_2$ are heart rate, contractility, and, most important, wall tension. Increases in wall tension occur as a direct consequence of Laplace's law in patients with relatively inadequate hypertrophy. The possibility of ischemic contractile dysfunction in the inadequately hypertrophied ventricle arises from increases in wall tension, which directly parallels the imbalance between the increased peak systolic pressure and the degree of mural hypertrophy. Although there is considerable evidence for supply-side abnormalities in the myocardial supply and demand relationship in patients with AS, clinical data also support increased $M\dot{V}O_2$ as important in the genesis of myocardial ischemia.

On the supply side, the greater left ventricular end-diastolic pressure (LVEDP) of the poorly compliant ventricle inevitably narrows the diastolic coronary perfusion pressure (CPP) gradient. With severe outflow obstruction, decreases in SV and resultant systemic hypotension may critically compromise coronary perfusion. A vicious cycle may develop because ischemia-induced abnormalities of diastolic relaxation can aggravate the compliance problem and further narrow the CPP gradient. This sets the stage for ischemic contractile dysfunction, additional decreases in SV, and worsening hypotension.

Difficulty of Low-Gradient, Low-Output Aortic Stenosis

A subset of patients with severe AS, left ventricular dysfunction, and low transvalvular gradient suffers a high operative mortality rate and poor prognosis. It is difficult to assess accurately the AVA in low-flow, low-gradient AS because the calculated valve area is proportional to forward SV and because the Gorlin constant varies in low-flow states. Some patients with low-flow, low-gradient AS have a decreased AVA as a result of inadequate forward SV rather than anatomic stenosis. Surgical therapy is unlikely to benefit these patients because the underlying pathology is a weakly contractile myocardium. However, patients with severe anatomic AS may benefit from valve replacement despite the increased operative risk associated with the low-flow,

Table 15.1 Pressure-Overload Hypertrophy

Beneficial Aspects	Detrimental Aspects
Increases ventricular work	Decreases ventricular diastolic distensibility
Normalizes wall stress	Impairs ventricular relaxation
Normalizes systolic shortening	Impairs coronary vasodilator reserve, leading to subendocardial ischemia

From Lorell BH, Grossman W. Cardiac hypertrophy: the consequences for diastole. *J Am Coll Cardiol.* 1987;9:1189.

low-gradient hemodynamic state. American College of Cardiology/American Heart Association (ACC/AHA) guidelines call for a dobutamine echocardiography evaluation to differentiate patients with fixed anatomic AS from those with flow-dependent AS with left ventricular dysfunction. Low-flow, low-gradient AS is defined as a mean gradient of less than 30 mm Hg and a calculated AVA less than 1.0 cm^2.

Timing of Intervention

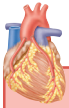
For asymptomatic patients with AS, it appears to be relatively safe to delay surgery until symptoms develop, but outcomes vary widely. Moderate or severe valvular calcification along with a rapid increase in aortic-jet velocity identify patients with a very poor prognosis. They should be considered for early valve replacement rather than delaying until symptoms develop.

Echocardiography and exercise testing may identify asymptomatic patients who are likely to benefit from surgery. In a study of 58 asymptomatic patients, 21 had symptoms for the first time during exercise testing. Guidelines for AVR in patients with AS are shown in Table 15.1.

Functional outcome after AVR for patients older than 80 years is excellent, operative risk is limited, and late survival rates are good. For patients with severe left ventricular dysfunction and a low transvalvular mean gradient, the operative mortality rate was increased, but AVR was associated with improved functional status. Postoperative survival rates were best for younger patients and those with larger prosthetic valves, whereas medium-term survival rates were correlated with improved postoperative functional class.

Anesthesia Considerations

The described pathophysiologic principles dictate anesthesia management based on avoidance of systemic hypotension, maintenance of sinus rhythm and an adequate intravascular volume, and awareness of the potential for myocardial ischemia (Box 15.1). In the absence of CHF, adequate premedication may reduce the likelihood of undue preoperative excitement, tachycardia, and exacerbation of myocardial ischemia and the transvalvular pressure gradient. In patients with critical outflow tract obstruction, however, heavy premedication with an exaggerated venodilatory response can reduce the appropriately increased LVEDV (and LVEDP) needed to overcome the systolic pressure gradient. In these patients, the additional precaution of administering supplementary oxygen may obviate the possibility of a similarly pronounced response to the sedative effects of the premedicant.



BOX 15.1 ***Aortic Stenosis***

Maintain preload and diastolic filling
Maintain sinus rhythm
Maintain or increase afterload
Avoid myocardial depression
Avoid tachycardia, hypotension, and increased myocardial oxygen demand situations

Intraoperative monitoring should include a standard five-lead electrocardiographic (ECG) system, including a V_5 lead, because of the LV's vulnerability to ischemia. A practical constraint in terms of interpretation is that these patients usually exhibit ECG changes because of preoperative LVH. The associated ST-segment abnormalities (ie, strain pattern) may be indistinguishable from or very similar to those of myocardial ischemia, making the intraoperative interpretation difficult. Lead II and possibly an esophageal electrocardiogram should be readily obtainable for assessing the P-wave changes in the event of supraventricular arrhythmias.

Hemodynamic monitoring is controversial, and few prospective data are available on which to base an enlightened clinical decision. The central venous pressure (CVP) is a particularly poor estimate of left ventricular filling when left ventricular compliance is reduced. A normal CVP can significantly underestimate the LVEDP or pulmonary capillary wedge pressure (PCWP). The principal risks, although minimal, of using a pulmonary artery catheter (PAC) in the patient with AS are arrhythmia-induced hypotension and ischemia. Loss of synchronous atrial contraction or a supraventricular tachyarrhythmia can compromise diastolic filling of the poorly compliant LV, resulting in hypotension and the potential for rapid hemodynamic deterioration. The threat of catheter-induced arrhythmias is significant for the patient with AS. However, accepting a low-normal CVP as evidence of good ventricular function can lead to similarly catastrophic underfilling of the LV on the basis of insufficient replenishment of surgical blood loss. To some extent, even the PCWP can underestimate the LVEDP (and LVEDV) when ventricular compliance is markedly reduced. Placement of a PAC also allows measurement of CO, derived hemodynamic parameters, mixed venous oxygen saturation (SvO_2), and possible transvenous pacing.

Intraoperative fluid management should be aimed at maintaining appropriately increased left-sided filling pressures. This is one reason why many clinicians think that the PAC is worth its small arrhythmogenic risk. Keeping up with intravascular volume losses is particularly important in noncardiac surgery, in which the shorter duration of the operation may make inhalation or potentially vasodilating regional anesthesia preferable to a narcotic technique.

Patients with symptomatic AS are usually encountered only in the setting of cardiovascular surgery because of their ominous prognosis without AVR. Few studies have specifically addressed the response of these patients to the standard intravenous and inhalation induction agents; however, the responses to narcotic and nonnarcotic intravenous agents are apparently not dissimilar from those of patients with other forms of VHD. The principal benefit of a narcotic induction is assurance of an adequate depth of anesthesia during intubation, which reliably blunts potentially deleterious reflex sympathetic responses capable of precipitating tachycardia and ischemia.

Many clinicians also prefer a pure narcotic technique for maintenance. The negative inotropy of inhalation anesthetics is a theoretical disadvantage for a myocardium

faced with the challenge of overcoming outflow tract obstruction. A more clinically relevant drawback may be the increased risk for arrhythmia-induced hypotension, particularly that associated with nodal rhythm and resultant loss of the atrium's critical contribution to filling of the hypertrophied ventricle.

Occasionally, surgical stimulation elicits a hypertensive response despite the impedance posed by the stenotic valve and a seemingly adequate depth of narcotic anesthesia. In these patients, a judicious trial of low concentrations of an inhalation agent, used purely for control of hypertension, may prove efficacious. The ability to concurrently monitor CO is useful in this situation. The temptation to control intraoperative hypertension with vasodilators should be resisted in most cases. Given the risk for ischemia, nitroglycerin seems to be a particularly attractive drug. Its effectiveness in relieving subendocardial ischemia in patients with AS is controversial; however, there is always the risk for transient episodes of overshoot. The hypertrophied ventricle's critical dependence on an adequate CPP may be unforgiving of even a momentary dip in the systemic arterial pressure.

Intraoperative hypotension, regardless of the primary cause, should be treated immediately and aggressively with a direct α-adrenergic agonist such as phenylephrine. The goal should be to immediately restore the CPP and then to address the underlying problem (eg, hypovolemia, arrhythmia). After the arterial pressure responds, treatment of the precipitating event should be equally aggressive, but rapid transfusion or cardioversion should not delay the administration of a direct-acting vasoconstrictor. Patients with severe AS in whom objective signs of myocardial ischemia persist despite restoration of the blood pressure should be treated extremely aggressively. This may mean the immediate use of an inotropic agent or accelerating the institution of cardiopulmonary bypass (CPB).

HYPERTROPHIC CARDIOMYOPATHY

Hypertrophic Obstructive Cardiomyopathy

Obstructive hypertrophic cardiomyopathy (HCM) is a relatively common genetic malformation of the heart with a prevalence of approximately 1 case in 500 births. The hypertrophy initially develops in the septum and extends to the free walls, often giving a picture of concentric hypertrophy. Asymmetric septal hypertrophy leads to a variable pressure gradient between the apical left ventricular chamber and the LVOT. The LVOT obstruction leads to increases in left ventricular pressure, which fuels a vicious cycle of further hypertrophy and increased LVOT obstruction.

Treatment modalities include β-adrenoceptor antagonists, calcium channel blockers, and surgical myectomy of the septum. For more than 40 years, the standard treatment has been the ventricular septal myotomy-myectomy of Morrow, in which a small amount of muscle from the subaortic septum is resected. Two new treatment modalities have gained popularity in recent years: dual-chamber pacing and septal reduction (ie, ablation) with ethanol.

Clinical Features and Natural History

The clinical presentation of patients varies widely. Echocardiography has unquestionably increased the number of asymptomatic patients who carry the diagnosis. Most patients with HCM are asymptomatic and have been seen by the echocardiographer because of relatives having clinical disease. Follow-up remains an important problem for

cardiologists because sudden death or cardiac arrest may occur as the presenting symptom in slightly more than one-half of previously asymptomatic patients.

Less dramatic presenting complaints include dyspnea, angina, and syncope. The clinical picture is often similar to valvular AS. The symptoms may share a similar pathophysiologic basis (eg, poor diastolic compliance) in the two conditions. The prognostic implications of clinical disease, however, are less certain for patients with HCM. Although cardiac arrest may be an unheralded event, some patients may have a stable pattern of angina or intermittent syncopal episodes for many years. Palpitations are frequently described and may be related to a variety of underlying arrhythmias.

Pathophysiology

In HCM, the principal pathophysiologic abnormality is myocardial hypertrophy. The hypertrophy is a primary event in these patients and occurs independently of outflow tract obstruction. Unlike in AS, the hypertrophy begets the pressure gradient, not the other way around. Histologically, the hypertrophy consists of myocardial fiber disarray, and anatomically, there is usually disproportionate enlargement of the interventricular septum.

HCM is characterized by a broad spectrum of obstruction, which is absent in some patients and varies from mild to severe in others. The most distinctive qualities of obstruction are its dynamic nature (ie, depends on contractile state and loading conditions), its timing (ie, begins early and peaks variably), and its subaortic location. Subaortic obstruction arises from the hypertrophied septum's encroachment on the systolic outflow tract, which is bounded anteriorly by the interventricular septum and posteriorly by the anterior leaflet of the mitral valve. In most patients with obstruction, exaggerated anterior (ie, toward the septum) motion of the anterior mitral valve leaflet during systole accentuates the obstruction. The cause of systolic anterior motion (SAM) is unclear. One possibility is that the mitral valve is pulled toward the septum by contraction of the papillary muscles, whose orientation is abnormal because of the hypertrophic process. Another theory is that vigorous contraction of the hypertrophied septum results in rapid acceleration of the blood through a simultaneously narrowed outflow tract. The generated hydraulic forces (consistent with a Venturi effect) can cause the anterior leaflet of the mitral valve to be drawn close to or in contact with the interventricular septum (Fig. 15.2). After the obstruction is triggered, the mitral valve leaflet is forced against the septum by the pressure difference across the orifice. However, the pressure difference further decreases orifice size and further increases the pressure difference in a time-dependent, amplifying feedback loop. This analysis is consistent with observations that the measured gradient directly correlates with the duration of mitral-septal contact. Although still controversial, there appears to be good correlation between the degree of SAM and the magnitude of the pressure gradient. The SAM-septal contact also underlies the severe subaortic obstruction characteristic of HCM of the elderly, although the narrowing usually is more severe and the contribution of septal movement toward the mitral valve is usually greater.

In addition to SAM, approximately two-thirds of patients exhibit a constellation of structural malformations of the mitral valve. Malformations include increased leaflet area and elongation of the leaflets or anomalous papillary muscle insertion directly into the anterior mitral valve leaflet. HCM is not a disease process confined to cardiac muscle alone because these anatomic abnormalities of the mitral valve are unlikely to be acquired or caused by mechanical factors.

Three basic mechanisms—increased contractility, decreased afterload, and decreased preload—exacerbate the degree of SAM-septal contact and produce the dynamic

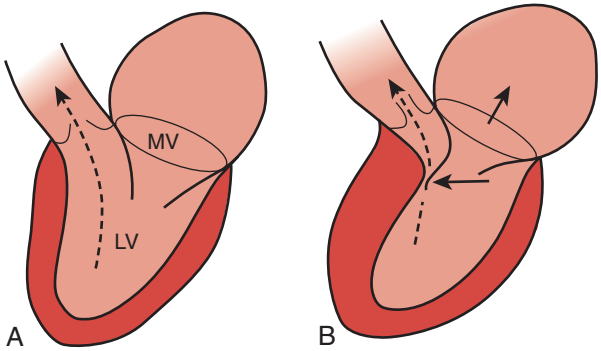


Fig. 15.2 Proposed mechanism of systolic anterior motion in hypertrophic cardiomyopathy. (A) Normally, blood is ejected from the left ventricle *(LV)* through an unimpeded outflow tract. (B) Thickening of the ventricular septum restricts the outflow tract, and the obstruction causes the blood to be ejected at a higher velocity and closer to the area of the anterior mitral valve *(MV)* leaflet. Owing to its proximity to the high-velocity fluid path, the anterior MV leaflet is drawn toward the hypertrophied septum by a Venturi effect *(left arrow)*. (From Wigle ED, Sasson Z, Henderson MA, et al. Hypertrophic cardiomyopathy: the importance of the site and the extent of hypertrophy—a review. *Prog Cardiovasc Dis*. 1985;28:1.)

obstruction characteristic of patients with HCM. The common pathway is a reduction in ventricular volume (actively by increased contractility directly or reflexively in response to vasodilation or passively by reduced preload), which increases the proximity of the anterior mitral valve leaflet to the hypertrophied septum. Factors that usually impair contractile performance, such as myocardial depression, systemic vasoconstriction, and ventricular overdistension, characteristically improve systolic function in patients with HCM and outflow tract obstruction.

Diagnostically, the paradoxes are exploited by quantifying the degree of subaortic obstruction after isoproterenol (eg, increased inotropy, tachycardia, decreased volume) and the Valsalva maneuver (eg, decreased venous return, ventricular volume), both of which reliably elicit increases in the pressure gradient. In the operating room, catheter-induced ectopy or premature ventricular contractions resulting from cardiac manipulation may transiently exacerbate the gradient by increased inotropy from postextrasystolic potentiation. Therapeutically, volume loading, myocardial depression, and vasoconstriction can minimize obstruction and augment forward flow.

Poor diastolic compliance is the most clinically apparent manifestation of the relaxation abnormalities. Left ventricular filling pressures are markedly increased despite enhanced systolic ejection and a normal or subnormal EDV. The reduced ventricular volume emphasizes the pivotal role played by the hypertrophied but intrinsically depressed myocardium. Reductions in afterload, which are mediated by hypertrophy, support the ventricle's systolic performance, resulting in increased emptying and a small diastolic volume. However, hypertrophy also impairs relaxation, resulting in poor diastolic compliance and an increased ventricular filling pressure. The high filling pressure does not reflect distension of a failing ventricle, although stress-volume relationships suggest that contractility is intrinsically depressed. This disease is characterized by systolic and diastolic dysfunction.

As in patients with valvular AS, relatively high filling pressures reflect the LVEDV (ie, degree of preload reserve) needed to overcome the outflow obstruction. Intervention with vasodilators is therefore inappropriate. The poor ventricular compliance also means that patients with HCM depend on a large intravascular volume and the maintenance of sinus rhythm for adequate diastolic filling. The atrial contribution

to ventricular filling is even more important in HCM than in valvular AS, and it may approach 75% of total SV.

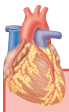
Another similarity between HCM and valvular AS is that the combination of myocardial hypertrophy, with or without LVOT obstruction, may precipitate imbalances in the myocardial oxygen supply and demand relationship. Angina-like discomfort is a classic symptom of patients with HCM, and its pathogenesis has been attributed to increases in $M\dot{V}O_2$, specifically the increased overall muscle mass and the high systolic wall tension generated by the ventricle's ejection against the dynamic subaortic obstruction. However, as in patients with AS, there is evidence of a compromise in myocardial oxygen supply.

β-Blockers and calcium channel blockers form the basis of medical therapy for HCM. β-Blockade is most useful for preventing sympathetically mediated increases in the subaortic gradient and for the prevention of tachyarrhythmias, which can exacerbate outflow obstruction. Disopyramide also has been used to reduce contractility and for its antiarrhythmic properties. Calcium channel blockers often prove clinically effective in patients with HCM, regardless of the presence or absence of systolic obstruction. The mechanism of action involves improvement in diastolic relaxation, allowing an increase in LVEDV at a lower LVEDP. The negative inotropy may attenuate the subaortic pressure gradient, but in selected patients the gradient may worsen because of pronounced and unpredictable degrees of vasodilation.

Surgery (ie, septal myotomy or partial myomectomy by the aortic approach) is reserved for patients who remain symptomatic despite maximal pharmacologic therapy. In a long-term retrospective study, the cumulative survival rate was significantly better in surgically than in pharmacologically treated patients. However, it is likely that pharmacologic therapy may be more appropriate for the patient with a dynamic component to their degree of subaortic obstruction. Further improvement in the clinical outcome of surgically treated patients may be achieved with the addition of verapamil, presumably reflecting a two-pronged attack on the systolic (ie, myomectomy) and diastolic (ie, verapamil) components of the disease. Enthusiasm continues for the therapeutic use of dual-chamber pacing in this disease, with some patients demonstrating reductions in their subaortic gradients. It is not an option for patients with atrial fibrillation (AF).

Anesthesia Considerations

Priorities in anesthesia management are to avoid aggravating the subaortic obstruction while remaining aware of the derangements in diastolic function that may be somewhat less amenable to direct pharmacologic manipulation (Box 15.2). It is necessary to maintain an appropriate intravascular volume while avoiding direct or reflex increases in contractility or heart rate. The latter goals can be achieved with a deep level of general anesthesia and the associated direct myocardial depression. Regardless of the



BOX 15.2 ***Hypertrophic Cardiomyopathy***

Preload is increased
Afterload is increased
Goal is myocardial depression
Avoid tachycardia, inotropes, and vasodilators

specific technique, preservation of an adequate CPP using vasoconstrictors rather than inotropes is necessary to avoid myocardial ischemia. Heavy premedication is advisable, with a view to avoiding anxiety-induced tachycardia or a reduction in ventricular filling. Chronic β-blockade or calcium channel blockade, or both, should be continued up to and including the day of surgery. These medications should be restarted immediately after surgery, particularly in patients undergoing noncardiac surgery.

Intraoperative monitoring should include an ECG system with the capability of monitoring a V_5 lead and each of the six limb leads. Inspection of lead II may be helpful in the accurate diagnosis of supraventricular and junctional tachyarrhythmias, which may precipitate catastrophic hemodynamic deterioration because of the potential for inadequate ventricular filling resulting from the reduction in diastolic time or loss of the atrial contribution to ventricular filling. The latter may be crucial in patients with significantly reduced diastolic compliance. Abnormal Q waves have been described on the electrocardiograms of 20% to 50% of patients with HCM. These waves should not raise concern about a previous myocardial infarction; instead, they probably represent accentuation of normal septal depolarization or delay in depolarization of electrophysiologically abnormal cells. Some patients exhibit a short PR interval with initial slurring of the QRS complex, and they may be at increased risk for supraventricular tachyarrhythmias due to preexcitation. Although the specific predisposing factors are unknown, patients with HCM are at increased risk for any type of arrhythmia in the operative setting.

Given the pronounced abnormalities in left ventricular diastolic compliance, the CVP is likely to be an inaccurate guide to changes in left ventricular volume. However, a CVP catheter is extremely useful for the prompt administration of vasoactive drugs if they become necessary. As in valvular AS, the information provided by insertion of a PAC is worth the small arrhythmogenic risk. The potential for hypovolemia-induced exacerbation of outflow tract obstruction makes it crucial that the clinician have an accurate gauge of intravascular filling. Reduced diastolic compliance means that the PCWP overestimates the patient's true volume status, and a reasonable clinical objective is to maintain the PCWP in the high-normal to elevated range. A PAC with pacing capability is ideal because atrial overdrive pacing can effect immediate hemodynamic improvement in the event of episodes of junctional rhythm. The absolute requirement of these patients for an adequate preload cannot be overemphasized because even abrupt positioning changes have resulted in acute hemodynamic deterioration, including acute pulmonary edema.

Intraoperative arrhythmias require aggressive therapy. During cardiac surgery, insertion of venous cannulas may precipitate atrial arrhythmias. Because the resultant hypotension may be severe, the surgeon should cannulate the aorta before atrial manipulation. Supraventricular or junctional tachyarrhythmias may require immediate cardioversion if they precipitate catastrophic degrees of hypotension. Although verapamil is one drug of choice for paroxysmal atrial and junctional tachycardia, it can disastrously worsen the LVOT obstruction if it elicits excessive vasodilation or it is used in the setting of severe hypotension. Cardioversion is preferable when the mean arterial pressure is already very low. The concurrent administration of phenylephrine also is advisable. This drug is typically a low-risk, high-yield choice for the hypotensive patient with HCM. It augments perfusion, may ameliorate the pressure gradient, and often elicits a potentially beneficial vagal reflex when used to treat tachyarrhythmia-induced hypotension.

The inhalation anesthetics commonly are used for patients with HCM. Their dose-dependent myocardial depression is ideal because negative inotropy reduces the degree of SAM-septal contact, which reduces LVOT obstruction. Hypotension is usually

the result of underlying hypovolemia, which is potentially exacerbated by anesthetic-induced vasodilation. Inotropes, β-adrenergic agonists, and calcium are contraindicated because they worsen the systolic obstruction and perpetuate the hypotension. In most cases, a beneficial response can be obtained with aggressive replenishment of intravascular volume and concurrent infusion of phenylephrine.

AORTIC REGURGITATION

Clinical Features and Natural History

AR may result from an abnormality of the valve itself, bicuspid anatomy, a rheumatic or infectious origin, or in association with any condition producing dilation of the aortic root and leaflet separation. Nonrheumatic valvular diseases commonly resulting in AR include infective endocarditis, trauma, and connective tissue disorders such as Marfan syndrome or cystic medial necrosis of the aortic valve. Aortic dissection from trauma, hypertension, or chronic degenerative processes also can result in dilation of the root and functional incompetence.

The natural history of chronic AR is that of a long asymptomatic interval during which the valvular incompetence and secondary ventricular enlargement become progressively more severe. When symptoms do appear, they are usually those of CHF, and chest pain, if it occurs, is often nonexertional in origin. The life expectancy for patients with significant disease has historically been about 9 years, and, in contrast with AS, the onset of symptoms because of AR does not portend an immediately ominous prognosis. In the absence of surgery, early recognition of AR and chronic use of vasodilators prolong the life span for this patient population.

A relatively unique and problematic feature of chronic AR is that the severity of symptoms and their duration may correlate poorly with the degree of hemodynamic and contractile impairment. The issue in surgical decision making is that many patients can remain asymptomatic, during which time they are undergoing progressive deterioration in myocardial contractility. Noninvasive diagnostic studies (ie, radionuclide cine angiography and two-dimensional and Doppler echocardiographic assessment of response to pharmacologic afterload stress) may facilitate the detection of early derangements in contractile function in relatively asymptomatic patients. These findings are important to the cardiologist when considering surgical referral because patients with depressed preoperative left ventricular function have greater perioperative mortality rates and are at increased risk for persistent postoperative heart failure (HF).

As in acute MR, the physiology of acute AR is quite different from chronic AR. Common causes include endocarditis, trauma, and acute aortic dissection. Because of a lack of chronic compensation, these patients usually have pulmonary edema and heart failure refractory to optimal medical therapy. Patients are often hypotensive and clinically appear to be on the verge of cardiovascular collapse.

Pathophysiology

Left ventricular volume overload is the pathognomonic feature of chronic AR. The degree of volume overload is determined by the magnitude of the regurgitant flow, which is related to the size of the regurgitant orifice, the aorta-ventricular pressure gradient, and the diastolic time.

Chronically, AR results in a state of left ventricular volume and pressure overload. Progressive volume overloading from AR increases end-diastolic wall tension (ie, ventricular afterload) and stimulates the serial replication of sarcomeres, producing

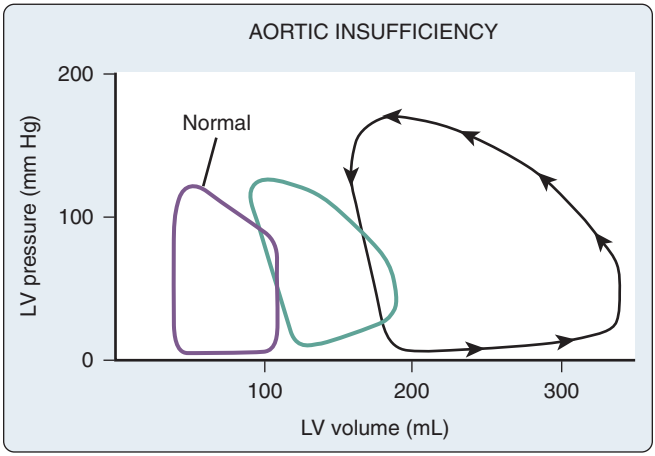


Fig. 15.3 Pressure-volume loop in aortic regurgitation (AR). Acute AR *(green loop)*; chronic AR *(black loop)*. *LV*, Left ventricular. (Modified from Jackson JM, Thomas SJ, Lowenstein E. Anesthetic management of patients with valvular heart disease. *Semin Anesth*. 1982;1:239.)

a pattern of eccentric ventricular hypertrophy. In accordance with Laplace's law, dilation of the ventricle increases the systolic wall tension, stimulating some concentric hypertrophy. The result is normalization of the ratio of ventricular wall thickness to cavitary radius. This process of eccentric hypertrophy results in the greatest absolute degrees of cardiomegaly seen in valve disease. EDV may be three to four times normal, and very high COs can be sustained.

Fig. 15.3 shows the pressure-volume loops for acute and chronic AR. In the chronic form, the diastolic pressure-volume curve is shifted far to the right. This permits a tremendous increase in LVEDV with minimal change in filling pressure, a property frequently described as high diastolic compliance.

Because the increase in preload is compensated for by ventricular hypertrophy, CO is maintained by the Frank-Starling mechanism, and cardiac failure is not seen despite probable decreases in contractility. There is virtually no isovolumic diastolic phase because the ventricle is filling throughout diastole. The isovolumic phase of systole also is brief because of the low aortic diastolic pressure. Minimal impedance to the forward ejection of a large SV allows performance of maximal myocardial work at a minimum of oxygen consumption. Eventually, however, progressive volume overload increases ventricular EDV to the point that compensatory hypertrophy is no longer sufficient to compensate, and a decline in systolic function occurs. As systolic function declines, the end-systolic dimension increases further, left ventricular wall stress increases, and left ventricular function is further compromised by the excessive ventricular afterload. At this point, the decline of ventricular function is progressive and can be quite rapid.

Despite the relatively normal $M\dot{V}O_2$, angina can occur in one-third of patients with severe AR, even in the absence of CAD. Patients with chronic AR may be at risk for myocardial ischemia caused by hypertrophy-induced abnormalities of the coronary circulation. The increase in total myocardial mass can increase baseline $M\dot{V}O_2$, and there is evidence that total coronary blood flow, although increased, fails to keep pace with the increase in myocardial mass. Evidence suggests that the insidious development of contractile dysfunction may in part have an ischemic basis.

Intraoperatively, patients with chronic AR may be at risk for acute ischemia with episodes of significant bradycardia. As bradycardia prolongs diastolic time, it increases

regurgitant flow, and left ventricular diastolic pressure and wall tension increase rapidly. Simultaneously, the CPP is decreased as aortic runoff occurs during diastole and diastolic ventricular pressure is increased. Under these conditions, myocardial perfusion pressure may be insufficient. Clinically, very rapid decompensation can occur. The ischemic ventricle can dilate rapidly such that progressively increased end-systolic dimensions are seen, and ischemia and ventricular failure become a positive feedback loop.

Surgical Decision Making

An accurate assessment of contractility is crucial to surgical decision making, because the clinical history of chronic AR may be an unreliable index of ventricular function. Asymptomatic patients may have ventricular dysfunction, whereas symptomatic patients may be free of myocardial depression. A variety of prognostic indicators have been used to identify early ventricular dysfunction as a trigger for surgical intervention. Clinical status, such as exercise capacity and New York Heart Association (NYHA) class, and noninvasive and invasive laboratory tests have been used. Hemodynamic parameters such as the end-systolic stress-volume relationship and estimates of the left ventricular contractile state have been evaluated as predictors of worsening left ventricular function.

Valve surgery is recommended for asymptomatic patients with left ventricular systolic dysfunction. Surgery also should be considered if ventricular dilation has occurred in the asymptomatic patient, even if the EF is normal. In patients who are symptomatic but have normal ventricular function, the ACC and AHA recommend further evaluation for an unrelated cause and observation. In these cases, serial echocardiographic assessment is appropriate. Symptomatic patients with left ventricular dysfunction should undergo surgery.

Acute Aortic Regurgitation

In acute AR, sudden diastolic volume overload of a nonadapted LV results in a precipitous increase in the EDP because the ventricle is operating on the steepest portion of the diastolic pressure-volume curve. In severe acute AR, the LVEDP can equilibrate with aortic diastolic pressure and exceed the left atrial pressure in late diastole. This may be sufficient to cause closure of the mitral valve before atrial systole. This is an important echocardiographic finding indicative of severe AR. Although this phenomenon initially shields the pulmonary capillaries from the full force of the dramatically increased LVEDP, the protection may be short lived. Severe left ventricular distension often follows and produces mitral annular enlargement and functional MR.

The inevitable decline in SV in acute decompensating AR elicits a reflex sympathetic response, making tachycardia and a high systemic vascular resistance common. Moderate tachycardia beneficially shortens the regurgitant time without reducing the transmitral filling volume. Vasoconstriction, however, preserves CPP at the expense of increasing the aortic-ventricular gradient and regurgitation.

Patients with acute AR may be at greater risk for myocardial ischemia. As with chronic AR and bradycardia, coronary perfusion may be compromised by the combination of a low diastolic arterial pressure and the precipitously increased LVEDP. Narrowing of CPP may be so severe that the phasic epicardial blood flow may change to a predominantly systolic pattern with severe acute AR. Dissection of the coronary ostia is rare but frequently causes the death of patients with acute AR. In addition to the structural impediment to myocardial oxygen delivery, catastrophic hypotension and

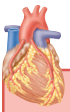
high LVEDP combine to cause accentuated ischemia and ventricular dilation. Immediate surgical correction is the only hope for salvaging these patients, who often prove refractory to inotropes and vasodilators. Attempts at stabilizing the ischemic component of their injury with an intraaortic balloon are usually contraindicated because augmenting the diastolic pressure worsens regurgitation.

Acute AR most commonly results from infective endocarditis or aortic dissection, and intraoperative transesophageal echocardiography (TEE) has assumed increasing importance in the diagnosis of acute AR and in decisions regarding its surgical management. Transesophageal echocardiographic studies are highly sensitive and specific for the diagnosis of infective endocarditis and are significantly more sensitive than transthoracic echocardiography. TEE is particularly useful in the diagnosis of abscesses associated with endocarditis and may detect previously unsuspected abnormalities.

Anesthesia Considerations

Intraoperative monitoring should include an ECG system for monitoring a lateral precordial lead because ischemia is a potential hazard (Box 15.3). For most valvular procedures, a PAC provides useful information. A PAC allows determination of basal filling pressures and CO, which is particularly useful in chronic AR given the potential unreliability of the clinical history and EF. Equally important is the ability to accurately monitor ventricular preload and CO response to pharmacologic interventions. The aggressive use of vasodilators often is appropriate therapy perioperatively for the failing ventricle, but their use can compromise the preload to which the ventricle has chronically adjusted. Concurrent preload augmentation, guided by the diastolic pulmonary arterial pressure (PAP) or PCWP, may be crucial to optimize CO when afterload is pharmacologically manipulated. The other requirement for a PAC is to allow for pacing when it is anticipated. The deleterious effects of significant bradycardia in AR have been described. In patients who arrive in the operating room with heart rates less than 70 beats per minute or patients for whom rapid epicardial pacing may be difficult to establish (eg, reoperations), placement of a pacing wire probably is indicated. Typically, only a ventricular wire is appropriate. It is more reliable than atrial pacing, and in AR, the atrial contribution to ventricular diastolic volume usually is not essential. Capturing the ventricle with a PAC-based, transvenous wire can be difficult because of the very large ventricular cavity size in patients with chronic AR.

Because patients with AR may have widely different degrees of myocardial dysfunction, anesthesia management must be appropriately individualized. For cardiac or noncardiac surgery, the hemodynamic goals are a mild tachycardia, positive inotropic state, and controlled reduction in systemic vascular resistance. For cardiac surgery, dopamine or dobutamine, pancuronium, ketamine, and nitroprusside infusions are excellent choices. For the patient with acute AR, the goals are the same, but urgency



BOX 15.3 ***Aortic Regurgitation***

Preload is increased
Afterload is decreased
Goal is augmentation of forward flow
Avoid bradycardia

must be stressed. It is essential to rapidly reduce end-diastolic and end-systolic ventricular volumes with the very aggressive use of inotropes (eg, epinephrine) and vasodilators. There is sometimes concern that inotropes may exacerbate the root dissection in acute AR by increasing the shear force on the aortic wall. Despite this theoretic concern, positive inotropes should not be withheld from the patient who deteriorates in the operating room because they may provide the precious additional minutes of hemodynamic stability needed to get on CPB.

In acute and chronic forms of AR, serial measurements of CO can indicate that ventricular size and CO have been optimized, regardless of the systemic pressure. TEE is useful to look at ventricular size, but probably maximizing CO under these conditions gets closer to the therapeutic goal than looking at ventricular size alone. With acute AR and premature closure of the mitral valve, PAPs may grossly underestimate the LVEDP, which continues to increase under the influence of the diastolic regurgitant jet from the aorta.

The early and late phases of CPB can be a problem, particularly in reoperations. Before cross-clamp placement, the ventricle is at risk for distension if it is not ejecting or being vented. If the ventricle dilates with AR during CPB, the intraventricular pressures may equilibrate with the aortic root pressures. Under these conditions, there is no coronary perfusion, and the ventricle may dilate rapidly and become profoundly ischemic. This can occur before cross-clamp placement with bradycardia, ventricular fibrillation, or tachycardia or with a rapid supraventricular rhythm that compromises organized mechanical activity. Correcting the rhythm, pacing, cross-clamping the aorta, or venting the ventricle addresses the problem. This also can occur in cardiac surgery for conditions other than AR. In patients with unknown or uncorrected AR, removal of the cross-clamp causes the same ventricular dilation and ischemia if the rhythm and ejection are not rapidly established. Ventricular venting or pacing may be essential until an organized, mechanically efficient, rhythm is established. This problem must be considered in patients referred for CABG alone, in those with mild or moderate AR not having AVR, and in patients for whom intraoperative TEE is not used.

MITRAL STENOSIS

Clinical Features and Natural History

Clinically significant MS in adult patients usually is a result of rheumatic disease. Congenital abnormalities of the mitral valve are a rare cause of MS in younger patients. Other uncommon conditions that do not directly involve the mitral valve apparatus but may limit left ventricular inflow and simulate the clinical findings of MS include cor triatriatum, large left atrial neoplasms, and pulmonary vein obstruction.

A decades-long asymptomatic period characterizes the initial phase of rheumatic MS. Symptoms rarely appear until the normal mitral valve area (MVA) of 4 to 6 cm^2 (Fig. 15.4) has been reduced to 2.5 cm^2 or less. When the MVA reaches 1.5 to 2.5 cm^2, symptoms usually occur only in association with exercise or conditions such as fever, pregnancy, or AF that lead to increases in heart rate or CO. After the MVA decreases to less than 1.5 cm^2, symptoms may develop at rest. Some patients are able to remain asymptomatic for long periods by gradually reducing their level of activity. Patients with MS commonly report dyspnea as their initial symptom, a finding reflective of increased left atrial pressure and pulmonary congestion. In addition to dyspnea, patients may report palpitations that signal the onset of AF. Systemic thromboembolization occurs in 10% to 20% of patients with MS and does not appear to be correlated

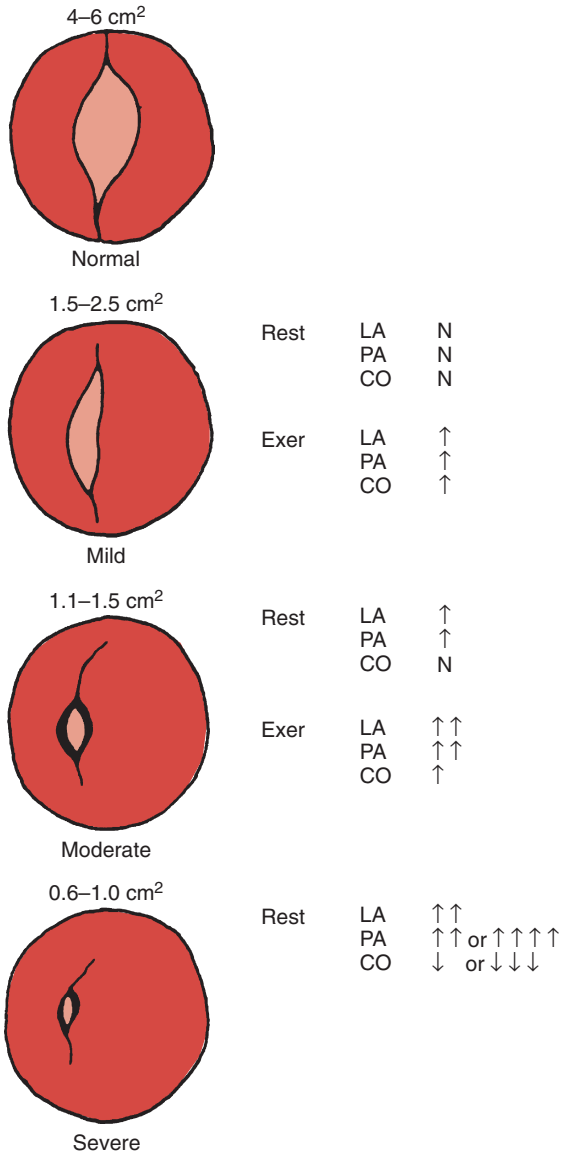


Fig. 15.4 Hemodynamic changes with progressive narrowing of the mitral valve. *CO*, Cardiac output; *Exer*, exercise; *LA*, left atrium; *N*, normal; *PA*, pulmonary artery; ↑, increased; ↓, decreased. (From Rapaport E. Natural history of aortic and mitral valve disease. *Am J Cardiol*. 1971;35:221.)

with the MVA or left atrial size. Chest pain that simulates angina occurs in a small number of patients with MS and may result from right ventricular hypertrophy (RVH) rather than CAD.

There has been a change in the typical age at which patients are diagnosed with MS. Previously, patients, often women, with MS were identified while in their 20s and 30s. Since the early 1990s, perhaps because of more slowly progressive disease in the United States, patients have been diagnosed in their 40s and 50s.

After symptoms develop, MS remains a slow, progressive disease. Patients often live 10 to 20 years with mild symptoms, such as dyspnea with exercise, before disabling NYHA class III and IV symptoms develop. The symptomatic state of the patient predicts the clinical outcome. For instance, the 10-year survival rate of patients with mild symptoms approaches 80%, but the 10-year survival rate of patients with disabling symptoms is only 15% without surgery.

Pathophysiology

Rheumatic MS results in valve leaflet thickening and fusion of the commissures. Later in the disease process, leaflet calcification and subvalvular chordal fusion may occur. These changes combine to reduce the effective MVA and limit diastolic flow into the LV. As a result of the fixed obstruction to left ventricular inflow, left atrial pressures increase. Elevated left atrial pressures limit pulmonary venous drainage and result in increased PAPs. Over time, pulmonary arteriolar hypertrophy develops in response to chronically increased pulmonary vascular pressures. Pulmonary hypertension may trigger increases in right ventricular end-diastolic volume (RVEDV) and pressure (RVEDP), and some patients may have signs of right ventricular failure such as ascites or peripheral edema. Left atrial enlargement is an almost universal finding in patients with established MS and is a risk factor for AF.

Patients with MS tolerate tachycardia particularly poorly. Left ventricular inflow, already limited by a mechanically abnormal valve, is further compromised by the disproportionate decline in the diastolic period that accompanies tachycardia. The flow rate across the stenotic valve must increase to maintain left ventricular filling in a shorter diastolic period. Because the valve area remains constant, the pressure gradient between the LA and LV increases by the square of the increase in the flow rate, according to the Gorlin formula, in which PG is the transvalvular pressure gradient:

$$\text{Valve area} = \text{Transvalvular flow rate}/\text{Constant} \times \sqrt{\text{PG}}$$

Tachycardia necessitates a significant increase in the transvalvular pressure gradient and may precipitate feelings of breathlessness in awake patients. In patients with AF, it is the increased ventricular rate that is most deleterious, rather than the loss of atrial contraction. Although coordinated atrial activity is always preferable, the primary goal in treating patients with MS and AF should be control of the ventricular rate.

MS results in diminished left ventricular preload reserve. As seen in the pressure-volume loop in Fig. 15.5, LVEDV and LVEDP are reduced, with an accompanying decline in SV. Controversy exists regarding the contractile state of the LV in these patients. Limited preload may contribute to a reduced EF in some of these patients. However, the observation that left ventricular contractile impairment persists after surgery in some patients suggests that other causes of left ventricular dysfunction may exist. Rheumatic myocarditis has been reported, although its role in producing left ventricular contractile dysfunction is uncertain.

Surgical Decision Making

Appropriate referral of patients for surgical intervention requires integration of clinical and echocardiographic data. Patients with severe symptoms (ie, NYHA class III and IV) should be immediately referred for surgery because the outcome is poor if treated medically. Patients with only mild MS and few or no symptoms may be treated conservatively with periodic evaluation. Patients who are asymptomatic but have

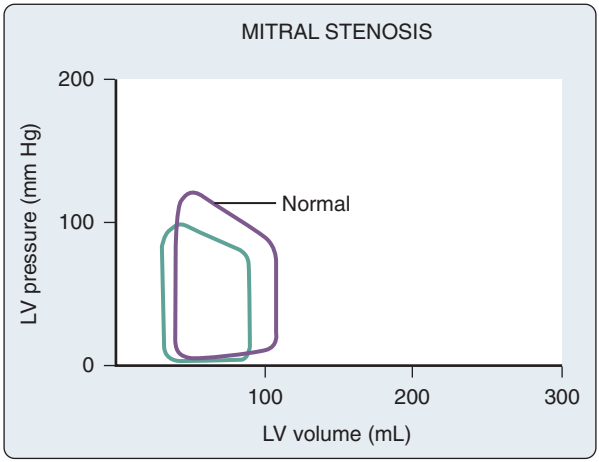


Fig. 15.5 Pressure-volume loop *(green)* for mitral stenosis. LV, Left ventricular. (From Jackson JM, Thomas SJ, Lowenstein E. Anesthetic management of patients with valvular heart disease. *Semin Anesth*. 1982;1:239.)

moderate MS (ie, MVA between 1.0 and 1.5 cm^2) require careful assessment. If significant pulmonary hypertension (ie, pulmonary artery systolic pressure >50 mm Hg) is identified, surgical intervention should be considered. Intervention also may be indicated if a patient becomes symptomatic or PAPs increase significantly during exercise testing.

The surgical options for treating MS continue to evolve. Closed commissurotomy, in which the surgeon fractures fused mitral commissures, was first performed in the 1920s. It became popular in the 1940s and still is used to treat MS in developing countries. With the advent of CPB in the 1950s, techniques of open commissurotomy developed, allowing the surgeon to directly inspect the valve before splitting the commissures. The common goals of closed and open mitral commissurotomy include increasing the effective MVA and decreasing the left atrial to left ventricular pressure gradient, with a resultant relief in the patient's symptoms.

Percutaneous mitral commissurotomy (PMC) allows a less invasive, catheter-based approach to MS. It was first reported by Inoue and coworkers in 1984, and clinicians worldwide perform PMC more than 10,000 times each year. The technique of PMC involves directing a balloon-tipped catheter across the stenotic mitral valve. Specifically designed balloons allow sequential inflation of the distal and proximal portions of the balloon, ensuring correct positioning across the mitral valve before the middle portion of the device is inflated to split the fused commissures. Patient selection for PMC requires careful echocardiographic evaluation.

Not all patients are candidates for surgical commissurotomy or PMC. Those with heavily calcified valves or significant MR are likely to experience suboptimal results after commissurotomy. Mitral valve anatomy unsuitable for PMC is more commonly encountered in Western countries, where patients with MS typically are diagnosed at an older average age. Mitral valve replacement commonly is recommended for these patients. The risk for mitral valve replacement depends on patient characteristics such as age, functional status, and other comorbid conditions. Surgical risk in younger patients with few coexisting medical problems usually is less than 5%. Conversely, surgical risk in elderly patients with severe symptoms related to MS and multiple comorbidities may be 10% to 20%.

MITRAL REGURGITATION

Clinical Features and Natural History

Unlike mitral stenosis (MS), which is usually the result of rheumatic valve disease, MR may result from a variety of disease processes that affect the valve leaflets, chordae tendineae, papillary muscles, valve annulus, or LV. MR can be classified as organic or functional. Organic MR describes diseases that result in distortion, disruption, or destruction of the mitral leaflets or chordal structures. In Western countries, degenerative processes that lead to leaflet prolapse with or without chordal rupture are the most common cause of MR. Other causes of organic MR include infective endocarditis, mitral annular calcification, rheumatic valve disease, and connective tissue disorders such as Marfan or Ehlers-Danlos syndrome. Much less common causes of organic MR include congenital mitral valve clefts, diet-drug or ergotamine toxicity, and carcinoid valve disease with metabolically active pulmonary tumors or right-to-left intracardiac shunting.

Functional MR describes MR that occurs despite structurally normal leaflets and chordae tendineae. Resulting from altered function or geometry of the LV or mitral annulus, functional MR often occurs in the setting of ischemic heart disease, and the term *ischemic MR* is sometimes used interchangeably with *functional MR*. However, the functional form can occur in patients without demonstrable CAD, such as those with idiopathic dilated cardiomyopathy and mitral annular dilation. The term *ischemic MR* probably best applies to functional cases with a known ischemic cause. Rupture of a papillary muscle with acute, severe MR is somewhat more difficult to classify. Although usually a sequela of acute myocardial infarction (AMI) with normal leaflets and chordae, there is an obvious anatomic disruption of the mitral apparatus.

The natural history of MR varies because it can be caused by a wide variety of disease processes. Even among patients with acute-onset disease, the clinical course depends on the mechanism of regurgitation and the response to treatment. For instance, patients with acute, severe MR caused by a ruptured papillary muscle have a dismal outcome without surgery. However, the clinical course of acute MR caused by endocarditis can be favorable if the patient responds well to antibiotic therapy. Although those with chronic MR usually enter an initial, often asymptomatic, compensated phase, the time course for progression to left ventricular dysfunction and symptomatic heart failure is unpredictable. The literature reflects the wide variation in the natural history of MR, with 5-year survival rates for patients with MR of 27% to 97%.

Pathophysiology

MR causes left ventricular volume overload. The regurgitant volume combines with the normal left atrial volume and returns to the LV during each diastolic period. The increased preload leads to increased sarcomere stretch and, in the initial phases of the disease process, augmentation of LV ejection performance by the Frank-Starling mechanism. Systolic ejection into the relatively low-pressure left atrium (LA) further enhances the contractile appearance of the LV.

The clinical presentation of patients with MR depends on the pathophysiology of the specific condition, including the mechanism, severity, and acuity of the disease. In cases of acute, severe MR, such as in patients with a ruptured papillary muscle after AMI, the sudden increase in preload enhances left ventricular contractility by the Frank-Starling mechanism. Despite the increased preload, the size of the LV is initially normal. Normal left ventricular size combined with the ability to eject into a low-pressure circuit (ie, the LA) results in decreased afterload in the acute setting.

The measured left ventricular ejection fraction (LVEF) in cases of sudden, severe MR may approach 75%, although forward SV is reduced. However, because the LA has not yet dilated in response to the large regurgitant volume, left atrial pressure increases acutely and may lead to pulmonary vascular congestion, pulmonary edema, and dyspnea.

Many patients with MR, particularly those whose valvular incompetence develops more slowly, may enter a chronic, compensated phase. In this phase, chronic volume overload triggers left ventricular cavity enlargement by promoting eccentric hypertrophy. Increased preload continues to augment left ventricular systolic performance. At the same time, the LA dilates in response to the ongoing regurgitant volume. Although left atrial dilation maintains a low-pressure circuit that facilitates left ventricular systolic ejection, the increased radius of the left ventricular cavity leads to increased wall tension according to Laplace's law.

With the eventual decline in left ventricular systolic function, patients enter a decompensated phase. Progressive left ventricular dilation increases wall stress and afterload, causing further deterioration in left ventricular performance, mitral annular dilation, and worsening of the MR. Left ventricular end-systolic pressure increases. The increased left ventricular filling pressures result in increased left atrial pressures and, given time, pulmonary vascular congestion, pulmonary hypertension, and right ventricular dysfunction. In addition to fatigue and weakness, patients with decompensated, chronic MR also may report dyspnea and orthopnea. It is difficult to predict when a patient with MR is likely to decompensate clinically. Progression of disease in any patient depends on the underlying cause of MR, its severity, the response of the LV to volume overload, and possibly the effect of medical management. Because of the combination of increased preload and the ability to eject into the low-pressure LA, a normally functioning LV should display an increased EF in the setting of significant MR. Conversely, an EF considered normal in a patient with competent valves may represent diminished left ventricular function in the setting of MR. In patients with severe MR, an EF in the range of 50% to 60% likely represents significant left ventricular dysfunction and is an indication for surgery.

Ischemic Mitral Regurgitation

Ischemic MR represents MR occurring in the setting of ischemic heart disease in patients without significant abnormalities of the valve leaflets or chordal structures. Myocardial ischemia may result in focal or global left ventricular bulging and, with time, ventricular remodeling to a more spherical shape. Geometric changes cause outward migration of the papillary muscles. The finding most strongly correlated with chronic ischemic MR is outward papillary muscle displacement. When the papillary muscles are displaced outward, the point of mitral leaflet coaptation moves apically and away from the mitral annulus, resulting in the appearance of valve tenting. Besides outward bulging of the LV, scarring and retraction of the papillary muscles may produce mitral leaflet tethering, with the net effect of incomplete leaflet coaptation and valvular incompetence. An additional potential mechanism of ischemic MR is decreased contractility of the posterior mitral annulus. During systole, annular contraction reduces the mitral orifice area by 25%. Because the anterior portion of the mitral annulus is more fibrous, posterior annular contraction plays a greater role in reducing the size of the mitral orifice. Loss of posterior annular contraction may contribute to MR in the setting of myocardial ischemia.

The clinical approach to ischemic MR depends on its underlying mechanism. Timely surgical intervention often is warranted in cases of papillary muscle rupture. For patients with an intact mitral apparatus who have ischemic MR in

the setting of AMI, early reperfusion therapy improves regional and global left ventricular function, reduces ventricular dilation, and decreases the likelihood of adverse remodeling and associated papillary muscle displacement. The resultant improvements in ventricular function and geometry combine to reduce the incidence of ischemic MR.

Surgical Decision Making

The surgical approach to MR has evolved as its pathophysiology has been clarified. High operative mortality rates associated with the surgical correction of MR in the 1980s led many clinicians to treat patients conservatively. Because favorable loading conditions and high left atrial compliance allow patients with significant MR to remain asymptomatic for long periods, it is likely that many patients did not undergo surgery until the onset of disabling symptoms. More severe preoperative symptoms are associated with a lower EF and a greater incidence of postoperative CHF. Historically, poor outcomes after surgery for MR might have occurred because clinicians did not appreciate the true degree of left ventricular dysfunction at the time of surgery in symptomatic patients. An EF of less than 60% in the setting of severe MR represents significant left ventricular dysfunction and predicts a worse outcome with surgery or medical management. Surgical techniques common in the 1980s probably also contributed to unfavorable postoperative outcomes. For instance, although the mechanisms are incompletely understood, resection of the subvalvular apparatus contributes to decreased left ventricular systolic performance after mitral valve replacement.

In part because of improved surgical techniques, the operative mortality rate for patients with organic MR who are younger than 75 years is about 1% in some centers. Besides preservation of the subvalvular apparatus, valve repair is another surgical technique associated with improved postoperative outcome. Although not applicable to all patients, such as those with advanced rheumatic disease, the popularity of valve repairs continues to grow.

Studies indicate numerous benefits associated with mitral repair. For instance, after accounting for baseline characteristics, patients who undergo mitral repair instead of replacement have lower operative mortality rates and longer survival times, largely because of improved postoperative left ventricular function. The survival benefit that accompanies valve repair also is observed among patients undergoing combined valve and CABG surgery. Valve repair does not increase the likelihood of reoperation compared with replacement. Although originally used most often for posterior leaflet disease, surgeons now routinely repair anterior mitral leaflets with good success. When repairing anterior leaflet prolapse, surgeons may insert artificial chordae. The approach to flail or prolapsing posterior mitral leaflet segments often involves resection of a portion of the leaflet. In addition to resecting a portion of the leaflet and plicating the redundant tissue, an annuloplasty ring often is placed to reduce mitral orifice size and return the annulus to a more anatomic shape. Some surgeons favor a flexible, partial, posterior annuloplasty band, which may allow improved systolic contraction of the posterior annulus and better postoperative left ventricular function.

Minimally Invasive Mitral Valve Surgery

The concept of minimally invasive mitral surgery usually refers to valve repairs accomplished through a 3- or 4-cm right inframammary incision in the fourth

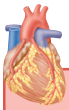
or fifth intercostal space. Several additional 1-cm incisions around the primary incision facilitate placement of robotic arms or other thoracoscopic instruments. The arterial cannula for CPB may be inserted directly or by a chimney graft into the femoral artery or into the ascending aorta through a thoracic incision under direct visualization. Venous drainage is accomplished by the femoral route using TEE guidance with a multiple side hole peripheral access cannula. Supplementary venous drainage is used in some centers by inserting either a 15- to 17-Fr right internal jugular vein cannula or specialized PAC with multiple end holes that drains to the venous reservoir during CPB.

Cardioplegia may be given antegrade into the aortic root or retrograde through the coronary sinus. Surgeons typically administer antegrade cardioplegia by one of two methods. The first involves the placement of a catheter tip into the ascending aorta through a right parasternal stab incision under thoracoscopic vision. This method is similar to standard antegrade cardioplegia administration in median sternotomy cases. A long-shafted aortic cross-clamp placed through a stab incision in the right lateral chest wall is used to occlude the aorta distal to the cardioplegia cannula. The second method of antegrade cardioplegia administration uses a specialized endoaortic cannula inserted into the femoral artery. A balloon near the distal end of this cannula is positioned in the ascending aorta using TEE guidance. Inflation of the balloon occludes the ascending aorta while antegrade cardioplegia delivery commences at the distal tip of the device.

Although referred to as *robotic*, systems such as the da Vinci are probably more appropriately described as telemanipulators. These devices receive direct input from the hands and feet of the surgeon who is seated at a remote console that translates these motions to end-effectors within the chest of the patient. When seated at the remote console of a robotic device, the surgeon has near-stereoscopic vision compared with viewing a two-dimensional image on a television screen. Robotic devices provide motion scaling and tremor filtration to smooth movements. Because the robotic arms have articulating "wrists" at their distal ends, the surgeon can achieve 7 degrees of freedom of movement within the chest, similar to open surgery. By comparison, long-handled thoracoscopic instruments, which are often oriented almost parallel to one another, afford only 4 degrees of freedom. Both thoracoscopic and robotically assisted approaches use the same operative techniques as standard open repairs. Techniques such as leaflet resection, chordal insertion or transfer, sliding plasties, edge-to-edge repair, and annuloplasty band insertion may be used by experienced surgeons.

Just as catheter-based techniques have been developed to treat valvular AS, efforts are under way to develop percutaneous interventions for MR. The device with the largest clinical experience is the MitraClip system (Abbott Laboratories, Abbott Park, IL). Leaflet plication is based on the open mitral repair technique reported by Alfieri and colleagues. It entails the creation of a double-orifice mitral valve by suturing the free edges of the leaflets at the site of regurgitation together to improve leaflet coaptation and reduce MR. MitraClip uses a percutaneous femoral venous transseptal delivery system to deploy a cobalt-chromium clip to secure the mitral leaflets under fluoroscopic and echocardiographic guidance.

Percutaneous techniques attempt to correct annular pathology by indirectly pushing the posterior annulus anteriorly using devices that exploit the anatomic relationship of the coronary sinus and mitral annulus. One device is the Carillon Mitral Contour System. It consists of self-expandable nitinol (ie, nickel-titanium alloy) proximal and distal anchors connected by a nitinol bridge. The application of tension on the system pulls the posterior mitral annulus anteriorly, reducing septal–lateral annular diameter.



BOX 15.4 ***Mitral Regurgitation***

Preload is increased
Afterload is decreased
Goal is mild tachycardia, vasodilation
Avoid myocardial depression

Anesthesia Considerations

Patients with MR may have significantly different risk factors for surgery, including duration of disease, symptoms, hemodynamic stability, ventricular function, and involvement of the right heart and pulmonary circulation (Box 15.4). For instance, a patient with severe MR caused by acute papillary muscle rupture may enter the operating room in cardiogenic shock with pulmonary congestion requiring intraaortic balloon pump augmentation. Another patient with a newly diagnosed flail posterior mitral leaflet may enter the surgical suite with relatively preserved left ventricular function and no symptoms; the compliance of the LA might have prevented pulmonary vascular congestion, pulmonary hypertension, and right ventricular dysfunction.

Despite differences in presentation, the general management goals remain similar and include maintenance of forward CO and reduction in the mitral regurgitant fraction. The anesthesiologist must optimize right ventricular function, in part by avoiding increases in pulmonary vascular congestion and pulmonary hypertension. Depending on the clinical presentation, various degrees of intervention are needed to achieve these hemodynamic management goals.

Invasive hemodynamic monitoring provides a wealth of important information. Arterial catheters are essential for monitoring beat-to-beat changes in blood pressure that occur in response to a variety of surgical and anesthesia manipulations. PACs facilitate many aspects of intraoperative patient management. Intraoperative use of a PAC allows careful optimization of left-sided filling pressures. Although the PCWP and diastolic PAP depend on left atrial and left ventricular compliance and filling, examination of intraoperative trends in these variables helps the anesthesiologist to provide appropriate levels of preload while avoiding volume overload. Periodic determination of CO allows a more objective assessment of the patient's response to interventions such as fluid administration or inotropic infusion. The presence or size of a v wave on a PCWP tracing does not reliably correlate with the severity of MR because this finding depends on left atrial compliance. As in the management of patients with AR, a benefit of PAC insertion is the ability to introduce a ventricular pacing wire to rapidly counteract hemodynamically significant bradycardia. In patients with right ventricular compromise, monitoring trends in the CVP recording may be helpful. Tricuspid regurgitation (TR) detected through analysis of the CVP tracing may suggest right ventricular dilation, which may be caused by pulmonary hypertension.

Intraoperative TEE provides invaluable information during the surgical correction of MR. It reliably identifies the mechanism of MR, thereby guiding the surgical approach, and it objectively demonstrates the size and function of the cardiac chambers. TEE can identify the cause of hemodynamic derangements, facilitating proper intervention. For instance, the appearance of SAM of the mitral apparatus immediately after valve repair allows the anesthesiologist to intervene with volume infusion and medications such as esmolol or phenylephrine as appropriate. In rare circumstances, when

hemodynamically significant SAM persists despite these interventions, the surgeon may elect to further repair or replace the mitral valve. TEE also identifies concomitant pathology that may warrant surgical attention, such as atrial-level shunts and additional valve disease.

Intraoperative TEE is essential during minimally invasive and robotically assisted mitral valve surgery. The use of a right minithoracotomy for these procedures precludes bypass cannulation in the chest. Instead, femoral arterial and venous cannulation with or without supplementary venous drainage from the superior vena cava or pulmonary artery is used. Real-time TEE imaging typically guides cannulation for CPB. If an endoaortic balloon clamp is used, the echocardiographer ensures that the balloon is correctly positioned in the ascending aorta.

In addition to TEE considerations related to cannulation procedures, the selection of a minimally invasive or robotically assisted approach to mitral repair necessitates other changes in anesthesia management. Although not universally used, one-lung ventilation is preferred in many centers. This may be achieved by the usual methods, such as a double-lumen endotracheal tube or bronchial blocker. Impaired oxygenation can occur when one-lung ventilation is used during the termination of CPB during these procedures.

Intraoperative care of patients with MR before the institution of CPB focuses on optimizing forward CO, minimizing the mitral regurgitant volume, and preventing deleterious increases in PAPs. Maintaining adequate left ventricular preload is essential. An enlarged LV that operates on a higher portion of the Frank-Starling curve requires adequate filling. At the same time, excessive volume administration should be avoided because it may cause unwanted dilatation of the mitral annulus and worsening of the MR. Excessive fluid administration may precipitate right ventricular failure in patients with pulmonary vascular congestion and pulmonary hypertension. Optimization of preload is aided by analysis of data obtained from PAC measurements and TEE images. Because significant left ventricular dysfunction is seen in many patients with MR, specific induction and maintenance regimens are selected to avoid further depressing left ventricular function. Large doses of narcotics have been popular in the past. Others have shown that smaller doses of narcotics combined with vasodilating inhalation anesthetics produce acceptable intraoperative hemodynamics. By reducing the amount of narcotics administered, the addition of a vasodilating inhalation agent to the anesthetic regimen may allow for faster extubation of the trachea after surgery. With the current trend toward early referral of asymptomatic patients for mitral repair, anesthetic regimens that reduce the duration of postoperative mechanical ventilation may be advantageous.

In patients with severe left ventricular dysfunction, infusions of inotropic medications such as dopamine, dobutamine, or epinephrine may be required to maintain an adequate cardiac output. Phosphodiesterase inhibitors such as milrinone also may augment systolic ventricular performance and reduce pulmonary and peripheral vascular resistances. By reducing pulmonary and peripheral vascular resistance, forward CO is facilitated. Nitroglycerin and sodium nitroprusside represent two additional options for reducing the impedance to ventricular ejection. If patients prove refractory to inotropic and vasodilator therapy, insertion of an intraaortic balloon pump should be strongly considered.

Because severe MR may result in pulmonary hypertension and right ventricular dysfunction, intraoperative management strategies should avoid hypercapnia, hypoxia, and acidosis. Mild hyperventilation may be beneficial in some patients.

Patients with severe right ventricular dysfunction after CPB can prove exceptionally difficult to treat. Besides avoiding the factors known to increase pulmonary vascular resistance (PVR), only a few options exist for these patients. Inotropic agents with

vasodilating properties such as dobutamine, isoproterenol, and milrinone augment right ventricular systolic performance and decrease PVR, but their use often is confounded by systemic hypotension. Prostaglandin E_1 (PGE_1) reliably reduces PVR and undergoes extensive first-pass metabolism in the pulmonary circulation. Although PGE_1 reduces PAPs after CPB, systemic hypotension requiring infusions of vasoconstrictors through a left atrial catheter has occurred.

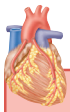
Inhaled nitric oxide is an alternative for the treatment of right ventricular failure in the setting of pulmonary hypertension. Nitric oxide reliably relaxes the pulmonary vasculature and is then immediately bound to hemoglobin and inactivated. Studies indicate that systemic hypotension during nitric oxide therapy is unlikely.

Left ventricular dysfunction may contribute to post-CPB hemodynamic instability. With mitral competence restored, the low-pressure outlet for left ventricular ejection is removed. The enlarged LV must then eject entirely into the aorta. Because left ventricular enlargement leads to increased wall stress, a condition of increased afterload often exists after CPB. At the same time, the preload augmentation inherent to MR is removed. The systolic performance of the LV often declines after surgical correction of MR. Treatment options in the immediate post-CPB period include inotropic and vasodilator therapy and, if necessary, intraaortic balloon pump augmentation.

Anesthesia Considerations

Several important goals should guide the anesthesia management of patients with significant MS. First, the anesthesiologist should prevent tachycardia or treat it promptly in the perioperative period (Box 15.5). Second, left ventricular preload should be maintained without exacerbation of pulmonary vascular congestion. Third, anesthesiologists should avoid factors that aggravate pulmonary hypertension and impair right ventricular function.

Prevention and treatment of tachycardia are central to perioperative management. Tachycardia shortens the diastolic filling period. An elevation in transvalvular flow rate is required, with a resultant increase in the left atrial–to–left ventricular pressure gradient to maintain left ventricular preload with a shortened diastolic period. Avoidance of tachycardia begins in the preoperative period. Anxiety-induced tachycardia may be treated with small doses of narcotics or benzodiazepines. However, excessive sedation is counterproductive because sedative-induced hypoventilation can result in hypoxemia or hypercarbia, potentially aggravating a patient's underlying pulmonary hypertension and because large doses of premedication can jeopardize the patient's already limited left ventricular preload. Appropriate monitoring and supplemental oxygen therapy should be considered for patients receiving preoperative narcotics or benzodiazepines. Medications taken by the patient before surgery to control heart rate, such as digitalis, β-blockers, calcium receptor antagonists, or amiodarone, should be continued in the perioperative period. Additional doses of β-blockers and calcium-receptor antagonists



BOX 15.5 ***Mitral Stenosis***

Preload is normal or increased
Afterload is normal
Goal is controlled ventricular response
Avoid tachycardia, pulmonary vasoconstriction

may be required intraoperatively, particularly to control the ventricular rate in patients with AF. Control of the ventricular rate remains the primary goal in managing patients with AF, although cardioversion should not be withheld from patients with atrial tachyarrhythmias who become hemodynamically unstable. Narcotic-based anesthetics often are helpful in avoiding intraoperative tachycardia. However, clinicians should realize these patients may be receiving other vagotonic drugs and that profound bradycardia is possible in response to large doses of narcotics. The selection of a muscle relaxant such as pancuronium may help prevent the unwanted bradycardia associated with high-dose narcotics.

Maintenance of preload is an important goal for treating patients who have a fixed obstruction to left ventricular filling. Appropriate replacement of blood loss and prevention of excessive anesthetic-induced venodilation help preserve hemodynamic stability intraoperatively. Invasive hemodynamic monitoring allows the anesthesiologist to maintain adequate preload while avoiding excessive fluid administration that can aggravate pulmonary vascular congestion. Placement of an arterial catheter facilitates timely recognition of hemodynamic derangements. PACs can be invaluable in treating patients with significant MS. Although the PCWP overestimates left ventricular filling and the pulmonary artery diastolic pressure may not accurately reflect left-heart volume in patients with pulmonary hypertension, trends and responses to intervention can be more readily assessed. Tachycardia increases the pressure gradient between the LA and LV. Increased heart rates widen the discrepancy between the PCWP and the true LVEDP. Despite these limitations, the PAC remains a useful monitoring tool, providing information on CO and PAPs.

Many patients with MS have pulmonary hypertension. Anesthesia techniques that avoid increases in PVR are likely to benefit these patients and prevent additional right ventricular embarrassment. Meticulous attention to arterial blood gas results allows appropriate adjustment of ventilatory parameters. Vasodilator therapy for patients with pulmonary hypertension usually is ineffective because the venodilation produced further limits left ventricular filling and does not improve cardiac output. The only MS patients who may benefit from vasodilator therapy are those with concomitant MR or those with severe pulmonary hypertension and right ventricular dysfunction in whom pulmonary vasodilation can facilitate transpulmonary blood flow and improve left ventricular filling. The treatment of right ventricular dysfunction was discussed earlier.

TRICUSPID REGURGITATION

Clinical Features and Natural History

Tricuspid disease is caused by a structural defect in the valve apparatus or a functional lesion. Primary disorders of the tricuspid valve apparatus that may lead to more significant degrees of TR include congenital disease (ie, Ebstein anomaly), rheumatic valve disease, prolapse, irradiation, carcinoid syndrome, blunt chest trauma, endomyocardial biopsy–related trauma, and right ventricular pacemaker/defibrillator lead trauma. Despite numerous potential causes of primary tricuspid disease, they account for only 20% of TR cases. The remainder of TR disease is functional in nature. Left-sided valvular disease, usually MR, most commonly is responsible. Functional tricuspid incompetence also can result from MS, AR, or AS and from isolated pulmonary hypertension. Causes of functional TR include dilation of the annulus or leaflet tethering from right ventricular dilation and remodeling, global right ventricular dysfunction from cardiomyopathy and myocarditis or CAD with resulting ischemia,

infarction, or rupture of the right ventricular papillary muscles. When mitral valve disease is severe enough to warrant valve repair or replacement, TR may be identified in 30% to 50% of patients.

Symptoms of isolated TR are usually minor in the absence of concurrent pulmonary hypertension. Intravenous drug abusers who develop tricuspid endocarditis are the classic example. In these patients, structural damage to the valve may be quite severe, but because they are free of other cardiac disease, they can tolerate complete excision of the tricuspid valve with few adverse effects. Excision of the tricuspid valve in endocarditis has been common because of the undesirability of placing a valve prosthesis in a region of infection. Surgical annuloplasty may be a better long-term option if the valve is structurally salvageable.

Another factor that broadly favors tricuspid repair rather than replacement is the high incidence of thrombotic complications with a valve in this position. The lower pressure and flow state on the right side of the heart are responsible for this phenomenon.

In chronic TR caused by right ventricular dilation, the clinical scenario often is much different from that of isolated tricuspid disease. The major hemodynamic derangements are usually those associated with mitral or aortic valve disease. The right ventricle (RV) dilates in the face of the afterload stress from long-standing pulmonary hypertension, and the resultant increase in end-diastolic fiber stretch (ie, preload reserve) promotes increases in SV mediated by the Starling mechanism. These increases are negated by a concurrently increasing right ventricular afterload because of relatively inadequate RVH. Regurgitation through the tricuspid valve reduces right ventricular wall tension at the price of a decrease in effective forward SV.

An important corollary to right ventricular chamber enlargement is the possibility of a leftward shift of the interventricular septum and encroachment on the left ventricular cavity. This phenomenon can reduce the left ventricular chamber size and the slope of the left ventricular diastolic pressure-volume curve, rendering the LV less compliant. Septal encroachment may mask left ventricular underfilling by decreasing left ventricular compliance, artificially increasing LVEDP. A failing RV underloads the left side by reduced effective SV and anatomic (ie, septal shift) mechanisms.

Surgical Decision Making

In cases of structural tricuspid insufficiency, the decision to repair or replace the valve is straightforward. The same cannot be said of functional TR. Because most functional cases are the consequence of left-sided valve lesions with right ventricular overload, the TR usually improves significantly (typically by at least one grade) after the aortic or mitral valve is repaired or replaced. It can be unclear in the operating room whether addition of a tricuspid procedure to the left-sided valve surgery is indicated. In this situation, intraoperative TEE plays an essential role. If the TR is severe in the pre-CPB assessment, tricuspid valve surgery is usually performed. The evidence is less clear when regurgitation is graded as moderate. Some surgeons choose to repair the tricuspid valve in cases of moderate TR, but others advocate observation. In the context of left-sided valve surgery, it is common with moderate or more severe TR to complete the left-sided procedure and then reassess the tricuspid valve with TEE when the heart is full and ejecting. If the TR remains more than moderate after the left-sided valve is fixed, many surgeons perform the tricuspid procedure. If regurgitation is moderate or less severe, the appropriate surgical course may remain unclear.

Some patients having left-sided valve procedures must return to the operating room for tricuspid surgery. Their morbidity and mortality rates are probably significantly higher than those for patients undergoing tricuspid valve repair at the time of the

aortic or mitral valve procedure. Decision making for cases of functional TR is made more complicated by the inability to rigorously quantify the severity of the regurgitation and right ventricular dysfunction.

Anesthesia Considerations

Because most tricuspid surgery occurs in the context of significant aortic or mitral disease, anesthesia management primarily is determined by the left-sided valve lesion. The exception is when significant pulmonary hypertension and right ventricular failure exist. Under these conditions, the primary impediment to hemodynamic stability after surgery is right ventricular failure rather than the left-sided process.

If right ventricular dysfunction is predicted, it is useful to place a PAC, even if the tricuspid valve will be replaced. If the PAC must be removed because of tricuspid valve replacement, it still can be helpful to obtain CO and PAPs before CPB to get insight into right ventricular function and anticipate the hemodynamic support that may be required. A PAC is of greater use than CVP alone because the CVP is a poor index of intravascular filling and the degree of TR. The right atrium and vena cavae are highly compliant and accept large regurgitant volumes with relatively little change in pressure.

A PAC also is useful when intraoperative TEE is used. As in AR within the LV, the RV in chronic TR is volume-overloaded and dilated and requires a large EDV to maintain forward flow. Because of the unreliability of the CVP as an indicator of filling, it is possible to volume-overload patients with TR and right ventricular failure. CO in right ventricular failure often can be augmented with the use of vasodilators, and although right ventricular dimensions can be followed intraoperatively with TEE, maximizing CO (sometimes at the cost of systemic arterial pressure) is best done with serial CO measurements (as in AR). When there is significant right ventricular distension, the possibility of septal shift and secondary deterioration of left ventricular diastolic compliance should be carefully considered. Echocardiography is uniquely helpful for this assessment.

Post-CPB treatment of the patient undergoing an isolated tricuspid valve procedure is usually straightforward. Patients usually do not have significant right ventricular failure or pulmonary hypertension and typically require only a brief period of CPB without aortic cross-clamping. A larger group of patients, particularly those with TR related to AS, typically come off CPB with little need for support of the RV. These patients often do well because the improvement in left ventricular function after AVR for AS is usually sufficient to reduce PAPs significantly and offload the right heart. When left-sided valve surgery is for mitral disease, the improvement usually is not as marked, and greater degrees of inotropic support of the RV often are indicated. The combination of a phosphodiesterase inhibitor with a vasodilator and a catecholamine infusion is useful. Serial CO measurements to balance systemic pressure and right ventricular output and filling are critical.

A few other practical points on tricuspid valve repair and replacement should be made. First, because right-sided pressures can be chronically increased with TR, it is important to look for a patent foramen ovale and the potential for right-to-left shunting before initiation of CPB. Second, intravascular volume may be quite high in this patient population, and it is often practical to avoid red blood cell transfusion by hemofiltration during bypass. Third, if the patient has significant right ventricular dysfunction or peripheral edema or ascites, there is the potential for a coagulopathy related to liver congestion, and the patient should be treated accordingly. Fourth, central catheters, particularly PACs, should not be entrapped by right atrial suture lines.

INNOVATIONS IN VALVE REPAIR

Interventional cardiology has had a significant impact on the volume of CABG, and it can be predicted that interventional cardiology will alter surgery for VHD over time. Many less invasive approaches to mitral valve repair are being assessed in animal studies or clinical trials, and tremendous inroads have been made in percutaneous replacement of the aortic valve. Innovations also are being made in surgical valve repair, including aortic valve repair and closed- and open-chamber procedures for MR.

Aortic Valve Repair

During the past several years, there has been a major shift from valve replacement to valve repair in patients with degenerative mitral valve disease. The same has not been true of the aortic valve because the valve disease is different in most patients and because of the high flow and pressure conditions across the aortic valve that make repair more prone to failure. However, aortic valve repair is being increasingly done as an appropriate patient population is defined. Although valve repair for AR has found broader use when regurgitation is associated with dissection or dilation of the aortic root, isolated valve repair has been less common. A growing body of data suggests that aortic valve repair may offer advantages over valve replacement in younger individuals with AR due to bicuspid valves. In contrast with AVR, aortic valve repair eliminates the need for anticoagulation for a mechanical valve and should delay the need for reoperation for a failed tissue valve. When regurgitation occurs with a bicuspid valve, the insufficiency usually is caused by retraction or prolapse, or both, of the conjoined cusp. Repair consists of a triangular incision to shorten and elevate the cusp to improve apposition. Although very long-term follow-up results have not been reported, late failure of the repair requiring reoperation does occur. Most of the failure was attributed to repairs done in the early experience of repairs.

As a result of this experience, aortic valve repair is likely to find increasing application in this patient population. For this group, anesthesia management usually is straightforward, although the clinical indications for valve repair in AR are the same as those for valve replacement. The compelling issue for the anesthesiologist in these cases is echocardiographic assessment of the valve for suitability of repair and the adequacy of the repair after the procedure.

Sutureless Valve Replacement

Surgical AVR continues to be the gold standard for patients with severe symptomatic aortic valve stenosis. Transcatheter aortic valve replacement (TAVR) reduced the rate of death and cardiac symptoms for patients deemed inoperable compared with medical therapy alone. These procedures have been associated with a decreased mortality rate at 1 year compared with open surgery in high-risk patients. However, these procedures are not without risk, including bradyarrhythmias requiring permanent pacemaker insertion, cardiac perforation, myocardial infarction, access-related complications, and other valve-related issues such as perivalvular leak and unknown long-term durability.

There is increased interest in the treatment of aortic valvular disease with sutureless AVR in patients who can benefit from a shorter cross-clamp time but are not truly inoperable. With the rapid technologic progress made in transcatheter valve technology and materials, sutureless AVR has been proposed as an additional therapeutic option for high-risk patients with severe AS. Potential advantages of sutureless AVR include

removal of the diseased and often calcific native aortic valve and reduction in aortic cross-clamp and CPB times in the setting of a potentially minimally invasive surgical approach.

NEW TECHNIQUES FOR MITRAL VALVE REPAIR

MR frequently is associated with CHF. In dilated and ischemic cardiomyopathy, enlargement of the mitral annulus results in a failure of coaptation of the mitral leaflets and valve incompetence. Although cardiac surgery is an effective treatment, morbidity can be high. Three approaches have been developed to address MR occurring in the absence of structural mitral pathology. They address the failure of leaflet coaptation at the level of the valve leaflets or valve annulus or by altering the anatomic relationship of the septal and lateral walls of the LV.

Altering Ventricular Anatomy to Reduce Mitral Regurgitation

Valve leaflet and annulus repair techniques are described in earlier sections of this chapter. The approach to closed mitral valve repair consists of altering the geometry of the lateral and septal left ventricular walls to bring the valve leaflets together. The commercial Coapsys device has entered clinical trials. It consists of anterior and posterior epicardial pads connected by a cord. With an open chest, the cord is placed transventricularly in a subvalvular position, and the tension on the cord is adjusted before the opposing epicardial pad is fixed in place. This effectively brings the ventricular walls together and improves leaflet coaptation. TEE is used to optimize cord length and pad positioning. In contrast to the leaflet-based and annulus-based approaches, the Coapsys approach is surgical, requiring an open chest but not CPB.

SUGGESTED READINGS

Alfieri O, Maisano F, De Bonis M, et al. The double-orifice technique in mitral valve repair: a simple solution for complex problems. *J Thorac Cardiovasc Surg*. 2001;122:674–681.

Augoustides JG, Wolfe Y, Walsh EK, et al. Recent advances in aortic valve disease: highlights from a bicuspid aortic valve to transcatheter aortic valve replacement. *J Cardiothorac Vasc Anesth*. 2009;23:569–576.

Bonow RO, Carabello B, de Leon AC, et al. ACC/AHA guidelines for the management of patients with valvular heart disease: executive summary. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Management of Patients with Valvular Heart Disease). *J Heart Valve Dis*. 1998;7:672–707.

Carabello BA. Evaluation and management of patients with aortic stenosis. *Circulation*. 2002;105:1746–1750.

Chaliki HP, Brown ML, Sundt TM, et al. Timing of operation in asymptomatic severe aortic stenosis. *Expert Rev Cardiovasc Ther*. 2007;5:1065–1071.

Filsoufi F, Rahmanian PB, Castillo JG, et al. Excellent early and late outcomes of aortic valve replacement in people aged 80 and older. *J Am Geriatr Soc*. 2008;56:255–261.

Grayburn PA, Eichhorn EJ. Dobutamine challenge for low-gradient aortic stenosis. *Circulation*. 2002;106:763–765.

Karon BL, Enriquez-Sarano M. Valvular regurgitation. In: Lloyd MA, Murphy JG, eds. *Mayo Clinic Cardiology Review*. 2nd ed. Philadelphia: Lippincott Williams & Wilkins; 2000:303–330.

Levine RA. Dynamic mitral regurgitation: more than meets the eye. *N Engl J Med*. 2004;351:1681–1684.

Matsunaga A, Duran CM. Progression of tricuspid regurgitation after repaired functional ischemic mitral regurgitation. *Circulation*. 2005;112(9 suppl):I453–I457.

Mohty D, Orszulak TA, Schaff HV, et al. Very long-term survival and durability of mitral valve repair for mitral valve prolapse. *Circulation*. 2001;104:I1–I7.

Monchi M, Gest V, Duval-Moulin AM, et al. Aortic stenosis with severe left ventricular dysfunction and low transvalvular pressure gradients: risk stratification by low-dose dobutamine echocardiography. *J Am Coll Cardiol*. 2001;37:2101–2107.

Mueller XM, Tevaearai HT, Stumpe F, et al. Tricuspid valve involvement in combined mitral and aortic valve surgery. *J Cardiovasc Surg (Torino)*. 2001;42:443–449.
Nishimura RA, Grantham JA, Connolly HM, et al. Low-output, low-gradient aortic stenosis in patients with depressed left ventricular systolic function: the clinical utility of the dobutamine challenge in the catheterization laboratory. *Circulation*. 2002;106:809–813.
Nishimura RA, Otto CM, Bonow RO, et al. AHA/ACC guideline for the management of patients with valvular heart disease: executive summary. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol*. 2014;2014(63):2438–2488.
Palep JH. Robotic assisted minimally invasive surgery. *J Minim Access Surg*. 2009;5:1–7.
Popovic AD, Stewart WJ. Echocardiographic evaluation of valvular stenosis: the gold standard for the next millennium? *Echocardiography*. 2001;18:59–63.
Roberts R, Sigwart U. New concepts in hypertrophic cardiomyopathies: part II. *Circulation*. 2001;104:2249–2252.
Roselli EE, Pettersson GB, Blackstone EH, et al. Adverse events during reoperative cardiac surgery: frequency, characterization, and rescue. *J Thorac Cardiovasc Surg*. 2008;135:316–323, 323 e1–6.
Shah PM, Raney AA. Tricuspid valve disease. *Curr Probl Cardiol*. 2008;33:47–84.
Siminiak T, Wu JC, Haude M, et al. Treatment of functional mitral regurgitation by percutaneous annuloplasty: results of the TITAN Trial. *Eur J Heart Fail*. 2012;14:931–938.
Skubas NJ, Shernan SK, Bollen B. An overview of the American College of Cardiology/American Heart Association 2014 Valve Heart Disease Practice Guidelines: what is its relevance to the anesthesiologist and perioperative medicine physician? *Anesth Analg*. 2015;121:1132–1138.