

MANAGEMENT OF VALVULAR HEART DISEASE - SURGICAL PERSPECTIVE

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Summary

Valves form an integral structure of the heart. Valvular deficiencies or damage comprise a wide spectrum of often lethal manifestation to heart disease that affects all ages. This chapter elucidates the four valves of the heart, its normal mechanism of function, etiology, and pathogenesis to disorder, clinical manifestation, methods of diagnosis and its treatment options.

After a brief introduction and pathophysiology of the valvular heart disease, the chapter discusses each of the individual valves – aortic, mitral, tricuspid and pulmonary, its maladies with regards to stenosis and regurgitation; and treatment with special emphasis on surgical perception to treatment modalities. A note on surgical perspective to infective endocarditis is made as well.

1. Introduction

The integrity of forward cardiac output is safeguarded by the presence of four unidirectional heart valves, which lie between chambers or between a chamber and its

outflow tract. Valvular heart disease comprises stenotic, regurgitant or mixed lesions of one or more valves. Valvular anatomical abnormalities may exist in a sub-clinical manner for many years. When clinical symptoms arise, medical management is aptly initiated. Surgical intervention is sought when medical management is not successful at controlling symptoms or when valvular disease is severe enough to warrant surgery to prevent death or progressive cardiac impairment. Occasionally, catastrophic valvular events may require earlier surgical intervention. This present chapter provides background information on valvular pathophysiology and guidelines regarding surgical management to treat valvular heart diseases.

2. Pathophysiology of Regurgitant and Stenotic Lesions

Valvular regurgitation (incompetence or insufficiency) implies leaking or retrograde flow of blood due to a diseased valve. To cope with the regurgitation, the involved ventricle (right for tricuspid disease and left for mitral disease) enlarges in order to maintain adequate forward flow. Enlargement of the affected ventricle represents a mechanism for maintaining adequate forward flow, as a good portion of the stroke volume leaks backward (Grossman, 1995). The atrium behind the leaking valve also enlarges. Regurgitant lesions represent a state of dilation or volume overload.

Valvular stenosis implies obstruction to the forward flow of blood and imposes a pressure overload on the cardiac chamber proximal to the affected valve. To cope with the increased afterload and to reduce wall stress by the law of Laplace, the left ventricle hypertrophies in the case of aortic stenosis (Setaro, 1993). In the case of mitral stenosis, the left atrium enlarges due to inadequate emptying of the atrial chamber through the stenotic mitral valve.

When valvular lesions are acute, no immediate cardiac adaptation is feasible and swift clinical decompensation may be witnessed. Even in the chronic state, ventricular dilation and/or hypertrophy exact a toll of increased myocardial oxygen demand, predisposing to ischemia and heart failure.

In broad terms, the etiology of valvular heart disease may involve either congenital defects or acquired pathology. Congenital defects may cause either stenosis or regurgitation. Description of individual congenital valvular defects and their pathophysiology would be beyond the scope of this chapter. Acquired causes of valve disease may be degenerative, rheumatic, infectious, or even traumatic or radiation-induced. These various causes can present either as stenotic, regurgitant or mixed lesions.

Clinical presentation may involve shortness of breath, lightheadedness, syncope, palpitations and chest pain. When the disease progresses further, development of leg swelling, abdominal swelling, or breathlessness even at rest can occur. Other systemic manifestations, such as a neurological event or functional impairment of other organs (especially liver or kidney) secondary to the valvular lesion may occur.

Disturbances of heart rhythm, rate, and electrical conduction often accompany valvular disease. Such phenomena can represent markers of severity of the pathologic process

and tend to complicate management because of their interference with hemodynamic function. Control of ventricular rate in atrial arrhythmias is extremely important in the face of acute or chronic valvular disease. Atrial arrhythmias and blood pooling proximal to stenotic valves tend to foster thrombus formation, with subsequent possibility of distal embolization.

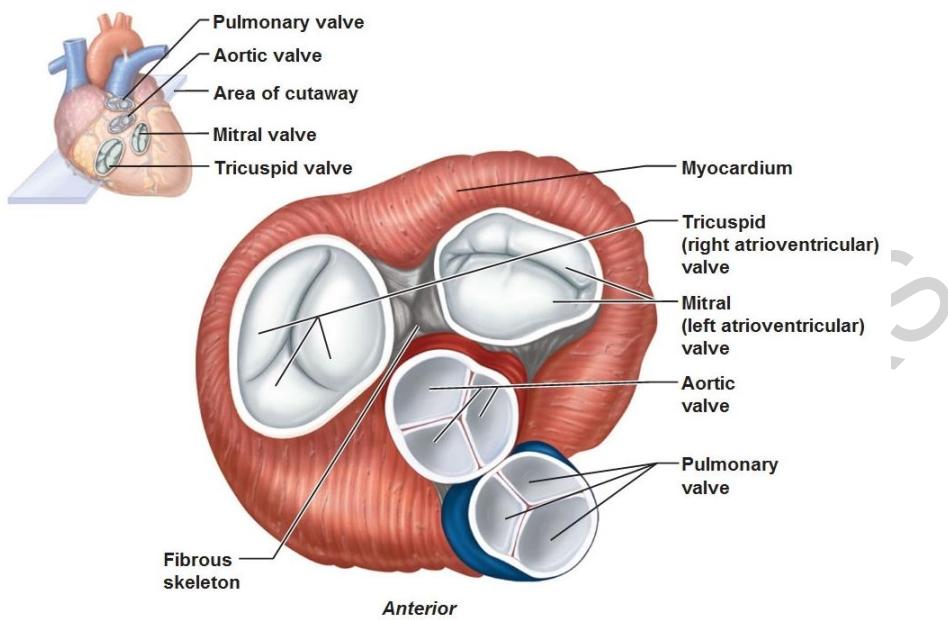


Figure 1. Fibro Skeleton of the heart and its relation to heart valves

3. Aortic Valve

The aortic valve separates the terminal part of the left ventricle outflow tract and the aorta. Its function is to maintain pressure in the aorta in diastole. The aortic valve is normally tricuspid. The aortic valve leaflets are attached to the aortic annulus within the expanded aortic sinuses. The right and the left coronary artery arise from these sinuses, termed the left and right coronary sinus and thus the leaflets are termed as left, right and non-coronary. The opening and closing of the aortic valve leaflets constitutes a passive mechanism dependent on the variation in the pressures, at different stages of the cardiac cycle between the left ventricle proximally and the aorta distally. As the pressure rises in the left ventricle during systole, the valve leaflets open to allow flow of blood to the aorta. As the ejection ceases, the aortic leaflets close in appropriate alignment to withstand and maintain the pressure of the aorta during diastole and prevent any regurgitation. Lesion of the aortic valve comprises stenosis, regurgitation and mixed lesions.

3.1. Aortic Valve Stenosis

Aortic stenosis arising due to degenerative changes from “wear and tear” of the leaflets is the most common cause for valve replacement (Passik et al, 1987). Degeneration can occur in anyone by the age of 75 or 80. Bicuspid aortic valve, which is prevalent in 2% of the population, is known to predispose to earlier degenerative changes, often by the

age of 40 or 50, if not earlier. The abnormal architecture of the bicuspid valve is believed to accelerate the wear and tear of the leaflets, leading to thickening, fibrosis, calcification, and obstruction. Bicuspid aortic valve accounts for more morbidity and mortality than all other congenital cardiac lesions combined (Friedman et al, 2008). Rheumatic aortic valve stenosis is commonly associated with mitral valve disease.

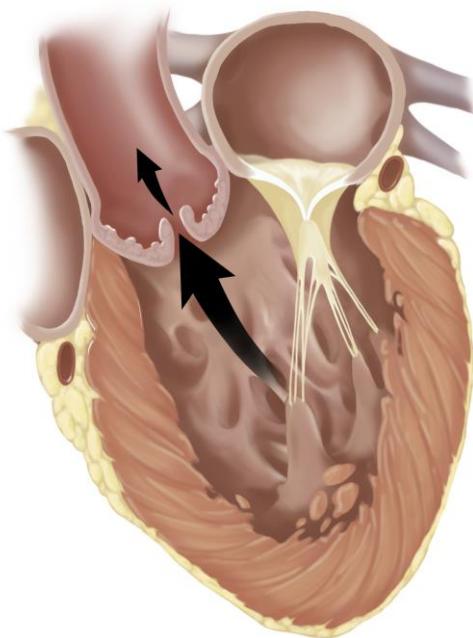


Figure 2. Sagittal section of the heart showing thickened and obstructive aortic valve.
[Used with the permission from: Elefteriades J; Cohen L. Your Heart—An Owner's Guide. Amherst, New York: Prometheus Books; 2007]

The cardinal symptoms associated with aortic stenosis are chest pain, shortness of breath and syncope. These symptoms eventually progress to heart failure if not treated (Bojar, 2011; Mihaljevic et al, 2007). The interval from the onset of symptoms to the patient's demise is usually 2 years for symptoms of heart failure, 3 yrs for syncope and 5 years in those presenting with angina, if not treated.

Findings on examination in aortic valvular stenosis include a harsh, crescendo-decrescendo murmur best heard at the right upper sternal border, radiating into the carotids and often associated with a single second heart sound. A thrill over the aortic area, a left ventricular lift, and a delayed carotid upstroke (and possible shudder), are other findings on physical examination (Mihaljevic et al, 2007). A cardiac gallop may be heard. Typically, the characteristic murmur is easily appreciated. Yet, as with any stenotic murmur, as the lesion progresses to near complete valve immobility, the murmur may become paradoxically quiet — an ominous sign of low forward flow.

The electrocardiogram in aortic stenosis often shows left ventricular hypertrophy from the chronic straining of the ventricle against the obstructed valve. So-called “repolarization changes” (T-wave inversions) are common accompaniments of the strain pattern. Conduction disturbance is not uncommon, due to calcific infiltration of the conducting system fibers where they pass in proximity to the aortic valve annulus.

Atrial fibrillation is an unfavorable rhythm in aortic stenosis, with loss of the atrial kick impairing filling of the stiff, hypertrophied left ventricle. Roentgenogram may reveal cardiomegaly, with a so-called “left ventricular contour” and may also show “post-stenotic” dilatation of the aorta. Calcification of the aortic valve may well be visualized within the cardiac contour.

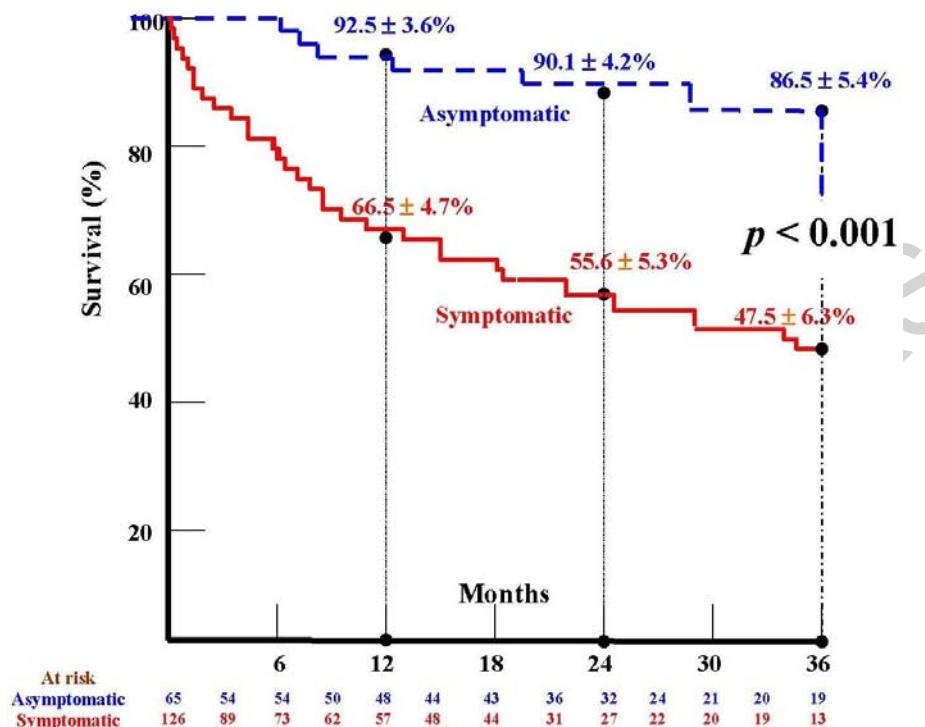


Figure 3. Kaplan-Meier survival estimates for non-operated patients with severe aortic stenosis. (The stepped curves are not as in the source but are drawn approximately to show trends. $p < 0.001$ denotes the statistically significant difference between the two Kaplan Meier curves - for survival between the asymptomatic and symptomatic patients at 36 months.) (Based on Bach et al, 2009).

Ultrasound examination of the heart is a valuable investigation and determines the morphology as well as the severity of the stenosis. Ultrasound reliably measures both the valve area and the pressure gradient across the aortic valve. The normal area of the aortic valve in adults varies from $3 - 4 \text{ cm}^2$. Current severity of aortic stenosis is graded as mild $> 1.5 \text{ cm}^2$, moderate $1 - 1.5 \text{ cm}^2$ and severe $< 1 \text{ cm}^2$ (Mihaljevic et al, 2007). We consider anything less than 0.75 cm^2 critical. One can also calculate the *aortic valve index*, or the aortic valve area divided by the body surface area (BSA). This allows one to take into account that large individuals need a larger aortic valve. An aortic valve index of $< 0.5 \text{ cm}^2$ is considered critical.

Based on mean pressure gradient, aortic stenosis is graded as mild for a mean gradient of $10 - 20 \text{ mmHg}$, moderate for $20 - 40 \text{ mmHg}$ and severe when gradient is $> 40 \text{ mmHg}$. We sometimes see peak gradients as high as 100 mmHg . In such circumstances, the left ventricle has to develop an internal blood pressure of 220 mmHg to achieve a normal systemic blood pressure of 120 mmHg beyond the aortic valve. This

consideration of the internal left ventricular pressure gives a sense of just how hard these ventricles need to work. One rarely sees a gradient of greater than 100 mmHg, probably because such circumstances are promptly lethal.

Cardiac Catheterization (Cath), Computerized Tomography (CT) and Magnetic Resonance Imaging (MRI) have also been used to assess the anatomy and severity of aortic stenosis along with left ventricular geometry.

The patient with severe or critical aortic stenosis is highly preload or volume sensitive. Low filling pressures will be inadequate to distend the stiff, hypertrophied left ventricle in diastole - leading to a depressed stroke volume and low forward output, with consequent systemic hypotension. This situation can create a downward spiral of progressive hemodynamic deterioration that may eventuate in cardiac arrest. This downward spiral leading to cardiac arrest is also commonly seen during induction of anesthesia, when loss of vasomotor tone diminishes cardiac preload. Patients with aortic stenosis are nearly impossible to resuscitate because of the "built-in" impediment to flow out of the left ventricle.

On the other hand, volume overload in patients with severe aortic stenosis will lead to pulmonary congestion, because of diastolic ventricular stiffness. Thus there exists a very narrow window of optimal filling pressures. In the setting of critical illness, a pulmonary artery catheter will be of major assistance in volume regulation. There is very little benefit that can be accomplished via administration of medications in severe aortic stenosis, where the mechanical abnormality requires a mechanical (surgical) correction (Bach et al, 2009). Vasodilators, nitrates, and even the intra-aortic balloon pump have very little role in critical aortic stenosis. In fact, nitrates are contraindicated in critical aortic stenosis, even for chest pain (usually from left ventricular strain against the stenotic valve; nitrates may lead to syncope or cardiac arrest by decreasing preload and dropping blood pressure).

Critical aortic stenosis requires prompt surgery. Apart from control of volume and heart rate manipulation as temporizing maneuvers, medical therapy has very little to offer. We prefer coronary arteriography in older patients, prior to valve replacement, in order to guide the placement of coronary artery bypass grafts in case of concomitant coronary occlusive disease.

As there is no effective medical therapy for aortic stenosis, aortic valve replacement is advised when patients are symptomatic with aortic stenosis or have severe aortic stenosis. Once symptoms occur, patient outlook, without corrective surgery, is severely compromised (Bach et al, 2009). Surgical indications for asymptomatic patients are controversial. AHA guidelines advise surgery for asymptomatic patients if valve area < 0.75 cm², mean gradient more than 40 mm Hg, or velocity of greater than 4 m/sec. (A simple rule of thumb is helpful: The gradient can be approximated as 4 times the velocity of flow; this is a result of the Bernoulli equation.)

Percutaneous aortic valvuloplasty is only of temporary utility appropriate only for very elderly patients who are not acceptable surgical candidates, or when urgent non-cardiac surgery is required; results do not last, as no amount of "stretching" of the valve by

balloon can completely undo the calcific obliteration of the valve lumen. (Block , 1998; Block and Palacios, 1990 ; McKay et al, 1991; Waller et al, 1991)

Class I

1. AVR is indicated for symptomatic patients with severe AS (*Level of Evidence: B*)
2. AVR is indicated for patients with severe AS undergoing coronary artery bypass graft surgery (CABG). (*Level of Evidence: C*)
3. AVR is indicated for patients with severe AS undergoing surgery on the aorta or other heart valves. (*Level of Evidence: C*)
4. AVR is recommended for patients with severe AS and LV systolic dysfunction (ejection fraction less than 0.50). (*Level of Evidence: C*)

Class IIa

1. AVR is reasonable for patients with moderate AS undergoing CABG or surgery on the aorta or other heart valves (*Level of Evidence: B*)

Class IIb

1. AVR may be considered for asymptomatic patients with severe AS and abnormal response to exercise (e.g., development of symptoms or asymptomatic hypotension). (*Level of Evidence: C*)
2. AVR may be considered for adults with severe asymptomatic AS if there is a high likelihood of rapid progression (age, calcification, and CAD) or if surgery might be delayed at the time of symptom onset. (*Level of Evidence: C*)
3. AVR may be considered in patients undergoing CABG who have mild AS when there is evidence, such as moderate to severe valve calcification progression may be rapid. (*Level of Evidence: C*)
4. AVR may be considered for asymptomatic patients with extremely severe AS (aortic valve area less than 0.6 cm^2 , mean gradient greater than 60mmHg, and jet velocity greater than 5.0 m per second) when the patient's expected operative mortality is 1.0% or less. (*Level of Evidence: C*)

Class III

1. AVR is not useful for the prevention of sudden death in asymptomatic patients with AS who have none of the above.

ACC – American College of Cardiology, AHA – American Heart Association, AVR – Aortic Valve Replacement, CABG – Coronary Artery Bypass Grafting, AS – Aortic Stenosis, LV – Left Ventricle, CAD – Coronary Artery Disease
(Nishimura et al. 2008)

Table 1. ACC/AHA Guidelines for Indications for Aortic Valve Replacement in Aortic Stenosis (Bonow et al, 2008)

Replacement of the valve with a biological or mechanical prosthesis is essential if the dismal natural history of critical aortic stenosis is to be averted. Life expectancy following surgery is restored. Percutaneously delivered aortic valves are under clinical investigation in the United States, and may represent another option for extremely elderly or frail patients in the future (Miller et al, 2012).

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Biographical Sketches

Bijoy G Rajbanshi, from Nepal, received his medical and initial cardiovascular thoracic training from Jawaharlal Nehru Medical College, Belgaum, India and National Institute of Cardiovascular Disease, Dhaka, Bangladesh. He joined as a registrar and junior staff at the National Heart Center in Kathmandu, Nepal and worked there for four years. He had advanced training as a Clinical Fellow in cardiovascular surgery at the Mayo Clinic in Rochester, Minnesota, USA from 2010 to June, 2012. Presently, he is doing an advanced clinical fellowship in adult cardiac surgery at Aortic Institute at Yale University and Yale New Haven Hospital, USA. His main research interest is focused on aortic diseases and surgical advances in the treatment of disease of the aorta. He is presently working at the aortic institute in a number of clinical research projects related to aortic disease and adult cardiac surgery.

John A. Elefteriades, is William W.L. Glenn Professor of Cardiothoracic Surgery and Director of the Aortic Institute at Yale University and Yale New-Haven Hospital. He served as Chief of Cardiac Surgery from 1995 to 2011, when he became Founding Director of the Aortic Institute at Yale-New Haven. He is among the most clinically active academic surgeons in the country.

Dr. Elefteriades graduated magna cum laude with a triple major in Physics, French, and Psychology from Yale University. He received his MD degree from the Yale University School of Medicine. He trained at

Yale in both general surgery and cardiothoracic surgery. After completing his training, he joined the faculty at the Yale University School of Medicine.

He performs all aspects of adult cardiac and thoracic surgery. He is a recognized authority in interventions for the failing left ventricle, including coronary artery bypass grafting, left ventricular aneurysmectomy, and artificial heart implantation. Dr. Elefteriades directs the Aortic Institute at Yale-New Haven Hospital, one of the nation's largest facilities for treatment of the dilated thoracic aorta. He conducts laboratory research in new techniques of heart transplantation. Among his research projects, he is working with Celera Diagnostics to identify the genetic mutations responsible for thoracic aortic aneurysms. Dr. Elefteriades' research work into brain and spinal cord protection via hypothermia has been supported by grants from the National Science Foundation.

At his Aortic Institute, Dr. Elefteriades and his team have achieved many milestones in the understanding and treatment of aortic diseases, including: recognition of the genetic nature of thoracic aortic aneurysm, characterization of the natural history of thoracic aortic aneurysm, determination that physical and emotional stress can precipitate aortic dissection, identification of the role of MMPs in the pathogenesis of thoracic aortic aneurysms, determination of the appropriate size for surgical resection, and identification of the correspondence of mechanical deterioration of the aortic wall with clinical behavior. His team has also developed an "RNA Signature" blood test for aneurysm disease.

Dr. Elefteriades serves on multiple scientific advisory and editorial boards. He is a past President of the Connecticut Chapter of the American College of Cardiology and member of the national Board of Governors of the College. He is also past President of the International College of Angiology. He serves on the editorial board of the *American Journal of Cardiology*, the *Journal of Cardiac Surgery, and Cardiology*. He is a member of the Thoracic Surgery Director's Association and has been named consistently in The Best Doctors in America. He is a frequently requested international lecturer, visiting professor, and guest surgeon. He is the author of over 300 scientific publications on a wide range of cardiac and thoracic topics. He was selected as one of the ten best doctors in America by *Men's Health* magazine. He has been featured in many dozens of print, radio, and television presentations. He has been awarded the Walter Bleifeld Memorial Award for Distinguished Contribution in Clinical Research in Cardiology, and the John B. Chang Research Achievement Award. In 2005 he was selected to deliver a lecture for the Leadership in Biomedicine Series at the Yale University School of Medicine. In 2006, he received the Socrates Award from the Thoracic Residents Association, Thoracic Surgery Directors' Association, and the Society of Thoracic Surgeons, recognizing exceptional achievement in teaching and mentorship of residents. In 2008, he was honored by the International Academy of Cardiology, with a Distinguished Fellow Award. In 2011, he received the AHEPA award for Achievements in Medicine. In 2012, he was selected as one of six "Stars" to lecture at the Reunion of his Yale University undergraduate class.

He was named the William W.L. Glenn Professor of Cardiothoracic Surgery in 2006. This endowed chair honors the memory of Dr. Elefteriades' mentor, Dr. Glenn. Dr. Elefteriades is the author of the books *House Officer Guide to ICU Care (1st, 2nd, and 3rd Editions)*, *Advanced Treatment Options for the Failing Left Ventricle*, *Diseases of the Aorta*, *Your Heart: The Owner's Guide*, *The Woman's Heart Book*, and *Acute Aortic Disease*, and *Controversies in Diseases of the Aorta*. He recently published his first fiction work, the medical mystery/ethics thriller *Transplant*, which was a finalist in the Next Generation Indie Book Awards as a best first novel. Dr. Elefteriades was featured in a documentary of the BBC Horizons television program for his innovative techniques applying deep hypothermia to surgery of the aortic arch. His hypothermia work has also been featured on the Italian RAI channel. His is featured again for hypothermia in a recently aired segment of the Science Channel's acclaimed program *Through the Wormhole with Morgan Freeman*.