A 78-year-old man was admitted with increasing shortness of breath. He had chest pain in the past but was able to continue with normal activities. He had passed out twice in the past year. On physical examination, a loud systolic murmur could be heard at the left sternal border radiating to the neck. His vital signs were: blood pressure 150/90 mm Hg, heart rate 88 beats per minute and irregular. The electrocardiogram (ECG) showed sinus rhythm with atrial premature contractions and left ventricular hypertrophy (LVH) with strain. A transthoracic echocardiogram showed a hypertrophied left ventricle (LV) and Doppler examination demonstrated severe aortic stenosis (AS) with a gradient of 64 mm Hg, mild aortic insufficiency (AI), and moderate mitral regurgitation (MR). He was scheduled for aortic valve replacement (AVR) and possible mitral valve (MV) repair or mitral valve replacement (MVR).

What are the major etiologies of AS, AI, MS, and MR?

AS occurs as a congenital lesion but more commonly as an acquired disease. Stenosis may develop on a previously normal valve following rheumatic fever (RF) or from progressive calcification. Congenitally bicuspid valves are also prone to calcification with eventual stenosis. Calcification of the leaflets can result in incomplete closure of the valve with associated insufficiency. AI is usually an acquired disease. The most common causes include bacterial endocarditis and rheumatic heart disease. Annular dilation may result from diseases such as cystic medial necrosis and collagen disorders or following aortic dissections with resultant insufficiency. When occurring as a congenital lesion, aortic insufficiency (AI) rarely occurs in the absence of other cardiac abnormalities.

Mitral stenosis (MS) is almost always caused by rheumatic fever (RF), although only half of patients will have a history of an acute febrile illness. The inflammatory process of RF results in thickening of the leaflets and fusion of the commissures. Other rare causes include congenital stenosis and other systemic diseases including systemic lupus erythematosus and carcinoid. Pathophysiology similar to that seen with valvular MS can occur with obstructing left atrial (LA) tumors and cor triatriatum. MS commonly occurs in conjunction with other
valvular heart disease; only 25% of patients present with isolated MS; approximately 40% have combined MS and mitral regurgitation (MR). MR can result from defects in the leaflets, the annular ring or the supporting chordae, the papillary muscles, or any combination of these. Primary leaflet dysfunction occurs with RF but can also follow bacterial endocarditis, connective tissue disorders, and congenital malformations. Annular dilation can follow ventricular dysfunction and left ventricular dilation. MV prolapse and/or rupture of papillary muscles results in incomplete leaflet closure or coaptation with resultant MR. Left ventricular ischemia can affect papillary muscle contraction and is the cause of postischemic or postinfarction MR.


**What are the three TEE vantage points for the comprehensive imaging of the LV? How are pressure gradients measured by echocardiography? How do the pressure gradients derived from Doppler echocardiography differ from those obtained in the catheterization laboratory by direct pressure measurement?**

The heart has two main axes, the longitudinal axis running from the base to the apex and the short axis perpendicular to that. Because the ultrasound beam can be thought of as a two-dimensional structure, multiple scan planes are required to completely image a three-dimensional structure. The left ventricle (LV) may be divided into 16 segments, 6 at the basal level, 6 at the midpapillary level, and 4 at the apical level. By moving the transesophageal echo cardiography (TEE) probe in the esophagus and by rotation of the crystal within the transducer tip, the LV can be imaged from three acoustic windows. These are the midesophageal four-chamber, the transgastric short axis, and the transgastric two-chamber view. Normal ventricular motion requires that the wall segment move centrally with systole and
similarly undergo thickening along this axis during contraction. Function is usually quantified as normal; mild, moderate, and severe hypokinesis; akinesis; and dyskinesis.

In addition to function, TEE permits the determination of wall thickness and chamber size, important parameters in understanding the pathophysiology of valvular heart disease. Pressure gradients are derived through analysis of the Doppler profiles of blood flow. A commonly determined echocardiography gradient is that which is present from the LV to the aorta (Ao) in the setting of aortic stenosis (AS). To obtain the change in blood flow velocity across the aortic valve with TEE, the probe is advanced far into the stomach and sharply anteflexed and left deflected to obtain the window ITom the apex of the heart (deep transgastric long axis) and align the ultrasound beam most parallel to the path of blood flow. From this window, the continuous wave Doppler cursor is directed across the left ventricular outflow tract and aortic valve. The large increase in blood flow velocity in this display occurs at the narrowest point along its path, which in this case is the aortic valve. Using the modified Bernoulli equation a gradient is calculated (100 mmHg in the example). This represents the maximum instantaneous pressure difference between the LV and the Ao. AS is also quantified at the time of catheterization by measuring the pressures from within the LV and the Ao as a rapid response pressure transducer catheter is withdrawn from the LV back to the Ao across the stenotic valve. The standard reported gradient is the difference between the maximum left ventricular and Ao pressures. Doppler-derived AS gradients are usually higher than those derived at the time of left heart catheterization.


What are the echocardiographic and cardiac catheterization criteria for the four valvular lesions?

Aortic stenosis

Echocardiographic criteria for AS include tw8-dimensional images demonstrating limited aortic valve opening and motion and left ventricular concentric hypertrophy. Doppler examination will reveal a turbulent, high-velocity jet across the aortic valve and color flow Doppler will demonstrate a turbulent, mosaic-appearing color map. The gradient across the aortic valve measured at cardiac catheterization is different from that measured by echo cardiography. Quantification of this Doppler-derived pressure gradient again relies on the modified Bernoulli equation. Because flow is an important determinant of pressure gradients, both these catheterization and Doppler-derived values are interpreted along with cardiac output. Calculations permit the determination of a valve area. Severe AS is present when the gradient exceeds 75 mmHg and/or the valve area is less than 0.8 cm².

Aortic insufficiency

Catheterization criteria for aortic insufficiency (AI) rely on the qualitative estimation of the regurgitation volume and an estimation of left ventricular size and EF. Similar quantification can be made from Doppler color echocardiography derived data. A commonly used echocardiographic criteria compares the width of the regurgitant jet at the level of the valve to the width of the left ventricular outflow tract. A ratio of greater than 0.66 corresponds with severe AI.

Mitral stenosis

The severity of mitral stenosis (MS) can be obtained by the direct measurement of a diastolic gradient between the left atrium and ventricle at the time of cardiac catheterization. However, this requires a trans atrial puncture, a procedure largely replaced by echocardiographic techniques. Echocardiographic diagnosis is based on gradient estimation by Doppler and by measuring the rate of decay in
the pressure with the time spent in diastole (pressure half-time). The MV area in cm² can be derived from an empirical formula wherein the MV area equals 220 divided by this pressure half-time (Hade constant). Severe MS is present when the end-diastolic gradient exceeds 12 mmHg corresponding to a valve area of less than 1.0 cm².

**Mitral regurgitation**

In the presence of mitral regurgitation (MR), ventriculography will demonstrate the reflux of dye from the LV into the left atrium. Severe MR is diagnosed when dye refluxes into the pulmonary veins. Color Doppler echocardiography permits similar quantification. Estimation relies on an estimation of regurgitant jet volume as compared with the left atrium and through analysis of pulmonary venous flow profiles. In every case, color Doppler echocardiography is often useful in identifying the cause of the valvular lesion, its extent of involvement within and around the valve, and the associated hemodynamic changes. Therefore, for many valvular lesions, it may be sufficient for the diagnosis. Catheterization, however, is often performed to assess the presence of concomitant coronary artery disease, especially in patients of advanced age.


**What are the presenting signs and symptoms of the four valvular lesions listed previously?**
Aortic stenosis

The triad of angina, syncope, and congestive heart failure represent the progression of symptoms associated with AS. These symptoms correlate directly with mortality; the 50% survival data for these symptoms are 5, 3, and 2 years from the onset of these symptoms, respectively. Angina results from both increased demand for and a decrease in supply of coronary blood flow. Increased muscle mass from left ventricular hypertrophy and the high energy requirements to generate increased (high) systolic pressure combine to increase demands for coronary blood flow. In addition, insufficient supply secondary to decreased perfusion gradients and a decrease in coronary vasculature relative to the large amount of myocardium sum to diminish relative myocardial blood supply. Therefore, up to one third of patients with aortic stenosis can have angina in the absence of significant coronary artery disease.

Aortic insufficiency

Patients with AI have variable clinical presentations, primarily depending on the rapidity with which the left ventricular volume overload develops. When the volume increase occurs gradually as in chronic AI, there is usually a long asymptomatic period. The onset of the symptoms of fatigability and dyspnea signals either reduced cardiac output or increased left ventricular end-diastolic pressure (LVEDP) indicative of impairment of left ventricular contractile function. When aortic insufficiency occurs acutely, the ventricular compliance is unchanged; increased left ventricular diastolic volumes from regurgitant flow, therefore, lead to rapid rises in LVEDP and the clinical picture of congestive failure.

Mitral stenosis

Mitral stenosis (MS) is a slowly progressive obstruction to flow across the MV with gradual increase in left atrial (LA) pressure and volume. Symptoms of pulmonary congestion result from elevations in LA pressures and not from poor left ventricular systolic function. Atrial fibrillation develops secondary to atrial dilation.

Mitral regurgitation
The time course for the development of mitral regurgitation determines the severity of the symptoms. When the volume of regurgitant flow from the left ventricle to the left atrium increases gradually, the left atrium compensates by gradual dilatation. In contrast, the onset of acute mitral regurgitation can lead to rapid increases in LA pressures and severe pulmonary congestion and congestive heart failure.


**What is the New York Heart Association (NYHA) classification of heart failure?**

Class I. No symptoms
Class II. Symptoms with ordinary activity
Class III. Symptoms with less than ordinary activity Class IV. Symptoms at rest


**Discuss the role of premedication for patients with the four different valvular lesions.**

The role of premedication is to allay the anxiety of the impending surgical procedure thereby controlling the sympathetic outflow that may accompany the stress response. However, acute changes in heart rate, venous return, and systemic resistance can have particularly profound effects on patients with valvular heart disease. Patients with aortic stenosis (AS) may benefit from premedication by preventing unnecessary increases in heart rate. Concern however must be taken
to ensure adequate venous return and preservation of sinus mechanism (see later).

Patients with aortic insufficiency (AI) can similarly benefit from premedication because any increases in afterload, which may accompany sympathetic stimulation, can increase regurgitant volume. Drug doses should be adjusted based on the severity of debilitation and degree of systemic hypoperfusion.

Patients with mitral stenosis (MS) should be premedicated with caution. Elevations in carbon dioxide resulting from narcotic-induced hypoventilation can dramatically elevate pulmonary pressures further compromising right ventricle output. Conversely, venodilation may excessively diminish filling pressures.

Patients with mitral regurgitation (MR) can respond similarly to those with MS, particularly when pulmonary hypertension is present. However, elevations in systemic pressure from stress can also compromise forward left ventricular output. Proper premedication can be delivered by careful dose selection and the provision of supplemental oxygen.


Outline the hemodynamic management goals for each of the four valvular lesions. What are the anesthetic goals with respect to heart rate and rhythm, preload, afterload, and contractility?

**Aortic stenosis**

Patients with aortic stenosis (AS) need the left ventricular filling obtained through a well-timed atrial contraction. Similarly, left ventricular hypertrophy renders the ventricle stiff and adequate preload is required. Reducing vascular tone will do little to relieve the fixed afterload increases from a stenotic valve but rather lower diastolic coronary perfusion gradients and should be avoided. Patients with AS experiencing angina may require the administration of an ~-
agonist such as phenylephrine rather than nitroglycerin to increase coronary perfusion pressure.

**Aortic insufficiency**

The severity of aortic insufficiency (AI) is determined by the size of the regurgitant orifice, the pressure gradient between the aorta and left ventricle during diastole, and the time spent in that phase of the cardiac cycle. Elevated heart rates decrease the time spent in diastole and can lead to a decrease in heart size. Afterload reduction can lessen the regurgitant driving forces but therapeutic maneuvers to accomplish this may be limited by resulting systemic hypotension.

**Mitral stenosis**

Patients with mitral stenosis (MS) can swiftly deteriorate in the setting of rapid heart rates. The decreased filling time necessitates the marked elevation of left atrial (LA) pressures and pulmonary edema can rapidly ensue. Whereas left ventricle (LV) contractility is generally preserve in mitral stenosis, use of β-blockade does result in decreased right ventricular (RV) contractility, which in the setting of pulmonary hypertension can further compromise the cardiac output and systemic blood pressure. However, the loss in RV contractility is more than offset by the beneficial effects of the reduction of heart rate. Slower heart rates permit adequate time for transfer of blood from the left atrium to the LV across the stenotic mitral valve (MV) to occur. In addition, the pressure gradient across the MV is also reduced; thereby lowering left atrial pressure and diminishing pulmonary congestion. Because there is some variability in the individual response, the use of short-acting β-blockers such as esmolol is prudent because an adverse response should be evanescent.

**Mitral regurgitation**

Patients with mitral regurgitation (MR) can rapidly deteriorate with marked increases in systemic blood pressure and afterload. As with other volume overload lesions such as aortic insufficiency (AI), slightly rapid heart rates (80 to 90 beats/minute) should result in smaller left ventricular volumes. This may lessen any component of MR secondary to annular dilation or chordal
malalignment. Importantly, tachycardia should be avoided in patients with ischemic MR.

**What are the hemodynamic goals for this patient with the combination of severe AS and MR?**

In the patient with combined aortic stenosis (AS) and MR, the situation is more complex than when only a singular valvular lesion is present. Careful examination of the hemodynamic goals for each of the two lesions will reveal that therapy beneficial to patients with AS may exacerbate the severity of the MR. Early aggressive intervention is the key to these combined lesions. There usually exists less of a margin for error because minor hemodynamic aberrations can rapidly lead to cardiac collapse. A good rule of thumb is to prioritize the management based on the character of the present symptoms. Patients with AS and MR who present with syncope or angina are best managed for their AS, whereas patients with dyspnea and pulmonary edema are best managed for their congestive symptoms. It is prudent to maintain the patient's own usual hemodynamics and avoid physiologic trespass. Transesophageal echo cardiography (TEE) evaluation of left ventricular performance can be helpful in separating pulmonary congestion secondary to left heart failure from that secondary to poor diastolic left ventricular compliance.


**Should the patient have a pulmonary artery (PA) catheter placed before induction?**

Volume status may be particularly difficult to assess in patients with valvular heart disease yet of critical importance in the management of these patients. Patients with stenotic lesions depend on adequate filling pressures for diastolic filling of the ventricle. Patients with the volume overload lesions of aortic insufficiency (AI) and mitral regurgitation (MR) can benefit from the careful reductions in pulmonary pressure guided by the simultaneous assessment of cardiac
performance. In these capacities, the PA catheter is useful. Patients with current hemodynamic stability, without severe respiratory distress, can be safely anesthetized before placement of the PA catheter. In all, there is no evidence that use of the PA catheter improves outcome in the setting of surgery.


What are the usual TEE findings in a patient with AS/AI/MR? How do you grade the severity of AS by TEE? How do you quantify the severity of MR? What is the impact of AS on the severity of MR?

The severity of aortic stenosis is usually stated in terms of aortic valve area (AVA). Normal AVA is 2.5 to 3.5 cm². Moderate stenosis is when the AVA is within the range of 0.8 to 1.2 cm² and severe stenosis when the AVA is less than 0.8 cm². A patient with a large peak pressure gradient (usually more than 75 mmHg) in the absence of excessively high cardiac output is usually considered to have severe AS as well. In the setting of low cardiac outputs, the pressure gradient may not be that great (20 to 30 mmHg), and determination of AVA is required. This can be accomplished with echocardiography.

Mitral regurgitation (MR) is graded by the amount of blood regurgitated backward into the left atrium during systole. Doppler color flow permits quantification of this flow. Common methods for MR quantification include the depth of MR jet extent into the left atrium (25% mild MR, 25% to 75% moderate MR, and more than 75% severe MR). Other methods of quantification include calculation of the area of the regurgitant jet. planimetry, by comparison of the MR jet area to the area of the left atrium, and by analysis of pulmonary vein flow profiles. It is important to remember that the amount of regurgitant blood flow in the setting of MR is determined by the amount of time spent in systole, the size of the defect in the mitral valve (MV), and the pressure gradient across the defect. Therefore, MR severity by Doppler color flow is load dependent. The lower pressure of the anesthetic state can often mask more severe degrees of MR seen when the patient is under his or her usual hemodynamic conditions. The left ventricular pressures are increased in the setting of aortic stenosis.
(AS). Therefore, the gradient across the MV is increased often leading to more severe MR. Following replacement of the stenotic aortic valve and elimination of the outflow tract obstruction, left ventricular pressures are markedly reduced. Moderate levels of MR without major structural defects in the MV apparatus usually revert to minimal or certainly less severe levels following reduction in the left ventricular outflow obstruction.


**How would you diagnose right heart failure and pulmonary hypertension? How would you treat it?**

Right heart failure is diagnosed by the elevations in right-sided filling pressures, specifically central venous pressure (CVP). Careful examination is required to rule out tricuspid insufficiency as the cause of the CVP elevation. A high CVP indicates the inability of the right heart to adequately propel the venous return volume into the pulmonary circulation. Elevation in the PA pressures is indicative of pulmonary hypertension. The combination of high CVP and high PA pressures indicates severe right heart failure. This scenario can be difficult to manage. Attempts to elevate systemic perfusion pressure with α-agonists can worsen pulmonary hypertension. Administration of vasodilators to lower pulmonary pressures results in systemic hypotension. In this setting, it is often prudent to return to cardiopulmonary bypass, relieve ventricular distention, and improve myocardial perfusion. During this "rest period," adjustments in inotropic therapy, ventilation, and cardiac rhythm can be instituted. Optimization of acid-base status and hemoglobin concentration should also be performed. Separation from bypass can then be reattempted. Typical inotropic agents effective in this setting are those with high degrees of β-adrenergic potency. Commonly employed agents include dobutamine, epinephrine, and/or the phosphodiesterase-III (PDE-III) inhibitors amrinone and milrinone. It is not uncommon to require the administration of α-agonists to counteract the systemic vasodilating effects of prostaglandin El and the PDE-III agents. Some selective
pulmonary vasodilating action and systemic vasoconstricting effects can often be achieved by administration of pulmonary vasodilating agents such as prostaglandin El through the right-sided access (CVP or PA catheter) and infusion of the a-agonists through the LA line. Therefore, the vasoconstriction of the pulmonary arterial bed can be minimized. Nitric oxide (NO) is a potent, inhaled pulmonary vasodilator. Its half-life in the systemic circulation is extremely short permitting its administration to the pulmonary vasculature with minimal systemic hypotensive effects. NO can selectively and effectively dilate the pulmonary vasculature. The exact method of delivery, scavenging of waste gases, and high cost remain as obstacles to its clinical application.


**How does the IABP work to benefit the failing heart?**

An IABP is a catheter with a large balloon (40 to 60 mL) at its tip. It is positioned in the thoracic Ao distal to the left subclavian artery origin and proximal to the take-off of the renal vessels. It is timed to inflate during diastole to increase diastolic perfusion pressure to the coronary arteries, great vessels, and major abdominal organs and to deflate just before systole to decrease afterload thereby increasing forward cardiac output. It is the unique modality, which can improve coronary perfusion pressures while reducing myocardial oxygen demand.


**What are the contraindications to the use of an IABP?**

The most common contraindications are severe AI and severe aortic disease, atheromatous, aneurysmal, or a dissection. Although often listed as absolute contraindications, there are reports of the effective use of IABP in these settings.


**In the intensive care unit (ICU) 4 hours later, the patient became hypotensive with a low cardiac output. How could you distinguish between cardiac tamponade and pump failure? How would the transesophageal echocardiography (TEE) images differ?**

The differentiation between cardiac tamponade and primary pump failure in the immediate postbypass-ICU setting can be difficult. Elevations in filling pressures, systemic hypotension, and low cardiac output are consistent with both diagnoses. The classic teaching of equalization of cardiac pressures seen in a "fluid" tamponade may not be present as areas of focal compression from clot can markedly reduce filling of only qpe chamber. Echocardiography can be beneficial in this setting by permitting visualization of chamber volume and function. The transesophageal approach has particular advantage over TTE in the postoperative setting in which the usual transthoracic window may be obscured by dressings and drainage tubing. Focal compression of the cardiac chambers from a clot or pericardial effusion can readily be distinguished from a volume-overloaded, failing heart with poor myocardial contractility. When the diagnosis is not clear, however, surgical reexploration may be indicated.
