Healthy Heart

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From the desk of Honorary Editor:

The cardiac valves have two functions. By opening, they control the direction in which blood flows and, by closing, they allow pressure differentials to exist in a closed system. Abnormal valve function produces either pressure overloading caused by restricted opening or volume overloading caused by inadequate closure. Valvular heart disease can be approached on the basis of the pathologic lesion-aortic stenosis or aortic regurgitation-or pathophysiologically, as pressure overloading versus volume overloading.

In this chapter, I summarize my current approach to aortic valve disease, aortic stenosis and regurgitation, with particular emphasis on the indications for valve surgery. In general, I adhere to the recommendations given by the American College of Cardiology-American Heart Association (ACC/AHA) 2012 guidelines for the management of patients with valvular heart disease.

- Dr. Dhaval Naik

Aortic valvular disease & current management approaches

Aortic Stenosis

Definition

Aortic stenosis refers to obstruction of flow at the level of the aortic valve and does not include the subvalvular and supravalvular forms of this disease. Aortic valve stenosis is usually defined by restricted systolic opening of the valve leaflets, with a mean transvalvular pressure gradient of at least 10 mm Hg. The cause of the stenosis can be further defined based on the anatomy and disease process affecting the valve. Calcific aortic stenosis ,congenital bicuspid aortic valve (Fig. 1) stenosis and rheumatic aortic stenosis account for the overwhelming majority of aortic stenosis cases.

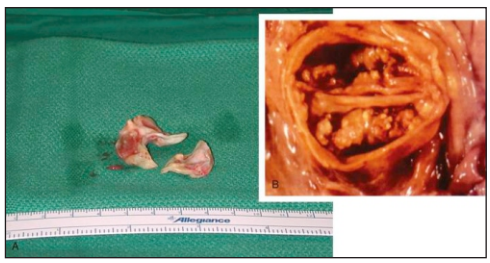

Figure 1 : Calcific Aortic Valve

Pathophysiology

Valvular aortic stenosis results in chronic left ventricular pressure overloading. At any stage of life, however, the natural history of aortic stenosis largely reflects the functional integrity of the mitral valve. As long as adequate mitral valve function is maintained, the pulmonary bed is protected from the systolic pressure overloading imposed by aortic stenosis. In contrast to mitral valve disease, in which the pulmonary circuit is directly involved, compensatory concentric left ventricular hypertrophy allows the pressure-overloaded ventricle to maintain stroke volume with modest increases in diastolic pressure, and patients remain asymptomatic for many years.

Eventually, however, left ventricular hypertrophy causes either diastolic dysfunction with the onset of congestive symptoms or myocardial oxygen needs in excess of supply with the onset of angina. Some patients might also experience exertional syncope, probably reflecting the inability to increase cardiac output and maintain blood pressure in response to vasodilation.

Signs and Symptoms

The onset of any of the classic symptoms of left ventricular outflow obstruction—angina, syncope, or heart failure—in a patient with valvular aortic stenosis indicates advanced valve disease and should be carefully and promptly evaluated.

On physical examination, the harsh systolic murmur of aortic stenosis, loudest at the base of the heart and radiating to the carotids, is often but not always prominent.The murmur may

radiate toward the cardiac apex, in which case the harsh component is lost; this finding may be mistaken for a second murmur. Other hallmarks of significant aortic valve stenosis include a single (pulmonic) component of the second heart sound and a sustained left ventricular apical impulse with a fourth heart sound. The slowly rising, low-volume carotid arterial pulse of severe aortic stenosis may be noted in younger patients, but changes in arterial compliance often mask these findings in older adults.

Diagnosis

The electrocardiogram often shows changes of left ventricular hypertrophy. The chest radiograph is seldom helpful, although occasionally heavy calcification of the valve or ascending aortic dilation may be seen. With their widespread availability, two-dimensional and Doppler echocardiography have become the tests of choice in the evaluation of patients with suspected valvular disease. Echocardiography allows assessment of the valve anatomy as well as of chamber size and ventricular function. Doppler studies permit estimation of pressure gradients and estimations of aortic valve area by using the continuity equation. Preoperative coronary angiography is indicated in men older than 35 years, women older than 35 years with risk factors, and all postmenopausal women, to exclude coronary artery disease.

Treatment

Patients with aortic stenosis fall into one of four categories of severity: mild, moderate, severe, or critical (Table 1). Current evidence indicates that calcific aortic stenosis progresses, on the average, at a rate of about 0.1 cm^2 per year decline in valve area. Asymptomatic patients should have an echocardiographic re-evaluation every 2 to 3 years for mild aortic stenosis, every 1 to 2 years for moderate stenosis, and every 6 to 12 months for severe stenosis. Patients with moderate to severe asymptomatic aortic stenosis should avoid strenuous or competitive activity, particularly postprandial exertion.

Table 1: Classification of Aortic Stenosis

Severity	Valve Area (cm2)	Maximum Aortic Velocity (m/sec)	Mean Pressure Gradient (mm Hg)
Mild	1.5-2.0	2.5-3.0	<25
Moderate	1.0-1.5	3.0-4.0	25-40
Severe	0.6-1.0	>4.0	>40
Critical	<0.6	-	-

Symptomatic patients (i.e., those with angina, syncope, or dyspnea) with severe aortic stenosis should undergo valve replacement (Class I indication). Additional indications for aortic valve surgery include patients with severe aortic stenosis undergoing coronary artery bypass grafting or surgery on the aorta or other heart valves (Class I indication); patients with severe aortic stenosis and left ventricular ejection fractions less than 0.50 (Class I indication); and patients with moderate aortic stenosis undergoing coronary artery bypass grafting or surgery on the aorta or other heart valves (Class IIa indication). Aortic valve surgery may be considered in asymptomatic patients who exhibit an abnormal response to exercise (e.g., drop in blood pressure, abnormal symptoms, or poor functional capacity; Class IIb indication). The preoperative evaluation should address any major comorbid conditions and optimize their management. A carotid duplex examination should be performed, because distinguishing a carotid bruit from a radiating murmur is difficult clinically. Coronary angiography is indicated to evaluate the need for coronary revascularization, because about one half will have significant coronary disease as indicated by the preoperative angiogram.

The consulting cardiologist, cardiac surgeon, or both should discuss the advantages and drawbacks of mechanical versus bioprosthetic valves (Fig. 2) with the patient and family during the presurgical evaluation. Often, the choice of prosthesis is straightforward, but younger patients in particular may have special needs, which should be addressed. Bioprosthetic valves offer the advantage of not requiring long-term oral anticoagulation, but have the drawback of relatively limited durability. In contrast, mechanical valves offer long-term durability but require lifelong warfarin therapy. The generally accepted risk of serious bleeding with warfarin is about 1% to 2% per year. Childbearing age (in women) and engaging in vigorous sports activities are factors that are relative contraindications to chronic oral anticoagulation with warfarin and may influence the choice of valves. In general, I favor bioprosthetic valves in patients older than 60 years and mechanical valves in those younger than 50 years. For male patients in their 50s, clinical outcomes with bioprosthetic valves are good; in this group, current estimates place the likelihood of reoperation for late (after 10 years) deterioration of a bioprosthesis at about 1 in 10, using competing outcomes analysis. In contrast, healthy women in their 50s should probably receive mechanical valves, because many can expect another 30 years of life. Homograft aortic valve

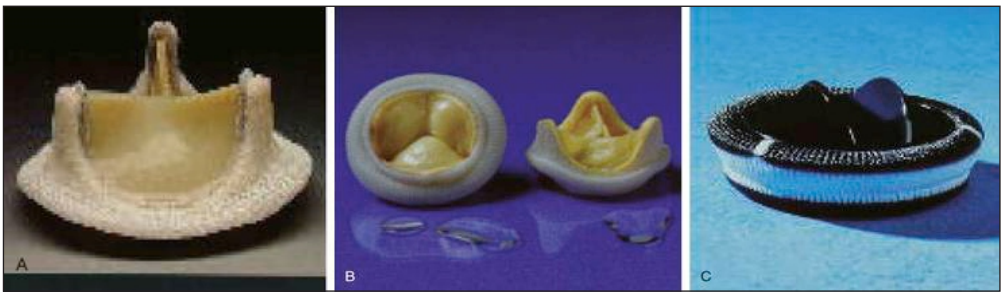

Figure 2 : Various prosthetic valves

replacement with a cryopreserved cadaveric valve may offer specific advantages for patients with infective endocarditis or diseases of the aortic root.

Reoperation may be required for malfunction of the prosthetic valve. In addition, a small but not insignificant subset of patients may require implantation of a permanent pacemaker after aortic valve surgery. Patients should clearly indicate their willingness to accept the limitations that valve replacement imposes before surgery. In addition, patients must understand that surgical risks include wound infection and stroke, as well as perioperative mortality.

Aortic regurgitation

Definition

Aortic regurgitation is defined by incompetence of the aortic valve, in which a portion of the left ventricular forward stroke volume returns to the chamber during diastole. The cause of the regurgitation, as for aortic stenosis, can be further defined based on the anatomy of the valve and aortic root and the disease process affecting the valve.

Causes and Pathophysiology

Aortic regurgitation can occur because of leaflet pathology or aortic root disease. As an isolated lesion, aortic regurgitation usually occurs because of a congenital bicuspid aortic valve, often resulting from leaflet prolapse. Infective endocarditis involving the aortic valve may result in aortic regurgitation because of loss of coaptation, leaflet retraction, or perforation (Fig. 3). However, any pathologic process that results in aortic root dilation and loss of leaflet coaptation can also result in aortic regurgitation. Examples include diseases of the aortic root, such as annuloaortic ectasia (Fig. 4), long standing hypertension, familial aortic aneurysmal disease, and hereditable diseases of connective tissue, such as Marfan syndrome. Additionally, ascending aortic dissections and congenital diseases, such as ventricular septal defects as seen in tetralogy of Fallot, can lead to aortic regurgitation. Other less common conditions include radiation heart disease, Ehlers-Danlos syndrome, and inflammatory aortitis and/or aortic valvulitis caused by giant cell aortitis, reactive arthritis, syphilitic aortitis, ankylosing spondylitis, and rheumatoid arthritis.

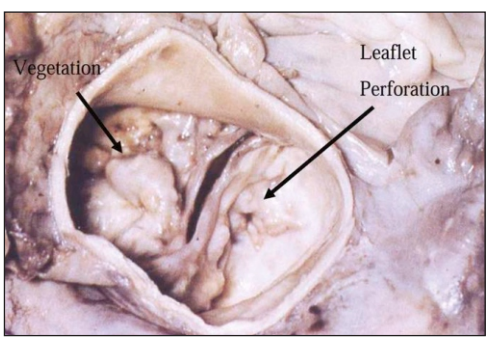

Figure 3 : Infective Endocarditis

Regardless of cause, chronic aortic regurgitation results in volume overloading of the left ventricle and, in contrast to mitral regurgitation, also causes a component of pressure overload. The volume overload usually is well tolerated for long periods, possibly even decades. The sequelae of aortic regurgitation reflect the severity of the diastolic leak; these include left ventricular dilation and hypertrophy, with remodeling of the left ventricle to a more spherical shape. The ejection fraction usually is preserved until the late stages of the disease.

Because patients may tolerate severe aortic regurgitation with minimal symptoms, management should include careful monitoring of left ventricular dimensions and systolic function. In addition, because aortic root and proximal ascending aortic dilation can coexist, careful monitoring of aortic enlargement is warranted in these patients. Surgical intervention is indicated, even in asymptomatic individuals, when left ventricular dilation reaches critical dimensions or ventricular dysfunction occurs.

Signs and Symptoms

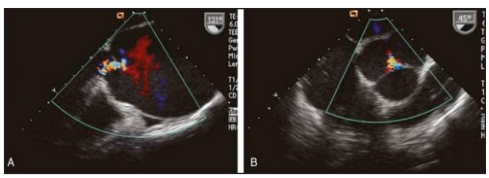

Figure 4 : Annuloaortic Ectasia

Symptoms of aortic regurgitation often begin with nonspecific fatigue. Patients might relate that their ability to get through a day's work is maintained, but they are exhausted after returning home. Palpitations, or awareness of a forceful heartbeat, is an early complaint. With further progression, typical heart failure symptoms follow. Angina pectoris and syncope are much less common with aortic regurgitation than with aortic stenosis. In contrast, palpitations and

ventricular premature beats are much more frequent, and nonsustained ventricular tachycardia has often been reported. Overt heart failure and cardiac chest pain are infrequent but, if present, may reflect a more acute process.

Careful physical examination may yield a host of eponymous signs (e.g., Hill's sign, Corrigan's pulse), almost all of which reflect a high stroke volume and wide pulse pressure. The wide pulse pressure, bounding arterial pulses, and hyperdynamic circulation of chronic moderately severe aortic regurgitation are easily noted. In contrast, the soft, blowing, diastolic murmur may be subtle, requiring careful auscultation, with the patient sitting forward in fully held expiration. The murmur is almost always best heard using the diaphragm of the stethoscope applied firmly to the upper right parasternal area of the anterior chest. A systolic murmur may be audible because of increased stroke volume. The duration of the diastolic murmur should be noted, because this reflects the severity of the leak until the late stages of disease, when the left ventricular diastolic pressure increases and shortens the diastolic murmur. An Austin-Flint apical diastolic murmur may also be present. This mid-diastolic murmur, best heard at the apex and often preceded by an S3 heart sound, occurs in the absence of organic mitral valve disease. It is likely the result of an antegrade flow across an incompletely opened mitral valve caused by the aortic regurgitant jet's effect on the anterior mitral leaflet. An Austin-Flint murmur usually indicates significant aortic regurgitation.

Diagnosis

The electrocardiogram of patients with aortic regurgitation commonly demonstrates generous voltage and upright T waves in the lateral chest leads have been referred to as "volume overload left ventricular hypertrophy." In addition, premature ventricular contractions may be present.

Echocardiography will, in almost all cases, define the functional anatomy of the valve and aortic root, and Doppler imaging will help assess the severity of the diastolic leak . In addition, the echocardiogram documents left ventricular diastolic dimensions, ejection fraction, and wall thickness. If transthoracic echocardiographic imaging is not adequate to define the pathoanatomy, transesophageal echocardiography should be performed. The anatomic consequences of aortic regurgitation include, as noted above, both left ventricular hypertrophy and dilation. Serial echocardiographic measurements of left ventricular systolic function and end-diastolic dimensions provide excellent objective parameters for long-term follow-up of asymptomatic patients.

Diagnostic coronary angiography should be performed as part of the presurgical evaluation when valve repair or replacement is planned.

Treatment

Asymptomatic patients with chronic severe aortic regurgitation and normal left ventricular systolic function should be assessed clinically and echocardiographically approximately every 6 to 12 months. Current guidelines suggest aortic valve surgery for chronic severe aortic regurgitation for patients with symptom onset (Class I indication), asymptomatic patients with left ventricular ejection fraction lower than 0.50 (Class I indication), patients undergoing coronary artery bypass grafting or surgery on the aorta or other heart valves (Class I indication), and patients with preserved ventricular function but left ventricular end-systolic dimension more than 55 mm or end-diastolic dimension more than 75 mm (Class IIa indication). Aortic valve surgery may be considered in asymptomatic patients with preserved ventricular function but left ventricular end-systolic dimension more than 50 mm or end-diastolic dimension more than 70 mm, patients with declining exercise tolerance, and patients with moderate aortic regurgitation undergoing coronary artery bypass grafting or surgery on the aorta or other heart valves (Class IIb indications).

Summary

- Calcific aortic stenosis(rheumatic) and congenital bicuspid aortic valve stenosis account for most aortic stenosis cases.
- Two-dimensional and Doppler echocardiography represent the gold standard in the evaluation of patients with suspected aortic valvular disease.
- Symptomatic patients with severe aortic stenosis should undergo valve replacement, as well as those with severe aortic stenosis undergoing cardiac surgery, severe aortic stenosis and left ventricular dysfunction, and moderate aortic stenosis undergoing cardiac surgery.
- Chronic aortic regurgitation may be caused by leaflet pathology, such as a congenital bicuspid aortic valve, or may be related to any pathologic process that results in aortic root dilation.
- Aortic valve surgery for chronic severe aortic regurgitation is indicated for those with symptom onset, asymptomatic patients with left ventricular dysfunction, patients undergoing cardiac surgery, and patients with preserved ventricular function but a left ventricular end-systolic dimension more than 50 to 55 mm or end-diastolic dimension more than 70 to 75 mm.

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