Cardiac Anaesthesia and Postoperative Care in the Twenty-First Century

Matias Caceres Quinones and Jacobo Moreno Garijo

Valvular Surgery

Valve heart surgery is one of most frequent and challenging procedures for the cardiovascular anesthesiologist. Different types and stages of valvular disease will influence the physiological conditions and the potential hemodynamic changes in the operating room.

During the perioperative period there are constant changes in the physiologic and hemodynamic conditions that are readily influenced by anesthesia, thus requiring a thorough understanding of the natural history and pathophysiology of valve defects.

Aortic Stenosis

History and Clinical Features

Aortic Stenosis (AS) is the most common valve lesion leading to surgery or catheter intervention in Europe and North America. It can be divided in congenital and acquired AS. The former can be classified as valvular, subvalvular and supravalvular AS based on the location. Valvular AS might occur in an unicuspid, bicuspid or tricuspid valve as a consequence of a degenerative process or secondary to rheumatic disease. Bicuspid aortic valve is found in 1–2% of the population,

M. Caceres Quinones · J. Moreno Garijo (✉)
Department of Anesthesia and Pain Medicine, University Health Network, Toronto General Hospital, Toronto, ON, Canada
e-mail: Jacobo.Moreno@uhn.ca

M. Caceres Quinones
e-mail: Matias.CaceresQuinones@one-mail.on.ca

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being the most common cause of AS below the age of 70. Clinically significant AS can develop in 2% of patients older than 65 years and this can increase to 5.5% in patients older than 85 years.

A normal aortic valve has an aortic valve area (AVA) that goes between 2.6 to 3.5 cm^2, and 2 cm^2/m^2 indexed with a rate of progression on average of a decrease of 0.1 cm^2/year. Clinical progression may manifest with classical symptoms like angina (initial symptom in two thirds of patients), syncope (first symptom in 15–30% of patients), and congestive heart failure (CHF), all signs of prognostic significance. Life expectancy when angina develops is about 5 years, 3–4 years for syncope, and 1–2 years once signs of left ventricle failure occur.[4]

Pathophysiology

The maintenance of normal stroke volume (SV) is associated with an increasing pressure gradient between the left ventricle (LV) and the aorta. This gradient results in pressure overload that triggers remodeling with compensatory concentric LV hypertrophy (Fig. 1). This induces changes in diastolic compliance with increases in left ventricle diastolic pressure (LVEDP) ultimately altering coronary perfusion pressure (CPP) and deterioration of exposing the LV to a higher risk of ischemia.

Preoperative Assessment

Echocardiography is the standard method for quantifying AS severity. This includes the measurement of AS peak jet velocity, mean transvalvular pressure gradient by Bernoulli equation and AVA by continuity equation as reflected in Table 1. Intraoperatively, transesophageal echocardiography (TEE) is the required method for the assessment of AS surgery as seen in Fig. 2.

Up to 30% of patients with severe AS may have transvalvular gradients and velocities bellow the range for severe stenosis, despite AVA < 1 cm^2. Intervention in these patients carries a higher risk of mortality and worse prognosis. The characteristics of this groups have been summarized in Table 2.

Timing for Intervention

Intervention is based upon the stage of the disease, and the stage of disease takes into account the severity and the presence or absence of symptoms. The four stages of AS as per the American Heart Association (AHA) guidelines recommendation are summarized in Tables 3 and 4. The less invasive nature of transcatheter aortic valve replacement (TAVR), as compared to surgical aortic valve replacement (AVR), has changed the landscape of patients presenting for AVR with symptomatic AS. TAVR has been proven superior to AVR even in low risk patients as

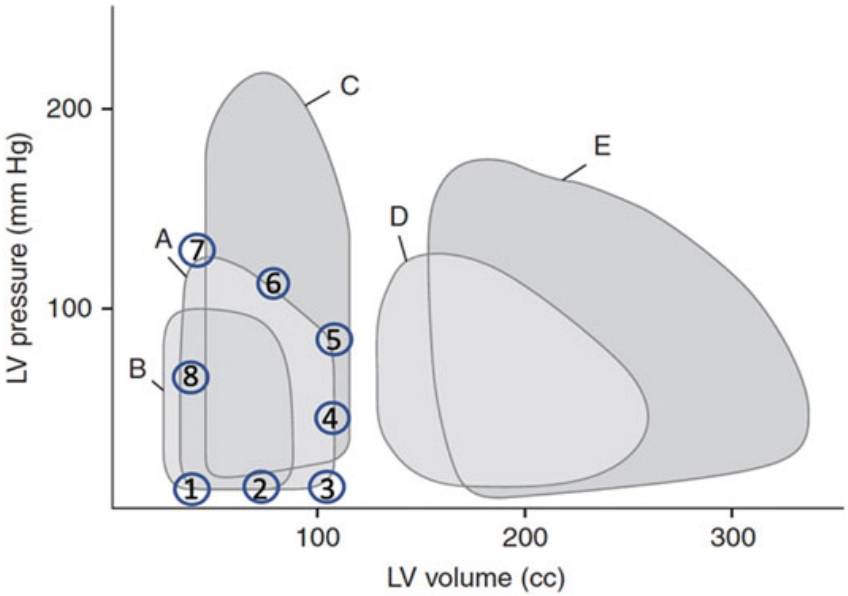


Fig. 1 Typical pressure-volume loop in a patient with AS (**c**). There is a higher peak pressure generated during systole compared to a normal LV (**a**) because of the high transvalvular pressure gradient. There is a preserved systolic function reflected in a normal stroke volume and ejection fraction. The diastolic limb is steeper, which reflects a reduced LV compliance, and thus, impaired diastolic function. Clinically, small changes in diastolic volume produce relatively large increases in ventricular filling pressure. This stiffness makes the contribution of atrial systole to ventricular filling account for up to 40% of the LV end-diastolic volume (LVEDV), rather than the 15–20% on a normal LV. 1. Mitral valve opening; 2. diastolic filling of the left ventricle; 3. mitral valve closure at end diastole; 4. isovolumetric contraction; 5. aortic valve opening; 6. ventricular ejection; 7. aortic valve closure at end systole; 8. isovolumetric relaxation of the LV. Modified from Jackson JM, Thomas SJ, Lowenstein E. Anesthetic management of patients with valvular heart disease. Semin Anesth. 1982;1:240

Table 1 AS grading as per the ASE

Parameters	Mild-moderate-severe
Peak velocity (m/s)	$< 2.6–2.9$ to ≥ 4.0
Mean gradient (mmHg)	< 20 to ≥ 40
AVA (cm^2)	> 1.5 to < 1.0
Indexed AVA ($cm^{2/}m^2$)	> 0.85 to < 0.6
Velocity ratio	> 0.5 to < 0.25

AS: Aortic Stenosis, ASE: American Society of Echocardiography, AVA: Aortic Valve Area. Modified from Baumgartner et al. *J Am Soc Echocardiogr*. 2017 Apr;30(4):372–392

per results of the PARTNER-3 and the EVOLUT low risk trial, suggesting that current guidelines might be modified in the future. Current recommendations for intervention in symptomatic AS patients are reflected in Fig. 3.

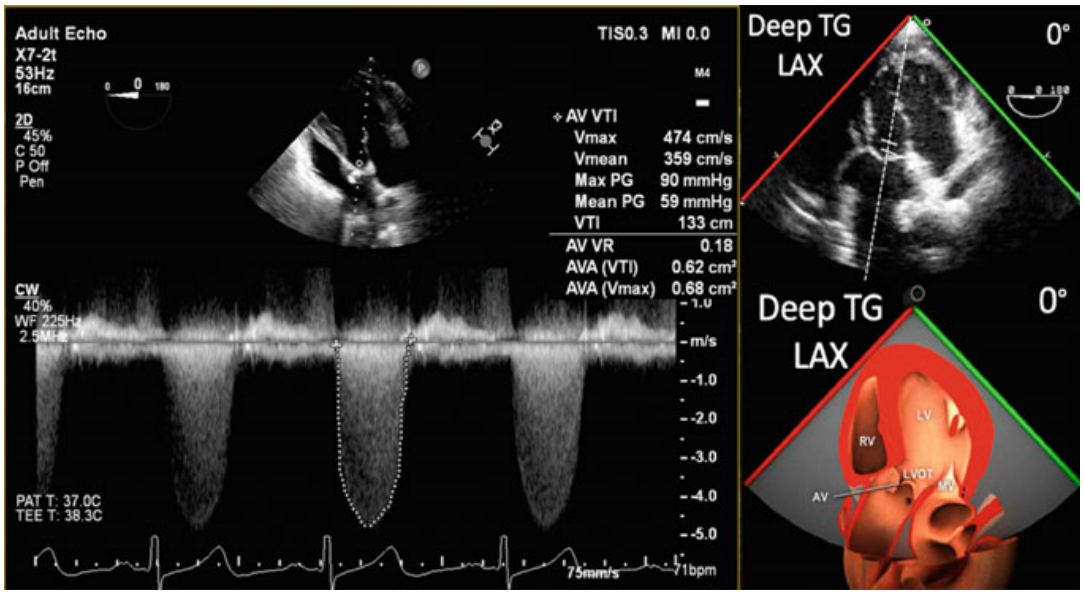


Fig. 2 Severe aortic stenosis with mean transvalvular pressure gradient (Mean PG) determined by Bernoulli equation derived from the aortic valve peak velocity (Vmax) and aortic valve area (AVA) by continuity equation (VTI). Parameters obtained from a long axis (LAX) aortic valve deep transgastric (TG) transesophageal echocardiography (TEE) view. Modified from pie.med.utoronto.ca

Table 2 Low flow, low gradient AS with reduced ejection fraction vs paradoxical low flow, low gradient AS with preserved ejection fraction

Low flow, low gradient AS with reduced ejection fraction	Paradoxical low flow, low gradient AS with preserved ejection fraction
• LV EF < 50% with a low-flow state defined as a stroke volume index (SVi) < 35 mL/m^2 along with mean gradient < 40 mm Hg, peak velocity < 4 m/s, and AVA < 1 cm^2. • Dobutamine stress echocardiography (DSE) should be performed in these cases to help differentiate two clinical situations: • Severe AS causing LV systolic dysfunction (true severe AS) • Moderate AS (pseudosevere AS) with another cause of LV dysfunction (ischemic cardiomyopathy, primary cardiomyopathy, etc.) • If the addition of inotropic support results in a mean gradient > 40 mm Hg and the calculated AVA remains less than 1 cm^2, the diagnosis of true severe AS is confirmed. • If the mean gradient remains less than 40 mm Hg, but the calculated AVA increases to greater than 1 cm^2, then a diagnosis of pseudosevere AS is made.	• The patient has a valve area < 1 cm^2 with a peak velocity <4 m/s and a mean pressure gradient <40 mmHg despite normal LVEF. • This is usually found in the setting of elderly patients with hypertrophied, small ventricles secondary to concentric remodeling and impaired diastolic filling that result in reduced transvalvular flow (SVi < 35 mL/m^2) despite normal EF. • Other more frequent reasons must be first excluded and may be more likely such as technical factors in AVA calculation. DSE is also helpful for diagnosis.

Table 3 Aortic stenosis classification

Stage (clinical)	Definition	
A	At risk of AS	
B	Progressive AS	
C	Asymptomatic severe AS	
	C1	LVEF $\geq 50\%$
	C2	LVEF > 50%
D	Symptomatic severe AS	
	D1	High Gradient AS
	D2	Low flow, Low gradient AS, LVEF < 50%
	D3	Low flow, Low gradient AS, LVEF $\geq 50\%$

Modified from Nishimura et al. *J Am Coll Cardiol*. 2014;63(22):2438–2488

Table 4 Recommendations for timing of AVR in aortic stenosis (AS)

Class I indications
• Symptomatic patients with severe high-gradient AS who have symptoms by history or on exercise testing • Asymptomatic patients with severe AS and LVEF <50% • Severe AS when undergoing other cardiac surgery
Class IIa indications
• Asymptomatic patients with very severe AS (aortic velocity ≥ 5 m/s) and low surgical risk • Asymptomatic patients with severe AS and decreased exercise tolerance or a fall in blood pressure with exercise • Asymptomatic patients with low-flow/low-gradient severe AS with reduced LVEF with low-dose dobutamine stress study showing an aortic velocity ≥ 4 m/s (mean pressure gradient ≥ 40 mm Hg) with a valve area $\leq 1 cm^2$ at any dobutamine dose • Symptomatic patients who have low-flow/low-gradient severe AS who are normotensive and have an LVEF $\geq 50\%$ if clinical, hemodynamic, and anatomic data support valve obstruction as the most likely cause of symptoms • Patients with moderate AS (aortic velocity 3–3.9 m/s) who are undergoing other cardiac surgery
Class IIb indication
• Asymptomatic patients with severe AS and rapid disease progression and low surgical risk
AVR, aortic valve replacement; LVEF, left ventricular ejection fraction. Adapted from Nishimura et al. *J Am Coll Cardiol*. 2014;63(22):2438–2488

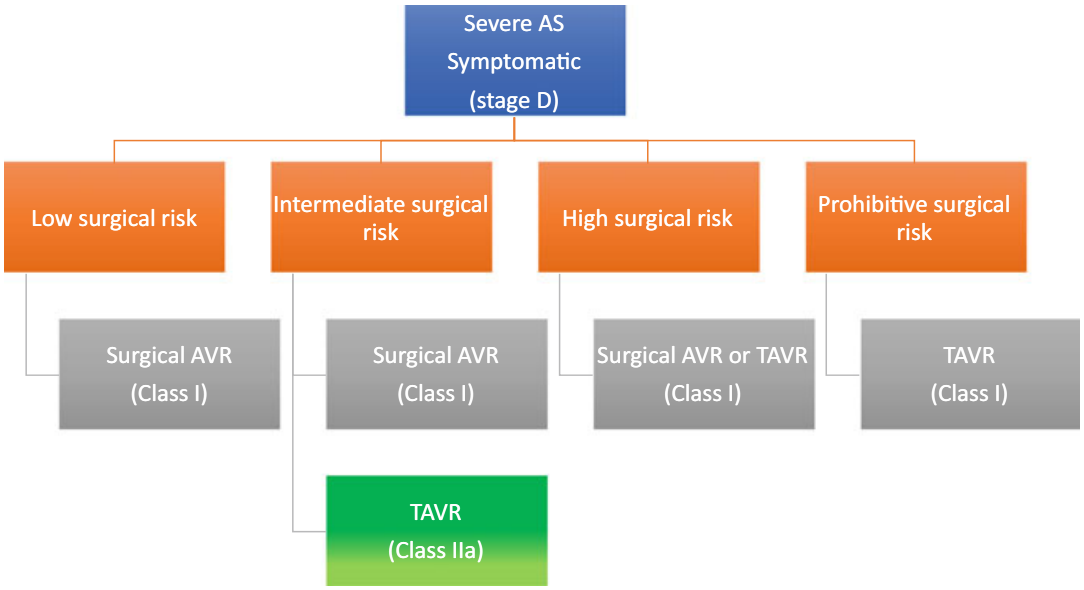


Fig. 3 Choice of TAVR versus surgical AVR in the patient with severe symptomatic AS. AS indicates aortic stenosis; AVR, aortic valve replacement; and TAVR, transcatheter aortic valve replacement. Nishimura, Rick A., et al. *J Am Coll Cardiol*. 2017; 70(2):252–289

Anesthetic Considerations

Regardless of the type of procedure, anesthetic management is based on the avoidance of systemic hypotension, maintenance of sinus rhythm, adequate rate avoiding tachycardia and bradycardia, preserving preload and contractility, and avoidance of potential myocardial ischemia due to impaired CPP and fixed forward flow. Considerations and goals for AS patient's management are summarized in Tables 5 and 6, respectively. Both, TAVR patients who receive general anesthesia and surgical patients can often be extubated shortly after arrival in the intensive care unit, as per fast-track protocols.

Hypertrophic Obstructive Cardiomyopathy (HOCM)

History and Clinical Features

Hypertrophic cardiomyopathy (HCM) is a genetic disorder with autosomal dominant inheritance and variable penetrance that affects around 0.2% of the general population (1:500 births). Around 25% of patients with HCM presents with dynamic obstruction (HOCM) to systolic outflow through the left ventricular outflow tract (LVOT) due to systolic anterior motion (SAM) of the mitral valve. The different types of HCM are summarized in Table 7 and anatomically represented in Fig. 4.

Table 5 Anesthetic considerations in AS

• The surgeon should be present during the induction period and the perfusionist ready to initiate emergency CPB in case of acute deterioration and blood must be readily available. • Premedication to reduce anxiety may be beneficial to prevent preoperative tachycardia and the potential for exacerbating myocardial ischemia due to increased gradient. Careful titration of narcotic-based anesthesia is usually chosen to meet hemodynamic goals, low concentrations of volatile anesthetics for maintenance are usually safe. • Additional to standard monitoring, five-lead ECG system with V5, large bore peripheral venous access, invasive arterial lineon pre-induction, external defibrillator pads and central line are recommended. • Pulmonary artery catheters (PACs) are helpful to assess cardiac output and mixed venous oxygen, capillary wedge pressures (PCWP) are not reliable in a non-compliant left ventricle (LV) and might overestimate LV end diastolic pressure (LVEDP).	• PACs carry the risk of inducing hypotension secondary to arrhythmias. The most conservative approach is leaving the catheter tip in a central venous position until the chest is open. • The post-bypass period is not likely to be marked by myocardial failure or low output states, and therefore PACs should be best reserved for patients with low LVEF preoperatively. • Transesophageal echocardiography (TEE) is useful for LV function, preload, and afterload assessment. Can also predict prosthetic aortic valve size based on the LV outflow tract width and AVA. Is also the method of choice for post-bypass assessment of the prosthetic valve to rule out paravalvular leaks or residual prosthetic valve gradient and assess other complications. • Myocardial function and stroke volume improve fast after the relief of the obstruction, but the hypertrophy will resolve more slowly over time, thus after surgery they still require elevated preload.

Table 6 Aortic stenosis hemodynamic goals

Preload	Increased
Contractility	Normal
Afterload	Increased
Rhythm/ Rate	Sinus rhythm, HR 50–70
Avoid	Hypotension, Tachycardia, Bradycardia and high myocardial oxygen demand situations

Table 7 HCM types

Non-obstructive	LVOT peak gradient < 30 mmHg (rest/provocation)
Obstructive (HOCM)	LVOT peak gradient $\geq$ 30 mmHg
Latent obstructive	LVOT peak gradient > 30 (provocation)

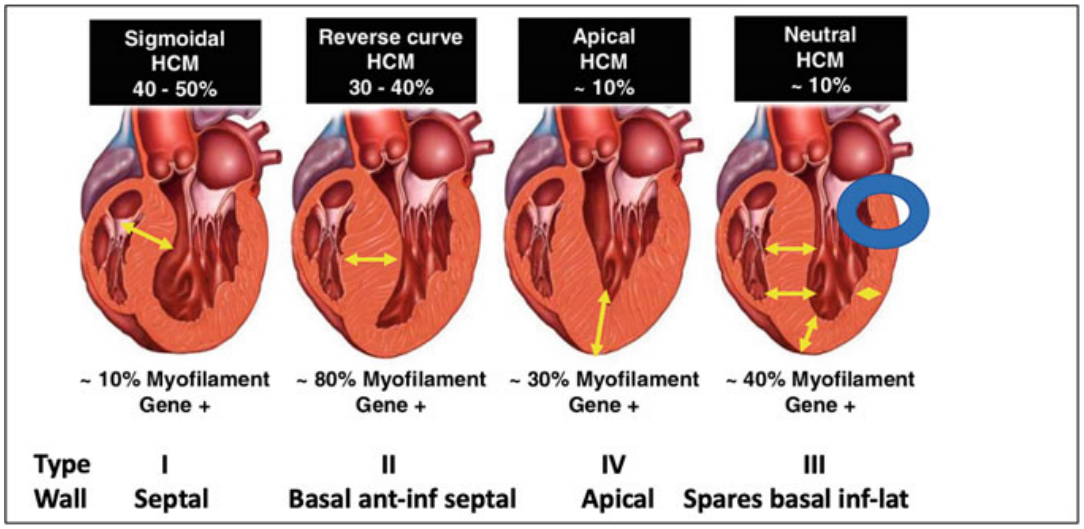


Fig. 4 Septal morphologies in HCM. Adapted from Bos et al. *J Am Coll Cardiol*. 2009;54:201–11

Clinically, it might be asymptomatic or present as dyspnea on exertion and poor exercise tolerance (most common symptom), syncope, chest pain, fatigue, or palpitations. There is no direct relationship between the degree of left ventricle outflow tract (LVOT) obstruction and the occurrence or severity of symptoms. Other symptoms might be secondary to diastolic dysfunction, dysrhythmias, mitral regurgitation (MR), and an imbalance of myocardial oxygen supply and demand. Sudden cardiac death or cardiac arrest may be the first manifestation in more than 50% of patients. The highest-risk group for sudden death include those with a family history of HCM and young patients with high physical demand (i.e., athletes), with an associated mortality of less than 1% a year.

Pathophysiology

HCM is characterized with asymmetrical hypertrophy of the myocardium without any identifiable cause for the hypertrophy, normal or even increased LV systolic function, impaired diastolic function and rarely dilated LV. Abnormal cellular architecture and interstitial fibrosis with patchy myocardial scarring contribute to impaired diastolic filling and relaxation and to derangement in the electrical tissue, putting them at risk of fatal dysrhythmias.

SAM of the anterior mitral valve leaflet (AML) is the underlying cause for dynamic outflow tract obstruction. The LV septum is narrowed by the severe hypertrophy, with further narrowing of the LVOT during systole that leads to an increase in the blood flow velocity and pressure gradient through the LVOT. There is also basal septal hypertrophy that shortens the distance between the AML and the

septum, along with hypertrophied anteriorly displaced papillary muscles and elongated mitral valve leaflets that causes a closer coaptation of the posterior mitral leaflet (PML) to the base of the AML and slack tissue extending beyond the coaptation point. Acceleration of flow through the LVOT creates a "Venturi effect" in which the hydraulic forces pulls the AML slack tissue into the LVOT generating a high gradient. Also, a drag force generated by the LV pulls the AML into the LVOT in early systole, currently being this mechanism the predominant cause of SAM. This leads to dynamic obstruction that varies with different loading conditions and contractility. This mid-late systole subaortic obstruction, increases with high contractility, higher rates, and decreases with high preload and afterload conditions. A posteriorly directed jet of MR results as a consequence of SAM, with its severity related to the degree of LVOT obstruction.

Preoperative Assessment

Echocardiography is the method of choice to assess the different types of HCM, severity and location of the obstruction and the presence of SAM. TEE exam at baseline is critical for the surgery providing different measurements to help guiding the extent of myectomy, including maximum basal septum thickness, distance from the AML impact into the septum to the AV annulus and depth of apical extent of the septal bulge as reflected in Fig. 5. SAM can be demonstrated with color flow Doppler showing a high-velocity turbulent flow (aliasing) in the LVOT. Predictor

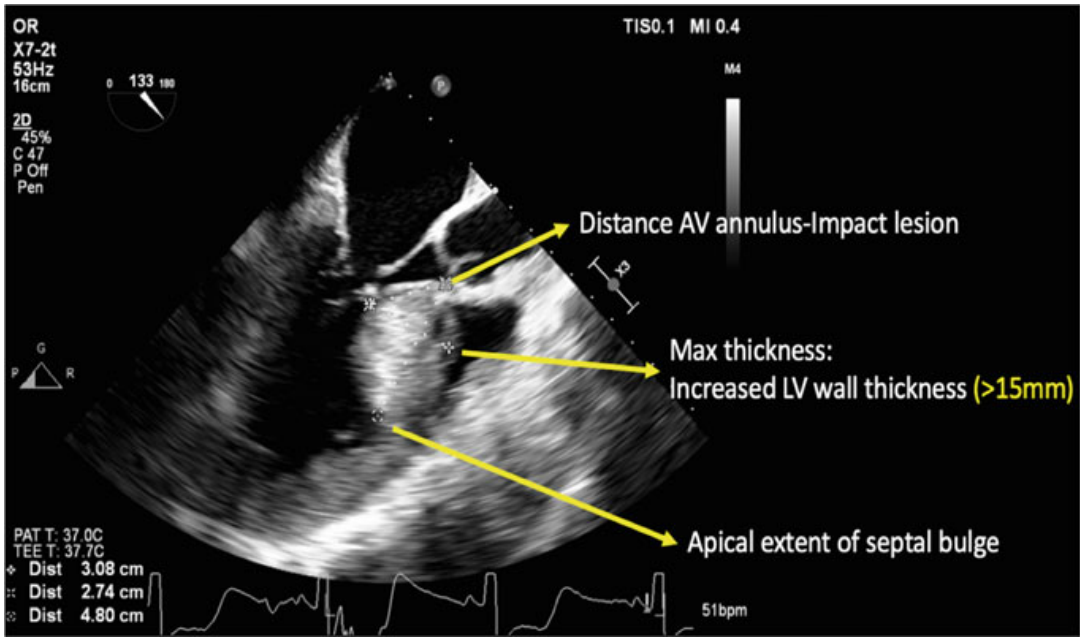


Fig. 5 TEE image of the mid-esophageal AV long axis view with the required measurements before septal myectomy in HOCM patients. AV: Aortic valve, Max thickness: maximum basal septum thickness

Table 8 Echocardiographic predictors of systolic anterior motion (SAM)

Measurement	Echocardiographic view	Time of cardiac cycle
Basal septal thickness >1.5 cm	ME LAX[a]	End diastole
C-sept distance < 2.5 cm[b]	ME LAX or ME 5 Chamber[c]	Onset of systole
PMVL length > 1.5 cm AMVL length > 2.0 cm AMVL:PMVL ratio < 1.3	ME LAX or ME 5 Chamber	Onset of systole
Mitral-aortic angle < 120°	ME LAX or ME 5 Chamber	Onset of systole

PMVL = posterior mitral valve leaflet; AMVL = anterior mitral valve leaflet
[a]Midesophageal long-axis view (ME LAX)
[b]Minimum distance from the coaptation point to the septum
[c]Midesophageal 5-chamber view
Adapted from Hymel et al. *Anesthesia & Analgesia, 118(6),2014:1197–1201.*

of SAM are summarized in Table 8. A characteristic finding of SAM due to dynamic subaortic obstruction of the LVOT is a late peaking, "dagger-shaped" of high-velocity Doppler flow due to the onset of obstruction in mid-to-late systole measured by continuous wave Doppler, as reflected in Fig. 6.

Anesthetic Considerations

The goals of management should be focused in avoiding aggravating the subaortic obstruction. It is critical to maintain an appropriate intravascular volume and prevent increases in contractility or heart rate. The considerations and goals are summarized in Tables 9 and 10.

Aortic Regurgitation

History and Clinical Features

Aortic regurgitation (AR) can be caused by a primary abnormality of the valve cusps and/or from abnormalities of the aortic root and ascending aortic geometry. About two-thirds are degenerative tricuspid and bicuspid AR. There may be also rheumatic or infectious origin, or it may occur in association with any condition producing dilatation of the aortic root and leaflet separation. A practical approach is based in a modification of the Carpentier classification for the mechanism of mitral valve regurgitation by El Khoury et al. reflected in Fig. 7.

Chronic AR remains asymptomatic for a long time during which valvular incompetence and secondary ventricular dilatation become more severe. Symptoms

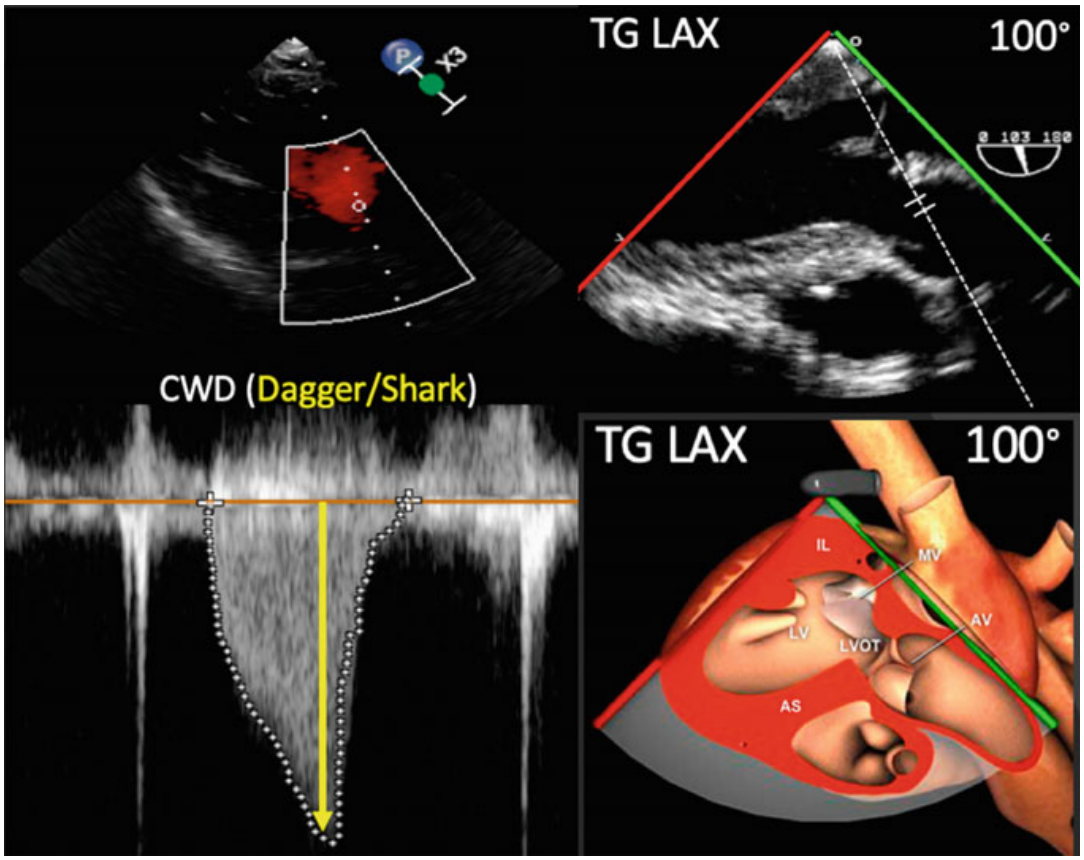


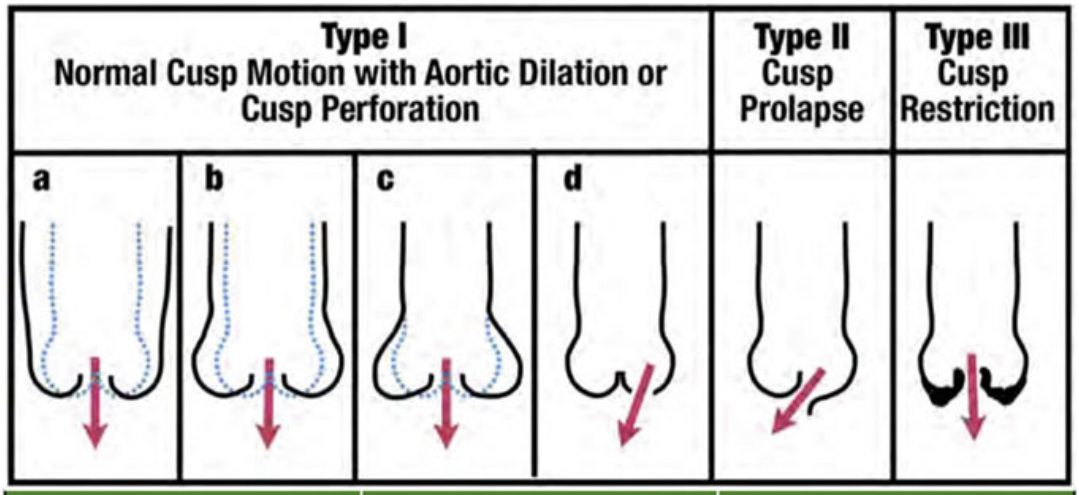
Fig. 6 Transesophageal echocardiography (TEE) long axis (LAX) transgastric view to determine dynamic subaortic obstruction of the left ventricle outflow tract (LVOT) due to systolic anterior motion (SAM) of the anterior mitral valve leaflet, by continuous wave Doppler (CWD). Modified from pie.med.utoronto.ca

Table 9 Anesthetic considerations in HOCM

• Most of patients with HOCM are treated with β-blockers to reduce LVOT obstruction due to their negative inotropic effects and reduction in heart rate and Calcium channel blockers for their effect on diastolic compliance. High risk patients for malignant dysrhythmias have an automated implantable cardioverter–defibrillator (AICD). In the preoperative period is better to keep those therapies and continue them throughout the perioperative period. • Additional to standard monitoring, five-lead ECG system with V5, large bore peripheral venous access, invasive arterial lineon pre-induction and central line are recommended. • AICDs can be deactivated once external defibrillator pads are available.	• Avoid sympathetic stimulation leading to increases in heart rate and contractility and avoid decreases in afterload. Narcotic based anesthesia along with volatile anesthesia might be of benefit due to his negative inotropic effect. • TEE allows to assess the location and extent of hypertrophy, the degree of SAM and LVOT obstruction, and degree of MR**.** It is also more reliable to accurately assess volume as opposed to CVP and PCWP since they overestimate volume status in the hypertrophied LV. More important, allows to assess the adequacy of surgical repair and possible complications. • Immediate postoperative complications of myectomy that must be ruled out include residual LVOT obstruction, residual SAM, residual MR, complete heart block, and ventricular septal defect.

Table 10 HOCM hemodynamic goals

Preload	Increased
Contractility	Normal or lower
Afterload	Increased
Rhythm/Rate	Sinus rhythm, lower rates 50–60 bpm
Avoid	Tachycardia, hypovolemia, and sympathetic stimulation



	Valve	Aorta
Acquired	Rheumatic Calcific Endocarditis Traumatic Radiotherapy Toxins	Aneurysm Aortic dissection Autoimmune (SLE, ankylosing spondylitis) Aortitis (Syphilis, Takayasu´s arteritis) Trauma
Congenital	Bicuspid Unicuspid Cuadricuspid	Annulo – aortic ectasia Connective tissue disorders (Marfan´s, Ehlers-Danlos)

Fig. 7 Common Etiologies and Classification of Aortic Regurgitation. Adapted from Zoghbi et al. J Am Soc Echocardiogr. 2017 Apr;30(4):303–371

are usually related to CHF like shortness of breath, palpitations, fatigue, and angina. They usually develop after significant dilatation and dysfunction of the LV. The risk of mortality at 10-years in asymptomatic patient is 5–15%. The prognosis after the onset of symptoms is about 10 years, and the severity and duration of those do not correlate well with the deterioration of contractility. Patients with depressed LV

Table 11 Echocardiographic assessment of aortic regurgitation

Grade	Mild-moderate-severe
Left Ventricular size	Normal to dilated
Jet deceleration rate by Pressure half-time (msec)	Slow (> 500) to Steep (< 200)
Diastolic flow reversal in descending thoracic aorta	Brief to prominent
Vena contracta width (cm)	< 0.3 to > 0.6
Jet width/LVOT width (%)	< 25 to ≥ 65
Jet CSA/LVOT CSA (%)	< 5 to ≥ 60
Regurgitation Volume (ml/beat)	< 30 to ≥ 60
Regurgitation Fraction (%)	< 30 to ≥ 50
EROA (cm^2)	< 0.1 to ≥ 0.3

CSA: cross sectional area, EROA: effective regurgitant orifice Area, LVOT: left ventricle outflow tract. Modified form Zoghbi et al. *J Am Soc Echocardiogr*. 2017;30(4):303–371

function have a higher perioperative mortality rate and higher risk for postoperative heart failure. Acute AR presents with severe symptoms like dyspnea, pulmonary edema and heart failure. Due to the lack of longstanding compensation as in chronic AR, they are not capable of maintaining sufficient forward stroke volume putting the patient at high risk of cardiovascular collapse. The assessment and grading of AR are performed by echocardiography. The parameters used are summarized in Table 11.

Pathophysiology

Left ventricular volume overload is the primary characteristic of aortic regurgitation. The degree of volume overload is determined by the magnitude of the regurgitant flow, which is related to the size of the regurgitant orifice, the aorto-ventricular pressure gradient, and the diastolic time.

Acute AR

There is a sudden major volume overload on the LV. To compensate and keep an adequate forward flow, there is an increased sympathetic tone that leads to tachycardia, an increased contractile state and fluid retention. However, this may not be sufficient to maintain a normal cardiac output and rapid deterioration of LV function can occur, emergency surgical intervention.

Chronic AR

In Chronic AR, there is gradual volume overload that leads to increased LEDV and compensatory eccentric hypertrophy that increases wall tension. The LVEDV increases slowly and the LVEDP remains relatively normal. Cardiac output and CPP is maintained at expenses of high LVEDV, peripheral vasodilation and large SV. As the dilatation progresses, coronary perfusion finally decreases leading to LV dysfunction followed by an increase in PA pressure that leads to CHF symptoms. As a compensatory mechanism for the poor cardiac output and poor coronary perfusion, sympathetic constriction of the periphery occurs to maintain blood pressure, which in turn leads to further decreases in cardiac output. These changes are summarized in Fig. 8.

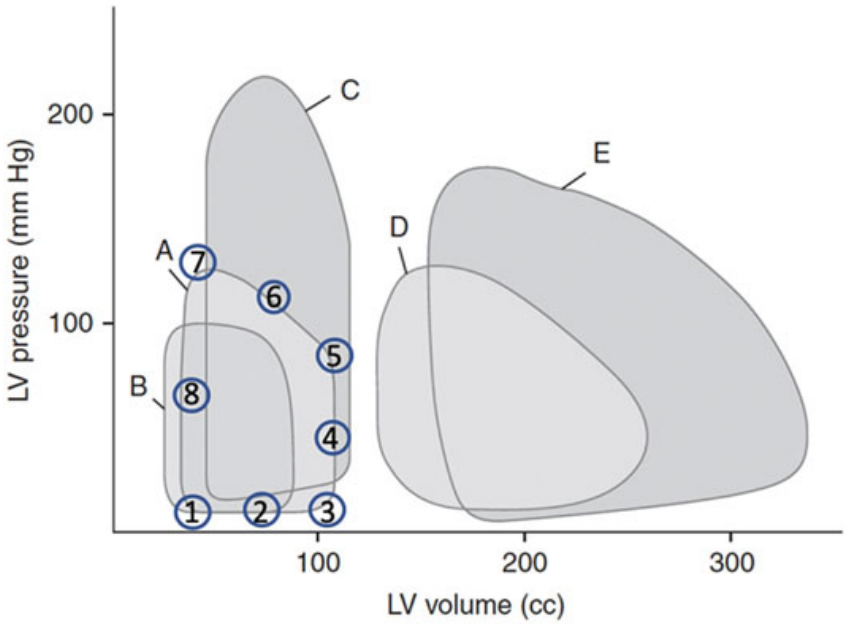


Fig. 8 Figure shows the pressure-volume loops for acute (D) and chronic (E) aortic regurgitation. In chronic AR, the pressure-volume curve is shifted far to the right. There is high diastolic compliance that allows an increase in LVEDV with minimal change in filling pressures. The increase in preload is compensated by ventricular hypertrophy and cardiac output is maintained by the Frank-Starling mechanism. Despite normal cardiac output, contractility is decreased. There is virtually no isovolumic diastolic phase and a brief isovolumic systolic phase. Eventually, with progressive increases in LVEDV, hypertrophy is no longer sufficient to compensate, and a decline in systolic function occurs. In acute AR LVEDV are also increased; however, the ventricle is not adapted to accommodate increased volumes and thus, elevation of filling pressures occurs. 1. Mitral valve opening; 2. diastolic filling of the left ventricle; 3. mitral valve closure at end diastole; 4. isovolumetric contraction; 5. aortic valve opening; 6. ventricular ejection; 7. aortic valve closure at end systole; 8. isovolumetric relaxation of the LV. Modified from Jackson JM, Thomas SJ, Lowenstein E. Anesthetic management of patients with valvular heart disease. Semin Anesth. 1982;1:240

Timing for Intervention

Acute AR is always an emergency and requires prompt surgical intervention. For chronic AR, surgical intervention is recommended in symptomatic patients with severe AR, regardless of LV systolic function. In asymptomatic patients with severe AR and LV systolic function (EF < 50%) should also undergo AVR. The asymptomatic patient with normal LV function should be closely followed clinically and with echocardiography. Surgical intervention is indicated with any sign of LV dysfunction and/or evidence of ventricular dilatation (LVEDD > 65 mm; LVESD > 50 mm). AR classification is summarized in Table 12.

Anesthetic Considerations

Patients with AR may differ widely in their degree of myocardial dysfunction and anesthetic management must be individualized. The general hemodynamic goals are a mild tachycardia, a positive inotropic state, and a controlled reduction in SVR. For the patient with acute AR, the goals are the same but in an "emergency setting" and thus, interventions should be prompt and aggressive. Anesthetic considerations and hemodynamic goals for AR are summarized in Tables 13 and 14 respectively.

Table 12 Aortic regurgitation classification

Stage (clinical)	Definition	
A	At risk of AR	
B	Progressive AR	
C	Asymptomatic severe AR	
	C1	Normal LVEF ($\geq$ 50%) and mild-to-moderate LV dilation (LVESD $\geq$ 50 mm)
	C2	Abnormal LVEF (< 50%) or severe LV dilatation (LVESD > 50 mm or indexed LVESD > 25 mm/m^2)
D	Symptomatic severe AR	

AR: aortic regurgitation, LVEF: left ventricle ejection fraction, LVESD: left ventricle end systolic diameter. Modified from Nishimura et al. *J Am Coll Cardiol*. 2014;63(22):2438–2488

Table 13 Anesthetic considerations in AR

• Light pre-medication is recommended, hemodynamic instability is less frequent during induction since most of the drugs will decrease in some degree the afterload, careful titration must be taken specially in the acute AR setting, since hemodynamic collapse is more likely to happen. • Patients with chronic AR are at risk for acute ischemia with bradycardia since it prolongs diastolic time, increases regurgitant flow, LV diastolic pressure and wall tension. • The CPP is decreased and myocardial perfusion pressure may be insufficient. All this can lead to rapid onset of heart failure and collapse. Inotropes and vasodilators should be promptly used to keep SV and CPP adequate. Pacing might also be considered to increase heart rate to above 70. • Additional to standard monitoring, five-lead ECG system with V5, large bore peripheral venous access, invasive arterial lineon pre-induction and central line are recommended.	• Pulmonary artery catheter (PAC) provides useful information since allows determination filling pressures, cardiac output and response to pharmacologic interventions. In acute AR, PCWP might underestimate LV filling pressures due to premature closing of the mitral valve caused by the regurgitant jet. • TEE is very helpful since it allows for assessment of the AR severity, LV function, preload and response to inotrope/ vasodilator therapy both during pre and post- bypass period. It is also critical to assess adequacy of surgical repair and the presence of valvular leaks and/or gradients after replacement of the valve. • Immediately after surgery there is a decrease in LVEDP and LVEDV but there might be a decline in LV function since the eccentric hypertrophy and LV dilatation takes time to improve. Inotropic or intra-aortic balloon pump support might be needed, especially in cases where surgery was not performed early enough.

Table 14 AR hemodynamic goals

Preload	Increased
Contractility	Maintain
Afterload	Decreased
Rhythm/Rate	Sinus rhythm, 70–90 bpm
Avoid	Bradycardia

Mitral Stenosis

History and Clinical Features

Clinically significant mitral stenosis (MS), more common in women than in men (2:1), can be acquired or congenital. Rheumatic disease accounts for up 10% of cases in Europe, being responsible for 99% of all MS surgeries. Congenital MS is a rare cause occurring in younger patients. Without surgery, 20% of all patients are likely to die within 1 year, and 50% die at 10 years of diagnosis.

The normal mitral valve area (MVA) is 4.0 to 6.0 cm^2 and valve index of 4 to 4.5 cm^2/m^2. Symptoms manifest with progressive decreases in valve area, of about