Anesthesia for the Patient With Valvular Heart Disease

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Maintaining cardiovascular stability with adequate hemodynamic parameters and systemic perfusion pressure during the anesthetic management of patients with valvular heart disease can be extremely challenging. With the high incidence and severity of complications in this subgroup of patients, a concise understanding of all relevant factors affecting myocardial performance with valvular pathology is necessary. The scheme depicted in Figure I is useful for focusing on relevant factors that maintain hemodynamic stability. Systemic blood pressure is the product of vascular resistance and cardiac output. The product of heart rate and stroke volume obtains the latter, where stroke volume represents the difference between end-diastolic and end-systolic ventricular volumes. Factors affecting end-diastolic volume are ventricular preload and diastolic compliance; end-systolic volume is affected by afterload and contractility. Each of these factors is variably affected by valvular heart disease and will be interrelated in this context to further understand the perioperative management of the patient with valvular heart disease presenting for noncardiac surgery.

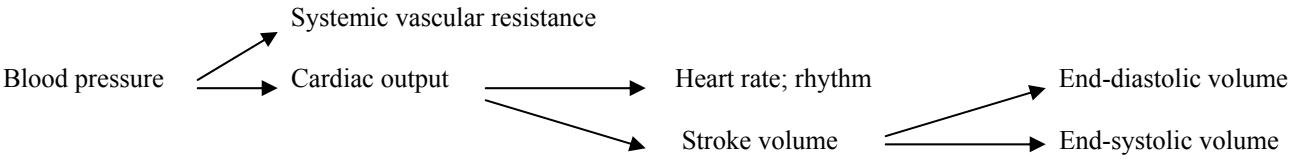

Figure 1. Relevant factors determining myocardial performance and systemic perfusion pressure.

MITRAL STENOSIS

The predominant causes of mitral stenosis include rheumatic heart disease > congenital > rheumatoid arthritis, lupus, and carcinoid syndrome. Normal mitral valve area is 4 to 6 cm2 with mild stenosis < 2 cm^2 and severe stenosis < 1 cm^2 (1,2). The severity of flow obstruction with mitral stenosis is affected by three factors: valve orifice area, mean diastolic pressure gradient across the valve, and duration of diastole (3). As mitral valve orifice decreases, the left atrial-ventricular pressure gradient must increase, thereby causing atrial dilation with atrial fibrillation seen in 30% - 70% of patients depending on patient age. Typical symptoms include congestive failure and pulmonary edema, particularly with elevated heart and transmitral flow rates seen with exercise, pregnancy, anemia, infection, or atrial fibrillation with rapid ventricular response. With mitral stenosis, valve area progressively decreases by approximately 0.09 - 0.17 cm^2/yr (4). Reactive pulmonary hypertension with a 5- to 10-fold increase in pulmonary vascular resistance from medial and intimal thickening of the pulmonary arteries and arterioles can occur secondarily, causing right ventricular failure and tricuspid regurgitation. Pregnant patients with mitral stenosis, pulmonary hypertension, and right heart failure are at increased risk from delivery and termination of pregnancy may require careful consideration. Symptomatic relief can be obtained by precutaneous mitral balloon valvulotomy, which may benefit high-risk patients with pure mitral stenosis. Catheter balloon commisurotomy for stenosis without regurgitation or calcification is effective in 90-95% of patients (5).

Anesthetic Implications

Echocardiography is the diagnostic examination of choice where mitral valve area can be derived from the Doppler half-time formula. This technique however tends to overestimate valve area and underestimate the severity of stenosis. Tachycardia can be particularly harmful because it shortens the period for diastolic filling. Since mitral valve orifice area is usually fixed, left atrial pressure must increase causing pulmonary congestion. Although sinus rhythm may contribute little to left ventricular end-diastolic volume with atrial dilation and valvular stenosis, atrial fibrillation can precipitate pulmonary edema from the increase in heart rate. Digoxin should be continued perioperatively with the use

of short-acting beta-blockers as necessary for rate control. If regional anesthesia is planned, careful consideration should be given to the additional hazard of chronic anticoagulation.

Due to longterm diuretic therapy, patients with mitral stenosis may require cautious volume expansion within limits imposed by the stenotic mitral valve. Reduced loading conditions from mitral stenosis plus atrial fibrillation are the primary determinants of impaired contractile performance although rheumatic myocarditis with fibrosis may further decrease myocardial compliance and ventricular filling. Any factor that further elevates pulmonary vascular resistance, such as hypoxia, hypercarbia, or nitrous oxide, can worsen right heart failure. Oversedation should be avoided, and supplemental oxygen in the preoperative period by nasal cannula is beneficial. If a pulmonary artery catheter is inserted, care should be taken to ensure proximal tip placement in order to avoid the possibility of pulmonary artery rupture with pulmonary hypertension. Due to the inherent limitation of preload reserve, inotropic infusion with dobutamine or epinephrine may be necessary to maintain stroke volume as long as tachycardia is avoided. Anticipate bleeding complications from chronic anticoagulation in patients with atrial fibrillation.

MITRAL REGURGITATION

Mitral regurgitation is the most commonly encountered valve lesion in modern clinical practice (6). Mitral regurgitation results primarily from abnormalities of the mitral valve, subvalvular apparatus, or cardiac skeleton caused by myxomatous degeneration and prolapse, rheumatic heart disease, fenfluramine diet suppressants, and endocarditis (7) or secondarily from functional lesions of the myocardium (ischemia, cardiomyopathy) (8). The amount of regurgitant flow depends on the pressure gradient between the left ventricle and atrium, the instantaneous mitral valve orifice size and duration of systole (9). The regurgitant orifice serves as an escape valve to reduce the impedance to left ventricular ejection so that there is little or no isovolumic contraction. Mitral regurgitation causes pure volume overload of the left ventricle which fails to elicit sarcomere replication as does pressure overload (10). With time and enhanced preload reserve, the left ventricle dilates and may decompensate secondary to the lack of hypertrophy (10). Global energy costs remain elevated due to increased fiber shortening with volume overload from forward and regurgitant ejection. No known medical therapy can directly alter the course of disease progression (11).

With experimental mitral regurgitation, mitral valve orifice area is not constant but varies directly with loading conditions. Volume expansion or afterload augmentation increases the regurgitant orifice area; afterload reduction or increased contractility reduces orifice area (9). Similar findings have been reported in patients where, in the absence of calcification, the area of the mitral valve regurgitant orifice parallels ventricular size (12). Afterload reduction with sodium nitroprusside reduces systemic vascular resistance but does not change the transvalvular systolic pressure gradient due to proportionate reductions in left ventricular systolic as well as left atrial pressures. Tachycardia from 90 bpm to 180 bpm reduces the regurgitant volume per beat by shortening systolic duration but, by increasing the number of beats per minute, has no overall effect on regurgitant fraction (13). As seen in Table 1, the effects of venous and arterial dilators in patients with mitral regurgitation produce complex but predictable interactions between preload and afterload effects. Preload reduction can allow more complete mitral valve closure that reduces regurgitant orifice area, particularly with dilated cardiomyopathy; afterload reduction can increase antegrade flow across the aortic valve.

Table 1. Net effects of venous and arterial dilators in patients with mitral regurgitation

Drug Action	Aortic pressure	Systemic vascular resistance	Forward stroke volume	Regurgitant volume	Regurgitant fraction	LV end-diastolic volume
Venodilation (nitroglycerin)	Slight ↓	No Δ	No Δ	No Δ or ↓	No Δ or ↓	↓
Arterial dilation (nifedipine, hydralazine)	No Δ	↓	↑	↓	↓	No Δ
Arterial and venous dilation (nitroprusside)	↓	↓	↑	↓	↓	No Δ

Anesthetic Implications

Since total stroke volume consists of both forward and regurgitant volumes, ejection fraction may overestimate left ventricular contractility. Favorable loading conditions tend to mask underlying contractile dysfunction; ejection fraction values less than 50% indicate impaired myocardial performance. If a pulmonary artery catheter is used, v-wave height may or may not indicate the severity of regurgitation, depending on size and compliance of the left atrium. Longstanding mitral regurgitation may cause reactive pulmonary hypertension and right heart failure. Vasodilator therapy can be acutely beneficial if the reduction in left ventricular pressure exceeds the reduction in left atrial pressure. With dynamic lesions, such as papillary muscle dysfunction, ruptured chordae, or dilated cardiomyopathy, the regurgitant orifice area as well as the transvalvular pressure gradient can decrease with vasodilators. In practice, under the influence of general or regional anesthesia, afterload reduction responds well to preload augmentation to enhance cardiac output and blood pressure (14). Although tachycardia may not change the regurgitant volume/min, bradycardia is poorly tolerated due to prolongation of left ventricular filling and should be avoided.

MITRAL VALVE PROLAPSE

Mitral valve prolapse is an inherited connective tissue disorder with myxomatous proliferation that causes thickening and redundancy of the mitral valve, thereby producing nonspecific symptoms of syncope, fatigue, palpitations, and atypical chest pain (16, 17). In this country, mitral valve prolapse is the most common cause of mitral valve disease. Primary risk factors for complications from mitral valve prolapse are ejection fraction < 50% and moderate-severe mitral regurgitation (17) and these complications occur most frequently in older, male patients (15). Any reduction in left ventricular volume during systole causes failure of prolapsing leaflets to coapt which worsens regurgitation. Atrial and ventricular arrhythmias are common. Prophylactic antibiotics are indicated with mitral valve prolapse with a murmur but not for a midsystolic click only (18). In general, the degree of mitral regurgitation with mitral valve prolapse corresponds with the extent of leaflet thickening and redundancy.

Anesthetic Implications

Patients with mitral valve prolapse should not be managed similar to patients with mitral regurgitation -- reductions in venous return and vascular resistance, tachycardia, or increased contractility are all poorly tolerated. In these patients, low intravascular volume causes greater systolic displacement of leaflets into the left atrium. General anesthesia using volatile agents with careful volume replacement, vasoconstrictors to support blood pressure, and short-acting beta-blockers to control heart rate is recommended to maintain end-diastolic and end-systolic ventricular volumes.

AORTIC STENOSIS

In the general population, the primary cause of aortic stenosis has changed from rheumatic heart disease to senile, calcific degeneration; patients with aortic stenosis are generally older and 50% have concomitant coronary artery disease (1, 19, 20). Less frequent is bicuspid valvular stenosis which is seen predominantly in men. Normal aortic valve area is 2.6 to 3.5 cm^2, with 1.0 to 1.4 cm^2 representing moderate stenosis and < 0.9 cm^2 representing severe stenosis. On average, the rate of progression of aortic stenosis further decreases aortic valve area by 0. 1 cm^2/yr or increases the transvalvular pressure gradient by 8-10 mmHg/year (20). Presenting symptoms with aortic stenosis include angina, failure (dyspnea), and syncope, with the average time of onset from symptoms to death without intervening therapy being 5, 3, and 2 years, respectively (21). Transvalvular flow depends on the systolic pressure gradient between the left ventricle and aorta, duration of systole, and aortic valve area, which may be fixed or dynamic. Valvular obstruction increases antegrade blood flow velocity and pressure gradient, although the magnitude of these increments varies with the volume flow rate across the valve. As stenosis becomes more severe, the maximal velocity and pressure gradient tend to occur later in systole. The severity of aortic stenosis can be followed by jet velocity where >3 m/sec indicates significant flow obstruction (19). In response to chronic pressure overload, the left ventricle undergoes concentric hypertrophy with parallel sarcomere replication and an increase in wall thickness to reduce wall stress. However, the increment in muscle mass has limited collateral perfusion and coronary vasodilator reserve that predisposes to subendocardial ischemia, particularly with the lengthening of systolic duration (21). Diastolic dysfunction may develop early in the course of disease giving less time for subendocardial blood flow. In addition, aortic regurgitation may coexist in 70% - 80% of patients with aortic stenosis.

Anesthetic Implications

Although patients with aortic stenosis have historically been at increased risk from surgery, these patients can undergo noncardiac surgery safely with special attention to optimizing and maintaining systemic perfusion pressure by avoiding hypotension (22, 23). With diastolic dysfunction and impaired relaxation, atrial contraction may contribute 25% - 40% of end-diastolic volume instead of the usual 15% - 20%. Consequently, maintaining volume status and sinus rhythm are essential; tachycardia must be avoided. Premedication should be light, and chest pain in the preoperative period should be managed with supplemental oxygen and not nitrates because of venodilation and reduced left ventricular filling. Volume depletion is poorly tolerated intraoperatively in patients with aortic stenosis. Systemic hypotension causing any reduction in coronary perfusion pressure should be vigorously managed with careful volume replacement and/or a vasoconstrictor. Postoperatively, effective pain management is essential, and patients should be appropriately monitored until fluid shifts have stabilized.

AORTIC REGURGITATION

Causes of aortic regurgitation include rheumatic heart disease, endocarditis, trauma, connective tissue disorders, and appetite suppressant medications containing dexfenfluramine. Unlike mitral regurgitation with predominantly volume overload, aortic regurgitation has combined pressure and volume overload, which causes eccentric ventricular hypertrophy with sarcomere replication in series (1, 10). Once preload reserve is exhausted and the hypertrophic response becomes inadequate, any further increase in afterload can reduce stroke volume and ejection fraction, thereby causing symptoms of exertional angina and failure with exercise intolerance (22). Symptoms and severity of aortic regurgitation correlate with an increase in left ventricular end-systolic size > 60 mL/m2 (7). Using angiography, regurgitant fraction < 0. 1 = trivial regurgitation, 0. 1 to 0.4 = mild regurgitation, 0.4 to 0. 6 = moderate regurgitation, and > 0. 6 = severe regurgitation.

The regurgitant volume depends directly on the aortic orifice area, the diastolic pressure gradient between the aorta and left ventricle, and the diastolic time interval. Theoretically, increasing heart rate should improve net forward flow by decreasing the diastolic time interval. However, any reduction in regurgitant volume/beat is offset by the

increase in beats/min so that regurgitant volume/min remains constant. In experimental animals, the effective aortic regurgitant orifice area appears dynamic and directly proportionate to the transvalvular pressure gradient - increasing or decreasing aortic pressure by 20 mmHg with dopamine or nitroprusside, respectively, caused directionally similar changes in orifice area by 30% (24). Consequently, a strong direct correlation exists between vascular resistance and regurgitant volume. Chronic therapy with afterload reduction can reduce the regurgitant fraction and progression of left ventricular dilation (1).

Anesthetic Implications

Ejection phase indices of contractility with aortic regurgitation tend to be unreliable, since the increase in preload and reduction in afterload (low diastolic blood pressure) can mask underlying impaired contractility. Similar to aortic stenosis, eccentric left ventricular hypertrophy elicits less development of coronary collateral vessels with reduced vasodilator reserve. Coupled with ventricular dilation, hypertrophy, tachycardia, and decreased diastolic coronary perfusion pressure, these patients are prone to subendocardial ischemia. Acute afterload reduction can be beneficial if coupled with preload augmentation. Patients with aortic regurgitation benefit intraoperatively from afterload reduction with sodium nitroprusside more than patients with mitral regurgitation; tachycardia is better tolerated than bradycardia. Although increasing heart rate does not alter total regurgitant flow or forward stroke volume, tachycardia decreases ventricular size and augments diastolic arterial pressure, and, consequently, coronary perfusion pressure.

HYPERTROPHIC CARDIOMYOPATHY

Hypertrophic cardiomyopathy is the most common genetically transmitted cardiac disorder. Inherited as an autosomal dominant trait, hypertrophic cardiomyopathy is a heterogeneous disease of the sarcomere with greater than 150 mutations of the 4 genes that encode protein composition (25). In 25% of patients with hypertrophic cardiomyopathy, dynamic left ventricular outflow results from mechanical impedance of the hypertrophied ventricular septum with abnormal systolic anterior motion of the mitral valve, although the majority of patients show no obstructive symptoms (26, 27). Diastolic dysfunction may ensue from impaired relaxation of hypertrophied muscle plus shortening of diastole from prolonged contraction. Patients can present with symptoms of congestive failure, angina, syncope, and palpitations. These patients are prone to subendocardial ischemia and sudden death from ventricular tachyarrhythmias. Chronic medical management consists of beta blockers to control heart rate and reduce outflow obstruction; calcium channel blockers to improve ventricular filling and reduce myocardial ischemia; disopyramide to reduce myocardial contractility; amiodarone for dysrhythmias or atrial fibrillation; dual chamber pacing which causes right ventricular preexcitation with asynchronous depolarization and contraction; ventricular septal myotomy or myectomy for severe outflow tract gradient >50 mmHg at rest; and alcohol injection of major septal perforator branches of the left anterior descending coronary artery to cause localized septal infarction. Although less invasive then surgery, alcohol injection may cause complete heart block requiring permanent pacemaker in 20% of patients (28).

Anesthetic Implications

Left ventricular outflow obstruction with hypertrophic cardiomyopathy is a dynamic process and worsened by any maneuver that reduces left ventricular volume. Clinical scenarios include hypovolemia, positive pressure ventilation, positive end-expiratory pressure, and tachycardia that reduce ventricular filling and end-diastolic volume, and increased contractility and systemic vasodilation that promote ventricular ejection to a lower end-systolic volume. Left atrial enlargement secondary to mitral regurgitation makes these patients prone to atrial fibrillation. Maintaining sinus rhythm is essential, since atrial contraction may contribute 50% to 60% of left ventricular end-diastolic volume. Careful volume replacement is necessary especially in the fasted patient, and transient episodes of hypotension should be aggressively managed with volume expansion and vasoconstriction (phenylephrine). These patients suffer significant perioperative morbidity with prolonged, major operations but surprisingly little mortality (29).

ANTIMICROBIAL PROPHYLAXIS AGAINST ENDOCARDITIS

Recent guidelines published by the American Heart Association now stratify different cardiac conditions, according to the probability of contracting endocarditis, as high-, moderate-, and negliable-risk categories (18). Patients with congenital cardiac defects, acquired valvular disease, hypertrophic cardiomyopathy, and mitral valve prolapse with regurgitation are in the moderate risk category and mandate perioperative antimicrobial therapy based on type, location, and severity of the operative procedure. In general, antimicrobial prophylaxis against *Streptococcus viridans* is necessary for dental, oral, and respiratory procedures, while antimicrobial prophylaxis against *Enterococcus faecalis* is necessary for genitourinary and gastrointestinal procedures.

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