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TIME IS TIGHT – AORTIC AND MITRAL STENOSIS

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Objectives

1. **Review the causes and anatomic responses to valvular stenosis**
2. **Recite the criteria for diagnosis of mitral and aortic stenosis**
3. **Describe the anesthetic management of valvular stenotic heart disease**

The importance of understanding stenotic valvular heart disease

Two of the most challenging valvular lesions for the anesthesiologist to consider are aortic stenosis and mitral stenosis. The added risk of patients with valvular abnormalities presenting for noncardiac surgery is unclear. Goldman considered aortic stenosis one of the variables associated with increased risk[1]. Detsky's modification in 1986 added even more weight to suspected critical aortic stenosis[2]. The added mortality risk of aortic stenosis with the revised Cardiac Risk Index (RCRI) was presented by Kertai and is significant[3]. Other studies have not shown similar risk[4]. The risk of noncardiac surgery in patients with mitral stenosis is even less well studied, although Goldman mentions it as a risk factor for congestive heart failure.

Aortic Stenosis

The most common cardiac valvular disease encountered by the anesthesiologist is aortic stenosis, occurring in nearly 4% of patients over the age of 85[5]. The elderly population is most commonly affected but even younger individuals may have significant disease. The development of symptomatic, severe stenosis is associated with a life expectancy of only 2-5 years unless treatment occurs.

Anatomy and Pathophysiology of Aortic Stenosis

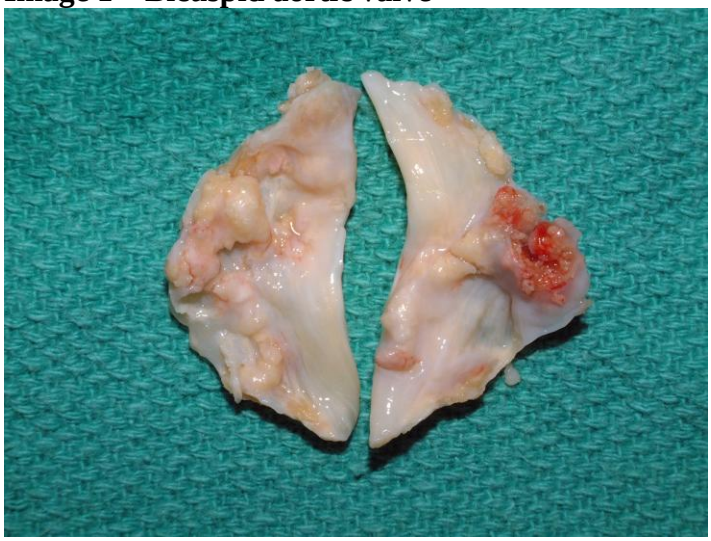
Stenosis may be either congenital, or more commonly acquired (Table XXX). Most is **degenerative**, associated with thickening, fibrosis, and calcification of valve leaflets, which decreases flexibility and orifice area (normal aortic valve area is 3-4 cm^2). Calcification and fibrosis begins at the commisures of the valves and progresses towards the tips of the leaflets. **Congenital abnormalities** lead to earlier degeneration. Adult patients with **unicuspid** aortic valves (rare) may require replacement for stenosis as early as their third decade of life, while those with **bicuspid** valves (1-2% of the population) usually present in the fourth to sixth decade (Image 1). Individuals requiring replacement of an abnormal aortic valve in the latest decades of life generally have trileaflet valves. Isolated **rheumatic aortic valvular disease** is uncommon in the United States but frequently noted overseas or in immigrants. As opposed to degenerative disease,

rheumatic valvular disease starts with thickening and calcification of the tips of the valve leaflets and progresses towards the commissures.

Table XXX- Causes of Aortic Stenosis

Congenital	Acquired	Rare
Unicuspid Bicuspid	Rheumatic Calcific	Obstructive Paget's disease of bone Systemic lupus Rheumatoid Irradiation

Image 1 – Bicuspid aortic valve

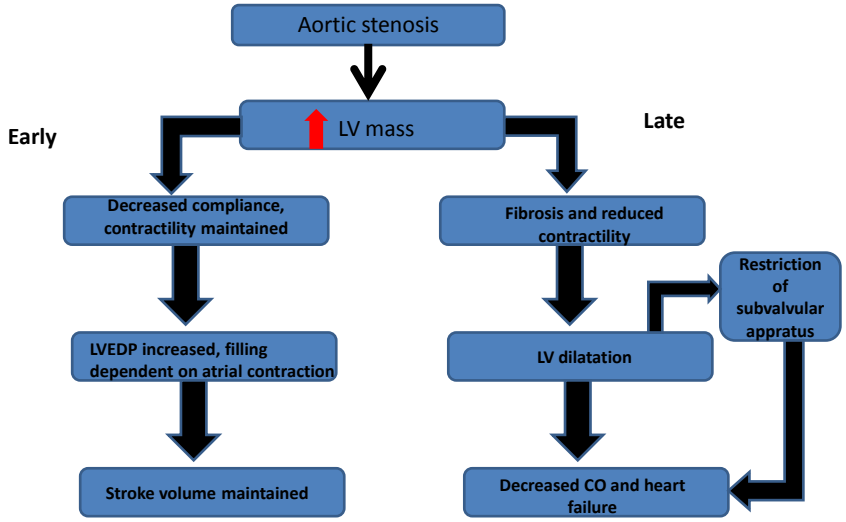


Progressive thickening and fibrosis leads to a decrease in the valve orifice area and a **pressure overload** on the left ventricle due to resistance to ejection of blood. The ventricle compensates with a **concentric hypertrophy** and a reduction in ventricular compliance (Table XXX). The concentric hypertrophy is a compensatory mechanism to maintain ejection. **Diastolic dysfunction** occurs as the thick ventricle looses the ability to relax appropriately. The phenomenon of "**atrial kick**" takes on an increased importance to maintain diastolic filling. The normal contribution to diastolic filling is 12-15% from the "atrial kick", but up to 40% with aortic stenosis. Stenosis seems to progress yearly with a reduction of orifice area by about 0.1 cm^2 to as high as 0.3 cm^2 per year. Clinically **critical aortic stenosis** develops when symptoms such as syncope, angina, or congestive heart failure occur. The atrium encounters a progressively elevated pressure and itself responds with dilatation. Enlargement results in the risk of atrial fibrillation. The ventricle weakens over time and ventricular dilatation occurs. Dilatation of the ventricle results in tethering of the components of the mitral subvalvular apparatus. This results in mitral regurgitation. Mitral regurgitation associated with ventricular dysfunction produces clinical heart failure (Figure XXX).

Table XXX- Effects of Reduction in Ventricular Compliance

Parameter	Effects
Importance of atrial contribution for ventricular filling	Increased
Sensitivity to volume depletion	Increased
Coronary perfusion pressure	Decreased
Response of LVEDP to ventricular filling	Increased

Figure XXX- Progression of Effects of Aortic Stenosis



Adapted from *Atlas of Anesthesia*, Miler RD, 1998

Grading Aortic Stenosis

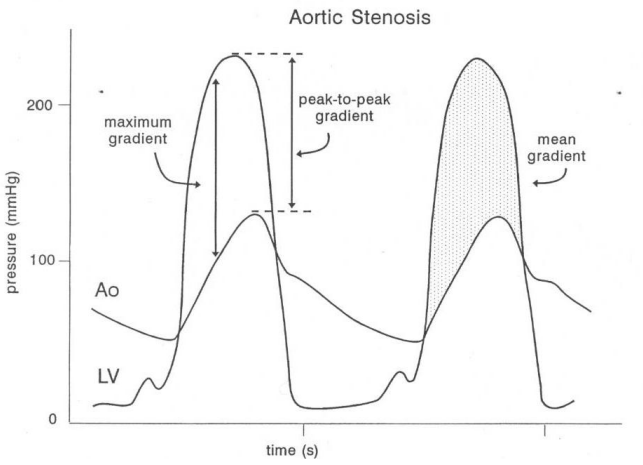
Aortic stenosis is graded by clinical, hemodynamic, and echocardiographic criteria (Table XXX). Although criteria are published[6], it must be remembered that an area of 1.0 cm^2 in a large healthy, active male may be critically symptomatic while a small elderly female may be fine with an area of 0.7 cm^2.

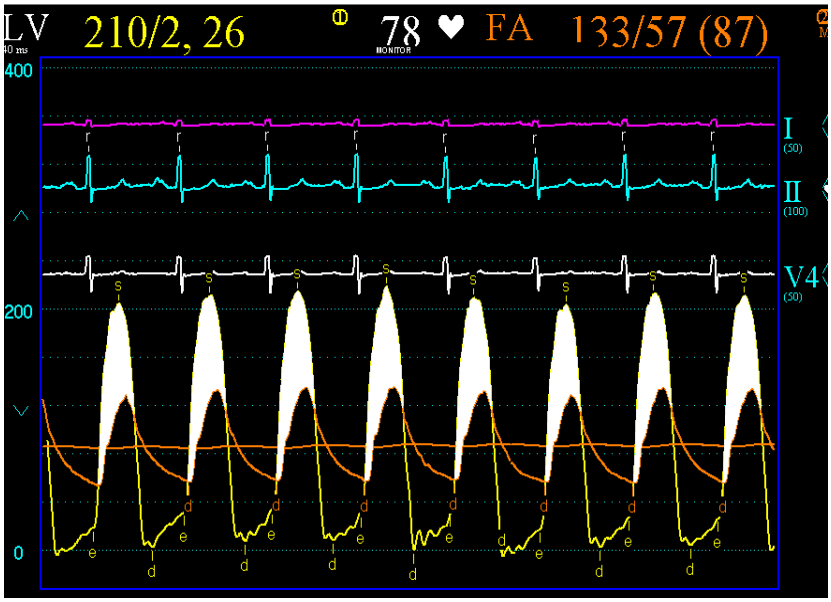
Table XXX- Aortic Stenosis Grading

Parameter	Mild	Moderate	Severe
Mean pressure gradient (mmHg)	< 25	25-40	>40
Aortic valve area (cm^2)	>1.5	1.0-1.5	<1.0

Cardiac catheterization allows measurement of a **peak-to-peak gradient** between the left ventricle cavity and the aorta. It is an accurate means to assess the gradient but is

invasive, expensive, and associated with some degree of morbidity. The area of the aortic valve is calculated with the **Gorlin equation**.





The gradient associated with aortic stenosis occurs during systole and is between the ventricle (yellow tracing) and the systemic circulation (in this case the femoral artery in orange)

Transesophageal echocardiography is a non-invasive means to assess both the aortic valve orifice area and gradient. It is fairly inexpensive and can easily be repeated. The gradient across the valve can be derived with a method called **continuous wave Doppler** (CWD). The peak and mean pressure gradients are calculated with the **Bernoulli equation**. Valve area can be assessed with a technique called **planimetry** (directly tracing the opening of the valve) or by the **continuity equation**. The simplified Bernoulli equation uses Doppler technology to determine a maximum velocity through an orifice. The maximum pressure gradient is calculated as Δ Pressure $_{max} = 4v_{max}^2$. The continuity equation utilizes the concept that flow through two orifices must be the same and that velocity must increase through a small orifice. Most modern echo machines have software allowing easy calculation.

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Low-Gradient Aortic Stenosis

Low-flow, low gradient aortic stenosis describes a subset of patients who are noted to have a severely stenotic valve (< 1.0 cm^2) yet low transaortic gradient (mean gradient commonly less than 30 mmHg) and poor ejection fraction (< 40%). These patients fall into three categories: Fixed AS, relative AS, and absence of contractile reserve. Patients with **fixed aortic stenosis** will demonstrate an increase in mean aortic gradient when the ventricle is subjected to an inotrope such as Dobutamine. The valve area does not change because it is "fixed". Patients with **relative aortic stenosis** demonstrate a calculated small aortic valve area. Essentially the ventricle is too weak to open the valve completely. When subjected to Dobutamine, the valve opens more completely. The calculated area is actually increased. Patients with **absent contractile reserve** simply do not respond to Dobutamine . These patients have a very high perioperative mortality associated with aortic valve replacement[7]. Patients with low-flow, low-gradient aortic stenosis presenting for general anesthesia should be considered to be at a further state of their disease and likely at more risk.

Clinical management

Patients diagnosed with aortic stenosis are initially evaluated with an ECG, chest x-ray, and echocardiogram. Exercise tolerance testing is generally discouraged, especially in symptomatic patients. Most patients are followed clinically and treated with beta-blockers until the classical symptoms of angina, dyspnea, or syncope develop. Certain patients without symptoms may benefit from valve replacement including severe hypertrophy, markedly decreased valve area, abnormal response to exercise (hypotension) or some degree of LV dysfunction. The care provider should also recognize the high rate of coronary artery disease in patients with stenosis of the aortic valve[8].

Anesthetic Management

1. **Adequate preload**. Compliance of the left ventricle is deceased in aortic stenosis. This results in an increase in LV end-diastolic pressure.
2. **Normal, slightly slow sinus rhythm**. Tachycardia may not allow adequate coronary perfusion. Bradycardia may result in an inadequate cardiac output due to a fixed stroke volume. It is important to maintain sinus rhythm in patients who are in sinus rhythm to maintain the important "atrial kick". Back up pacing modalities such as transesophageal, pacing wires, or external pacing pads, may be considered in the setting of extreme bradycardia or escape ventricular rhythms. Very light anesthesia or use of medications such as pancuronium may result in tachycardia.
3. **Maintain the contractile state**.
4. **Adequate systemic vascular resistance**. Most of the resistance to left ventricle ejection comes from the stenotic aortic valve. Afterload reduction does little to increase cardiac output and may compromise perfusion to a hypertrophied left ventricle.

Neuraxial Blockade and Aortic Stenosis

One of the classic controversies in our specialty is whether spinal or epidural anesthesia can safely be performed in a patient with aortic stenosis. The potential sudden and profound decrease in systemic vascular resistance can result in hypotension and a decrease in coronary perfusion to a hypertrophied myocardium. Additionally, the potential for significant bradycardia can decrease cardiac output. There is no clear answer concerning the safety of neuraxial anesthesia[9]. Case reports do exist describing successful spinal, epidural, and combined techniques[10,11]. Appropriate monitoring to detect hemodynamic compromise early and allow intervention is imperative.

Mitral Stenosis

Mitral stenosis (MS) is uncommon in the United States but is still primarily a result of **acute rheumatic fever** (RF) (Table XXX). Nearly 40% of cases occur in patients without a history of rheumatic fever. Women are more commonly affected than men. Still, the anesthesiologist must be prepared to manage these patients presenting for surgery or labor and delivery.

Table XXX- Causes of Mitral Stenosis

Cause	Involved Structures		
	Leaflets	Chordae	Commissure
Rheumatic fever	+	+	+
Congenital	+	+	-
Endocarditis	+	-	-
Neoplasm	-	-	-
Annular calcification	+	-	-
SLE	+	+	-

Anatomy and Physiology of Mitral Stenosis

The normal opening of the mitral valve orifice is 4-6 cm^2. The thickening, decreased mobility, and calcification of the mitral valve associated with rheumatic heart disease starts at the tips of the leaflets and progresses towards the commissures of the valve and usually commonly involves the chordae tendinae and tips of the papillary muscle (Image 2). The time from the episode of RF until the appearance of symptoms may be 20-40 years or longer. As the orifice of the mitral valve decreases with time, a gradient develops between the left atrium and left ventricle. Symptoms initially start with exercise or high cardiac output states. The constant pressure overload on the left atrium results in progressive dilatation and atrial fibrillation. A patient may remain asymptomatic as long as the heart rate is low enough to allow filling of the left ventricle and emptying of the atrium (during diastole). Periods of stress associated with increases in cardiac output and heart rate decrease LA emptying and result in symptomatic pulmonary edema. Over time the pulmonary vasculature hypertrophies resulting in pulmonary hypertension, right

ventricular dilitation, tricuspid dilatation, and peripheral congestion, the classic symptoms of right ventricular failure (Figure XXX).

Image 2 – Mitral Stenosis

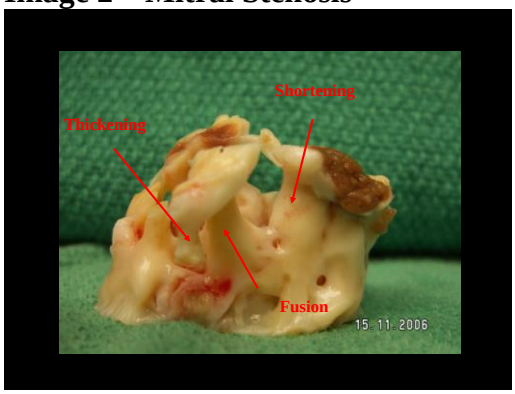
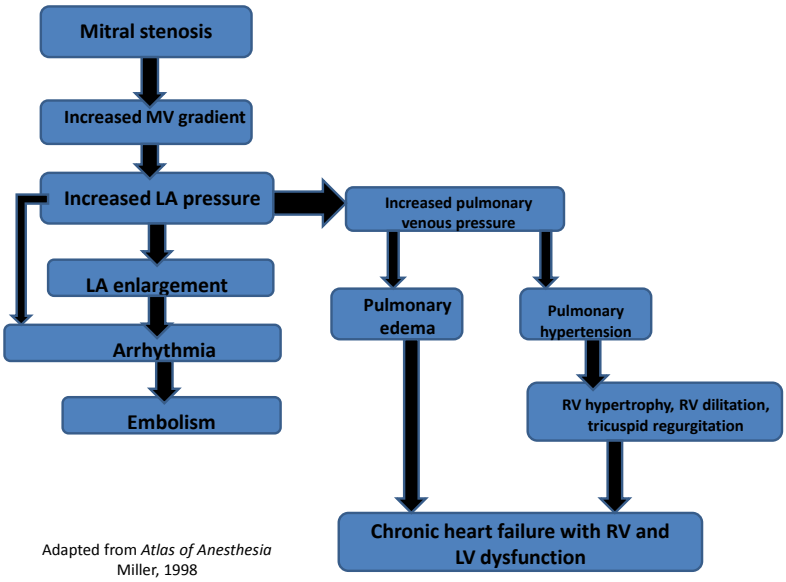


Figure XXX – Progression of Mitral Stenosis



Adapted from *Atlas of Anesthesia*
Miller, 1998

Grading Mitral Stenosis

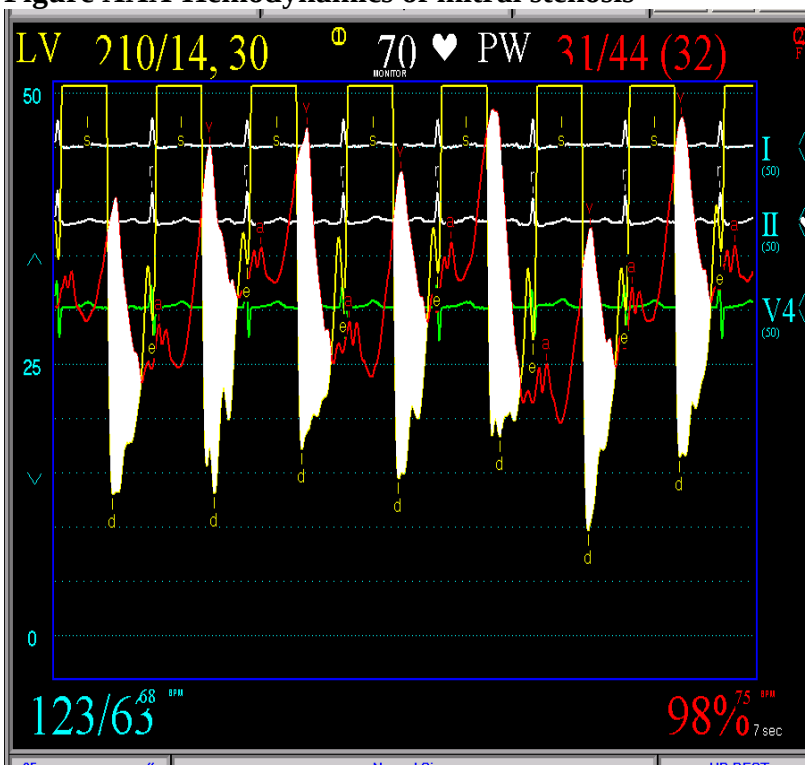
Mitral stenosis may be graded by clinical, hemodynamic, echocardiographic criteria. Clinical progression is usually associated with the decrease in orifice area (Table XXX). Moderate exercise produces symptoms in the setting of a valve area of 1.5-2.0 cm^2 (stage 1). Mild to moderate exercise or situations of physiologic stress such as anemia or pregnancy may cause symptoms when the valve area is 1.0-1.5 cm^2 (stage 2). As the valve area decreases further (less than 1 cm^2) symptoms occur even at rest (stage 3).

Cardiac catheterization may reveal a diastolic pressure gradient across the mitral valve such that the left atrial pressure will be higher than the left ventricular end diastolic pressure. Transthoracic and transesophageal echocardiography may be utilized to measure a gradient across the valve utilizing continuous wave Doppler and the Bernoulli equation. Two flow waves across the mitral valve may be noted, the first wave is referred to as the early diastolic filling wave or "E wave". The second wave is associated with the "atrial kick" and is referred to as the "A" wave. The valve orifice may be measured by two means. **Planimetry** directly measures the opening of the valve orifice while the **pressure half-time technique** is utilized to calculate the valve area mathematically.

Table XXX-Mitral Stenosis Grading

Parameter	Mild	Moderate	Severe
Stage	1	2	3
Mean pressure gradient (mmHg)	<5	6-10	>10
Pulmonary artery systolic pressure (mmHg)	<30	30-50	>50
Mitral valve area (cm^2)	>1.5	1.0-1.5	<1.0

Figure XXX-Hemodynamics of mitral stenosis



The gradient associated with mitral stenosis is between the ventricle (yellow tracing) and left atrium (red tracing) and occurs during diastole.

Additional long-term cardiovascular complications associated with mitral stenosis include pulmonary hypertension, right ventricular dilatation and potential failure, tricuspid regurgitation, and atrial fibrillation.

Clinical management

Medical management of mitral stenosis is directed at controlling the symptoms but does little to control the progression of the disease. Initial treatment starts with prevention by

appropriate **antibiotic prophylaxis.** Symptoms generally do not occur until the valve area is less than 2-2.5 cm^2 and then generally with exertion. Beta-blockers or calcium channel blockers may **limit the increase in heart rate,** a common cause of dyspnea. Atrial fibrillation occurs in 30-40% of patients and results in a decrease in cardiac output due to the loss of "atrial kick". It also is associated with a decreased survival rate. Rate control and cardioversion is the mainstay. Prevention of systemic embolization with appropriate anticoagulation is imperative when a patient is in AF. In our experience, most patients with mitral stenosis ultimately undergo **valve replacement.** Indications include moderate to severe MS with NYHA functional class III-IV heart failure symptoms or patients with less severe symptoms but severe pulmonary hypertension (PA systolic pressure > 60-80 mmHg). **Percutaneous mitral valvuloplasty** is less invasive than surgery and associated with a high success rate (80-95%) as defined by a decrease in trans mitral valve gradient and larger valve area.

Anesthetic management

Six factors considered for successful management of a patient with mitral stenosis:

1. **Balanced preload**. Patients with MS require adequate filling of the left ventricle to maintain a good cardiac output. Filling primarily occurs during diastole. High heart rates decrease the time for filling.
2. **Slow heart rate**. Patients with MS do not tolerate tachycardia or atrial fibrillation well as the "atrial kick" contributes significantly to filling of the ventricle. A slow heart rate allows appropriate filling.
3. **Maintain the current contractile state**. The left ventricle is chronically underfilled and this leads to a degree of dysfunction. Progressive pressure overload on the right ventricle leads to dysfunction with ultimate impairment of LV filling.
4. **Maintain afterload**. Afterload reduction does not help in MS and may impair perfusion to a right ventricule hypertrophied in response to elevated pulmonary pressures..
5. **Do not worsen pulmonary hypertension**. These patients are known to have pulmonary hypertension. Hypercarbia, hypoxemia, acidosis, and light anesthesia in particular will result in an exaggerated response.
6. **Be aware of co-existing valvular disorders**. Patients with MS frequently have aortic stenosis and tricuspid regurgitation.

Key Points

1. The anesthesiologist must recognize the physiologic manifestations of aortic and mitral stenosis.
2. The onset of symptomatic aortic and mitral stenosis is associated with a significantly reduced survival unless surgical treatment is initiated.
3. As a general rule, stenotic valves are generally replaced when symptoms develop.
4. Adequate preload, maintenance of a slow sinus rhythm along with adequate afterload, and avoidance of factors that exacerbate pulmonary hypertension is the key to successful anesthetic management.

5. Neuraxial anesthetic techniques may be used successfully in patients with valvular stenosis but the anesthesiologist must plan to quickly manage complication such as hypotension and profound bradycardia.

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