Valvular Heart Disease and Postoperative Considerations

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Abstract
Despite increasing trends in catheter-based cardiac surgical procedures, more than 278 000 Americans had traditional cardiac surgery in 2013. Of those surgical procedures, approximately 133 000 involved valvular repair or replacement. Aortic valve replacement was by far the most common valvular operation, followed by mitral valve repair or replacement. This review article will discuss characteristics of valvular pathologies and postoperative concerns for each the 4 cardiac valves.

Keywords
cardiac surgery, aortic valve replacement, mitral valve, postoperative care, postoperative complications

Introduction

Although there are only 4 valves in the human heart, there are many pathologies that can occur with those 4 valves. Accordingly, each valve lesion has innate caveats that need to be recognized when caring for these patients in a postoperative setting. This review will highlight the physiologic changes and specific management points of care concerning common valvular lesions.

Aortic Stenosis

Aortic valve sclerosis and aortic valve stenosis (AS) are the most common valve diseases in Europe and North America, with sclerosis present in about 25% of all people older than 65 years and stenosis present in 2% to 7% of this population.[1-3] AS occurs earlier in patients with bicuspid aortic valve leaflets, likely due to years of shear stress. Other causes of AS include rheumatic heart disease and senile calcification. Overall, operative mortality rates are low for aortic valve replacement at 5% to 15%, even in octogenarians.[3]

Pathology of Aortic Stenosis

Aortic stenosis creates a chronic pressure overload on the left ventricle (LV) that will increase wall tension as described by Laplace's law:

$$\text{Wall tension} = \frac{\text{Pressure} \times \text{Radius}}{2 \times \text{Wall thickness}}$$

In attempts to decrease wall tension, the ventricle duplicates fibers, which increases wall thickness and decreases radius. This creates a concentric hypertrophy that results in LV pressure overload. Figure 1 demonstrates a typical LV pressure–volume loop for a control versus a patient with AS. The pressure generated by the LV during systole must be increased to overcome the aortic transvalvular pressure gradient. The more stenotic the lesion, the more pressure the LV must generate in order to create forward blood flow to the body.

Postoperative Considerations for Aortic Stenosis

Patients presenting with lone AS typically have maintained systolic function with coexisting diastolic heart failure (DHF) or a relaxation abnormality (reduced lusitropy). Because there is no significant regression of concentric hypertrophy for 6 to 12 months after aortic valve replacement, the compliance characteristics of the LV will be unchanged in the postoperative period.[4]

For several reasons, patients with concentric hypertrophy benefit from higher filling volumes. The ventricular hypertrophy causes a loss of compliance that results in failure to relax properly and failure to fill during diastole. Thus, adequate preload is necessary in order to fill the ventricle before systole begins. Coronary perfusion is dependent on end-aortic diastolic blood pressure, which is a preload dependent pressure. The thickened myocardium

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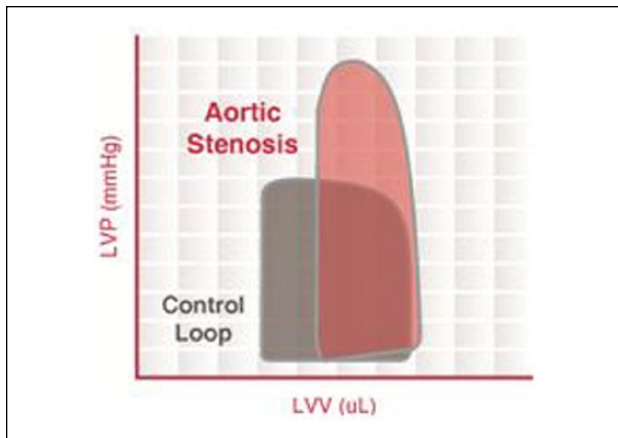Email: brigidcflynn@yahoo.com

Figure 1. Aortic stenosis (AS): AS leads to increased left ventricular systolic pressure. Development of concentric hypertrophy (smaller radius) allows normalization of wall stress. However, if systolic heart failure develops, a reduction in stroke volume and ejection fraction will be seen. Surgical correction should be imminent at this point.
Abbreviations: LVP, left ventricular pressure; LVV, left ventricular volume.
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will consume more oxygen and require more oxygen delivery in the form of coronary blood flow to function. "Adequate" preload may be assessed clinically by the hemodynamic response to passive leg raising and the systolic pressure variation in mechanically ventilated patients. Along with higher filling volumes, it is helpful to have longer diastolic filling times, or decreased heart rate. Increased heart rate results in less time for coronary perfusion, especially in the LV, which is perfused solely during diastole. Tachycardia should be treated in order to avoid subendocardial ischemia and hemodynamic compromise.[4] Attempts to ensure comfort and adequate volume resuscitation should be made when patients are tachycardiac.

Beta-blockade may be required in select patients to treat underlying tachycardia in an effort to improve hemodynamics. In the acute postoperative period, esmolol has the advantage of a short half-life (9 minutes) and can be terminated quickly should bradycardia or hypotension occur. If the patient is tachycardiac and hypotensive, phenylephrine is the pressor of choice because of its actions of increasing systemic vascular resistance and coronary perfusion pressure, while also causing a reflex bradycardia. However, phenylephrine may not be potent enough alone as a pressor following cardiopulmonary bypass. Immediately, postoperatively, many surgeons request the systolic blood pressure be kept low-normal. This should be done without compromising renal or cerebral perfusion, especially in patients who have coexisting carotid disease. The concern is that a forceful systolic ejection may cause a dehiscence of the newly placed valve. A large valve dehiscence would present as acute heart failure associated with a new murmur, while a small leaking dehiscence may present insidiously as unexplained hypotension and poor end-organ perfusion.

Not only is adequate filling time necessary but also maintenance of sinus rhythm is paramount. The atria supply as much as 40% of the cardiac output. Loss of atrial synchrony will have devastating consequences on the cardiac output. Patients with DHF decompensate with atrial fibrillation because of less preload filling time in a ventricle with preexisting relaxation problems.

For many of the above reasons, patients with AS physiology typically do not benefit from inotropic support. Systolic function is typically preserved in lone AS and the tachycardia and increased contractility of an inotrope may have deleterious effects on a thick LV. Also, reduction of systemic vascular resistance through use of a vasodilator is also not typically indicated. In fact, reduction of the diastolic coronary perfusion gradient will decrease oxygen supply to the thick myocardium. Table 1 elucidates differences in management goals among several valvular lesions.

Coexisting Cardiopulmonary Dysfunction

One of the most difficult clinical scenarios involves the patient with DHF due to AS, along with right or left ventricular systolic dysfunction. Whereas patients with isolated systolic heart failure often benefit from lower filling pressures via the use of diuretics, lower systemic (or pulmonary, in the case of the right ventricle) vascular resistance and inotropic support; this is not always the approach in DHF. Coexisting pulmonary hypertension is common and may require pulmonary vasodilators, which decreases the pulmonary vascular resistance along with the systemic vascular resistance. Both effects are detrimental to patients with DHF. All of these management goals are in stark contrast to the goals in DHF. In situations of mixed heart failure etiology, the cardiac lesion creating the worst instability needs to be treated more aggressively without sacrificing care of the other lesions. The use of a pulmonary artery catheter and echocardiography may prove particularly useful to guide management when competing lesions exist.

A pulmonary artery catheter can be used to monitor the central venous pressure, the pulmonary artery diastolic pressure and the pulmonary artery occlusion pressure, or wedge, as surrogates of intravascular volume. Obviously, an understanding of pertinent physiology and the shortcomings of a pulmonary artery catheter need to be recognized prior to making management decisions based on these numbers, as several caveats exist.

Ventricular hypertrophy and loss of compliance will create a higher "starting" left ventricular end-diastolic pressure (measured via pulmonary artery occlusion pressure, or wedge) than in a compliant ventricle. Hence, a high

Table 1. Goals of Care for Specific Valvular Pathologies and Cardiac Abnormalities.[a]

	LV Preload	Heart Rate	Contractility	SVR
Aortic stenosis	↑	↓, sinus	Maintain	↑
Aortic regurgitation	Maintain or ↑	↑	Maintain (may need support)	↓
Mitral stenosis	↑	↓	Maintain	Maintain
Mitral regurgitation	Maintain	↑	Maintain (may need support)	↓
Tricuspid stenosis	Maintain	↓	Maintain	Maintain or ↑
Tricuspid regurgitation	Maintain or ↑	↑	↑	Maintain
Pulmonic stenosis	↑	↓	Maintain	Maintain
Pulmonic regurgitation	↑	↑	↑	Maintain or ↓
Left heart failure	↓	Normal	↑	↓
Right heart failure	↓	↑	↑	Maintain
Pulmonary hypertension	↓	↑	↑	Maintain

Abbreviations: LV = left ventricle; SVR = systemic vascular resistance.
[a]Note that each pathology is benefited by certain hemodynamics, which makes optimization difficult when a patient has multiple competing pathologies.

pulmonary artery occlusion pressure may not represent fluid overload. Also, if the patient has pulmonary artery hypertension or coexisting right ventricular (RV) failure, the pulmonary artery diastolic pressure and/or central venous pressure, will have a higher baseline. Again, these indices do not necessarily represent fluid overload. If there is tricuspid regurgitation, the cardiac output is falsely elevated. In these cases, total body oxygen delivery versus consumption can be trended by obtaining a mixed venous saturation from the pulmonary artery catheter, which also carries a certain margin of error. While the exact numbers extracted using the pulmonary artery catheter are important, the trend of changes in these numbers as management strategies are initiated is crucial. Obtaining "baseline" filling pressures while the patient is hemodynamically stable may be prudent.

If the patient requires pulmonary vasodilation due to pulmonary hypertension and/or RV failure, inhaled agents such as prostacyclin analogs or nitric oxide are recommended in order to avoid or minimize decreases in systemic vascular resistance. Pulmonary hypertension and RV failure both greatly benefit from inodilators, such as milrinone and dobutamine, which increase RV contraction and also act as pulmonary vasodilators. Unfortunately, the concomitant increases in heart rate and LV contractility are deleterious for patients with DHF. Despite these negative effects, these agents may be required to treat coexisting systolic heart failure. The positive lusitropy provided by milrinone and dobutamine may actually help to relax the hypertrophied, low compliant LV.[5] As with any intervention, clinical parameters such as lactate, mixed venous saturation, and urine output should be monitored to assess the benefit of inotropic support.

Reliance on clinical acumen, along with monitoring tools, is imperative when patients have confounding data. For example, elevated pulmonary artery pressures, hypotension, and decreased cardiac output can all be due to several life-threatening problems. These findings could be associated with systolic dysfunction of either ventricle, systolic anterior motion of the mitral valve (SAM) or tamponade physiology. However, there are often subtle clues as to which abnormality is occurring. Systolic dysfunction is usually identified in the operating room. While it may worsen postoperatively, completely new onset systolic dysfunction is rare without an underlying cause. Similarly, SAM is usually identified in the operating room, but this lesion can definitely worsen postoperatively if certain therapies are instituted. Therapies such as inotropy, tachycardia and diuresis may bring about an otherwise quiescent SAM physiology. (SAM will be discussed below.) Pulmonary hypertension may be diagnosed by unexplained increases in central venous pressure, decreased cardiac output and/or decreased mixed venous saturation, especially if accompanied by hypoxia.

Finally, tamponade commonly follows copious chest tube output that may or may not subside. Pulsus paradoxus is pathognomonic of states of hypovolemia, however, is striking in true tamponade. Also, of all of the lesions, tamponade brings about equalization of pressures (left and right atrial and ventricular diastolic) moreso than the others. Last, large V-waves seen on the central venous pressure tracing can result from tamponade or right ventricular failure, but are not pronounced with SAM or LV failure.

Atrial Fibrillation

For reasons indicated earlier, maintaining normal sinus rhythm is important for effective cardiac output in patients with AS. These patients may be a particular population in

which prophylactic amiodarone is beneficial as the loss of "atrial kick" can lead to rapid decompensation.[6]

While institutions may vary in treatment of new onset atrial fibrillation (AF), it is agreed that attempts at restoring sinus rhythm need to be made. If the patient is unstable, immediate direct current cardioversion (DCCV) is necessary. If the patient is stable, many clinicians will attempt an amiodarone infusion. If sinus rhythm does not return, DCCV should be considered. Per American College of Cardiology/American Heart Association guidelines, if the AF has been present for ≤48 hours, cardioversion can be attempted even if systemic anticoagulation or transthoracic echocardiography have not been performed.[7]

Postoperative Conduction Disturbances

Conduction disturbances following AS surgery can occur due to ischemia to the atrioventricular (AV) node or surgical trauma to conduction system. Local edema of the conduction system can also contribute to conduction delays, but these are usually not permanent once the edema resolves. Because the aortic valve is located in close proximity to the conduction system, the incidence of conduction disturbances following aortic valve replacement is higher than with other valves with an incidence of permanent pacemaker requirement in about 1% of patients.[8] However, this percentage may be as high 8.5%, especially in patients with preoperative conduction disturbances.[9] Conduction delays in patients with left ventricular hypertrophy warrant special attention since the atrial kick is so important for LV ejection and filling. Following aortic valve surgery, most surgeons will place ventricular, and possibly atrial, epicardial pacing wires. Use of AV sequential pacing may be necessary, and is preferred over ventricular pacing alone, if an AV conduction abnormality causes hemodynamic instability. Pacing wires are typically removed in a few days following surgery if there are no signs of conduction disturbance.

Cerebral Ischemia

All patients receiving cardiopulmonary bypass need to have neurologic examinations postoperatively. The risk of a cerebrovascular event is greater when the heart is opened during valvular surgery, unlike coronary artery bypass grafting surgery. Left-sided heart valve replacement carries a higher risk than right-sided valve surgeries. In a recent prospective study, clinical strokes were detected in 17% and transient ischemic attacks in 2% of patients undergoing aortic valve replacement. Interestingly, in 54% of the "stroke-free" subjects, postoperative magnetic resonance imaging demonstrated a silent infarct.[10]

Transcatheter Aortic Valve Replacement. Because of the advanced age and high incidence of coexisting morbidities
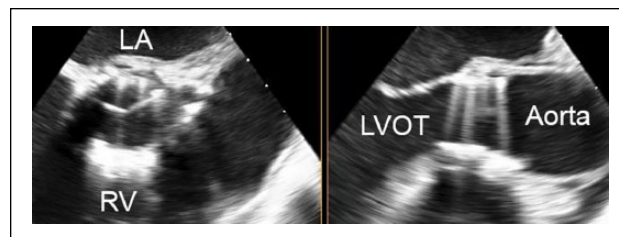

Figure 2. Images of a catheter deployed newly placed aortic valve. The left image is a midesophageal, aortic valve, short axis view (45°) and the right image is a midesophageal, aortic valve, long axis view (135°).
Abbreviations: LA, left atrium; RV, right ventricle; LVOT, left ventricular outflow tract.

in many patients with AS, a large number of patients are considered high-risk surgical candidates. Currently, there is a paradigm shift in that many patients considered to have prohibitive risks for surgical aortic valve replacement (AVR) are now being managed with the less invasive transcatheter AVR (TAVR) procedure (Figure 2). A TAVR can be performed via a transfemoral, transaortic, transapical (through the left ventricular apex) or less commonly via a transaxillary or subclavian approach.

There are 2 TAVR grafts currently used: the self-expanding CoreValve (Medtronic) and the balloon-expandable Sapien/Sapien XT (Edwards Lifesciences). Studies have not shown differences in mortality between the 2 valve systems. However, patients receiving a CoreValve have a higher incidence of permanent pacemaker requirement than with the Sapien valve system (37% vs 17%, respectively).[11,12] Temporary pacing wire implantation is a necessity for all TAVR procedures because the AV node and its left bundle branch lie adjacent to the noncoronary cusp of the aortic valve, leading to a potential risk of AV conduction block postintervention. The self-expanding CoreValve graft will continue to expand for 7 to 10 days, possibly explaining the higher risk of AV nodal blockade with this valve. A recent meta-analyses found that male patients, presence of a baseline conduction abnormality, or intraprocedural AV block during the procedure are risk factors for permanent pacemaker implantation following TAVR.[13]

Postoperatively, TAVR patients may be hemodynamically unstable due to significant preexisting comorbid conditions. Hypotension can be treated in typical fashion with fluids and vasopressors, however, significant paravalvular leak must be ruled out with echocardiography if the hypotension is not easily corrected or explained. Most clinicians avoid AV nodal blockers, such as beta-blockers, in the immediate postoperative period. Hypertension can be treated with hydralazine and/or dihydropyridine calcium channel blockers, such as nicardipine and amlodipine.

Evidence Behind TAVR

The largest trial evaluating TAVR is the PARTNER (Placement of AoRtic TraNscathetER Valves) trial that used the Sapien heart valve system and produced favorable results.[14,15] The trial had 2 cohort groups. Cohort A randomized high-risk patients with severe symptomatic aortic stenosis to TAVR versus standard surgical AVR and cohort B randomized patients with severe symptomatic AS deemed too high risk for standard surgery to either transfemoral TAVR or best medical management (including balloon valvuloplasty). The primary end-point was all cause mortality at one year.

Cohort A demonstrated mortality at one year of 24% in the TAVR patients compared with 26% in the AVR group (*P* = .001 for noninferiority). Additionally, there was no statistically significant difference in 30-day all-cause mortality. However, patients in the TAVR group did have a higher incidence of major vascular complications and stroke or transient ischemic attack, while major bleeding and new onset AF were more common in the AVR group.

Cohort B demonstrated a 20% absolute reduction in mortality in patients receiving TAVR versus best medical management, in which 78% of patients underwent balloon valvuloplasty. The 1-year mortality was reported to be 30.7% in the TAVR group compared with 50.7% with medical therapy. The number needed to treat was 5 patients treated with TAVR to save 1 life. There was also a significant improvement in quality of life in the TAVR group both at 30 days and at 1 year.

Aortic Regurgitation

Aortic regurgitation (AR) is characterized by increases in left ventricular volume and pressures; however, management of chronic versus acute AR differs considerably. Chronic AR is most commonly caused by a bicuspid AV, calcific valve disease or dilation of the aortic root (annuloaortic ectasia).[3] Aortic root dilation is the cause of about half of AR cases and timing of AV surgery depends on the pathology of the aortic root disease (Figure 3). In 80% of these cases, the cause is idiopathic ectasia, but can also be caused by aging, Marfan syndrome, Ehlers–Danlos syndrome, Behcet's disease, ankylosing spondylitis, Takayasu arteritis, systemic hypertension,[16] and fulminant infective endocarditis. Acute, severe AR may result in volume overload of the LV, pulmonary edema and shock with the need for immediate surgical intervention. Depending on the valve lesion, various replacement options are available, including bioprosthetic, mechanical, and composite valve/root replacement.

Pathology of Aortic Regurgitation

Chronic AR leads to increased LV volume and resultant eccentric hypertrophy meaning an increase in ventricular
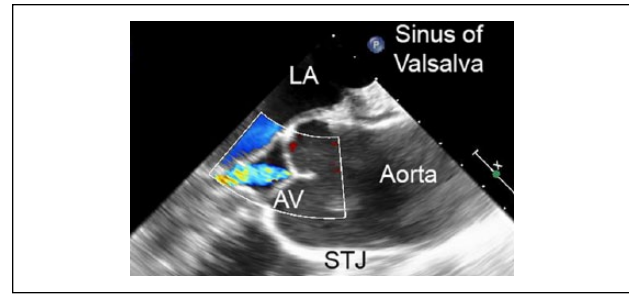

Figure 3. This midesophageal, aortic valve, long axis image (150°) shows the aortic valve with central insufficiency as the result of a dilated root. With dilation there is also effacement of the sinuses of Valsalva and resultant loss of a distinct sinotubular junction.
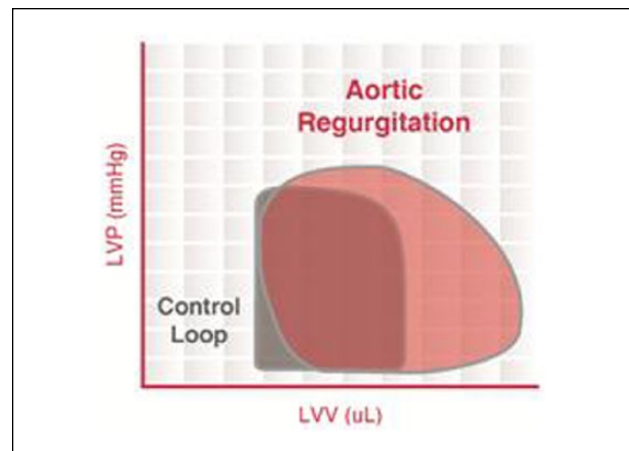Abbreviations: LA, left atrium; AV, aortic valve; STJ, sinotubular junction.

Figure 4. Aortic regurgitation: Both left ventricular (LV) end-systolic volume and end-diastolic volumes are increased and the entire curve is shifted to the right. The shift of the diastolic pressure–volume curve allows low diastolic pressure to be maintained at large end-diastolic volumes. The large increase in LV radius elevates wall stress.
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radius. Concomitantly, patients may develop concentric hypertrophy due to elevated afterload from years of increased stroke volume required to compensate for the regurgitant flow, especially if their blood pressure was not adequately managed. Figure 4 demonstrates a typical LV pressure–volume curve for AR. The constant backflow of blood through a leaky aortic valve does not allow for a true isovolemic relaxation or isovolemic contraction phase.

The increased end-diastolic volume activates the Frank–Starling mechanism to increase the force of contraction, LV systolic pressure and stroke volume. However, a heart cannot maintain this burden forever and will eventually succumb to heart failure. The goal is to correct the AR prior to or at the onset of LV dysfunction. If this is

done, LV size is likely to return to normal weeks to months postoperatively. Finally, many patients with AR develop functional mitral regurgitation due to LV dilation or alternatively, functional mitral stenosis due to impaired diastolic opening of the mitral valve.

Postoperative Considerations

The hemodynamic goals for patients with AR are summarized in Table 1. Faster heart rates compensate for regurgitant flow as time of regurgitation is minimized by less time spent in diastole. This heart rate response occurs as a result of increased catecholamine production. The renin–angiotensin–aldosterone system is also activated, and thus many patients with chronic AR are maintained preoperatively on angiotensin-converting enzyme inhibitors in an effort to slow the rate of LV dilation. Since the LV takes months to remodel after valve replacement, a high normal heart rate (90/min) is beneficial, and sometimes requires the use of external pacing. Lower systemic vascular resistance, adequate preload and maintenance of contractility are additional hemodynamic goals.

Conduction abnormalities are similar to those seen following AS surgery. Inotropy is not discouraged in these patients, as the increased heart rate and contractility may provide necessary support. If the aortic root was also replaced, coagulopathy, myocardial stunning and systemic inflammatory response are usually more pronounced. Patients who undergo circulatory arrest with a root replacement should have frequent neurologic assessments and possibly receive steroids for prolonged circulatory arrest.[17]

Mitral Stenosis

The most common cause of mitral stenosis (MS) remains rheumatic heart disease (RHD) caused by rheumatic fever. In fact, rheumatic involvement is found in 99% of stenotic mitral valves at the time of replacement.[18] RHD is caused by Group A β-hemolytic streptococci, usually after an episode of pharyngitis in a genetically susceptible person. RHD is characterized by inflammation, fibrosis and scarring of the valve and valve apparatus. This leads to abnormalities that can result in pure stenosis in approximately 25% of patients, while an additional 40% of patients have combined MS and mitral regurgitation.[19] Data indicate that 65% of all patients with rheumatic fever develop RHD.[20]

Other causes of MS are rare and include severe annular calcification, congenital heart disease (usually requires correction in infancy or early childhood), cleft mitral valve, radiation treatment to the chest, diseases of serotonin metabolism, systemic autoimmune disease (eg, systemic lupus erythematous) and some medications (methysergide).[21]
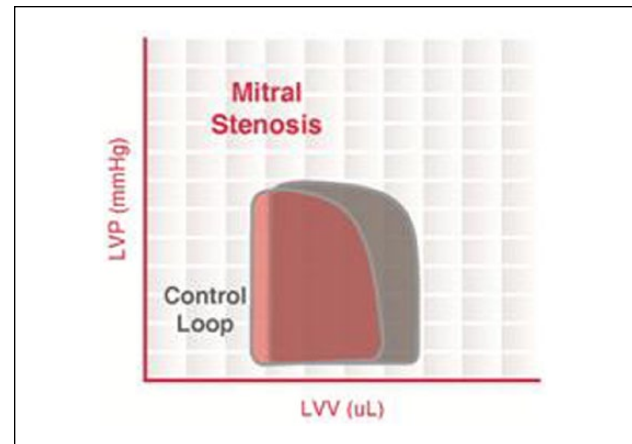

Figure 5. Mitral stenosis: Because of a fixed restriction of forward flow across the mitral valve, the left ventricular end-diastolic volume is greatly reduced. This leads to a decrease in cardiac output and endaortic end-diastolic pressure.
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Pathology of Mitral Stenosis

Mitral stenosis is a lesion of ventricular underloading. As the mitral valve orifice decreases, the left atrium must increase the pressure gradient in order to provide forward flow. If diastole is long enough, that is, slow heart rate, complete LV filling can be accomplished. As the valve area decreases, the elevated left atrial pressure will not be adequate for maintaining normal LV end-diastolic volume and LV underfilling will occur. The pressure–volume loop for MS is shown in Figure 5.

Postoperative Considerations

The more severe and long-standing the MS, the more likely the patient will have decreased ventricular function, AF, embolic events or pulmonary hypertension (PH). The presence and severity of each of these conditions guide postoperative therapies. Typically, patients with repaired MS benefit from maintaining the same hemodynamic parameters as prior to the repair (Table 1). Maintenance of sinus rhythm by avoiding tachycardia and other predisposing AF risk factors, such as increased sympathetic stimulation via excessive inotropy, anxiety, pain, and hypothermia, is key. Maintenance of intravascular volume while avoiding increases in left atrial pressure is essential; central venous and pulmonary artery pressure monitoring, dynamic pulsatility indices and/or echocardiographic imaging of the left ventricle can be helpful in guiding fluid therapy.[22]

Atrial Fibrillation

Approximately 60% of patients with MS older than 50 years have AF due to increased left atrial pressure and left

atrial enlargement.[23] Patients with chronic AF will actually tolerate the conduction disturbance better after the transvalvular gradient has been relieved by surgery. It is unlikely that electrical cardioversion will convert long standing AF to normal sinus rhythm; thus rate control should be the focus. Beta-blockers and nondihy-dropyridine calcium channel antagonists are appropriate first line therapies. Digoxin[24] and amiodarone can also be useful in this setting. A common practice is to initiate the rate control agent that the patient had been taking successfully preoperatively at a lower dose, depending on postoperative hemodynamics.

New onset, postoperative AF leads to clinical decompensation due to loss of atrial contraction resulting in loss of cardiac output. As discussed with aortic stenosis valve surgery, chemical and/or electrical cardioversion should be considered.

Anticoagulation with warfarin, enoxaparin or heparin is indicated in patients who have MS and any of the the following: paroxysmal, persistent or permanent AF; a prior history of an embolic event; or a known left atrial appendage (LAA) thrombus.[22] If warfarin is contraindicated, aspirin should be given postoperatively. Preoperative anticoagulation may lead to postoperative coagulopathy. Once the bleeding risk is low, systemic anticoagulation should be resumed with intravenous heparin, enoxaparin, or warfarin. Newer antithrombotic agents are not yet approved for anticoagulation for AF in the setting of valvular abnormalities.

Patients with known AF undergoing mitral valve surgery may receive a concomitant maze procedure. The maze procedure involves using radiofrequency to create a lines of scar tissue within the atrial muscle that in principle contains the conduction of aberrant electrical current. Patients who receive maze therapy should continue to be anticoagulated for 3 months[25] to several months,[26] as the atria may be stunned, and a nidus for thrombus formation. Furthermore, many patients have paroxysmal AF for several months. Additionally, excision of the LAA may be performed in patients with severe MS and a history of embolic events.[22] Excision is preferred over ligation as one study showed as many as 60% of patients will still have communication between the LAA and left atrium after ligation alone.[27] Currently, there are no practice guidelines concerning anticoagulation after such procedures,[24] but the objective in many centers is to eliminate the need for anticoagulation.

Pulmonary Hypertension

To ensure forward flow against a stenotic mitral valve, the left atrial pressure must increase, often to the point of pulmonary congestion. This creates a back pressure in the pulmonary vasculature and eventually pulmonary venous hypertension. Pure pulmonary venous hypertension due to MS is reversible following valve replacement. One study showed pulmonary artery systolic pressures decreasing from 55 mm Hg at baseline to 49 mm Hg 1 week postoperatively and then to 32 mm Hg at 3-year follow-up.[28]

However, long-standing pulmonary venous hypertension leads to pulmonary endothelial changes resulting in concomitant pulmonary arterial hypertension. Distinguishing between arterial and venous pulmonary hypertension is essential to selecting appropriate therapy. The two can be differentiated by the transpulmonary pressure gradient (mean pulmonary artery pressure less the pulmonary capillary occlusion pressure; normal <12 mm Hg) or the diastolic pulmonary pressure gradient (diastolic pulmonary artery pressure less the mean pulmonary capillary wedge pressure, normal <7 mm Hg). Both of these gradients will be elevated in pulmonary arterial hypertension and normal in pulmonary venous hypertension. Calculation of pulmonary vascular resistance is also extremely helpful.

Nonpharmacologic interventions that reduce pulmonary arterial vasoconstriction should be undertaken in most cardiac surgical patients, especially those with known PH. These include the avoidance of hypercarbia, hypoxia, overzealous positive pressure ventilation, and alpha-receptor stimulation (pain, hypothermia, and anxiety). With arterial hypertension pulmonary vasodilators may also be necessary. Inhaled pulmonary vasodilators have the advantage of less systemic vasodilatory effects and include inhaled nitric oxide[29] and prostacyclin analogs, such as iloprost, epoprostenol, and treprostinil. Prostacyclin analogs, can be given intravenously, but are often limited by systemic vasodilation.[30] Treprostinil has also been approved for subcutaneous administration. It is imperative that inhalational pulmonary vasodilators are not abruptly discontinued as rebound pulmonary hypertension may occur. Rebound is especially problematic with nitric oxide; use of another pulmonary vasodilating agent such as sildenafil, during the weaning process has been shown to decrease this rebound.[31] Pulmonary vasodilators should not be used in patients with lone pulmonary venous hypertension because these agents may, in fact, worsen the pulmonary venous congestion. By decreasing right ventricular afterload via pulmonary vasodilation, the pulmonary blood flow will increase and create more venous congestion.

Right ventricular failure may accompany pulmonary hypertension, and is recognized by an increasing central venous pressure and a large c-v-wave on the central venous pressure tracing indicating tricuspid regurgitation. Milrinone and dobutamine both increase chronotropy, lusitropy and pulmonary vasodilation by increasing cellular cyclic adenosine monophosphate. Milrinone accomplishes this through inhibition of phosphodiesterase-3 while dobutamine increased adenylyl cyclase activity via stimulation

of β1 and β2 receptors. Sildenafil, tadalafil, and vardenafil are oral agents that decrease pulmonary vascular resistance through inhibition of phosphodiesterase-5. Endothelin blockers are used sparingly in the postsurgical population because of the side effects of hepatotoxicity, teratogenicity, and peripheral edema.

Prosthetic–Patient Mismatch

If the mitral valve has been replaced, PH may be due to prosthetic–patient mismatch. In prosthetic–patient mismatch, the effective orifice area index of the prosthetic valve is small in relation to the patient's body size. This leads to transvalvular pressure gradients and may contribute to persistent PH. These patients typically have continued, unexplained post-operative hypotension, especially if attempts at diuresis are made. Prosthetic-patient mismatch should be evaluated echocardiographically, if suspected.

Anticoagulation

Patients with mechanical mitral valve replacement (MVR) should be systemically anticoagulated for life. Anticoagulation should begin as soon as safe to do so from a bleeding risk standpoint, usually 24 to 48 hours postoperatively, with intravenous heparin and/or warfarin. Patients receiving a bioprosthetic MVR and mitral valve repairs may receive only aspirin, unless they require anticoagulation for another purpose.[22] However, some centers may opt for systemic anticoagulation for 3 months following bioprosthetic MVR in order to lessen the thrombotic risk.

Mitral Regurgitation

Mitral regurgitation (MR) results from an abnormality in at least 1 of the 4 components of the mitral valve: (*a*) leaflets, (*b*) annulus, (*c*) chordae tendinae, and (*d*) papillary muscles/LV myocardium. Primary MR refers to abnormalities of the leaflets and is most commonly due to myxomatous degeneration. The most common cause of primary MR in the United States is mitral valve prolapse. In younger patients, MR is typically due to gross redundancy of the anterior and posterior leaflets, or Barlow's valve. Older patients likely have fibroelastic deficiency. Other causes include infectious endocarditis, rheumatic heart disease, cleft mitral valve, and radiation exposure. Mitral valve repair, with or without ring annuloplasty, is preferred over replacement in primary MR as long-term LV function, morbidity, and mortality have been shown to be improved.[32,33] However, certain conditions associated with MR such as extensive calcifications, prolapse of more than one third of the leaflet tissue, extensive chordal fusion, or papillary muscle rupture may require replacement.
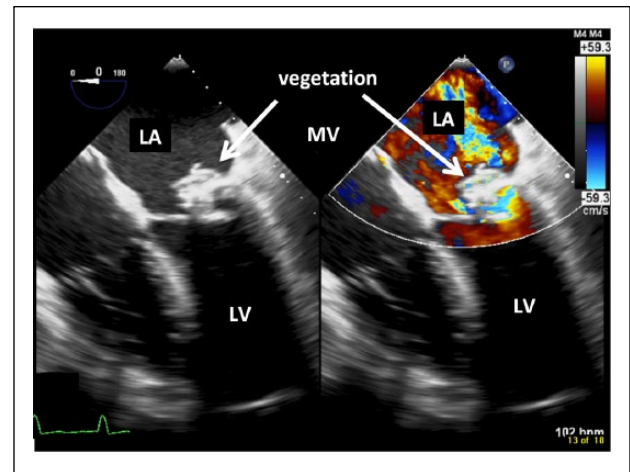

Figure 6. Midesophageal (0°), 4-chamber view showing a large vegetation on the atrial side of the posterior leaflet of the mitral valve due to infective endocarditis. The right image displays the color flow Doppler representing severe mitral regurgitation.
Abbreviations: LA, left atrium; LV, left ventricle; MV, mitral valve.
Image attributable to Dr. Nathaen Weitzel.

In secondary, or functional MR, the leaflets are usually normal and the regurgitation is due to adverse LV remodeling with papillary muscle displacement, leaflet tethering, and annular dilation.[34] Ischemic heart disease, LV systolic dysfunction, and hypertrophic cardiomyopathy are common causes of functional MR. Acute, life-threatening MR may occur suddenly due to leaflet perforation due to infectious endocarditis (Figure 6). Alternatively, chordal rupture due to acute myocardial infarction would necessitate urgent intervention.

Pathology of Mitral Regurgitation

Patients with MR typically have an eccentric ventricular hypertrophy similar to that seen in aortic regurgitation. As in aortic regurgitation, the increase in ventricular radius elevates wall stress and stimulates some degree of concentric hypertrophy.[4] Figure 7 demonstrates a typical pressure–volume loop in MR. Notably, an left ventricular ejection fraction (LVEF) merely compares end-diastolic volume to end-systolic volume and does not differentiate if the volume went forward or backward. Hence, a "supranormal" LVEF in MR of 70% is likely necessary to maintain a normal stroke volume.

Decompensated Mitral Regurgitation

Patients who are symptomatic and/or those with LV dysfunction, defined as an ejection fraction <60% or left ventricular end-systolic diameter ≥40 mm are considered to have decompensated MR. Patients with decompensated
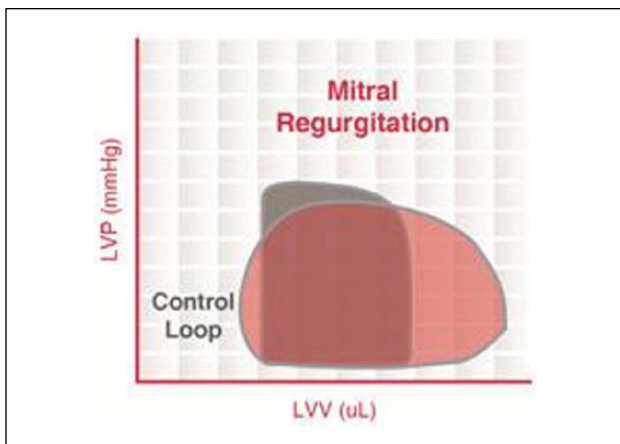
Figure 7. Mitral regurgitation (MR): The eccentric hypertrophy seen in MR leads to a right-shifted curve with a reduced isovolemic contraction phase—even shorter than that seen in aortic regurgitation. The low impedance outflow tract to the left atrium provided by an incompetent valve allows wall stress to remain low. Thus, despite possible systolic dysfunction, the ejection fraction is maintained near normal. Image attributable to Creative Commons Attribution 3.0 Unported.

MR present more challenges postoperatively than those with compensated MR, and may require aggressive diuresis, inotropic support, and in some cases, placement of an intra-aortic balloon pump. An intra-aortic balloon pump helps patients with decompensated MR in 2 ways: (*a*) balloon deflation decreases aortic afterload, hence eases cardiac ejection and increases output and (*b*) balloon inflation increases aortic end diastolic pressure. A lower LV end-diastolic volume and pressure from increased performance, combined with the higher diastolic pressure increases coronary perfusion.

Symptomatic patients with acute MR are the most tenuous and require urgent surgical correction. Acute MR causes a sudden increase in left atrial and pulmonary venous pressures, leading to pulmonary congestion, and hypoxia. Dyspnea and low cardiac output both lead to massive catecholamine release. The resulting systemic vasoconstriction perpetuates a vicious cycle of regurgitation, dyspnea and diminished forward flow, and creates a rapidly evolving form of shock.[22]

Postoperative Considerations

After mitral valve repair or replacement for chronic MR, the ventricle that has undergone compensation for a volume overload lesion is now is faced with a relative pressure overload.[4] Instead of having a pressure "pop-off" valve in the form of mitral regurgitation, the ventricle must eject against a competent valve. This increases the total myocardial oxygen consumption and could create a milieu

for new systolic dysfunction. Additionally, even with chordal sparing procedures, MVR reduces the hemodynamic efficiency of the LV contraction.[35] While these patients need adequate preload volume postoperatively, too much volume will increase the left ventricular end-diastolic volume to a point that may cause systolic failure. These patients benefit from afterload reduction and inotropic support. The goal of vasodilator use is to reduce the impedance to ejection through the aorta as much as possible.

In acute MR, although the ventricle is not remodeled, the postoperative management is much the same with mainstays of therapy being afterload reduction and inotropic support. Inotropic support may be especially necessary if the acute MR was due to ischemia.

Patients undergoing mitral valve surgery with secondary MR have LV remodeling, by definition and thus, should receive heart failure therapy such as beta-blockers, aldosterone antagonists, and angiotensin-converting enzyme inhibitors when safe to do so postoperatively. Patients with secondary MR are at increased risk of both perioperative and long-term mortality following mitral valve surgery.[36] Inotropes may be required to maintain cardiac output and can typically be weaned off in the perioperative period as the ventricle adapts to the new or repaired mitral valve. Perioperative fluid management requires recognition that a dilated LV may require more volume to maintain adequate filling pressures (Table 1).

Pulmonary Hypertension

Patients who presented to surgery with pulmonary systolic pressures approaching 50 mm Hg have increased postoperative risk. Despite the dramatic reduction in left atrial pressures following mitral valve correction, patients may continue to have elevated pulmonary pressures. This PH is due to not only pulmonary arterial endothelial changes but also from reactive pulmonary vasoconstriction. The same groups of pulmonary vasodilators used in PH and RV dysfunction associated with mitral stenosis are recommended for patients with corrected MR.

Systolic Anterior Motion of the Mitral Valve

Systolic anterior motion of the mitral valve occurs in 4% to 5% of patients after prosthetic ring mitral valve repair. The paradoxical anterior motion of the mitral valve during systole leads to LV outflow tract obstruction (Figure 8). This dynamic outflow obstruction is caused by narrowing of the left ventricular outflow tract commonly due to septal hypertrophy or an inappropriately sized or positioned mitral prosthesis.

Factors predisposing to SAM are the presence of myxomatous mitral valve with redundant leaflets, a nondilated,
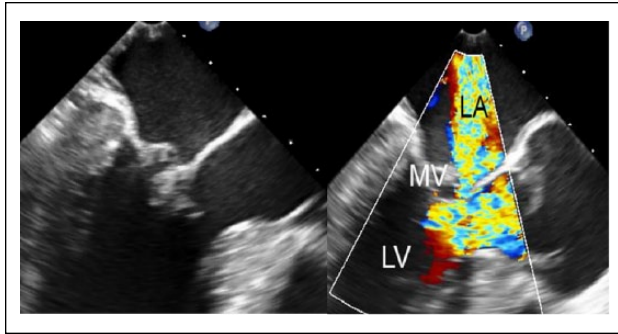
Figure 8. This side-by-side midesophageal, aortic valve, long axis demonstrates severe mitral regurgitation in the setting of SAM (systolic anterior motion). The left image shows the subvalvular apparatus and anterior leaflet being "pulled" into the left ventricular outflow tract. The right image shows the increased turbulent flow in the left ventricular outflow tract consistent with obstruction, and a central jet of mitral regurgitation due to noncoaptation.
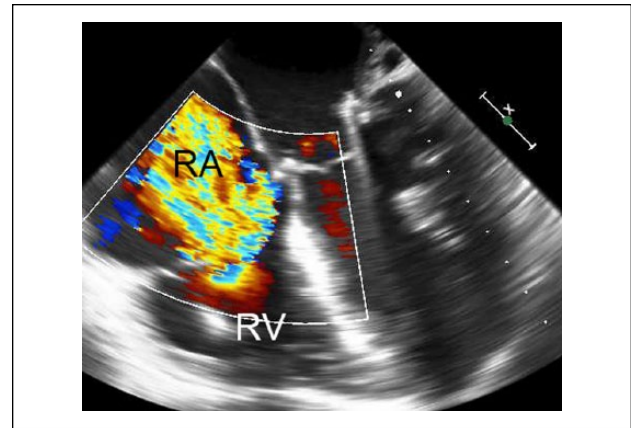Abbreviations: LA, left atrium; MV, mitral valve; LV, left ventricle.

Figure 9. This is a midesophageal, 4-chamber view (10°) with color flow Doppler illustrating severe, central, tricuspid regurgitation secondary to annular dilatation.
Abbreviations: RA, right atrium; RV, right ventricle.

hyperdynamic LV and a short distance between the MV coaptation point and ventricular septum after mitral repair.[37] Typically, SAM is noted on intraoperative echocardiography, and management should be optimized under visual guidance. SAM should also be suspected in patients who become hemodynamically unstable following initiation of inotropic support, hypovolemia, or vasodilation. If suspected, an echocardiogram should be obtained to differentiate SAM from other etiologies of heart failure. Avoidance of inotropic agents and tachycardia are essential in managing this obstructive lesion. Maintenance of higher filling volumes also aids in maintaining proper leaflet position during ejection.

Other Postoperative Concerns

Atrial fibrillation is common in patients with MR due to elevated left atrial pressures and left atrial enlargement. Patients with preoperative AF will rarely convert postoperatively, so rate control is the goal. The management strategies are the same as postoperative AF in mitral stenosis patients.

Similar to mitral stenosis surgery, issues associated with a prosthetic-patient mismatch following prosthetic valve replacement should be evaluated echocardiographically, if suspected.

Transcatheter Approaches to the Mitral Valve. Due to the success of transcatheter aortic techniques, it is likely that transcatheter approaches for MR will become more prominent. Currently, the only device approved for transcatheter repair of MR is the MitraClip (Abbott Vascular). It is approved for the reduction of significant (≥3+), symptomatic, degenerative MR in highly anatomically selected patients considered to be at prohibitive risk for MV

surgery.[34] The mainstay of postoperative care of these patients revolves not only around the ventricular changes due to chronic mitral regurgitation, but also the numerous comorbidities of each patient.

Tricuspid Regurgitation

Tricuspid regurgitation (TR) in trace to mild degrees is common and of no physiologic concern. There are numerous causes of more significant TR (Figure 9). Primary causes include: RHD, prolapse, congenital disease (Ebstein's), infective endocarditis, radiation, carcinoid, trauma, and intra-annular RV pacemaker or implantable cardioverter-defibrillator leads.[22] However, the majority (up to 80%) of significant TR cases have a secondary or functional etiology. As RV pressure overload and remodeling occurs, tricuspid annular dilation, and leaflet tethering will result in functional TR.[22] Patients with structural TR usually to not have pulmonary hypertension, however many cases of functional TR arise from PH.

Indications for Surgery

Repair of an incompetent tricuspid valve is preferred over replacement and is typically addressed at the time of mitral or aortic valve surgery. Tricuspid regurgitation may not improve and may worsen after left-sided heart surgery.[38] Tricuspid annuloplasty is indicated in patients with significant tricuspid annular dilation or PH, even if there is only mild to moderate TR.

Surgical correction of isolated primary TR carries inherent risks with a postoperative mortality rate of 20%,[39] most commonly due to heart failure.[40] Ideally, surgery is performed before the onset of RV dysfunction and congestive hepatopathy in order to improve outcomes.

Tricuspid valve replacement is the surgery of choice in carcinoid heart disease affecting the TV. Prosthetic valves are at risk for premature valve degeneration due to carcinoid disease, thus a somatostatin analog, such as octreotide, should be used postoperatively not only to control symptoms, but also to protect the valve. Mechanical valves are also not ideal, as these patients may need to have various tumor debulking surgeries in the future.

Postoperative Considerations

The main postoperative concern after tricuspid valve surgery is RV dysfunction. Dilation and failure of the RV leads to LV underfilling and decreased cardiac output. Maintenance of euvolemia with diuretics if necessary is beneficial to a hypokinetic RV (Table 1). Additionally, reduction of pulmonary vascular resistance with pulmonary vasodilators such as prostacyclin analogues, inhaled nitric oxide and sildenafil may be beneficial. Inodilators such as dobutamine and milrinone support RV function with increased contractility and pulmonary vasodilation. Lastly, damage to the atrioventricular node during surgery can result in complete heart block, so functioning pacing wires should be placed and checked often.

Tricuspid Stenosis

The most common etiology of TS is RHD. Correction of severe TS should occur at the time of other valve surgery if TS is due to RHD. Isolated, symptomatic TS is also a class I indication for surgery.[22] Because TR is often associated with TS, surgical correction may be superior to percutaneous balloon tricuspid commissurotomy, which may either create or worsen regurgitation. Perioperative considerations focus on right ventricular support as with TR surgery.

Pulmonic Valve Disease

Mild to moderate amounts of pulmonic regurgitation are not uncommon and are generally well tolerated. Significant PR may manifest after congenital heart disease surgical repairs. Another cause of significant PR is carcinoid disease, which should be managed similarly to carcinoid involvement of the TV. Secondary PR is often due to pulmonary hypertension, hence management of pulmonary hypertension is paramount. Pulmonic stenosis is most often a congenital cardiac disease.

Summary

Postoperative care of patients following valvular surgery requires an understanding the characteristic pressure and volume dynamics that characterize ventricular function in each abnormality. Hemodynamic compromise should be approached with the knowledge of factors complicating each type of valvular surgery, as well as the preferred hemodynamic profile for each type of lesion. Right ventricular failure and pulmonary hypertension accompany several valvular abnormalities, especially mitral and tricuspid diseases and may require inhaled medications and other nontraditional agents for stabilization. Conduction abnormalities and arrhythmias are common in both open and transcatheter valve replacements. When competing lesions exist, such as multiple valvular abnormalities or biventricular failure, management may require use of pulmonary artery catheterization or serial echocardiographic examination. Postoperative complications such as bleeding and tamponade should always be suspected in hemodynamically unstable postoperative patients.

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