

Valvular Heart Diseases

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Patients who have valvular heart disease coming for surgery present many challenges to the anesthesiologist. Over the past three decades there has been a persistent improvement in our understanding of the pathophysiology of valvular heart disease and in the surgical techniques for correcting it. With the development of efficient and safe noninvasive monitoring of cardiac function, new surgical techniques, better designs of prosthetic valves, and with the development of useful guidelines for choosing the proper timing of surgical intervention, patients who have valvular disease with varying physiology can be encountered in the perioperative period. The perioperative physician has to be aware of the varying effects of hemodynamic variables on this subpopulation of patients. In general, the goal in stenotic lesions is to enhance forward flow, whereas in regurgitant lesions the goal is to decrease regurgitant flow. The timing for surgery has also undergone significant changes. Now it is no longer necessary or even advisable to delay surgery until advanced symptoms are present; thus surgery is timed earlier today than it was even a decade ago. On the other hand, many, but not all patients who have advanced disease, once considered inoperable, are now often helped substantially by valve surgery [1]. According to the American College of Cardiology/American Heart Association (ACC/AHA) guidelines, severe valvular disease is considered a major clinical predictor of cardiac risk, and should lead to consideration of delay or cancellation of elective noncardiac surgery and consideration of echocardiography, cardiac catheterization, or possible valve surgery [2]. The five variables in dealing with valvular heart diseases are: (1) preload, (2) afterload, (3) myocardial contractility, (4) heart rate, and (5) rhythm. The goals of anesthesia for each valvular disease are described below in terms of these five variables. The monitoring for patients who have any of the valvular

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abnormalities depends on the severity of the lesion, the associated comorbidities, and the type of the surgery. For patients who have mild disease and minor surgery, noninvasive monitors may be all that is needed, whereas in patients who have severe disease and surgery with huge fluid shifts, invasive monitors, including arterial line, pulmonary artery (PA) catheter or intraoperative transesophageal echocardiography (TEE), may be needed.

Aortic stenosis

General

Aortic stenosis (AS) is the most common cardiac valvular lesion affecting US adults, and its incidence is increasing, in part because AS is a disease of aging and the US population is getting older. The most common cause of AS is degeneration and calcification in the elderly population, whereas stenosis of a bicuspid aortic valve is more common in the younger population. The normal aortic valve area is 2.6 to 3.5 cm². Although AS was considered to be a “degenerative” disease, it is now seen that the early lesion is an active inflammatory process with some similarities to atherosclerosis [3]. It has also been seen that asymptomatic patients who have moderate to severe AS have a good prognosis during their “latent” period, and are at low risk for cardiac or sudden death. Data indicate that asymptomatic patients who have AS can be followed medically as long as they remain free of symptoms [4]. Survival is nearly normal until the symptoms of angina, syncope, or heart failure develop, after which survival abruptly declines; in fact, about 75% of symptomatic patients will succumb within 3 years after the onset of symptoms without valve replacement. In general, symptoms can be attributed to AS if valve area is less than 1.0 cm² or if mean transvalvular gradient exceeds 50 mm Hg [1]. On an average, the rate of progression of AS further decreases aortic valve area by 0.1 cm²/year, or increases the transvalvular pressure gradient by 8 to 10 mmHg/year. The onsets of angina, syncope, and dyspnea have been shown to correlate with average times to death of 5, 3, and 2 years, respectively [5]. Patients who have AS have been considered to have a higher than average risk of perioperative complications ever since Goldman and colleagues [6] in 1977 reported major cardiac complications in 13% of 23 patients who had significant AS. More recent studies have reinforced this concept, and patients who have AS are seen to have an increased risk of perioperative mortality and nonfatal myocardial infarction independent of other risk factors (14% versus 2% in the general population) [7].

Preoperative evaluation

The preoperative assessment of the patient who has aortic stenosis includes a careful history focusing on the symptoms of angina, syncope,

and dyspnea. A detailed history and physical examination is key in identifying the high-risk patient when confronted with a patient who has AS. In patients who have known bicuspid aortic valves, one should keep in mind the association with coarctation of the aorta. It is not feasible or practical to request an echocardiographic examination on every patient who has AS. With aortic stenosis, angina results from increased pressure demands and limited coronary blood flow reserve. Syncope is caused by ventricular dysrhythmias or baroreceptor dysfunction with a relatively fixed cardiac output. Heart failure results from diastolic dysfunction more frequently than systolic dysfunction. Sudden death occurs predominantly in patients who have symptomatic AS.

The physical findings in patients who have AS are

1. A slow rising pulse, “parvus et tardus,” is usually associated with a notch on the upstroke (anacrotic pulse) or a systolic shudder or thrill.
2. Sustained cardiac apical impulse and a double impulse is sometimes felt because the fourth heart sound or atrial “kick” maybe palpable. A systolic thrill may be felt in the aortic area.
3. A soft or inaudible second heart sound when the aortic valve becomes immobile
4. Systolic ejection click, unless the valve has become immobile and calcified
5. Reversed splitting of the second heart sound (splitting on expiration)
6. Prominent fourth heart sound, unless coexisting mitral stenosis prevents this
7. Ejection systolic murmur that is usually “diamond-shaped” (crescendo-decrescendo). The murmur is usually longer when the disease is more severe, because a longer ejection time is needed. The murmur is usually rough in quality and is best heard over the aortic area. It radiates to the carotid arteries and also the precordium. The intensity of the murmur is not a good guide to the severity of the condition, because it is lessened by reduced cardiac output, and in severe cases the murmur may be inaudible.

In symptomatic patients, patients who have poor functional capacity, or patients who have conflicting data regarding the severity of the stenosis, it is helpful to get an echocardiographic evaluation of the heart, focusing on the morphology and gradients across the valve.

The concomitant presence of coronary artery disease with AS should be kept in mind, and a careful history and physical exam should be conducted to detect any evidence of this association [8,9]. In addition, relevant carotid artery stenosis should be included in the preoperative risk evaluation. There are also no clear data regarding the timing of valve repair/replacement before noncardiac surgery. Conventional thinking is that the indication is the same as in the absence of noncardiac surgery [10]. Controversial categories are the asymptomatic patient who has severe AS and the patient

who has low ejection fraction and low gradient. Echocardiography and exercise testing may identify the asymptomatic patient who might benefit from valve surgery. Exercise testing in a monitored setting is safe for patients who have moderate to severe stenosis, and may identify patients who have reduced reserve [11]. Patients who have low ejection fraction and low gradients have a high risk, and only half of such patients are alive 3 to 4 years after surgery. Echocardiography can also help to distinguish patients who have true stenosis from those who have pseudostenosis, in which the valve does not open because of the poor cardiac output. In these patients, if the cardiac output is increased pharmacologically, the valve area increases substantially to greater than 1 cm². In patients who have true stenosis, the increased output increases the gradient. Patients who have low gradient and no response to inotropic stimulation have a poor outcome because of advanced disease process [11].

Echocardiographic measures of the severity of aortic stenosis

Echocardiography is a noninvasive and cost-effective method of assessing the presence and severity of AS. It has replaced cardiac catheterization for the assessment of AS in many centers. It helps to assess the morphology of the aortic valve (bicuspid versus tricuspid), presence of left ventricular hypertrophy (LVH), systolic or diastolic dysfunction, and need for antibiotic prophylaxis.

Aortic blood flow velocity proportionately increases with the severity of aortic stenosis, so that patients can be followed up for the severity of their disease by periodic echocardiographic examinations. More recent studies indicate that risk factors for morbidity with AS are aortic jet flow velocity greater than 4.5 m/second, and left ventricular ejection fraction less than 50% [11a]. Also, aortic regurgitation (AR) may coexist in 50% to 70% of patients with AS [12]. Grading of the severity is done using several measures, including estimating valve area by planimetry and continuity equations, and measuring the pressure gradients across the valve (Table 1).

Table 1
Measures to determine severity of aortic stenosis

Measurement	Normal	Mild	Moderate	Severe
Valve area (cm ²)	2.6–3.5	1.0–1.5	0.8–1.0	<0.8
Peak gradient (mm Hg)		<36	> 50	> 80
Mean gradient (mm Hg)		<20	20–50	> 50
Peak velocity (m/s)	1.0–1.7			> 4

Data from Cohen IS. Aortic stenosis. In: Perrino AC, Reeves S, editors. A practical approach to transesophageal echocardiography. Philadelphia: Lippincott Williams and Wilkins; 2003. p. 189.

Anesthetic implications

Aortic valve replacement is the preferred treatment for patients who have severe AS and who require elective noncardiac surgery. It has been shown, however, that when this is not feasible, it is possible for patients who have moderate to severe AS to undergo elective noncardiac surgery with acceptable risk with aggressive intra- and postoperative monitoring, prompt recognition and treatment of hypotension, and careful control of the five hemodynamic variables mentioned in Table 2 [13]. Attention should be paid to the maintenance of normal sinus rhythm, because the contribution of the atrial contraction to left ventricular filling becomes important in these patients who have a high, left-ventricular, end-diastolic pressure (LVEDP) (Table 3). Any unstable arrhythmia should be promptly treated with cardioversion. Maintenance of the heart rate close to baseline is also desirable, because an adequate diastolic time is necessary for adequate left ventricular filling, and any tachycardia compromises this time. On the other hand, bradycardia can lead to an acute decrease in cardiac output in these patients who have a fixed stroke volume, and may cause an acute distension of the left ventricle, with precipitous deterioration in the patient's condition. Similarly, the systemic vascular resistance should be maintained close to normal, because any decrease will be associated with decrease in the coronary perfusion pressure, whereas an increase causes a fall in the stroke volume. Both are associated with decrease in cardiac output. The increased left ventricular, end-diastolic volume (LVEDV) and LVEDP lead to increased myocardial work load and oxygen demand. At the same time, myocardial oxygen supply is decreased by the increased LVEDP, causing a decrease in coronary perfusion pressure (the coronary perfusion pressure is equal to aortic diastolic pressure minus LVEDP). Finally, a Venturi effect of the jet of blood flowing through the aortic valve and past the coronary arteries is created, and this may lower the pressure in the coronary ostia enough to reverse systolic coronary blood flow. In the event of CPR, external cardiac massage in

Table 2
Classification of severity of aortic regurgitation

Method of evaluation	Trivial	Mild	Moderate	Severe
AR jet height/LVOT diameter (%)	1–24	25–46	47–64	>65
Vena contracta height				>6 mm
Aortic diastolic flow reversal				Holodiastolic reversal in descending aorta
Slope of AR jet decay			>2 m/s	>3 m/s
Pressure half-time (ms)		>500	200–500	<200

Abbreviations: AR, aortic regurgitation; LVOT, left ventricular outflow tract.

Data from Cohen IS. Aortic stenosis. In: Perrino AC, Reeves S, editors. A practical approach to transesophageal echocardiography. Philadelphia: Lippincott Williams and Wilkins; 2003. p. 180.

Table 3
Goals for anesthetic management in common valvular diseases

Disease	Preload	Afterload	Rate	Rhythm	Contractility
Aortic stenosis	Normal to high	Normal to high	Maintain baseline (70–80)	Sinus	Normal
Aortic regurgitation	Normal	Decrease	90–100	Sinus	Normal to high
Mitral stenosis	Normal	Normal	65–80	Usually atrial fibrillation	Normal to high
Mitral regurgitation	Normal	Decrease	90–100	Sinus	Normal to high

unlikely to be effective because of difficulty in establishing flow across a stenotic valve. Arrhythmias should therefore be treated early and aggressively. In patients who have significant AS, it may be prudent to have a defibrillator on hand before induction of anesthesia.

Preoperative medication

Medication is administered judiciously, keeping in mind the hemodynamic goals (see Table 2). Prophylactic antibiotics are administered according to the guidelines published by the AHA [14].

Intraoperative management

General anesthesia is usually selected over a pure regional technique, although with careful control of the systemic vascular resistance, both spinal and epidural anesthesia can be performed with good results [7]. If succinylcholine is chosen as relaxant, the bradycardia that is sometimes associated with its administration must be kept in mind, and a plan to deal with it should be in place. A balanced anesthetic technique with nitrous oxide, volatile agent, and opioid seems to work well with minimal cardiac depression. Prompt treatment of hypovolemia and blood loss is essential to maintain the perfusion pressure and oxygen-carrying capacity of the blood. Any unstable arrhythmias should be promptly terminated by electrical cardioversion. Ventricular ectopy also should be treated aggressively, because patients whose rhythm deteriorates into ventricular fibrillation often cannot be successfully resuscitated.

The use of invasive monitoring should be guided by the severity of the stenosis and the type of surgery.

Aortic regurgitation

General

AR may be caused by primary disease of the aortic valve leaflets or the wall of the aortic root, or both [15]. Common causes of leaflet abnormalities

are infective endocarditis and rheumatic fever. Diseases involving the aortic root are annuloaortic ectasia, Marfan's syndrome, syphilis, ankylosing spondylitis, rheumatoid arthritis, and systemic hypertension. Acute AR is usually caused by infective endocarditis. The basic derangement is a decrease in forward stroke volume through the aortic valve, caused by regurgitation of part of the ejected stroke volume back into the left ventricle during diastole. A long diastolic time is associated with greater time for regurgitation and consequent decrease in effective cardiac output. Similarly, an increase in resistance to forward flow because of an increase in systemic vascular resistance also decreases effective cardiac output.

With a low operative mortality rate for patients undergoing isolated elective aortic valve replacement in the current era (<2.5%), and with an acceptable incidence of late valve-related death (5.2% at 10 years), aortic valve replacement earlier in the course of chronic AR may be indicated to prevent irreversible myocardial damage [16].

Preoperative evaluation

Patients who have AR can have symptoms of exertional dyspnea, exercise intolerance, and congestive heart failure. Angina pectoris is a frequent complaint, arrhythmias are relatively uncommon. Physical examination would reveal bounding or collapsing peripheral pulse (Water-hammer or Corri-gan's pulse), decreased diastolic pressure (secondary to aortic runoff during diastole), wide pulse pressure, and a blowing diastolic murmur best heard at the left sternal border with the patient leaning forward and the breath held in expiration. Also the Austin Flint murmur from fluttering of the anterior mitral valve in mid-diastole may be heard. Other signs that indicate a hyperdynamic circulation may also be present:

Quincke's sign (capillary pulsation in the nail beds)

De Musset's sign (head nodding with each heart beat)

Duroziez's sign (a to-and-fro murmur heard when the femoral artery is auscultated with pressure applied distally; if found, it is a sign of severe AR)

Left ventricular enlargement may be seen on the chest radiograph or EKG. In contrast to AS, sudden death from AR is rare [17]. Medical therapy of AR is based on decreasing any associated systolic hypertension in order to reduce wall stress and improve LV function. Vasodilators such as hydralazine and nifedipine have been shown to have a beneficial effect, and may delay the need for surgery in asymptomatic patients who have normal LV function or in patients who have severe AR who are not candidates for surgery [18].

Echocardiographic measures of the severity of aortic regurgitation

Echocardiography is helpful in studying the morphology of the aortic valves, to identify leaflet perforation, cusp fibrosis, and leaflet prolapse,

and to identify the pattern of aortic root dilation. Quantifying the degree of regurgitation is also possible to evaluate the severity of AR. Surgery should be considered in patients who have left ventricular, end-systolic diameter more than 55 mm or ejection fraction of less than 50% [19]. The dimensions of the aortic root are usually taken at the annulus, the sinus of Valsalva, the sino-tubular junction, and the ascending aorta. There is no single method that is accepted as a gold standard for the quantification of aortic regurgitation. The aortic regurgitant jet is studied in different views by echocardiography and an attempt is made to quantify the size of the regurgitant jet using a combination of techniques, including calculating the ratio of the diameter of the AR jet to aortic annulus, pressure half-time, and deceleration slope, calculating proximal iso-velocity surface area (PISA), the presence of diastolic mitral regurgitation, reversed doming of anterior mitral valve leaflet, regurgitant volume, and holodiastolic aortic flow reversal (see Table 2).

Anesthetic implications

The goal of anesthesia in patients who have AR is to maintain adequate forward flow across the aortic valve and decrease the fraction of the stroke volume that flows back into the left ventricle. This is achieved by avoiding bradycardia and any sudden increases in systemic vascular resistance (see Table 3). It is advantageous in these patients to keep the heart rate higher than normal, because bradycardia increases the diastolic time during which regurgitation occurs and causes left ventricular volume overload. Similarly, a decrease in the systemic vascular resistance can also promote forward flow across the aortic valve. Although the induction of peripheral vasodilation from a regional anesthetic seems to be an attractive option in these patients, the uncontrolled nature of this response may detract from its appeal in this patient population. Any drug-induced depression of myocardial contractility is avoided.

Preoperative medication

Premedication includes antibiotic prophylaxis when appropriate [14] and anxiety relief if needed.

Intraoperative management

Although any of the intravenous agents can be used safely, ketamine may be preferred in patients who are intravascularly volume-depleted, because the tachycardia that it induces could be of advantage. Measures to avoid bradycardia with succinylcholine administration (eg, glycopyrrolate premedication) may have to be considered.

A balanced maintenance technique is preferred, with nitrous oxide, volatile anesthetic, and opioids. The vagolytic effects of pancuronium can be used to advantage in the choice of muscle relaxants in these patients. Close

attention should be paid to maintain blood volume, and blood loss should be replaced promptly to maintain adequate cardiac output and oxygen-carrying capacity of the blood. Any rhythm disturbances should also be treated promptly before a downward spiral results from inadequate cardiac output. In the presence of severe AR, monitoring with TEE or PA catheter may be needed to identify any myocardial depression or hypovolemia early and institute treatment. Use of intra-aortic balloon pump is contraindicated in the presence of AR, because the augmentation of diastolic pressure increases the amount of regurgitant volume.

Mitral valve prolapse

General

Mitral valve prolapse (MVP) is an inherited connective tissue disorder with myxomatous proliferation resulting in thickening and redundancy of the mitral valve [12]. It is one of the most prevalent cardiac valvular abnormalities, affecting 5% to 10% of the population [15]. It is more commonly seen in young women than in men or older women, and it has a familial incidence. Mitral valve prolapse may be associated with Marfan's syndrome, rheumatic endocarditis, myocarditis, thyrotoxicosis, and systemic lupus erythematosus. Mild mitral valve prolapse is so common that some authorities regard it as a normal variant.

There has been a lot of discussion regarding the distinction between "billowing mitral valve," "prolapse of mitral valve," "floppy mitral valve," and "flail mitral valve." Barlow [20] attempted to define these terms in 1985. He called the exaggeration of the normal movement of the mitral valve leaflets into the left atrial cavity during systole "billowing." This, then by definition, is not associated with regurgitation. Once this progresses and there is disruption of leaflet edge convergence, MVP is the result, and this is associated with mitral regurgitation (MR). If the mitral valve leaflets are enlarged with elongated chordae, the term "floppy mitral valve" is used. MVP is always present with a floppy valve. When this progresses to rupture or at least grossly elongated chordae, then part of the leaflet will be flail and the term "flail mitral valve" is used [20]. Most of these terms are used interchangeably in the literature, however, and the definition has to be verified in each case.

Preoperative evaluation

The majority of patients are asymptomatic. Preoperative evaluation should focus on distinguishing patients who have purely functional disease from those who have structurally abnormal valve with associated severe MR. Patients may suffer from anxiety and other nonspecific symptoms, such as fatigability, palpitations, postural orthostasis, neuropsychiatric

symptoms, and autonomic dysfunction. Any complaints of chest pain must be probed to rule out any associated coronary artery disease. The chest pain of MVP often has been described as atypical for angina pectoris, because it is left precordial, sharp, cyclic, unrelated to exertion, and unrelieved by nitroglycerin. Proposed etiologies for this chest pain are: excessive stretch on chordae tendinae causing focal areas of decreased subendocardial blood flow and coronary vasospasm, microembolism to the coronary circulation, decreased diastolic perfusion with tachycardia and increased inotropy, and esophageal motility disorders [21]. These patients may be on beta blockers to control palpitations, and these should be continued throughout the perioperative period. An increased incidence of embolic events in younger patients who have MVP has been described [22]. There also has been reported an increase in incidence of migraine in patients who have MVP [23]. On physical examination, auscultation of the chest may reveal a midsystolic click with or without a late systolic murmur. If there is associated severe MR, this may be accompanied by pansystolic murmur and other signs of failure such as S₃ gallop, rales, and peripheral edema. The electrocardiogram usually shows nonspecific ST/T wave changes in about 15% to 30% of cases, but there is no evidence that this more than in the general population.

Echocardiographic signs of mitral valve prolapse

When using transthoracic echocardiography, the diagnosis of MVP should be made only with parasternal views, because it is generally accepted that the apical views would result in "over-calling" MVP. This phenomenon is caused by the peculiar shape of the mitral valve annulus, which is described as being "saddle-shaped." The annulus is farthest from the LV apex in its anterior and posterior portions, which is seen in the parasternal views. In the apical view the plane cuts across the annulus at the medial and lateral portions, and may give the impression of the leaflets being excessively displaced into the left atrial chamber, consistent with the diagnosis of MVP [24,25]. Generally accepted criteria include a posterior systolic motion of at least 2 mm in late systole or at least 3 mm for holosystolic prolapse [26]. Color Doppler can be used to quantify any MR that is present.

Anesthetic implications

The choice of anesthetic agents depends on the particular physiologic abnormality present in the patient. MVP has been associated with increased incidence of embolism, especially in patients younger than 45 years of age, and measures to prevent this may be warranted in this patient population [27]. The degree of MR plays an important role in the choice of anesthetics.

Premedication

MVP is reported to be the most common cause of infective endocarditis in this country, responsible for 11% to 29% of all cases [28]. It has been recommended that patients who have prolapse of mitral valves with no documented regurgitation do not need antibiotic prophylaxis, whereas patients who have prolapse of mitral valves with associated mitral regurgitation receive prophylactic antibiotics [14]. Men older than 45 years who have MVP may deserve prophylaxis, even in the absence of resting regurgitation, because they have been shown to have increased risk for developing endocarditis [29–31].

Intraoperative management

Most patients who have MVP tolerate regional anesthesia well, provided they are adequately prehydrated and do not have significant myocardial dysfunction. For patients who have hemodynamically significant MR, the myocardial depressant effect of volatile anesthetics at higher concentration has to be kept in mind.

Factors that determine regurgitant flow in MR are: the systolic pressure gradient between the two chambers, the size of the regurgitant orifice, and the duration of ventricular systole. Therefore, maintaining good filling pressures, a higher heart rate, and moderate vasodilation are desirable. The choice of monitors again depends on the severity of the physiologic derangement and the surgery. An arterial line and PA catheter can be used in patients who have significant MR and myocardial dysfunction. The V wave on the wedge tracing can be used to estimate the extent of regurgitation into the LA and the pulmonary veins, especially in patients who have acute MR [21]. In patients in whom the data are inconclusive, TEE has been seen to be useful to assess the degree of MR and to calculate the hemodynamic variables [32,33].

Mitral stenosis

General

Although rheumatic heart disease was the most common cause of mitral stenosis (MS) historically, the incidence has decreased recently [34]. Approximately 25% of all patients who have rheumatic heart disease have pure MS. Rheumatic MS is more common in women. An additional 40% have combined MS and MR [15]. The rheumatic process affects all layers of the heart, the endocardium, myocardium, and pericardium; however, the endocardium is the most affected, leading to inflammation and scarring of the cardiac valves. The mitral valve is involved in almost all cases with stenosis occurring from leaflet thickening, commissural fusion, and chordal

shortening. The other common causes for MS are congenital, rheumatoid arthritis, lupus, and carcinoid syndrome. In the elderly, a syndrome similar to MS develops because of calcification and fibrosis of the valve, valve ring, and subvalvular apparatus. The normal mitral valve area is 4 to 6 cm². The basic derangement in MS is that there is a restriction in free flow of blood from the left atrium to the left ventricle, and a pressure gradient develops between the two chambers. The resulting increase in the left atrial pressure causes left atrial enlargement and pulmonary congestion. The right ventricle is the primary chamber that generates the force necessary to drive blood across the stenotic mitral valve, and therefore there is right ventricular pressure overload. In severe MS there is usually associated pulmonary vasoconstriction, which has an additive effect on the pulmonary hypertension leading to right heart failure [34].

Preoperative evaluation

Most patients who have mild disease may be entirely asymptomatic, and these patients start to become symptomatic only after the valve is moderately stenosed (ie, has an area of 2 cm²). Progressively patients may exhibit dyspnea on exertion, orthopnea, and paroxysmal nocturnal dyspnea with advancing disease. Some patients may have hemoptysis caused by pulmonary venous hypertension. Pressure symptoms from the enlarged left atrium may manifest as dysphagia or hoarseness (Ortner's syndrome). Atrial fibrillation is a common finding in this population of patients. Physical examination may reveal reduced pulse pressure indicative of reduced stroke volume. The typical mitral facies, or malar flush, is plethoric upper cheeks with bluish patches that are caused by arteriovenous anastomoses and vascular stasis. Jugular venous distension is seen if there is right heart failure.

Physical examination reveals a parasternal "heave" if pulmonary hypertension has developed. A diastolic thrill may be palpated in the left lateral decubitus position. Auscultation reveals a loud first heart sound if the mitral valve is pliable, but it will not occur in calcific mitral stenoses. After the second heart sound, the mitral valve opens with a snap. The time between these two events is a good indicator of the severity of the MS. An S₂-opening snap interval of less than 0.08 seconds usually indicates severe disease [34]. A low-pitched, rumbling mid-diastolic murmur follows the opening snap, and if the patient is in normal sinus rhythm, the murmur becomes louder at the end of diastole as a result of atrial contraction (presystolic accentuation). Sometimes a high-pitched murmur (Graham-Steell) may be present at the cardiac base, which could be caused by pulmonary regurgitation, or more often by aortic regurgitation [35]. As the valve cusps become immobile, the loud S₁ softens and the opening snap disappears. When pulmonary hypertension occurs, the S₂ sound is increased in intensity and the mitral diastolic murmur may become quieter because of the reduction in the cardiac output.

Echocardiographic assessment of the severity of mitral stenosis

Echocardiography is used to delineate the anatomy of the valve, usually described as being “hockey-stick” shaped. The valve area can be determined by planimetry. Doppler interrogation of the valve can establish the pressure gradient. Dividing 220 by the pressure half-time gives the mitral valve area, with higher-pressure half times being associated with more severe MS. In a small percentage of patients, cardiac catheterization may be needed to determine the valve area and cardiac output when conflicting data are obtained by noninvasive measurements.

Anesthetic implications

Because most of these patients present with atrial fibrillation, the use of oral anticoagulants with them must be kept in mind when considering regional anesthesia. These patients have a fixed mitral valve orifice, and so need adequate preload to maintain cardiac output. Any tachycardia can be detrimental because it shortens the diastolic filling time. On the other hand, fluid overload is also poorly tolerated by these patients, precipitating pulmonary edema. These patients are usually on digoxin which is continued perioperatively.

Premedication

Sedatives should be used cautiously in these patients. Any respiratory depression leading to hypercarbia can increase pulmonary vascular resistance and worsen the severity of right heart failure. Antibiotic prophylaxis is given as per guidelines [14]. Avoidance of anticholinergics should be considered to minimize tachycardia.

Intraoperative management

Careful management of fluids is necessary in these patients, because most of them are on chronic diuretic therapy and need volume expansion preoperatively and intraoperatively. Overloading can push the patient into pulmonary edema, however, and thus careful monitoring, if necessary with a pulmonary arterial catheter, is needed in patients who have severe MS because they have very little, if any, flow reserve [36]. The pulmonary artery catheters should be inserted further than usual because of the dilated pulmonary arteries; because of the risk of pulmonary artery rupture, final placement of the catheter in a wedge position is not necessary. Many of these patients have pulmonary hypertension, and therefore careful insertion and estimation of wedge pressures are imperative with this group. Avoiding factors that increase pulmonary vascular resistance, such as hypoxia, hypercapnia, and acidosis, is also important. Avoiding tachycardia is important to maintain adequate cardiac output, using short-acting

beta blockers if necessary. Patients who have severe MS also tolerate decreases in systemic vascular resistance poorly, because the only way to maintain a cardiac output is by increasing heart rate. Support with sympathomimetic agents may be necessary to maintain the stroke volume. Under conditions of light anesthesia, patients can have an increase in systemic and pulmonary vascular resistance, and this can decrease the stroke volume. Nitroprusside infusion has been used to decrease systemic vascular resistance and increase left ventricular stroke volume, especially when patients also have pulmonary hypertension [37]. An attempt should be made to maintain these patients in normal sinus rhythm at all times, with immediate use of cardioversion if new atrial fibrillation should occur.

Mitral regurgitation

General

Normal mitral valve function depends on perfect function of the complex interaction between the mitral leaflets, the subvalvular apparatus, and the mitral annulus. An imperfection in any one of these components can cause the valve to leak. The major causes of MR include MVP (20%–70% of cases), rheumatic heart disease, infective endocarditis, annular calcification, cardiomyopathy, and ischemic heart disease [15,38]. Rare causes include collagen vascular diseases (eg, Marfan's syndrome and Ehlers-Danlos syndrome), trauma, the hypereosinophilic syndrome, carcinoid, and exposure to certain appetite suppressant drugs (eg, fenfluramine). Acute MR is usually caused by acute myocardial infarction or rupture of chordae tendinae secondary to infective endocarditis.

Preoperative evaluation

Most patients who have mild to moderate MR are asymptomatic and are diagnosed on the basis of the presence of a systolic murmur on auscultation of the precordium. Patients who have significant MR may present with symptoms of heart failure, atrial fibrillation, or endocarditis. Symptoms can also be precipitated in previously asymptomatic patients by superimposed stress such as pregnancy, anemia, or infection [38]. On physical examination, an apical holosystolic murmur that radiates to the axilla can be typically heard. The intensity of the murmur seems to correlate well with the degree of regurgitation in patients who have organic MR, whereas in ischemic or functional MR the correlation is weak [39]. Prominent S₃ is present (sometimes a short mid-diastolic flow murmur may follow the S₃ sound) owing to the sudden rush of blood back into the dilated left ventricle in early diastole. The onset of atrial fibrillation has a much less dramatic effect on symptoms than in MS.

Echocardiographic assessment of the severity of mitral regurgitation

Echocardiographic assessment of the mitral valve is indicated in patients who have symptoms suggestive of a valve abnormality, with physical examination revealing a systolic murmur (> grade 3/6) or other cardiac findings. Echo allows accurate evaluation of the presence or absence or severity of MR, and to an extent can establish the cause. Although there are many methods of estimating the severity of MR by Doppler echocardiography, none of them has been shown to predict outcome. Therefore many practitioners employ a combination of methods, using color flow, continuous, and pulsed-wave Doppler imaging techniques to grade MR. Left atrial pressures can be indirectly estimated by pulsed-wave Doppler examination of the pulmonary veins. In patients who have atrial fibrillation, the left atrial appendage is examined for evidence of thrombus.

Anesthetic implications

Management of anesthesia in these patients is centered around decreasing the amount of regurgitant volume and facilitating forward stroke volume. The heart rate is maintained at normal or slightly higher rates. Sudden bradycardia is not well-tolerated, because it leads to acute left ventricular volume overload. Mild vasodilation is beneficial to increase forward stroke volume. Patients who have chronic MR usually have associated myocardial dysfunction; therefore even minimal drug-induced myocardial depression may be undesirable. Factors that increase systemic vascular resistance should be avoided.

Premedication

Prophylactic antibiotics are instituted before surgical procedures per guidelines [14].

Intraoperative management

Regional anesthesia can be considered in patients who have MR, keeping in mind that although decrease in systemic vascular resistance is desirable in these patients, the uncontrolled nature of this response can compromise cardiac function. General anesthesia is the usual choice for patients who have significant MR. Succinylcholine-induced bradycardia may be undesirable in these patients, and a plan to deal with this if it occurs must be in place before induction. Among the nondepolarizing muscle relaxants, pancuronium seems to be an attractive choice because it produces a modest increase in heart rate. Maintenance can be done with a combination of nitrous oxide, opioids, and volatile agent. Maintenance of intravascular fluid volume with prompt replacement of blood loss is important to maintaining the preload and cardiac output. Vasodilators have been used to decrease afterload

and increase forward stroke volume and decrease regurgitant flow; however, vasodilators have been seen to be most effective in patients who have ventricular dilation and associated systolic dysfunction [40]. The use of invasive monitoring again depends on the severity of MR and the proposed surgery. A pulmonary artery catheter can be used to measure cardiac outputs and follow trends in the wedge pressure as an indicator of left atrial pressure. Transesophageal echocardiography can be considered for intraoperative monitoring of mitral valve function and assessment of myocardial function and regurgitant volume, provided the anesthesiologist is familiar with the technique. In patients who have acute MR, it has been postulated that maintaining a small left ventricle with sustained myocardial contractility will reduce the amount of MR for a given lesion, whereas dilation of the ventricles increases the effective regurgitant orifice independent of the pressure gradient [41].

Prosthetic valves

General

An increasing number of patients who have prosthetic heart valves are presenting for noncardiac surgery. More than 60,000 valve replacements are performed annually in the United States. Prosthetic heart valves can be mechanical or bioprosthetic. Mechanical valves are made of metal or carbon alloy, and are named according to their structure as caged ball, tilting disc, or bileaflet tilting disc valves. Bioprosthetic valves are usually heterografts and are made of bovine or porcine tissue and mounted on a metal support; however they are highly thrombogenic, and patients need to be placed on long-term anticoagulant therapy. On the other hand, the bioprosthetic valves have low thrombogenic potential, and thus do not need long term anticoagulation; however they have a short life span of close to 10 years. They are consequently used in elderly patients and those who cannot take long-term anticoagulants for other reasons. The presence of prosthetic valves in the patient prompts us to look for associated complications, the most common of which are thromboembolism and anticoagulant related bleeding. Hemolysis and endocarditis are also frequently seen in patients who have prosthetic valves.

Preoperative evaluation

Every attempt must be made to obtain information about the type of prosthetic valve the patient has and its current function. On auscultation of the precordium, the character of the heart sounds should be ascertained. Mechanical valves produce crisp, high-pitched opening and closing sounds, whereas bioprosthetic valves produce sounds that are similar to native valve.

A change in the intensity or quality of previously audible sounds, the appearance of a new murmur, or change in the characteristics of an existing murmur usually indicates valve dysfunction. Although not commonly used, cinefluoroscopy can be used to assess the structural integrity of mechanical valves [42]. Echocardiography can usually give information regarding sewing ring stability and leaflet motions of bioprosthetic valves. Mechanical valves need good windows for examination because of echo reverberations on passing through metal. MRI can be safely done in patients who have prosthetic valves, except those who have Pre-6000 Starr-Edwards caged-ball prosthesis [43]; however it is not as useful as echo in assessing prosthetic valve structure, and should be reserved for those cases in which prosthetic valve regurgitation or paravalvular leakage is suspected but not visualized by echocardiography.

Anesthetic implications

An important consideration in these patients is the discontinuation of anticoagulants perioperatively for the surgery. Elective surgery should be avoided in the first month after an arterial embolism. On an average, the rate of major thromboembolism in patients who have mechanical heart valves and who are not given anticoagulant therapy is estimated at 3% to 5%. Anticoagulation is thought to reduce this risk by 75% [44]. The temporary discontinuation of anticoagulants in patients who have mechanical heart valves exposes them to the risk of thromboembolism. This is compounded by the prothrombotic effect of surgery, although this is predominantly venous and not arterial. This has to be balanced against the risk of bleeding in an anticoagulated surgical patient. Postoperative therapy with intravenous heparin has been recommended for patients undergoing minor surgery in whom the risk of bleeding is low [45]; however, for patients who have had major surgery, intravenous heparin is not recommended because of the high risk of bleeding. Subcutaneous low-dose heparin or low molecular-weight heparin is recommended for hospitalized patients who have low risk of arterial embolism [45].

Premedication

Antibiotic prophylaxis is recommended in these patients and should be administered according to guidelines [14].

Intraoperative management

The choice of drugs depends on the valve involved and the function of the valve at the time of surgery. The general principles outlined earlier should hold good in guiding therapy. The possibility of excessive bleeding in these patients from residual anticoagulation must be kept in mind, and

replacement of losses should be performed promptly. There may be associated clotting factor deficiencies that may have to be corrected.

Diseases of the tricuspid and pulmonary valves occurring in isolation are not uncommon. A brief overview of diseases affecting these valves is presented below.

Tricuspid regurgitation

Functional tricuspid regurgitation (TR) may occur whenever the right ventricle dilates, as in cor pulmonale, pulmonary hypertension, and myocardial infarction. Organic TR may occur with rheumatic heart disease, infective endocarditis, carcinoid syndrome, blunt chest trauma, Ebstein's anomaly, and other congenital abnormalities of the atrioventricular valves, and is secondary to disease of the valvular apparatus (leaflets, annulus, chordae, and papillary muscle). The most common cause of TR is probably functional valvular regurgitation secondary to annular dilatation, which in turn is the result of pulmonary hypertension of any cause. A final cause of TR is tricuspid valve prolapse, seen in myxomatous valve syndrome; in most cases it is seen in conjunction with mitral valve prolapse. Tricuspid regurgitation causes volume overload of the right atrium and right ventricle, and will cause right heart dilatation. Isolated tricuspid regurgitation is well-tolerated because the right ventricle can compensate for volume overload, and actually small physiologic degrees of TR are often encountered in the normal disease-free individual.

Preoperative evaluation

The regurgitation gives rise to high right-atrial and systemic venous pressure. Patients may complain of the symptoms of right heart failure. On examination, these patients often demonstrate a large jugular venous CV wave and a palpable liver that tends to pulsate in systole. Usually a right ventricular impulse may be felt in the left sternal border, and there is a blowing pansystolic murmur that is best heard on inspiration at the lower sternal edge. Atrial fibrillation is not uncommon.

Anesthetic implications

Antibiotic prophylaxis is recommended in these cases too. The goals of hemodynamic management in these cases are to provide adequate forward flow achieved by augmenting the right ventricular preload, to keep the heart rate normal to high in order to prevent peripheral congestion, to maintain contractility of the myocardium, and to decrease the pulmonary vascular resistance.

Tricuspid stenosis

General

An uncommon valvular lesion, seen more commonly in women, this is usually secondary to rheumatic heart disease and is frequently associated with mitral or aortic valve disease. Tricuspid stenosis (TS) is also seen in carcinoid syndrome (these patients usually will have predominantly tricuspid regurgitation with some element of tricuspid stenosis). TS is easily missed when evaluating a patient who has rheumatic heart disease, and a markedly dilated right atrium should serve as a clue to possible TS. Other causes of TS include exceptionally rare cases of congenital stenosis, as a result of foreign body such as a transvenous pacemaker wire across the tricuspid annulus causing fibrosis of the wire and the valve, and right atrial tumors (primary cardiac tumors or metastatic lesions) causing obstruction of the tricuspid flow. TS is commonly associated with some degree of TR.

TS causes a reduction in cardiac output that usually does not occur until the valve orifice decreases to less than 1.5 cm^2 (with resultant mean gradient across the diseased valve of 3 mm Hg), down from the normal of 7 to 9 cm^2 , and this is restored toward normal when the right atrial pressure increases. The resulting systemic venous congestion produces hepatomegaly, hepatic dysfunction, ascites, and dependent edema and jugular venous distension, with giant A waves visible on central venous pressure recording.

Anesthetic implications

Antibiotic prophylaxis is recommended. The goals of hemodynamic management are to maintain right ventricular preload and to maintain the patient in sinus rhythm, because supraventricular dysrhythmias are poorly tolerated and should be treated with cardioversion. The contractility should be maintained, because adequate forward cardiac output depends on it. Keeping both systemic and pulmonic vascular resistances within normal limits is desirable.

Pulmonary valve

Pulmonary regurgitation

Pulmonary regurgitation (PR) is the most common acquired lesion of the pulmonary valve. Dilatation of the pulmonary annulus can be idiopathic or can be caused by pulmonary artery dilatation, which in turn can be secondary to pulmonary hypertension, Marfan's syndrome, or other connective tissue disorders. Other causes include infective endocarditis, carcinoid syndrome, trauma, surgical interventions (such as correction of tetralogy of Fallot), rheumatic heart disease, and congenital malformations. Mild degrees of PR are commonly encountered in the normal disease-free

individual, and do not necessarily imply anatomic diseases of the valve or elevated pulmonary artery pressures. PR is characterized by a decrescendo diastolic murmur, beginning with the pulmonary component of the second heart sound, that is difficult to distinguish from the murmur of aortic regurgitation. Pulmonary regurgitation usually causes no symptoms and treatment is rarely necessary.

Pulmonary stenosis

Pulmonary stenosis (PS) is usually a congenital lesion, but it may rarely result from rheumatic heart disease or carcinoid syndrome. Congenital PS maybe associated with an intact ventricular septum or with a ventricular septal defect (Fallot's tetralogy). The stenosis can be valvular or infundibular, subvalvular, or supra-valvular. Multiple congenital pulmonary arterial stenosis is usually caused by infection with rubella during pregnancy. The obstruction to the right ventricle results in right ventricular hypertrophy, which in turn leads to right atrial hypertrophy. Severe PS may be incompatible with life, but lesser degrees of obstruction may present with fatigue, angina, syncope, and the symptoms of right heart failure. Mild degrees of PS may go unnoticed. On examination, a harsh midsystolic ejection murmur is best heard on inspiration in the second intercostal space to the left of the sternum; this is usually associated with a thrill. The pulmonary closure sound is usually delayed and soft. A fourth heart sound and a prominent A wave are present when the stenosis is moderately severe. A right ventricular heave may be felt. Atrial fibrillation from resultant TR is common.

Perioperative management

All patients who have pulmonary stenosis require endocarditis prophylaxis. Patients who have mild degrees of obstruction should otherwise have no anesthetic problems. Patients who have moderate to severe degrees of stenosis require preload augmentation. Increase in heart rate usually provides increased flow across the valve. Myocardial contractility should be maintained. Afterload should be maintained to provide adequate coronary perfusion to the hypertrophied right ventricle. The pulmonary vascular resistance should be kept in the low-to-normal range, because major increases in the former can lead to right ventricular dysfunction.

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