

Cardiac Valve Disease and Anesthetic Management

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Aortic Valve	2-4 cm ²
Mitral Valve	3-6 cm ²
Tricuspid Valve	7-9 cm ²
Pulmonary Valve	2-4 cm ²

Table 1. Normal cardiac valve areas.

Aortic Stenosis

Aortic stenosis (AS) is obstruction to outflow of blood from the left ventricle (LV), which may be at the valve, above the valve (supravalvular), or below the valve (subvalvular). Supravalvular AS is a congenital lesion. Subvalvular AS results either from a congenital discrete fibromuscular obstruction or from a muscular obstruction (hypertrophic obstructive cardiomyopathy HOCM).

The most common causes of AS are congenital, rheumatic, and calcific (degenerative). At the present time, calcific ("degenerative") AS in the older patient is the most common valve lesion requiring aortic valve replacement (AVR). Among patients younger than age 70 years, congenital bicuspid valve accounted for one-half of the surgical cases; "degenerative" changes were the cause in 18 percent. In those age 70 years or older, degenerative changes accounted for almost one-half of the surgical cases and a congenital bicuspid valve accounted for approximately one-quarter of the cases.

Pathophysiology: The aortic valve area (AVA, normal value 2-4 cm²) has to be reduced by approximately 50 percent of normal before a measurable gradient can be demonstrated in humans. When a pressure gradient develops between the LV and the ascending aorta, LV pressure rises. As LV pressure rises, ventricular wall stress increases, which leads to impaired LV function. Hypertrophy develops in proportion to increased intraventricular pressure, and myocardial stress remains normal (Laplace's Law). The ventricle becomes hypertrophied and stiff in response to the increased pressure load. Coordinated atrial contraction becomes critical to maintaining adequate ventricular filling and stroke volume. Adequate preload also becomes critical and hypovolemia is not well tolerated. The ventricle is susceptible to ischemia due to increased muscle mass and decreased coronary perfusion in the setting of increased intraventricular pressure. A valve area <1.0 cm² defines severe stenosis. Critical aortic stenosis is defined as a valve area ≤0.75 cm². A mean gradient across the aortic valve between 25-50 mmHg is consistent with moderate stenosis, a mean gradient higher than 50 mmHg defines severe aortic stenosis.

Patients with heart failure can show lower pressure gradients on echocardiographic examination. In aortic stenosis patients symptoms include angina, dyspnea, syncope and congestive heart failure. Symptoms appear late in the disease process. In the absence of surgical intervention, life expectancy is 5 years after the onset of angina and 2 years after the development of CHF.

Anesthetic Considerations: Aortic stenosis is the only valvular lesion directly associated with an increased risk of perioperative ischemia, MI, and death. Normal sinus rhythm, low to normal heart rate and adequate volume status should be maintained. Contractility should not be increased and afterload must be maintained normal to elevated. Hypotension, tachycardia (decreasing filling and increasing oxygen demands), and severe bradycardia (decreasing cardiac output) are poorly tolerated and should be aggressively treated to maintain coronary perfusion pressure. Cardiac pacing capabilities should be considered to treat bradycardia. Supraventricular tachydysrhythmias should be treated aggressively with direct current cardioversion. Pulmonary artery catheters may be useful to assess baseline filling pressures (caution: volume might be underestimated), ventricular function, and response to pharmacologic interventions, fluid therapy, and changes of heart rate and rhythm. Nitrates and peripheral vasodilators should be administered with extreme caution because small reductions of ventricular volume can markedly reduce cardiac output. The treatment of ischemia in these patients is directed at increasing oxygen delivery by raising coronary perfusion pressure and decreasing oxygen consumption (usually by lowering heart rate). Options for anesthetic management in patients with severe/critical aortic stenosis should start by considering general anesthetic the technique of choice. Spinal anesthetic is a high risk technique. A gradual epidural could be a choice if the benefit truly outweighs the elevated risk. If regional anesthetic is considered blocks are usually both safe and suitable. Use of intraaortic balloon pump in emergencies can be life saving. If there is enough time, consideration can be given to a percutaneous valvuloplasty as a temporary relief to the obstruction.

Hypertrophic Cardiomyopathy

Genetic cardiac disorder characterized by asymmetric LV hypertrophy (it means either not all the walls of the LV are hypertrophic, or the degree of hypertrophy is not the same in all of them).

Pathophysiology: Although most patients with hypertrophic cardiomyopathy do not have an increased LV outflow tract gradient at rest, many of them develop dynamic outflow tract obstruction with increased cardiac output. The mechanism of subaortic LV outflow tract obstruction is systolic anterior motion of the mitral valve leaflets leading to ventricular septal contact. Factors that worsen the outflow obstruction include decreased arterial pressure, decreased intraventricular volume, increased contractility, and increased heart rate. Clinical implications and treatment are similar to those for aortic stenosis.

Anesthetic considerations: Maintain normal sinus rhythm. Heart rate low to normal. Consider cardioversion for supraventricular tachycardia. Continue β -adrenergic and calcium channel blocker therapy. Maintain preload and afterload is very important. Maintain normal volume status. Correct vasodilation with α -adrenergic agonists to avoid tachycardia and marked changes in contractility. Use inotropes with caution because they may exacerbate the outflow obstruction. Use nitrates and peripheral dilators only with extreme caution. Spinal anesthetic is a high risk technique in these patients. A gradual epidural could be a choice if the benefit truly outweighs

the elevated risk. If regional anesthetic is considered blocks are usually both safe and suitable.

Aortic Insufficiency

Etiologies include rheumatic heart disease, endocarditis, trauma, collagen vascular diseases, and processes that dilate the aortic root (e.g., aneurysm, Marfan).

Pathophysiology: Chronic aortic regurgitation leads to LV dilation and eccentric hypertrophy. Symptoms may be minimal until late in the disease process when left heart failure occurs. Early referral for valve replacement has been associated to better outcomes. Acute aortic regurgitation may cause sudden LV volume overload with increased LV end-diastolic pressure and pulmonary capillary occlusion pressure. Manifestations may include decreased cardiac output, CHF, tachycardia, and vasoconstriction. Angina has also been described during acute aortic regurgitation, it is due to decreased myocardial perfusion due to the sudden increase in left ventricular end diastolic pressure.

Anesthetic considerations: Maintain a normal to slightly increased heart rate to minimize regurgitation and maintain aortic diastolic and coronary artery perfusion pressure. Maintain adequate volume status. Improve forward flow, decrease LV end-diastolic pressure and myocardial wall tension with vasodilators. Avoid peripheral arterial constrictors. They may worsen regurgitation. Consider pacing. These patients have an increased frequency of conduction abnormalities. Both general and regional anesthetic techniques are suitable to manage these patients.

Mitral stenosis

The etiology is almost always rheumatic. Congenital Mitral stenosis is an uncommon cause.

Pathophysiology: In Mitral stenosis increased left atrial pressure and volume overload increases left atrial size and may lead to atrial fibrillation. Elevated left atrial pressure increases pulmonary venous pressure and pulmonary vascular resistance. In turn, right ventricular (RV) pressure is increased for a given cardiac output. Chronic pulmonary hypertension produces pulmonary vascular remodeling. Pulmonary hypertension may lead to tricuspid regurgitation, RV failure, and decreased cardiac output. Tachycardia is poorly tolerated because it decreases diastolic filling time, decreases cardiac output, and increases left atrial pressure.

Anesthetic considerations: Avoid tachycardia. Maintain/restore sinus rhythm whenever possible. Control ventricular response pharmacologically or consider cardioversion for patients with atrial fibrillation. Normal preload and afterload should be kept. Contractility should be kept equal. Continue digoxin, calcium channel blockers, and β -adrenergic blockers perioperatively. Avoid pulmonary hypertension. Hypoxia, hypercarbia, acidosis, atelectasis, and sympathomimetics increase pulmonary vascular resistance. Oxygen, hypocarbia, alkalosis, nitrates, prostaglandin E₁, and inhaled nitric oxide decrease pulmonary vascular resistance. Hypotension may be caused by hypovolemia; however, one must have a high suspicion for RV failure. Inotropes and agents that decrease pulmonary hypertension may be useful (e.g., dopamine, dobutamine, milrinone, amrinone, nitrates, prostaglandin E₁, and inhaled nitric oxide). A pulmonary artery catheter

may assist in perioperative evaluation of volume status, intracardiac pressures, and cardiac output (caution of underestimating volume status). Premedication should be adequate to prevent anxiety and tachycardia. Exercise caution when prescribing sedation to patients with pulmonary hypertension, or low cardiac output. Spinal anesthetic is high risk in these patients. A gradual epidural is considered safe.

Mitral Regurgitation

Etiologies include mitral valve prolapse and degeneration, chronic ischemic heart disease, endocarditis, and post-MI papillary muscle rupture.

Pathophysiology: Mitral regurgitation allows blood to be ejected into the left atrium during systole. The amount of regurgitant flow depends on the ventricular-atrial pressure gradient, size of the mitral orifice, and duration of systole. Acute mitral regurgitation usually occurs either in the setting of MI or bacterial endocarditis.

Acute volume overload of the left heart leads to LV dysfunction with increased wall tension. Chronic mitral regurgitation causes gradual left atrial and LV overload and dilation with compensatory hypertrophy. Measurement of ejection fraction does not quantify forward versus backward flow, because the incompetent valve permits immediate bidirectional ejection with systole.

Anesthetic considerations: Relative tachycardia is desirable to decrease ventricular filling time and ventricular volume. Bradycardia is associated with increased LV volume and regurgitation. Afterload reduction is beneficial. Intra-aortic balloon counterpulsation may be life-saving. Increased systemic vascular resistance will increase regurgitation. Maintain preload. Careful titration of myocardial depressants is indicated. Special consideration must be given to adequate analgesia intraoperatively, a sudden elevation of systemic blood pressure in response to surgical stimulation can lead to acute heart failure and pulmonary edema. Both spinal and epidural anesthetic are well tolerated.

Tricuspid Stenosis

Uncommon lesion. Usually associated to other valve conditions, Tricuspid stenosis causes include congenital, rheumatic heart disease, carcinoid syndrome.

Pathophysiology: increased pressure in the right atrium is the main problem as a progressive gradient develops across the valve. Right atrial hypertrophy develops to compensate. Both diastolic time and atrial contraction become more important to fill the right ventricle and maintain cardiac output.

Anesthetic considerations: bradycardia and sinus rhythm are desirable. Good preload and normal contractility besides normal afterload are the goals in management. Aggressive treatment of tachycardia and tachyarrhythmias must be remembered.

Tricuspid Regurgitation

Mild Tricuspid regurgitation (TR) is a common finding in otherwise asymptomatic people. Significant Tricuspid regurgitation is more commonly due to valve annulus dilatation caused by left heart disease (Mitral valve disease, decreased LV function); pulmonary hypertension can also cause TR. Degenerative disease of the valve is another cause of TR and usually is associated to Mitral regurgitation. Carcinoid

syndrome, rheumatic heart disease and congenital problems (Ebstein anomaly the most common) are other causes of TR.

Pathophysiology: increased volume load on the right ventricle is the main problem, the right ventricle will handle the volume overload and will dilate leading to increased TR. Venous congestion (edema, ascites, liver enlargement) will ensue.

Anesthetic considerations: management aimed to keep sinus rhythm and maintain a normal to elevated heart rate, maintain preload and contractility and prevent increases in pulmonary vascular resistance.

Pulmonary Stenosis

Very uncommon lesion. Usually a congenital lesion that might be associated to a VSD (as in Fallot's tetralogy). Rheumatic and carcinoid are other possible etiologies.

Symptomatic pulmonary stenosis can lead to fatigue and syncope.

Pathophysiology: increased RV pressure and delayed emptying of the RV during systole are the most prominent features. Secondary TR is usually seen as well as signs of RV failure.

Anesthetic considerations: management is aimed to maintain sinus rhythm and normal to increased heart rates (to maintain forward flow) and preserve contractility (to preserve cardiac output), a normal preload and low to normal afterload by preventing increases in pulmonary vascular resistance. Systemic vascular resistance must be preserved to prevent RV ischemia.

Lesion	Rate	Rhythm	Preload	Contractility	Afterload LV	Afterload RV
AS	LOW	SINUS (CV)	HIGH	NORMAL	N TO HIGH	NORMAL
AI	HIGH	SINUS	NORMAL	N TO HIGH	NORMAL	NORMAL
MS	LOW	SINUS	NORMAL	N TO HIGH	NORMAL	N TO LOW
MR	HIGH	SINUS	NORMAL	N TO HIGH	LOW	N TO LOW
TS	LOW	SINUS	NORMAL	NORMAL	NORMAL	N TO LOW
TR	HIGH	SINUS	NORMAL	NORMAL	NORMAL	N TO LOW
PS	LOW	SINUS (CV)	HIGH	INCREASE	N TO HIGH	N TO LOW
PI	HIGH	SINUS	NORMAL	NORMAL	NORMAL	N TO LOW
HOCM	LOW	SINUS (CV)	HIGH	DECREASE	N TO HIGH	NORMAL

TABLE 2. Hemodynamic goals in cardiac valve disease. (CV): low threshold for cardioversion; LOW heart rate: 60-80, HIGH heart rate 80-90; HIGH preload CVP 6-10 or PCWP 10-15; N TO HIGH: normal to elevated; N TO LOW: normal to decreased. Modified from different sources. (Barasch, Fleisher, Miller, Kaplan, and Anesthesiol Clin 2006).

Prosthetic Valves

About 100,000 new prosthetic cardiac valves are implanted each year worldwide. Prosthetic valves can be either mechanical or bioprosthesis. 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; they are highly thrombogenic, and patients need to be placed on long-term anticoagulant therapy. Bioprosthetic valves are usually heterografts and are made of bovine or porcine tissue and mounted on a metal support; 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.

Anesthetic considerations: The presence of prosthetic valves in the patient prompts us to look for associated complications related to the cardiac condition that forced to replace the valve and also complications related to the presence of the prosthetic valve, the most common of which are thromboembolism and anticoagulant related bleeding. Hemolysis and endocarditis are also seen in patients who have prosthetic valves. Preoperative assessment must focus on the pre-existing cardiac condition that lead to valve replacement and associated complications (pulmonary hypertension, dilated cardiomyopathy) that were present before the valve replacement, the impact caused by the valve replacement on cardiac function recovery and/or improvement in pulmonary pressures is usually documented through clinical history and echocardiogram. Patients on anticoagulation will need bridge therapy switching to heparin before surgery, consultation and advice from Hematologists is usually wise. A patient with normal functioning prosthetic valve and preserved (or improved) ventricular function is treated as a general patient and considerations are limited to anticoagulation and bacterial endocarditis prophylaxis. Patients with pre valve replacement cardiomyopathy and/or pulmonary hypertension that did not improve after the valve replacement must be dealt with caution as high risk patients due to the underlying cardiovascular abnormalities. Invasive monitoring in patients with prosthetic cardiac valves will yield better information and allow better management, a pulmonary artery catheter is contraindicated in patients with prosthetic tricuspid or pulmonary valve, other contraindications to pulmonary artery catheters are shown in table 3.

Insertion site:	Infection
Catheter placement:	ICD/permanent pacing leads <2 months Right atrial/ventricle mass, vegetation Critical Tricuspid and Pulmonary stenosis Prosthetic tricuspid/pulmonary valve
Caution with placement (pacing pads on):	LBBB Severe aortic stenosis

Table 3. Contraindications to pulmonary artery catheters

Cardiac Valve Disease during pregnancy, labour and C-section

The basic principles of obstetric anesthesia management always apply: 1- provisions for the maintenance of uteroplacental perfusion by the avoidance of

aortocaval compression; 2- minimizing sympathetic blockade coupled with intravascular volume maintenance; 3- monitoring of the parturient and the fetus; 4- provision for aspiration prophylaxis; 5- assess associated conditions. Asymptomatic and stable patients with cardiac valve disease will have lower rates of complications. Anesthetic assessment specifically oriented to the valve condition must include clinical history (functional class, symptoms, heart failure, arrhythmias, anticoagulation, previous echocardiograms) and physical examination aimed to assess the impact of the cardiac valve condition on the patient's physiological reserve.

Five principal changes in the cardiovascular system during pregnancy that present unique problems to the parturient with underlying heart disease have been well delineated:

- 1- 50% increase in intravascular volume that generally peaks by the early-to-middle third trimester.
- 2- Progressive decrease in systemic vascular resistance (SVR) throughout pregnancy, thanks to this MAP is preserved at normal values, despite a 30%–40% increase in cardiac output (CO).
- 3- Marked fluctuations in CO during labor. Pain and apprehension may precipitate an increase in CO to as much as 40%–50% over those levels seen in the late second stage of labor.
- 4- Each uterine contraction serves as an autotransfusion to the central blood volume, an increase in CO of 10%–25% is seen
- 5- Hypercoagulability associated with pregnancy and the possible need for appropriate anticoagulation, especially in those patients at increased risk for arterial thrombosis and embolization (prosthetic heart valve).