Chapter 14

Valvular Heart Disease: Replacement and Repair

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Valve surgery is very different from coronary artery bypass grafting (CABG). Over the natural history of valvular heart disease (VHD), the physiologic characteristics change markedly and, in the operating room, physiologic and hemodynamic conditions are quite variable and are readily influenced by anesthetic interventions. For some types of valve lesions it can be relatively difficult to predict preoperatively 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 each of the major adult-acquired valve defects and how the pathophysiologic conditions evolve. Surgical decision making regarding valve repair or replacement must also be understood, because a valve operated on at the appropriate stage of its natural history will have a good and more predictable outcome than one operated on at a late stage, when the perioperative result can be quite poor. Because pathophysiologic conditions are dynamic and differ significantly among valve lesions, understanding the physiology and natural

history of individual valve defects is the foundation of developing an anesthetic plan that includes various requirements for pacing rate and rhythm, use of inotropes (or negative inotropes), and use of vasodilators or vasoconstrictors to alter loading conditions.

Although valvular lesions impose various physiologic changes, all VHD is characterized by abnormalities of ventricular loading. The status of the ventricle changes over time as ventricular function and the valvular defect are influenced by the progression of volume or pressure overload. The clinical status of patients with VHD therefore can be complex and dynamic. It is possible to have clinical decompensation in the context of normal ventricular contractility or ventricular decompensation 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. This divergence between cardiac performance and inotropy occurs as a result of compensatory physiologic mechanisms specific to each of the ventricular loading abnormalities.

AORTIC STENOSIS

Clinical Features and Natural History

Aortic stenosis is the most common cardiac valve lesion in the United States. One to 2 percent of the population is born with a bicuspid aortic valve, which is prone to stenosis with aging. Calcific aortic stenosis 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. There is clinical evidence of an atherosclerotic hypothesis for the cellular mechanism of aortic valve stenosis. There is a clear association between clinical risk factors for atherosclerosis and the development of aortic stenosis: elevated lipoprotein levels, increased low-density lipoprotein (LDL) cholesterol, cigarette smoking, hypertension, diabetes mellitus, increased serum calcium and creatinine levels, and male gender.[1] 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 rate of progression is on average a decrease in aortic valve area (AVA) of 0.1 cm^2/yr, and the peak instantaneous gradient increases by 10 mmHg/yr. The rate of progression of aortic stenosis in men older than age 60 years is faster than in women, and it is faster in women older than age 75 years 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 aortic stenosis is associated with a life expectancy of only 2 to 5 years. There is evidence that patients with moderate aortic stenosis (i.e., valve areas of 0.7 to 1.2 cm^2) are also at increased risk for the development of 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 aortic stenosis; and about one half of symptomatic patients are found to have anatomically significant CAD.

The preoperative assessment of aortic stenosis 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 presence of aortic stenosis. This maximal velocity (v) is then inserted in the modified Bernoulli equation to determine the pressure gradient (PG) between the left ventricle and the aorta:

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

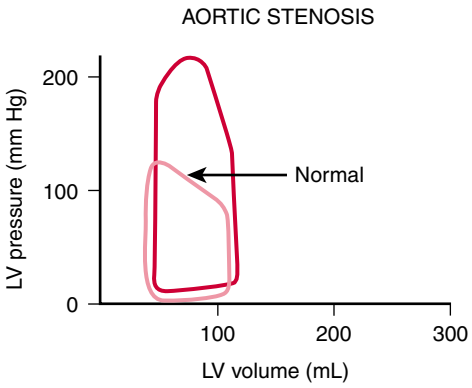

Figure 14-1 Pressure-volume loop in aortic stenosis. LV = left ventricular. (From Jackson JM, Thomas SJ, Lowenstein E: Anesthetic management of patients with valvular heart disease. Semin Anesth 1:239, 1982.)

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. Generally accepted criteria for critical outflow obstruction include a systolic pressure gradient greater than 50 mmHg, with a normal cardiac output and an AVA of less than 0.4 cm^2. In view of the ominous natural history of severe aortic stenosis (AVA < 0.7 cm^2), symptomatic patients with this degree of aortic stenosis are generally referred for immediate aortic valve replacement. 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:

$$\text{AVA}\left(\text{cm}^2\right) = \frac{\text{CO}}{\sqrt{\text{PG}}}$$

An obvious corollary of the previously described relationship is that "minimal" pressure gradients may actually reflect critical degrees of outflow obstruction when the cardiac output is significantly reduced (i.e., the 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 (i.e., minimal transvalvular flow) as the aortic stenosis worsens.

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

$$\sigma = \frac{\text{P} \times \text{R}}{2\text{h}}$$

in which P is the intraventricular pressure, R is the inner radius, and h is the wall thickness.

This elevation of wall tension is believed 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 this LV hypertrophy 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.

Figure 14-1 shows a typical pressure-volume loop for a patient with aortic stenosis. Two differences from the normal curve are immediately apparent. First, the peak

pressure generated during systole is much higher because of the high transvalvular pressure gradient. Second, the slope of the diastolic limb is steeper, reflecting the reduced left ventricular (LV) diastolic compliance that is associated with the increase in chamber thickness. Clinically, this means that small changes in diastolic volume produce relatively large increases in ventricular filling pressure.

This increased chamber stiffness places a premium on the contribution of atrial systole to ventricular filling, which in patients with aortic stenosis may account for up to 40% of the LV end-diastolic volume (LVEDV), rather than the 15% to 20% characteristic of the normal left ventricle. Echocardiographic and radionuclide studies have documented that diastolic filling and ventricular relaxation are abnormal in patients with hypertrophy from a variety of causes, with significant prolongation of the isovolumic relaxation period being 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 higher mean left atrial (LA) pressure is necessary to distend the left ventricle 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). It is likely that use of preload reserve and adequate LV hypertrophy are 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 relationship. LV hypertrophy can be viewed as a compensatory physiologic response; however, severe afterload stress and proportionately massive LV hypertrophy could decrease subendocardial perfusion and superimpose a component of ischemic contractile dysfunction.

In aortic stenosis, signs and symptoms of CHF usually develop when preload reserve is exhausted, not because contractility is intrinsically or permanently impaired. This contrasts to mitral and aortic regurgitation, 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 elevates 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 the latter 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 elevated 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 aortic stenosis, clinical data also support increased $M\dot{V}O_2$ as important in the genesis of myocardial ischemia.

On the supply side, the higher LV 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 aortic stenosis, LV dysfunction, and low transvalvular gradient suffers a high operative mortality rate and poor prognosis.[2] It is difficult to accurately assess the AVA in this low-flow, low-gradient aortic stenosis 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 aortic stenosis 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 aortic stenosis may benefit from valve replacement despite the increased operative risk associated with the low-flow, low-gradient hemodynamic state. Guidelines from the American College of Cardiology (ACC) and American Heart Association (AHA) call for a dobutamine echocardiography evaluation to distinguish patients with fixed anatomic aortic stenosis from those with flow-dependent aortic stenosis with LV dysfunction. Low-flow, low-gradient aortic stenosis is defined for a mean gradient of less than 30 mmHg and a calculated AVA less than 1.0 cm^2.

Timing of Intervention

In asymptomatic patients with aortic stenosis, it appears to be relatively safe to delay surgery until symptoms develop, but outcomes vary widely. The presence of moderate or severe valvular calcification along with a rapid increase in aortic-jet velocity identifies patients with a very poor prognosis. These patients 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.[3] In a study of 58 asymptomatic patients, 21 had symptoms for the first time during exercise testing. Guidelines for AVR in patients with aortic stenosis are shown in Table 14-1.

Functional outcome after aortic valve replacement in patients older than 80 years is excellent, operative risk is limited, and late survival rates are good. In patients with

Table 14-1 Recommendations for the Use of Aortic Valve Replacement in Patients with Aortic Stenosis

Replacement Indicated

- Patients with severe aortic stenosis and any of its classic symptoms (e.g., angina, syncope, dyspnea)
- Patients with severe aortic stenosis who are undergoing coronary artery bypass surgery
- Patients with severe aortic stenosis who are undergoing surgery on the aorta or other heart valves

Replacement Possibly Indicated

- Patients with moderate aortic stenosis who require coronary artery bypass surgery or surgery on the aorta or heart valves
- Asymptomatic patients with severe aortic stenosis and at least one of the following: ejection fraction of no more than 0.50, hemodynamic instability during exercise (e.g., hypotension), ventricular tachycardia; not indicated to prevent sudden death in asymptomatic patients who have none of the findings listed

Adapted from the American Heart Association web site (www.americanheart.org).

BOX 14-1 *Aortic Stenosis*

Preload:	Increased
Afterload:	Increased
Goal:	Sinus rhythm
Avoid:	Hypotension, tachycardia, bradycardia

severe LV dysfunction and low transvalvular mean gradient, operative mortality is increased, but aortic valve replacement was associated with improved functional status. Postoperative survival was best in younger patients and with larger prosthetic valves, whereas medium-term survival was related to improved postoperative functional class.

Anesthetic Considerations

The foregoing pathophysiologic principles dictate that anesthetic management be based on the avoidance of systemic hypotension, maintenance of sinus rhythm and an adequate intravascular volume, and awareness of the potential for myocardial ischemia (Box 14-1). In the absence of CHF, adequate premedication may reduce the likelihood of undue preoperative excitement, tachycardia, and the resultant potential for exacerbating myocardial ischemia and the transvalvular pressure gradient. In patients with truly critical outflow tract obstruction, however, heavy premedication with an exaggerated venodilatory response can reduce the appropriately elevated LVEDV (and LVEDP) needed to overcome the systolic pressure gradient. In these patients in particular, the additional precaution of administering supplementary oxygen may provide worthwhile insurance.

Intraoperative monitoring should include a standard five-lead ECG system, including a V_5 lead, because of the left ventricle's vulnerability to ischemia. A practical constraint in terms of interpretation is that these patients usually exhibit ECG changes because of preoperative LV hypertrophy. The associated ST-segment abnormalities (i.e., strain pattern) may be indistinguishable from or at least very similar to those of myocardial ischemia, making the intraoperative interpretation difficult. Lead II 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 LV filling when LV 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 (PA) catheter in the patient with aortic stenosis are arrhythmia-induced hypotension and ischemia. Loss of synchronous atrial contraction or a supraventricular tachyarrhythmia can compromise diastolic filling of the poorly compliant left ventricle, resulting in hypotension and the potential for rapid hemodynamic deterioration. The threat of catheter-induced arrhythmias is significant for the patient with aortic stenosis. However, accepting a low-normal CVP as evidence of good ventricular function can lead to similarly catastrophic underfilling of the left ventricle 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 PA catheter also allows for measurement of cardiac output, derived hemodynamic parameters, mixed venous oxygen saturation $S\bar{v}o_2$, and possible transvenous pacing.

Intraoperative fluid management should be aimed at maintaining appropriately elevated left-sided filling pressures. This is one reason why many clinicians believe that the PA catheter is worth its small arrhythmogenic risk. Keeping up with intravascular volume losses is particularly important in noncardiovascular surgery.

Patients with symptomatic aortic stenosis are usually encountered only in the setting of cardiovascular surgery because of their ominous prognosis without aortic valve replacement. Few studies have specifically addressed the response of these patients to the standard intravenous and inhalation induction agents; however, the responses to narcotic and non-narcotic intravenous agents are apparently not dissimilar from those of patients with other forms of VHD. The principal benefit of a narcotic induction is the 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 the 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 of 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 such 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 cardiac output is useful in this situation. The temptation to control intraoperative hypertension with vasodilators should be resisted in most cases. Given the risk of ischemia, nitroglycerin seems to be a particularly attractive drug. Its effectiveness in relieving subendocardial ischemia in patients with aortic stenosis is controversial; however, there is always the risk of even transient episodes of "overshoot." The hypertrophied ventricle's critical dependence on an adequate CPP may be very 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 (e.g., 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 aortic stenosis 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 simply accelerating the institution of cardiopulmonary bypass (CPB).

HYPERTROPHIC CARDIOMYOPATHY

Hypertrophic cardiomyopathy (HCM, formerly known as hypertrophic obstructive cardiomyopathy) is a relatively common genetic malformation of the heart with a prevalence of approximately 1 in 500. 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 LV chamber and the LV outflow tract (LVOT). The LVOT obstruction leads to increases

in LV pressure, which fuels a vicious cycle of further hypertrophy and increased LVOT obstruction.[4] Various treatment modalities include β-adrenoceptor antagonists, calcium channel blockers, and surgical myectomy of the septum. For more than 40 years, the traditional standard treatment has been the ventricular septal myotomy-myomectomy 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 (ablation) therapy with ethanol.

Clinical Features and Natural History

Patients vary widely in their clinical presentation. The contribution of echocardiography to the diagnosis 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.[5]

Less dramatic frequently presenting complaints include dyspnea, angina, and syncope. The clinical picture is often similar to that of valvular aortic stenosis. The symptoms may share a similar pathophysiologic basis (e.g., 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, other patients may have a stable pattern of angina or intermittent syncopal episodes for many years. Palpitations are also 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 aortic stenosis, 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.

A consensus exists that the disease is characterized by a wide spectrum of the severity of obstruction. It is totally absent in some patients, may be variable in others, or may be critically severe. Its most distinctive qualities are its dynamic nature (depending on contractile state and loading conditions), its timing (begins early, 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 (i.e., toward the septum) motion of the anterior mitral valve leaflet during systole accentuates the obstruction. The cause of this 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. This could generate hydraulic forces consistent with a Venturi effect whereby the anterior leaflet of the mitral valve would be drawn close to or within actual contact with the interventricular septum (Fig. 14-2). This means that after the obstruction is triggered the mitral valve leaflet is forced against the septum by the pressure difference across the orifice.

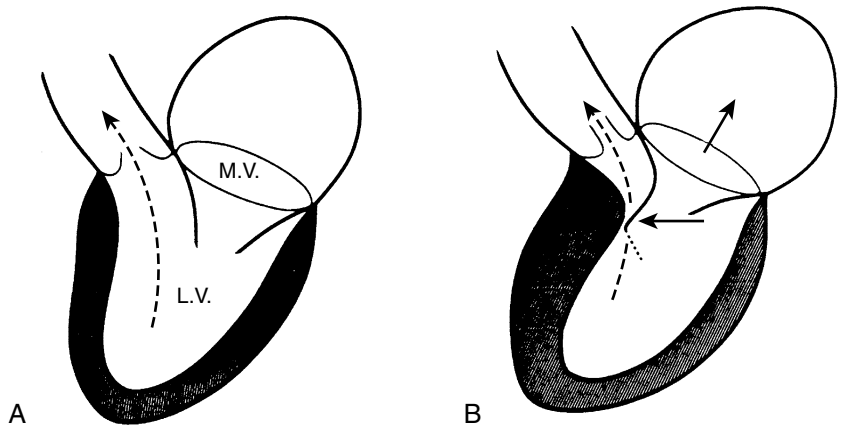

Figure 14-2 Proposed mechanism of systolic anterior motion (SAM) in hypertrophic cardiomyopathy (HCM). **A,** Normally, blood is ejected from the left ventricle through an unimpeded outflow tract. **B,** Thickening of the ventricular septum results in a restricted outflow tract, and this obstruction causes the blood to be ejected at a higher velocity, closer to the area of the anterior mitral valve leaflet. As a result of its proximity to this high-velocity fluid path, the anterior mitral valve leaflet is drawn toward the hypertrophied septum by a Venturi effect (*arrow*). M.V. = mitral valve; L.V. = left ventricle. (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 28:1, 1985.)

However, the pressure difference further decreases orifice size and further increases the pressure difference in a time-dependent amplifying feedback loop. This analysis is also consistent with observations that the measured gradient is directly correlated with the duration of mitral-septal contact. 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 is usually 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. These 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 secondary to mechanical factors.

Three basic mechanisms—increased contractility, decreased afterload, and decreased preload—exacerbate the degree of SAM-septal contact and produce the dynamic obstruction characteristic of patients with HCM. The common pathway is a reduction in ventricular volume (actively by increased contractility, directly or reflexly 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 overdistention, characteristically improve systolic function in patients with HCM and outflow tract obstruction. Diagnostically, these paradoxes are exploited by quantifying the degree of subaortic obstruction after isoproterenol (e.g., increased inotropy, tachycardia, and decreased volume) and the Valsalva maneuver (e.g., decreased venous return and 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 also

transiently exacerbate the gradient by increased inotropy from postextrasystolic potentiation. Therapeutically, volume loading, myocardial depression, and vasoconstriction should minimize obstruction and augment forward flow.

Poor diastolic compliance is the most clinically apparent manifestation of the relaxation abnormalities. LV filling pressures are markedly elevated despite enhanced systolic ejection and the normal or subnormal end-diastolic volume. This reduced ventricular volume reemphasizes the pivotal role played by the hypertrophied but intrinsically depressed myocardium. Reductions in afterload, 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 elevated ventricular filling pressure. The key point is that the high filling pressure does not reflect distention of a failing ventricle, even though stress-volume relationships suggest that its contractility is intrinsically depressed. This disease is characterized by systolic and diastolic dysfunction.

As in patients with valvular aortic stenosis, relatively high filling pressures reflect the LVEDV (i.e., 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 aortic stenosis, and it may approach 75% of total SV.

Another similarity between HCM and valvular aortic stenosis 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 one of the classic symptoms 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 aortic stenosis, there is also evidence of a compromise in myocardial oxygen supply.

Hemodynamic derangements peculiar to the disease may aggravate the ventricle's anatomic vulnerability to ischemia. The increased LVEDP for any LVEDV (i.e., poor compliance) inevitably narrows the diastolic CPP gradient. This may precipitate subendocardial ischemia in some patients with HCM, particularly those faced with the increased oxygen demand of overcoming late-systolic obstruction. There is evidence that hypertrophy-induced myocardial ischemia may underlie the diastolic dysfunction characteristic of HCM. As in patients with valvular aortic stenosis, ischemia-induced abnormalities of diastolic calcium sequestration may further exacerbate relaxation abnormalities, initiating a vicious cycle.

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 also exacerbate outflow obstruction. Disopyramide has also 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 relatively lower LVEDP. The negative inotropy may attenuate the subaortic pressure gradient, although in selected patients, the gradient may worsen because of pronounced and unpredictable degrees of vasodilation.

Surgery—a septal myotomy or partial myomectomy by the aortic approach—is reserved for those 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

BOX 14-2 *Hypertrophic Cardiomyopathy*

Preload:	Increased
Afterload:	Increased
Goal:	Myocardial depression
Avoid:	Tachycardia, inotropes, vasodilators

quite 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 (myomectomy) and diastolic (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 in atrial fibrillation.

Anesthetic Considerations

Priorities in anesthetic 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 14-2). It is therefore 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 specific technique, the 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 those 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 due to 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 ECGs in 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 on the basis of 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 LV diastolic compliance, the CVP is likely to be an inaccurate guide to changes in LV volume. However, a CVP catheter is extremely useful for the prompt administration of vasoactive drugs if they become necessary. As in valvular aortic stenosis, the information provided by insertion of a PA catheter 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. The reduced diastolic compliance means that the PCWP will overestimate 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 the venous cannulae may precipitate atrial arrhythmias. Because the resultant hypotension may be severe, the surgeon should cannulate the aorta before any atrial manipulations. 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 has the potential of disastrously worsening the LVOT obstruction if it elicits excessive vasodilation or if 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 is also advisable. This drug is almost always 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 are commonly used for patients with HCM. Their dose-dependent myocardial depression is ideal because negative inotropy reduces the degree of SAM-septal contact, which results in LVOT obstruction. Hypotension is almost always the result of underlying hypovolemia, which is potentially exacerbated by anesthetic-induced vasodilation. Inotropes, β-adrenergic agonists, and calcium are all 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

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

The natural history of chronic aortic regurgitation 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 to aortic stenosis, the onset of symptoms due to aortic regurgitation

does not portend an immediately ominous prognosis. In the absence of surgery, early recognition of aortic regurgitation and chronic use of vasodilators appear to be prolonging life span in this patient population.

A relatively unique and problematic feature of chronic aortic regurgitation 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 their myocardial contractility. Noninvasive diagnostic studies may facilitate the detection of early derangements in contractile function in relatively asymptomatic patients. This finding is important to the cardiologist when considering surgical referral, because patients with depressed preoperative LV function have a higher perioperative mortality rate and are at increased risk for persistent postoperative heart failure.

As in acute mitral regurgitation, the physiology of acute aortic regurgitation is quite different from chronic aortic regurgitation. Common causes include endocarditis, trauma, and acute aortic dissection. Because of a lack of chronic compensation, these patients usually present with pulmonary edema and heart failure refractory to optimal medical therapy. These 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 aortic regurgitation. 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, aortic regurgitation results in a state of LV volume and pressure overload. Progressive volume overloading from aortic regurgitation increases end-diastolic wall tension (i.e., ventricular afterload) and stimulates the serial replication of sarcomeres, producing a pattern of eccentric ventricular hypertrophy.[6] This dilation of the ventricle, in accordance with Laplace's law, also elevates the systolic wall tension, stimulating some concentric hypertrophy. This process of eccentric hypertrophy results in the greatest absolute degrees of cardiomegaly seen in valve disease. End-diastolic volume may be three to four times normal, and very high cardiac outputs can be sustained.

Figure 14-3 shows the pressure-volume loops for acute and chronic aortic regurgitation. 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, cardiac output is maintained by the Frank-Starling mechanism, and cardiac failure is not seen. This is true despite probable decreases in contractility. There is virtually no isovolumic diastolic phase, because the ventricle is filling throughout diastole. The isovolumic phase of systole is also brief because of the low aortic diastolic pressure. This minimal impedance to the forward ejection of a large SV allows for the performance of maximal myocardial work at a minimum of oxygen consumption. Eventually, however, progressive volume overload increases ventricular end-diastolic volume to the point that compensatory hypertrophy is no longer sufficient to compensate, and a decline in systolic function occurs. As systolic function declines, end-systolic dimension increases further, LV wall stress increases, and LV function is further compromised by the excessive ventricular afterload. At this point, the decline of ventricular function is progressive and can be quite rapid.

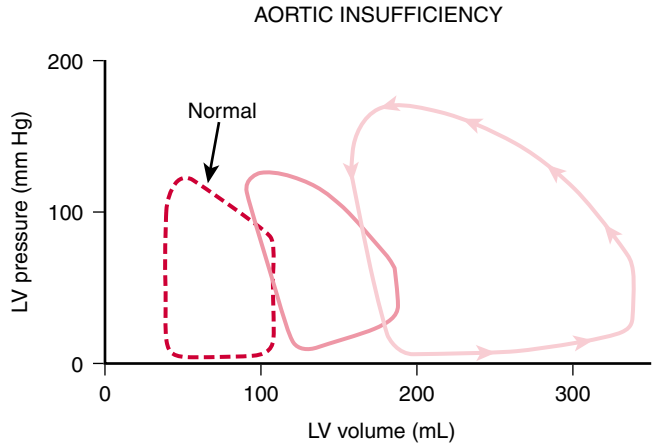

Figure 14-3 Pressure-volume loop in acute (*middle loop*) and chronic (*right loop*) aortic regurgitation. (Adapted from Jackson JM, Thomas SJ, Lowenstein E: Anesthetic management of patients with valvular heart disease. Semin Anesth 1:239, 1982.)

Despite the relatively normal $M\dot{V}O_2$, angina can occur in one third of patients with severe aortic regurgitation, even in the absence of CAD. Patients with chronic aortic regurgitation 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 (CBF), 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 aortic regurgitation may be at risk for acute ischemia with episodes of significant bradycardia. Because bradycardia prolongs diastolic time, it increases regurgitant flow, and LV diastolic pressure and wall tension rise 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 rapidly dilate such that progressively increased end-systolic dimensions are seen, and ischemia and ventricular failure become a positive feedback loop.

Acute Aortic Regurgitation

In acute aortic regurgitation, sudden diastolic volume overload of a nonadapted left ventricle results in a precipitous rise in the end-diastolic pressure because the ventricle is operating on the steepest portion of the diastolic pressure-volume curve. In severe acute aortic regurgitation, the LVEDP can equilibrate with aortic diastolic pressure and exceed the LA 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 aortic regurgitation. Although this phenomenon initially shields the pulmonary capillaries from the full force of the dramatically elevated LVEDP, the protection may be short-lived. Severe LV distention often follows and produces mitral annular enlargement and functional mitral regurgitation.

The inevitable fall in SV in acute decompensating aortic regurgitation elicits a reflex sympathetic response so that tachycardia and a high systemic vascular resistance

BOX 14-3 *Aortic Regurgitation*

Preload:	Increased
Afterload:	Decreased
Goal:	Augmentation of forward flow
Avoid:	Bradycardia

(SVR) are common. Moderate tachycardia beneficially shortens the regurgitant time without reducing the transmitral filling volume. Vasoconstriction, however, preserves CPP at the expense of increasing the aorta-ventricular gradient and regurgitation.

As may be expected, patients with acute aortic regurgitation may be at greater risk for myocardial ischemia. As with chronic aortic regurgitation and bradycardia, coronary perfusion may be compromised by the combination of a low diastolic arterial pressure and the precipitously increased LVEDP. This narrowing of CPP may be so severe that the phasic epicardial blood flow may change to a predominantly systolic pattern with very severe acute aortic regurgitation. Dissection of the coronary ostia is rare but frequently causes fatality in patients with acute aortic regurgitation. 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 the intra-aortic balloon are usually contraindicated, because augmenting the diastolic pressure worsens regurgitation.

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

Anesthetic Considerations

Intraoperative monitoring should include an ECG system with the capability of monitoring a lateral precordial lead, because ischemia is a potential hazard (Box 14-3). For most valvular procedures, a PA catheter provides very useful information. A PA catheter allows determination of basal filling pressures and cardiac output, which is particularly useful in chronic aortic regurgitation given the potential unreliability of the clinical history and EF. Equally important is the ability to accurately monitor ventricular preload and cardiac output response to pharmacologic interventions. The aggressive use of vasodilators is often 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 pulmonary artery diastolic pressure or PCWP, may be crucial to optimize cardiac output when afterload is pharmacologically manipulated. The other requirement for a PA catheter is to allow for pacing when it is anticipated. The deleterious effects of significant bradycardia in aortic regurgitation have been described. In patients who arrive in the operating room with heart rates less than 70 or in patients for whom rapid epicardial pacing may be difficult to establish (e.g., redo operations), placement of a pacing wire is probably indicated. Typically, only a ventricular wire would be appropriate; it is

more reliable than atrial pacing and, in aortic regurgitation, the atrial contribution to ventricular diastolic volume usually is not essential. Capturing the ventricle with a PA-based transvenous wire can be difficult because of the very large ventricular cavity size in patients with chronic aortic regurgitation.

Because patients with aortic regurgitation may differ widely in their degree of myocardial dysfunction, anesthetic management must be appropriately individualized. The hemodynamic goals are a mild tachycardia, a positive inotropic state, and a controlled reduction in SVR. For cardiac surgery, dopamine or dobutamine, pancuronium, ketamine, and nitroprusside infusions are excellent choices. For the patient with acute aortic regurgitation, the goals are the same but urgency must be stressed. It is essential to try to rapidly reduce end-diastolic and end-systolic ventricular volumes with the very aggressive use of inotropes (e.g., epinephrine) and vasodilators. Positive inotropes should not be withheld from the patient whose condition 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 aortic regurgitation, serial measurements of cardiac output can indicate that ventricular size and cardiac output have been optimized, regardless of the systemic pressure. TEE is also useful to look at ventricular size, but probably maximizing cardiac output under these conditions gets closer to the therapeutic goal than looking at ventricular size alone. With acute aortic regurgitation and premature closure of the mitral valve, the PA pressures may grossly underestimate the LVEDP, which continues to rise under the influence of the diastolic regurgitant jet from the aorta.

The early and late phases of CPB can be a real problem in aortic regurgitation, particularly in repeat operations. Before cross-clamp placement, the ventricle is at risk for distention if it is not ejecting or being vented. If the ventricle dilates with aortic regurgitation during bypass, the intraventricular pressures may equilibrate with the aortic root pressures. Under these conditions, there is no coronary perfusion, and the ventricle may rapidly dilate and become profoundly ischemic. This can occur before cross-clamp placement with bradycardia, ventricular fibrillation, or tachycardia, or even with a rapid supraventricular rhythm that compromises organized mechanical activity. Correcting the rhythm, pacing, cross-clamping the aorta, or venting the ventricle addresses this problem. This can also occur in cardiac surgery for conditions other than aortic regurgitation. In patients with unknown or uncorrected aortic regurgitation, removal of the cross-clamp causes the same ventricular dilation and ischemia if a 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 coronary surgery alone, in those with mild or moderate aortic regurgitation not having aortic valve replacement, and in patients in whom intraoperative TEE is not used.

MITRAL REGURGITATION

Clinical Features and Natural History

Unlike mitral stenosis, which is almost always the result of rheumatic valve disease, mitral regurgitation may result from a variety of disease processes that affect the valve leaflets, the chordae tendineae, the papillary muscles, the valve annulus, or the left ventricle. Mitral regurgitation can be classified as organic or functional. Organic mitral regurgitation 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 represent the most common cause of mitral regurgitation. Other causes of organic mitral regurgitation

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 mitral regurgitation 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 mitral regurgitation describes cases in which mitral regurgitation occurs despite structurally normal leaflets and chordae tendineae. Resulting from altered function or geometry of the left ventricle or mitral annulus, functional mitral regurgitation often occurs in the setting of ischemic heart disease, and the term *ischemic mitral regurgitation* is sometimes used interchangeably with *functional mitral regurgitation*. However, the functional form can occur in patients without demonstrable CAD, such as those with idiopathic dilated cardiomyopathy and mitral annular dilatation.

Because it can be caused by a wide variety of disease processes, the natural history of mitral regurgitation is quite variable. Even among patients with acute-onset disease, the clinical course depends on the mechanism of regurgitation and the response to treatment. For instance, patients presenting with acute, severe mitral regurgitation due to a ruptured papillary muscle have a dismal outcome without surgery. However, the clinical course of acute mitral regurgitation due to endocarditis could be favorable if the patient responds well to antibiotic therapy. Although those with chronic mitral regurgitation usually enter an initial, often asymptomatic, compensated phase, the time course for progression to LV dysfunction and symptomatic heart failure is unpredictable. The literature reflects the wide variability in the natural history of mitral regurgitation, with published 5-year survival rates for patients with mitral regurgitation of 27% to 97%.[7]

Pathophysiology

Mitral regurgitation causes LV volume overload. The regurgitant volume combines with the normal LA volume and returns to the left ventricle during each diastolic period. This elevated 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 further enhances the contractile appearance of the left ventricle.

The presentation of patients with mitral regurgitation varies depending on the pathophysiology of the specific condition, which is affected by the mechanism, severity, and acuity of the mitral regurgitation. In cases of acute, severe mitral regurgitation, such as patients with a ruptured papillary muscle after acute myocardial infarction, the sudden increase in preload enhances LV contractility by the Frank-Starling mechanism. Despite the increased preload, LV size is initially normal. Normal LV size combined with the ability to eject into a low-pressure circuit (i.e., the left atrium) results in decreased afterload in the acute setting. The measured LVEF in cases of sudden, severe mitral regurgitation may approach 75%, although forward SV is reduced. However, because the left atrium has not yet dilated in response to the large regurgitant volume, LA pressure rises acutely and may lead to pulmonary vascular congestion, pulmonary edema, and dyspnea.

Many patients with mitral regurgitation, particularly those whose valvular incompetence develops more slowly, may enter a chronic, compensated phase. In this phase, chronic volume overload triggers LV cavity enlargement by promoting eccentric hypertrophy. Elevated preload continues to augment LV systolic performance. At the same time, the left atrium dilates in response to the ongoing regurgitant volume. Although LA dilatation maintains a low-pressure circuit that facilitates LV systolic ejection, the increased radius of the LV cavity leads to increased wall tension.

With the eventual decline in LV systolic function, patients enter a decompensated phase. Progressive LV dilatation increases wall stress and afterload, causing further deterioration in LV performance, mitral annular dilatation, and worsening of the mitral regurgitation. LV end-systolic pressure increases. The increased LV filling pressures result in elevation of LA pressures and, given time, pulmonary vascular congestion, pulmonary hypertension, and RV dysfunction. In addition to fatigue and weakness, patients with decompensated, chronic mitral regurgitation may also report dyspnea and orthopnea. It is difficult to predict when a patient with mitral regurgitation is likely to decompensate clinically. The progression of disease in any given patient depends on the underlying cause of mitral regurgitation, its severity, the response of the left ventricle to volume overload, and possibly the effect of medical management.[8]

Surgical Decision Making

Just as progress in the understanding of the pathophysiology of mitral regurgitation has evolved, so too has the surgical approach to this disease process. A high operative mortality associated with the surgical correction of mitral regurgitation in the 1980s led many clinicians to manage patients conservatively. Because favorable loading conditions and high LA compliance allow even patients with significant mitral regurgitation to remain asymptomatic for long periods, it is likely that many patients did not undergo surgery until the onset of disabling symptoms. Studies show that more severe preoperative symptoms are associated with a lower EF and a higher incidence of postoperative CHF. Historically, poor outcomes after surgery for mitral regurgitation might have occurred because clinicians did not appreciate the true degree of LV dysfunction at the time of surgery in symptomatic patients. An EF of less than 60% in the setting of severe mitral regurgitation represents significant LV 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 LV systolic performance after mitral replacement.

In part because of improved surgical techniques, the operative mortality rate for patients with organic mitral regurgitation who are younger than 75 years is about 1% in some centers. Besides preservation of the subvalvular apparatus, valve repair represents another surgical technique associated with improved postoperative outcome.[9] 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 experience lower operative mortality and better long-term survival largely because of improved postoperative LV function. The survival benefit that accompanies valve repair is also observed among patients undergoing combined valve and coronary artery surgery. Valve repair does not increase the likelihood of reoperation when 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 is often 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 LV function.

BOX 14-4 ***Mitral Regurgitation***

Preload:	Increased
Afterload:	Decreased
Goal:	Mild tachycardia, vasodilation
Avoid:	Myocardial depression

Anesthetic Considerations

Patients who present to the operating room with mitral regurgitation may differ significantly with respect to duration of disease, symptoms, hemodynamic stability, ventricular function, and involvement of the right heart and pulmonary circulation (Box 14-4). For instance, a patient presenting with severe mitral regurgitation due to acute papillary muscle rupture may enter the operating room in cardiogenic shock with pulmonary congestion requiring intra-aortic balloon pump (IABP) augmentation. Another patient with a newly diagnosed flail posterior mitral leaflet may enter the surgical suite with relatively preserved LV function and no symptoms whatsoever. In the latter patient, the compliance of the left atrium may have prevented pulmonary vascular congestion, pulmonary hypertension, and RV dysfunction. Despite the differences in presentation, the general management goals remain similar and include maintenance of forward cardiac output and reduction in the mitral regurgitant fraction. The anesthesiologist must also seek to optimize RV function, in part by avoiding increases in pulmonary vascular congestion and pulmonary hypertension. Various degrees of intervention are needed to achieve these hemodynamic management goals depending on the patient's presentation.

Invasive hemodynamic monitoring provides the anesthesiologist with 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 anesthetic manipulations. PA catheters facilitate many aspects of intraoperative patient management. Intraoperative use of a PA catheter allows the anesthesiologist to more carefully optimize left-sided filling pressures. Although the PCWP and PA diastolic pressure depend on LA and LV compliance and filling, examination of intraoperative trends in these variables enhances the ability of the anesthesiologist to provide appropriate levels of preload while avoiding volume overload. Periodic determination of cardiac output 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 mitral regurgitation, because this finding depends on LA compliance. Just as in the management of patients with aortic valvular regurgitation, another benefit of PA catheter insertion is the ability to introduce a ventricular pacing wire to rapidly counteract hemodynamically significant bradycardia. In patients with RV compromise, monitoring trends in the CVP recording may also be helpful. Tricuspid regurgitation detected through analysis of the CVP tracing may suggest RV dilatation, which may be caused by pulmonary hypertension.

Intraoperative TEE provides invaluable information during the surgical correction of mitral regurgitation. It reliably identifies the mechanism of mitral regurgitation, thereby guiding the surgical approach,[10] and it objectively demonstrates the size and function of the cardiac chambers. TEE can readily 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 even replace the mitral valve. TEE also identifies concomitant pathology that may warrant surgical attention, such as atrial level shunts and additional valve disease.

The intraoperative management of patients with mitral regurgitation before the institution of CPB focuses on optimizing forward cardiac output, minimizing the mitral regurgitant volume, and preventing deleterious increases in pulmonary artery pressures. Maintaining adequate LV preload is essential. An enlarged left ventricle that operates on a higher portion of the Frank-Starling curve requires adequate filling. At the same time, excessive volume administration is to be avoided because it may cause unwanted dilatation of the mitral annulus and worsening of the mitral regurgitation. Excessive fluid administration may precipitate RV failure in patients with pulmonary vascular congestion and pulmonary hypertension. Optimization of preload is aided by analysis of data obtained from PA catheter measurements and TEE images. Because significant LV dysfunction is present in many patients with mitral regurgitation, anesthesiologists often select specific induction and maintenance regimens to avoid further depressing LV function. For this reason, large doses of narcotics have been popular in the past. Others have shown that smaller doses of narcotics combined with vasodilating inhalation anesthetics also 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 postoperatively. 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 LV dysfunction, infusions of inotropic medications such as dopamine, dobutamine, or even epinephrine may be required to maintain an adequate cardiac output. Phosphodiesterase inhibitors such as milrinone may also augment systolic ventricular performance and reduce pulmonary and peripheral vascular resistances. By reducing pulmonary and peripheral vascular resistance, forward cardiac output 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 IABP should be strongly considered.

Manipulation of the heart rate may be necessary in some patients to optimize hemodynamics. Bradycardia should generally be avoided because slower heart rates allow for larger filling volumes, potentially resulting in LV distention and mitral annular dilatation. Regurgitant volumes may increase at slower heart rates. Slightly increased heart rates, especially when combined with increased LV contractility, favor a smaller mitral annular area and may decrease the regurgitant fraction. Sinus rhythm and preserved atrial contraction are less important in patients with mitral regurgitation compared with patients with stenotic valves. Mitral annular dilatation accompanies most cases of long-standing mitral regurgitation. Patients with pure mitral regurgitation generally have no impedance to LV filling, and atrial fibrillation is usually better tolerated than in patients with stenotic lesions.

Because severe mitral regurgitation may result in pulmonary hypertension and RV dysfunction, anesthesiologists should tailor their intraoperative management strategies accordingly. Hypercapnia, hypoxia, and acidosis elevate pulmonary artery pressures and should be avoided. Mild hyperventilation may be beneficial in some patients.

Patients with severe RV dysfunction after CPB can prove exceptionally difficult to manage. Besides avoiding the factors known to increase peripheral vascular resistance (PVR), only a few options exist for these patients. Inotropic agents with vasodilating properties such as dobutamine, isoproterenol, and milrinone augment RV systolic performance and decrease PVR, but their use is often 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 pulmonary artery pressures after CPB, systemic hypotension requiring infusions of vasoconstrictors through an LA catheter has also occurred. Inhaled nitric oxide represents another alternative available for the treatment of RV 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.

LV dysfunction may also contribute to post-CPB hemodynamic instability. With mitral competence restored, the low-pressure outlet for LV ejection is removed. The enlarged left ventricle must then eject entirely into the aorta. Because LV enlargement leads to increased wall stress, a condition of elevated afterload often exists after CPB. At the same time, the preload augmentation inherent to mitral regurgitation is removed. It is therefore not surprising that the systolic performance of the left ventricle often declines after surgical correction of mitral regurgitation. Treatment options in the immediate post-CPB period include inotropic and vasodilator therapy and, if necessary, IABP augmentation.

MITRAL STENOSIS

Clinical Features and Natural History

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

A decades-long asymptomatic period characterizes the initial phase of rheumatic mitral stenosis. Symptoms rarely appear until the normal mitral valve area of 4 to 6 cm^2 (Fig. 14-4) has been reduced to 2.5 cm^2 or less. When the mitral valve area reaches 1.5 to 2.5 cm^2, symptoms usually occur only in association with exercise or other conditions, such as fever, pregnancy, or atrial fibrillation, that lead to an increase in heart rate or cardiac output. After the mitral valve area falls below 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 mitral stenosis commonly report dyspnea as their initial symptom, a finding reflective of elevated LA pressure and pulmonary congestion. In addition to dyspnea, patients may report palpitations that signal the onset of atrial fibrillation. Systemic thromboembolization occurs in 10% to 20% of patients with mitral stenosis and does not appear to be correlated with the mitral valve area or LA size. Chest pain that simulates angina is present in a small number of patients with mitral stenosis and may result from RV hypertrophy rather than CAD.

There has been a change in the typical age of presentation of patients with mitral stenosis. Previously, patients, often women, presented with mitral stenosis while in their 20s and 30s. In the past 15 years, perhaps because of more slowly progressive disease in the United States, patients have been presenting in their 40s and 50s. After symptoms develop, mitral stenosis remains a slow, progressive disease. Often, patients 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.

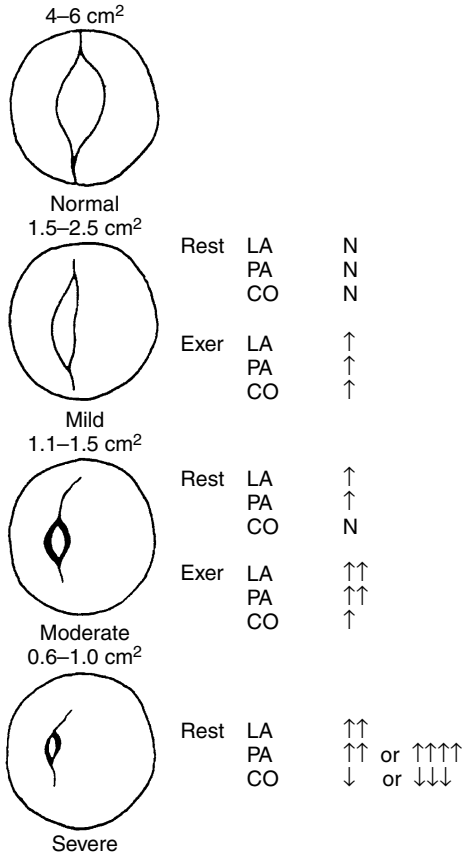

Figure 14-4 Hemodynamic changes with progressive narrowing of the mitral valve. LA = left atrium; PA = pulmonary artery; CO = cardiac output. (From Rapaport E: Natural history of aortic and mitral valve disease. Am J Cardiol 35:221, 1971.)

Pathophysiology

Rheumatic mitral stenosis 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 mitral valve area and limit diastolic flow into the left ventricle. As a result of the fixed obstruction to LV inflow, LA pressures rise. Elevated LA pressures limit pulmonary venous drainage and result in elevated PA pressures. Over time, PA hypertrophy develops in response to chronically elevated pulmonary vascular pressures. Pulmonary hypertension may trigger increases in RV end-diastolic volume and pressure, and in some patients, signs of RV failure such as ascites or peripheral edema may appear. LA enlargement is an almost universal finding in patients with established mitral stenosis and is a risk factor for the development of atrial fibrillation.

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

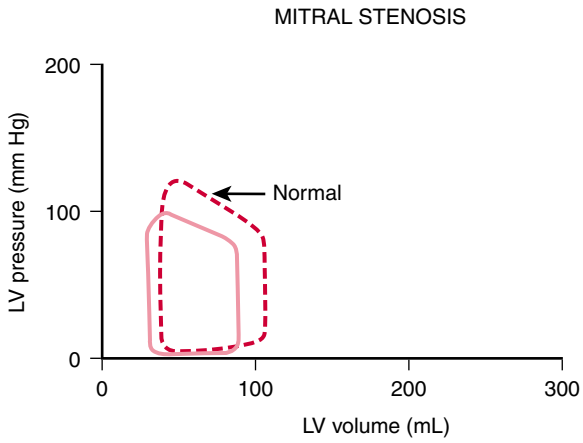

Figure 14-5 Pressure-volume loop in mitral stenosis. LV = left ventricular. (From Jackson JM, Thomas SJ, Lowenstein E: Anesthetic management of patients with valvular heart disease. Semin Anesth 1:239, 1982.)

$$\text{Value area} = \frac{\text{Flow}}{\sqrt{\text{PG}}}$$

Tachycardia necessitates a significant increase in the transvalvular pressure gradient and may precipitate feelings of breathlessness in awake patients. In patients with atrial fibrillation, it is the elevated 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 mitral stenosis and atrial fibrillation should be control of the ventricular rate.

Mitral stenosis results in diminished LV preload reserve. As seen in the pressure-volume loop in Figure 14-5, LVEDV and LVEDP are reduced with an accompanying decline in SV. Controversy exists, however, regarding the contractile state of the left ventricle in these patients. Limited preload may contribute to a reduced EF in some of these patients. However, the observation that LV contractile impairment persists postoperatively in some patients suggests that other causes of LV dysfunction may exist. Rheumatic myocarditis has been reported, although its role in producing LV contractile dysfunction is uncertain.

Assessment of Mitral Stenosis

As for patients with mitral regurgitation, echocardiography represents the diagnostic modality of choice for patients with suspected mitral stenosis.[11] Two-dimensional and Doppler echocardiographic techniques are able to accurately and noninvasively measure the transvalvular pressure gradient and mitral valve area. Because the pressure gradient varies with the flow rate and diastolic period, the assessment of mitral stenosis severity ideally should be based on the measured or calculated mitral valve area. Echocardiographic methods used to obtain mitral valve area include the pressure half-time technique, the continuity equation, planimetry of the valve orifice, and Proximal Isovelocity Surface Area (PISA) analysis. Other invaluable information obtained during an echocardiographic study includes the size and function of the ventricles and an estimation of the pulmonary artery pressure.

Surgical Decision Making

Appropriate referral of patients for surgical intervention requires integration of clinical and echocardiographic data. Patients presenting with severe symptoms (i.e., NYHA class III and IV) should be immediately referred for surgery because their outcome is poor if treated medically. Patients with only mild mitral stenosis and few or no symptoms may be managed conservatively with periodic evaluation. Patients who are asymptomatic but have moderate mitral stenosis (i.e., mitral valve area between 1.0 and 1.5 cm^2) require careful assessment. If significant pulmonary hypertension (i.e., PA systolic pressure > 50 mmHg) is present, surgical intervention should be considered. Intervention may also be indicated if a patient becomes symptomatic or PA pressures increase significantly during exercise testing.

The surgical options for treating mitral stenosis 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 is still used to treat mitral stenosis 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 mitral valve area and decreasing the LA-to-LV pressure gradient with a resultant relief in the patient's symptoms.

Percutaneous mitral commissurotomy (PMC) allows a less invasive, catheter-based approach to mitral stenosis. First reported by Inoue in 1984,[12] 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. For instance, those with heavily calcified valves or significant mitral regurgitation are likely to experience suboptimal results after commissurotomy. Mitral valve anatomy unsuitable for PMC is more commonly encountered in Western countries, where patients with mitral stenosis typically present at an older average age. Mitral valve replacement is commonly recommended for these patients. The risk of 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 generally is less than 5%. Conversely, surgical risk in elderly patients with severe symptoms related to mitral stenosis and multiple comorbidities may be 10% to 20%.

Anesthetic Considerations

Several important goals should guide the anesthetic management of patients with significant mitral stenosis. First, the anesthesiologist should seek to prevent tachycardia and treat it promptly if it develops in the perioperative period (Box 14-5). Maintenance of LV preload without exacerbation of pulmonary vascular congestion represents a second management goal. Third, anesthesiologists should avoid factors that aggravate pulmonary hypertension and impair RV function.

Prevention and treatment of tachycardia are central to the perioperative management of these patients. Tachycardia shortens the diastolic filling period. To maintain LV preload with a shortened diastolic period, an elevation in transvalvular flow rate is required with a resultant increase in the LA-to-LV pressure gradient. Avoidance of

BOX 14-5 ***Mitral Stenosis***

Preload:	Normal or increased
Afterload:	Normal
Goal:	Controlled ventricular response
Avoid:	Tachycardia, pulmonary vasoconstriction

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 LV preload. Appropriate monitoring and supplemental oxygen therapy should be considered for patients receiving preoperative narcotics or benzodiazepines. Medications taken by the patient preoperatively 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 may be required intraoperatively, particularly to control the ventricular rate in patients with atrial fibrillation. Control of the ventricular rate remains the primary goal in managing patients with atrial fibrillation, although cardioversion should not be withheld from patients with atrial tachyarrhythmias who become hemodynamically unstable. Narcotic-based anesthetics are often 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 another important goal for managing patients who have a fixed obstruction to LV 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 could aggravate pulmonary vascular congestion. Placement of an arterial catheter facilitates timely recognition of hemodynamic derangements. PA catheters can be invaluable in the management of patients with significant mitral stenosis. Even though the PCWP overestimates LV filling and the pulmonary artery diastolic pressure may not accurately reflect left-sided heart volume in patients with pulmonary hypertension, examination of trends and responses to intervention can be more readily assessed. Tachycardia increases the pressure gradient between the left atrium and left ventricle. Elevated heart rates widen the discrepancy between the PCWP and the true LVEDP. Despite these limitations, the PA catheter remains a useful monitoring tool, providing information on cardiac output and pulmonary artery pressures. As anesthesiologists gain an increasing appreciation for the role of intraoperative TEE, this powerful imaging modality will no doubt be used more frequently to assess ventricular filling and function.

Many patients with mitral stenosis present with pulmonary hypertension. Anesthetic techniques that avoid increases in PVR are likely to benefit these patients and prevent additional RV embarrassment. Meticulous attention to arterial blood gas analysis results allows appropriate adjustment of ventilatory parameters. Vasodilator therapy in patients with pulmonary hypertension generally is ineffective as the venodilation produced further limits LV filling and does not improve cardiac output.

The only mitral stenosis patients who may benefit from vasodilator therapy are those with concomitant mitral regurgitation or those with severe pulmonary hypertension and RV dysfunction in whom pulmonary vasodilation can facilitate transpulmonary blood flow and improve LV filling. The treatment of RV dysfunction has been discussed in preceding sections.

TRICUSPID REGURGITATION

Clinical Features and Natural History

Surgical tricuspid disease is caused by a structural defect in the valve apparatus or is a functional lesion. Functional tricuspid regurgitation is far more common and usually results from RV overload and tricuspid annular dilation. Left-sided valvular disease, usually mitral regurgitation, is most commonly responsible. Functional tricuspid incompetence can also result from mitral stenosis, aortic regurgitation or stenosis, or from isolated pulmonary hypertension. When mitral regurgitation is severe enough to warrant valve repair or replacement, tricuspid regurgitation may be present in 30% to 50% of patients.[13]

Tricuspid regurgitation may also be caused by structural defects as in rheumatic valve disease, carcinoid syndrome, endocarditis, Epstein's anomaly, or trauma.[14] In rheumatic disease, histologic involvement of the tricuspid valve may occur in 46% of patients, but it is rarely clinically severe, and in these cases, the valve is usually also stenotic. Tricuspid regurgitation has also been described in association with CAD as a result of ischemia, infarction, or rupture of the RV papillary muscles.

Symptoms of isolated tricuspid insufficiency 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.

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

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

Surgical Decision Making

In structural tricuspid insufficiency, the decision to repair or replace the valve is straightforward. The same cannot be said of functional tricuspid regurgitation. Because most functional cases are the consequence of left-sided valve lesions with RV overload, the tricuspid regurgitation usually improves significantly after the aortic or mitral valve is repaired or replaced, typically at least one grade. 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 tricuspid regurgitation is severe in the pre-CPB assessment, tricuspid valve surgery is almost always performed.[14] However, the evidence is less clear when the regurgitation is graded as moderate. Some surgeons choose to repair the tricuspid with moderate regurgitation, but others advocate observation.[15] It is common with moderate or moderate to severe tricuspid regurgitation, in the context of left-sided valve surgery, to complete the left-sided procedure and then reassess the tricuspid valve with TEE when the heart is full and ejecting. If the regurgitation remains more than moderate after the left-sided valve is fixed, many surgeons then do the tricuspid procedure. If the regurgitation is moderate or less, the appropriate surgical course may remain unclear. Some patients having left-sided valve procedures must return to the operating room in the future for tricuspid surgery. When this occurs, the morbidity and mortality rates are probably significantly increased over what would have been experienced were the tricuspid valve fixed at the time of the aortic or mitral valve procedure. Decision making in functional tricuspid regurgitation is made more complicated by the inability to rigorously quantify the severity of the regurgitation and RV dysfunction.

Anesthetic Considerations

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

If RV dysfunction is predicted, it is useful to place a PA catheter, even if the tricuspid valve will be replaced. Even if the PA catheter has to be removed because of tricuspid valve replacement, it still can be helpful to obtain cardiac outputs and pulmonary artery pressures before CPB to get insight into RV function and anticipate the hemodynamic support that may be required. A PA catheter is also of greater use than a central venous catheter alone because the CVP is a poor index of intravascular filling and the degree of tricuspid regurgitation. This is true because the atrium and vena cavae are highly compliant and will accept large regurgitant volumes with relatively little change in pressure. A PA catheter is also useful even if intraoperative TEE is used. As in aortic insufficiency with the left ventricle, the right ventricle in chronic tricuspid regurgitation is volume overloaded and dilated and requires a large end-diastolic volume to maintain forward flow. At the same time, because of the unreliability of the CVP as an indicator of filling, it is possible to volume overload patients with tricuspid regurgitation and RV failure. Cardiac output in RV failure can often be augmented with the use of vasodilators; and even though RV dimensions can be followed intraoperatively with TEE, maximizing cardiac output (sometimes at the cost of systemic arterial pressure) is best done with serial cardiac output measurements (as in aortic regurgitation). Whenever there is significant RV distention, the possibility of septal shift and secondary deterioration of LV diastolic compliance should be carefully considered. Echocardiography is uniquely helpful for this assessment.

The post-CPB management of the patient undergoing an isolated tricuspid valve procedure is usually straightforward. These patients usually do not have significant RV 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 tricuspid regurgitation related to aortic stenosis, typically come off CPB with little need for support of the right ventricle. These patients often do well because the improvement in LV function after AVR for aortic stenosis is usually sufficient to reduce pulmonary artery pressures significantly and offload the right side of the heart. When the left-sided valve surgery is for mitral disease, the improvement is usually not as marked and greater degrees of inotropic support of the right ventricle are often indicated. The combination of a phosphodiesterase inhibitor with a vasodilator and a catecholamine infusion is useful. Serial cardiac output measurements to balance systemic pressure and RV 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 elevated with tricuspid regurgitation, 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 significant RV dysfunction is present or there is peripheral edema or ascites, there is the potential for a coagulopathy related to liver congestion, and the patient should be managed accordingly. Fourth, it is important to ensure that central venous catheters, particularly PA catheters, are not entrapped in right atrial suture lines.

INNOVATIONS IN VALVE REPAIR

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

Aortic Valve Repair

Over the past several years there has been a major shift from valve replacement to valve repair in degenerative mitral valve disease. The same has not been true of the aortic valve, in part because the valve disease is different in most patients, but also because the high flow and pressure conditions across the aortic valve might make repair more prone to failure. That said, aortic valve repair is being increasingly done as an appropriate patient population is being defined. Valve repair for aortic regurgitation has found broader use when regurgitation is associated with dissection or dilation of the aortic root, but isolated valve repair has been less common.[16] A growing body of data suggests that aortic valve repair may offer advantages over valve replacement in younger individuals with aortic insufficiency caused by bicuspid valves. In contrast to aortic valve replacement, this eliminates the need for anticoagulation for a mechanical valve and may delay the need for reoperation if a tissue valve has been placed. When regurgitation occurs with a bicuspid valve, the insufficiency is usually caused by retraction or prolapse of the conjoined cusp and repair consists of a triangular incision to shorten and elevate that cusp to improve apposition. Although very

long-term follow-up has not been reported, in a large series from the Mayo Clinic with a mean follow-up of 4.2 years, late failure of the repair requiring reoperation occurred in 14 of 160 consecutive patients, with most of that failure occurring from repairs done in the first decade of the 15-year experience.

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

Techniques for Mitral Valve Repair

Mitral regurgitation is frequently 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 different approaches have been developed to address mitral regurgitation occurring in the absence of structural mitral pathology. These approaches address the failure of leaflet coaptation at the level of the valve leaflets or the valve annulus or by altering the anatomic relationship of the septal and lateral walls of the left ventricle.

Mitral Leaflet Repair

Alfieri and associates showed that mitral regurgitation could be improved using an edge-to-edge technique by which mitral valve leaflets are brought together by a central suture. This surgical approach led to a catheter-based technology, which results in edge-to-edge repair by apposing the edges of a regurgitant mitral valve. The Evalve device consists of a catheter-mounted clip that is threaded using a femoral and transseptal approach.[17] With the use of general anesthesia and echocardiographic and fluoroscopic guidance, the clip is placed to achieve apposition of the central portion of the anterior and posterior leaflets. If the severity of mitral regurgitation is not reduced, the device can be opened and repositioned.

Percutaneous Mitral Annuloplasty

The second type of approach to functional mitral regurgitation is alteration of the mitral annulus, as might occur with a traditional open mitral repair; however, with the percutaneous approach the annulus is downsized by an extracardiac restraint.[18] With this technique, interventional cardiologists percutaneously thread a wire from the venous system into the right atrium and the coronary sinus. The relationship between the coronary sinus and posterior leaflet allows downsizing of the septal-lateral diameter from this position. Under echocardiographic guidance, tension is applied to cinch the mitral annulus smaller and an anchor is deployed to maintain position.

Altering Ventricular Anatomy to Reduce Mitral Regurgitation

The third approach to closed mitral valve repair consists of altering the geometry of the lateral and septal LV walls to bring the valve leaflets together. The commercial Coapsys device has entered clinical trials. This device 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.[19] This effectively brings the ventricular walls together and in doing so improves leaflet coaptation. TEE is used to optimize cord length and pad positioning. In contrast to the leaflet-based and

annular-based approaches, the Coapsys approach is surgical, requiring an open chest but not CPB. The position of the epicardial vessels and the relationship of the submitral apparatus could pose significant risk, but the device has been used successfully in animal models and preliminary clinical trials.

Percutaneous Valve Replacement

Although surgery, particularly for aortic valve disease, has expanded to include a much older population in recent years, there remains a subset of patients for whom cardiac surgery may entail unacceptable risks. For this population, less invasive techniques such as percutaneous aortic valve replacement are being developed, with the initial clinical experience with percutaneous replacement having been reported.[20]

The study population included patients with severe aortic stenosis and class IV heart failure who had been denied surgery because of excessive risk. The mean AVA was 0.49 cm^2, with a low transvalvular gradient reflecting ventricular decompensation. Patients had a mean EF of 24%. With the use of local anesthesia with sedation, the femoral vein was catheterized, and, using a transseptal approach, a balloon-tipped catheter was placed across the stenotic aortic valve. After valvuloplasty, the aortic valve stent (with incorporated leaflets) was seated at the native valve over a second balloon. In one patient, the valve was ejected into the ascending aorta on placement, and the patient died shortly thereafter. In two patients, hemodynamic collapse occurred during the initial valvuloplasty but both patients were resuscitated.

It is likely that many of these technologies will come to clinical trial and will demonstrate various degrees of efficacy. However, it is unclear what the long-term benefits of these less invasive interventions will be or how they will compare with each other or with traditional surgical approaches. Some may find use in high-risk patients as temporizing procedures, in place of reoperations, or in conjunction with percutaneous approaches for coronary disease.[21] These technologies will get better, and there will be pressure for clinical application. The most important factors will be case selection and long-term follow-up of outcomes; otherwise, these innovations and others like them will add markedly to the burden of health care costs without clear social benefit. For any of the percutaneous valves to succeed, they must demonstrate long-term successful clinical outcomes similar to the excellent results seen with mechanical or tissue valves for more than 10 to 15 years.[22]

SUMMARY

- Although various valvular lesions generate different physiologic changes, all valvular heart disease is characterized by abnormalities of ventricular loading.
- The left ventricle normally compensates for increases in afterload by increases in preload. This increase in end-diastolic fiber stretch or radius further elevates 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.
- Aortic stenosis is the most common valvular heart abnormality. Angina, syncope, and congestive heart failure are the classic symptoms and the indications for surgery.
- 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 (ablation) therapy with ethanol.

- 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.
- Mitral regurgitation causes left ventricular volume overload. Treatment depends on the underlying mechanism and includes angiotensin-converting enzyme inhibitors and surgical repair or replacement of the mitral valve.
- 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.
- Most tricuspid surgery occurs in the context of significant aortic or mitral disease, and anesthetic management is primarily determined by the left-sided valve lesion.
- Innovations in surgical valve repair include aortic valve repair and closed- and open-chamber procedures for mitral regurgitation.

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