

Aortic Stenosis in the Elderly

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Since the incidence of aortic stenosis increases with age, physicians are likely to encounter this valvular disorder with greater frequency as populations continue to age. This paper provides a comprehensive overview of the etiology, natural history, pathophysiology, diagnosis, and management of aortic stenosis in the elderly. Echocardiography is the diagnostic modality of choice, suitable for the serial assessment of disease progression. Cardiac catheterization should be reserved mainly for the evaluation of possible concomitant coronary artery disease prior to cardiac surgery. Aortic valve replacement represents the only proven treatment modality for symptomatic, hemodynamically significant aortic stenosis. Advanced age is not a contraindication for surgery, and valve replacement can be performed in any patient with an acceptable surgical risk. (AJGC. 2000;9:321-329,345)

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Valvular aortic stenosis represents a family of related disorders in which left ventricular emptying is impeded due to progressive narrowing of the aortic valve orifice. The disease is characterized by two clinical stages: latent and symptomatic. During the latent stage, which may last decades, there is a progressive rise in the pressure gradient across the aortic valve, with no apparent clinical manifestations. In the symptomatic stage, which may last several years, three hallmarks of the disease develop: angina pectoris, syncope, and congestive heart failure. Once symptomatic, severe aortic stenosis is usually fatal in the absence of surgical correction. Because the disease has a very long natural course, and as the population in industrialized countries continues to age, aortic stenosis in the elderly will become more important.

ETIOLOGY

Calcific degeneration of either the tricuspid or bicuspid aortic valve accounts for most aortic stenosis cases in the elderly. Postinflammatory (including rheumatic) forms of aortic stenosis are becoming less common in industrialized countries (Figure).

Senile Calcific Aortic Stenosis. In the elderly, calcification of an apparently normal tricuspid aortic valve is the most important cause of aortic stenosis. The condition is usually referred to as senile calcific aortic stenosis. The older the patient, the higher the likelihood that the aortic stenosis is due to calcific degeneration of the tricuspid aortic valve. In individuals aged 70 years and older, this condition accounts for about half of all cases of aortic stenosis. Because even among octogenarians the overall prevalence of aortic stenosis is about 20%,¹ there must be additional agents that cause calcific aortic valve degeneration in the elderly.

Preexisting valve abnormalities seem to work in concert with calcification-enhancing processes (such as atherosclerosis, end-stage renal disease, or Paget's disease) to produce calcific aortic stenosis. Postmortem studies have shown that in some patients the tricuspid aortic valve may have slight congenital irregularities, such as unequal cusp and/or commissure size, and these patients may be over-represented in cohorts with aortic stenosis compared with the general population.² The notion that calcific aortic stenosis in some cases may be

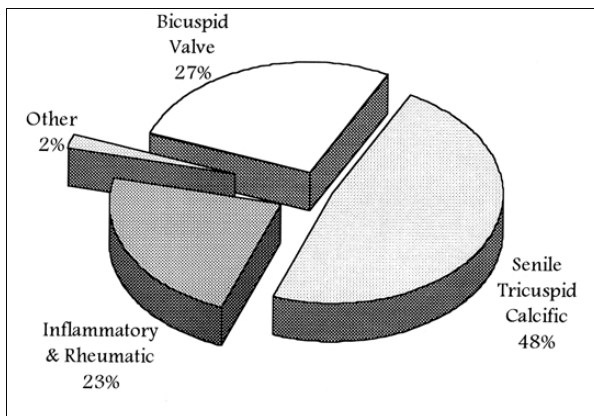


Figure. Etiology of aortic stenosis in persons aged 70 years. Based on surgical series data from Passik et al.⁹

atherogenic in origin is supported by epidemiologic studies showing a higher than expected prevalence of diseases such as coronary artery disease³ or carotid stenosis⁴ in elderly patients with valvular aortic stenosis. End-stage renal disease, with its attendant abnormalities in calcium and phosphate metabolism, leads rapidly to aortic stenosis in susceptible individuals.⁵ Likewise, in Paget's disease, the prevalence of calcific aortic stenosis is four times higher than in the general population.⁶

Bicuspid Valve. A bicuspid valve is the most common congenital cardiac defect, occurring in 0.4%–2% of live births.^{7,8} The bicuspid valve is not stenotic at birth. However, due to abnormal flow through the malformed valve, repetitive jet injuries lead to progressive calcification and orifice narrowing. It is rare to encounter an elderly person with a bicuspid valve and no significant aortic stenosis. Although most cases of bicuspid calcific aortic stenosis present in late middle age, this congenital anomaly still accounts for about one fourth of aortic stenosis cases in patients older than age 70.⁹

Rheumatic Aortic Stenosis. Even at a time when rheumatic fever was still prevalent in the Western industrialized world, a rheumatic etiology was found only in a minority of aortic stenosis cases. In several surgical or postmortem series from the 1970s, the prevalence of presumably rheumatic aortic stenosis was around 25% of all aortic stenosis cases.^{2,10} By the 1980s, the share of rheumatic aortic stenosis dropped in some surgical

Table I. Hemodynamic Degrees of Aortic Stenosis

	MILD	MODERATE	SEVERE
Mean pressure gradient (mm Hg)	<25	25–45	>45
Peak instantaneous pressure gradient (mm Hg)	<40	40–70	>70
Aortic valve area (cm ²)	>1.3	0.8–1.3	<0.8

Note: The normal aortic valve in adults has an area of 3–4 cm² and no appreciable pressure gradient. The pressure gradient is proportional to the degree of aortic stenosis, as long as the cardiac output is normal. In patients with low cardiac output, however, the pressure gradient may be deceptively low. In such instances, one should rely on the aortic valve area for the assessment of aortic stenosis severity.

series to as low as 5.4%.¹¹

NATURAL HISTORY

During the latent period, aortic stenosis progresses gradually over the years, from mild to moderate to severe (Table I). Patients with less than severe aortic stenosis are rarely symptomatic in the absence of comorbid conditions. Initially, the rising transvalvular pressure gradient increases left ventricular wall stress. The left ventricle adapts to this chronic systolic pressure overload by developing concentric left ventricular hypertrophy. According to the Laplace law, the increased thickness of the ventricular wall decreases the wall stress. Normalization of wall stress through left ventricular hypertrophy is the primary reason why aortic stenosis can be clinically latent for decades.

Asymptomatic Patients. Even patients with hemodynamically severe aortic stenosis are initially asymptomatic. Retrospective postmortem studies performed in the 1960s led to the initial impression that these asymptomatic patients were nevertheless at increased risk of sudden cardiac death. At the time, it was estimated that sudden cardiac death accounted for 3%–5% of all deaths in patients with asymptomatic-acquired aortic stenosis.¹²

However, in a prospective study performed in 1990 in patients whose mean age was 72, and in whom the severity of

aortic stenosis was objectively measured by echocardiography, no cases of sudden cardiac death were observed in asymptomatic patients with severe aortic stenosis.¹³ The likelihood that the asymptomatic patient with severe aortic stenosis will die suddenly is less than the expected surgical mortality for aortic valve replacement. The results of this study provide a powerful argument against prophylactic replacement of the aortic valve in asymptomatic patients with severe aortic stenosis.

Symptomatic Patients. The natural history in patients with symptomatic aortic stenosis and objectively documented transvalvular gradients has not been sufficiently studied in any adult age group. Only a few studies have specifically targeted elderly populations.^{1,14} Therefore, for natural history, we have to rely mostly on studies performed in middle-aged individuals.

In the 1960s, cardiac catheterization (then the only modality capable of measuring transvalvular gradients) coincided with the introduction of the first surgical techniques aimed at correcting aortic stenosis. Since symptomatic patients with severe aortic stenosis were generally referred to surgery, inferences about the natural history of severe uncorrected aortic stenosis were made from either postmortem studies or studies in the few patients who refused or were denied surgery.^{15,16} Such studies have shown that once symptoms develop, the patient is expected to live less than 5 years on average, and that survival beyond 10 years is unlikely.

In 1968, Ross and Braunwald¹² argued in their landmark review of postmortem studies that survival in medically treated individuals is strongly dependent on the type of symptom. In their opinion, angina carried the best prognosis and congestive heart failure the worst prognosis. Survival in patients with syncope was thought to be somewhere between these two.

PATHOPHYSIOLOGY

Progressive narrowing of the aortic valve, which in the elderly is always associated with extensive valve calcification, produces

not only hemodynamic consequences, but also puts the patient at an increased risk of systemic embolism, bacterial endocarditis, conduction abnormalities, and possibly gastrointestinal bleeding from angiodysplastic lesions.

Hemodynamic Aspects. Angina pectoris, syncope, and congestive heart failure represent the classic symptom triad of significant aortic stenosis. Because most patients with symptomatic aortic stenosis undergo operations, the true prevalence of each of the three symptoms can be deduced more accurately from the current surgical series than from the older, natural history studies performed in patients who refused or were denied surgery. Surgical series, for instance, revealed that among octogenarians who underwent aortic valve replacement for aortic stenosis, about two thirds had congestive heart failure, about half had angina, and about one third had syncope preoperatively.^{17,18}

Angina in aortic stenosis is due to a significant myocardial demand/coronary supply mismatch that is particularly evident during exercise. There is both an increase in myocardial demand (from an often massively hypertrophied left ventricle) and a decrease in coronary flow. In normal individuals and in patients with aortic stenosis, the magnitude of coronary perfusion is determined by the pressure gradient between the ascending aorta and the left ventricle. In aortic stenosis, there is an increase in the left ventricular pressure, both in systole and diastole. In systole, the ventricular pressure is high as a result of a chronically elevated resistance imposed by the narrowed aortic valve. In diastole, an elevated ventricular pressure is required for proper filling of the stiff, hypertrophied left ventricle. As a result, the diminished aortoventricular pressure gradient in diastole impairs coronary blood, as does the high systolic pressure in the left ventricle.

Furthermore, concomitant coronary artery disease is common in the elderly. For instance, at least 50% of octogenarians with aortic stenosis, who are referred for aortic valve replacement, have angiographic signs of significant coronary artery disease.

The average survival of medically treated patients with aortic stenosis and angina is about 5 years, as opposed to 3 years for patients with aortic stenosis and syncope, and 2 years for patients with aortic stenosis and congestive heart failure. The better survival of aortic stenosis patients with angina may reflect the fact that concomitant coronary artery disease, which has a better prognosis than symptomatic aortic stenosis, is the primary cause of angina in many patients with aortic stenosis.

Syncope and Sudden Death. The exact mechanism of syncope in aortic stenosis, which often occurs during exertion, remains controversial. The classic explanation is that the arterial pressure is a product of cardiac output and systemic vascular resistance. During exercise, there is normally a drop in systemic vascular resistance due to peripheral vasodilation. Normal individuals can augment their cardiac output as much as is needed to prevent hypotension. However, in patients with advanced aortic stenosis, the maximum cardiac output is limited by the fixed aortic valve orifice. Some argue that it is this limited cardiac output reserve that is responsible for exercise-induced hypotension, cerebral hypoperfusion, and syncope in patients with aortic stenosis.

However, experimental data in animals suggest that the activation of the Bezold-Jarisch reflex is the most plausible explanation for exertional syncope in aortic stenosis. A very high left ventricular pressure, which develops during exercise in patients with aortic stenosis, activates baroreceptors in the walls of the left ventricle. This, in turn, leads to reflex vasodilation, bradycardia, and syncope. Alternatively, malignant ventricular arrhythmias may be the cause of syncope in some patients with aortic stenosis, especially if the syncope occurs at rest.¹⁹

Sudden death rarely occurs in asymptomatic patients with severe aortic stenosis, but this is not the case with symptomatic patients. In the presurgical era, the incidence of sudden death in patients with symptomatic aortic stenosis was estimated to be as high as 15%–20%.¹² However, the incidence of sudden death may have decreased in modern times due to early surgical intervention in patients with symptomatic aortic stenosis.

Congestive Heart Failure. Initially, diastolic

dysfunction due to left ventricular hypertrophy predominates. As previously noted, left ventricular hypertrophy is an adaptive mechanism responsible for normalization of wall stress in patients with aortic stenosis. However, increased left ventricular stiffness results, with a subsequent rise in left ventricular filling pressure, and pulmonary congestion develops, especially during exercise. As the left ventricular wall stiffens, the importance of normal sinus rhythm and atrial systole for the diastolic filling of the left ventricle increases. Therefore, the onset of atrial fibrillation often precipitates overt heart failure in patients with aortic stenosis.

Unfortunately, the incidence of atrial fibrillation seems to be higher in elderly patients with aortic stenosis than in the general elderly population, presumably due to a chronically elevated left atrial pressure in the former group. The prevalence of atrial fibrillation in individuals older than 70 is about 6%–8%,²⁰ whereas the prevalence of aortic stenosis in patients of similar age is reported to be as high as 16.8%.²¹

Systolic dysfunction may also be responsible for heart failure in patients with significant aortic stenosis. Left ventricular scarring and chamber dilatation often result from not only the chronic systolic pressure overload imposed by the aortic stenosis, but also the coexistent contractile dysfunction due to myocardial infarctions, hibernating myocardium, or cardiomyopathy.

Nonhemodynamic Aspects. Endocarditis. All patients with aortic stenosis are at risk for bacterial endocarditis and should, therefore, receive standard antibiotic prophylaxis prior to procedures that may lead to bacteremia.

Embolism. Rarely, fragments of the calcific valve may embolize into the systemic circulation. Endocarditic vegetations represent a more important cause of systemic embolism, including stroke in patients with aortic stenosis. In addition, atrial fibrillation and aortic atheromas are common in the elderly, and both carry a high embolic risk.^{20,22}

Conduction Abnormalities. Aortic valve calcification may extend into the perivalvular tissue, infiltrate the conduction system, and

cause various forms of heart block.

Gastrointestinal Bleeding. Some 40 years ago, an association between aortic stenosis and gastrointestinal (GI) bleeding from angio-dysplastic lesions was first reported.²³ It has since been argued that the incidence of GI bleeding from angiodysplasia in patients with aortic stenosis may be higher than in the general elderly population.

An acquired form of von Willebrand's disease may be the link between the two conditions. Patients with aortic stenosis are hypothesized to have lower plasma levels of von Willebrand factor, which predispose them to blood loss from preexisting GI angiodysplasias.²⁴ There is some evidence that aortic valve replacement normalizes plasma levels of von Willebrand factor, and corrects the GI bleeding diathesis.²⁵ However, some argue that no causality between aortic stenosis and GI angiodysplasia exists, and that the apparent relationship between these two entities is merely the consequence of both conditions being common in the elderly.²⁶

DIAGNOSIS

Although the physical exam, chest radiography, and electrocardiography have relatively low sensitivity and specificity, these routine tests are nonetheless essential in detecting and following patients with aortic stenosis. However, for objective hemodynamic assessment of aortic stenosis severity, one has to rely on either echocardiography or cardiac catheterization.

Routine Tests. Physical Exam. The classic findings include a harsh, late-peaking, crescendo-decrescendo murmur best heard above the aortic valve, absent A₂, prominent S₄, narrow pulse pressure, and a delayed carotid upstroke (pulsus parvus et tardus). However, one has to bear in mind that the so-called classic physical findings of aortic stenosis were first described in young and middle-aged patients and may not be encountered in the elderly.

Cardiac output is as important a determinant of the intensity of the murmur as the degree of valve narrowing. Consequently, patients with left ventricular dysfunction (whether from aortic stenosis or from other causes) will often have a soft murmur, even in the presence of severe aortic stenosis. Since heart failure from any cause is prevalent in the elderly, a soft

murmur in association with severe aortic stenosis is a frequent finding in this age group.

A prominent S₄ follows atrial systole in any patient who is in normal sinus rhythm and has a noncompliant left ventricle. Because the degree of left ventricular hypertrophy correlates with the severity of aortic stenosis, and because left ventricular hypertrophy leads to a noncompliant ventricle, the presence of S₄ in younger patients is suggestive of severe aortic stenosis.

In the elderly, S₄ is less specific for aortic stenosis, because hypertension, coronary artery disease, and other disorders common in older individuals can diminish left ventricular compliance independent of aortic stenosis. In addition, atrial fibrillation, which is also prevalent in the elderly, eliminates atrial systole—the crucial requirement for the generation of S₄. The pulse pressure may be normal, or even wide, and the carotid upstroke may be rapid in the elderly due to concomitant atherosclerosis of the arterial tree.²¹

Electrocardiography. Left ventricular hypertrophy is the most common finding on the electrocardiograph of patients with severe aortic stenosis. In elderly patients, left ventricular hypertrophy is present electrocardiographically in about two thirds of patients (and echocardiographically in nearly all patients).¹ Less common findings include left atrial enlargement, first degree atrioventricular block, or bundle branch block. Each occurs in no more than one fourth of elderly patients with significant aortic stenosis.²⁷

Chest Radiography and Fluoroscopy. Calcifications in the region of the aortic valve represent the most relevant chest radiography finding in the elderly, since calcific degeneration is the hallmark of aortic stenosis in this age group. However, the absence of aortic valve calcifications on chest radiography does not exclude the diagnosis of severe aortic stenosis.²⁷ Valvular calcifications are more easily seen on fluoroscopy, as this modality provides higher image quality than standard roentgenography or real-time imaging.

Calculation of Pressure Gradient and Aortic

Valve Area. The objective assessment of aortic stenosis severity requires the calculation of hemodynamic parameters, such as transvalvular pressure gradients and the computation of the actual aortic valve orifice area. Only two techniques can accomplish such a task: cardiac catheterization and echocardiography. Both use the same fundamental hemodynamic principle for the assessment of the aortic valve area (AVA). This principle states that the AVA is a ratio between the transvalvular volumetric flow and blood velocity at the level of the aortic valve. The transvalvular volumetric flow across the aortic valve is equivalent to the forward cardiac output.

$$\text{Equation 1: AVA} = \text{cardiac output} \div \text{blood velocity}$$

However, during cardiac catheterization, blood velocities are not measured. Instead, pressures in the left ventricle and the aorta are obtained and the transvalvular pressure gradient calculated. In the 1950s, Gorlin and Gorlin²⁸ developed a method (Equation 2) of substituting pressure gradients for blood velocities in Equation 1.

$$\text{Equation 2: AVA} = \text{cardiac output} \div (44.3 \times \text{HR} \times \text{SEP} \times \text{MPG})$$

where HR stands for heart rate, SEP for systolic ejection period, and MPG for the mean pressure gradient.

Assuming a normal cardiac output and a normal heart rate, AVA is therefore inversely proportional to the square root of the mean transvalvular pressure gradient. Subsequently, clinicians have become accustomed to expressing the severity of aortic stenosis in terms of peak and mean pressure gradients. The higher the peak (or mean) gradient, the more severe the aortic stenosis (Table I).

Doppler echocardiography, which was first applied to aortic stenosis assessment in the late 1970s, is capable of directly measuring blood velocities. Ironically, echocardiographers had to devise methods of converting blood velocities back to pressure gradients to compare their results with cardiac catheterization data.^{29,30} The modified Bernoulli equation provides for such a conversion.

$$\text{Equation 3: pressure gradient} = 4 \times (\text{velocity})^2$$

Patients with diminished cardiac output often have deceptively low pressure gradients, even in the presence of severe aortic stenosis. Therefore, pressure gradients alone cannot be used to assess the degree of aortic stenosis in the presence of left ventricular dysfunction or mitral regurgitation. Instead, one has to calculate the valve orifice area in such patients.

During cardiac catheterization, cardiac output can be measured and AVA calculated using the Gorlin equation. Echocardiographers, however, prefer to use the so-called continuity equation to calculate AVA.³¹ This equation obviates the need for cardiac output measurement. Instead, this method assumes that the left ventricular outflow tract has a circular cross-sectional area, and that the amount of blood flow across that area is identical to the amount of blood flow crossing the aortic valve. Because the flow through any orifice is a product of the cross-sectional area of the orifice and the blood velocity at that orifice (Equation 1),

$$\text{LVOT velocity} \times \text{LVOT area} = \text{aortic valve velocity} \times \text{AVA}$$

where LVOT stands for left ventricular outflow tract.

Pulsed-wave and continuous-wave Doppler can be used to determine velocities across the LVOT and aortic valve, respectively. From 2-D echo, one can obtain the diameter of the LVOT, and then calculate its presumably circular area. AVA can then be calculated.

$$\text{Equation 4: AVA} = \text{LVOT area} \times (\text{LVOT velocity} \div \text{aortic valve velocity})$$

Echocardiography vs. Cardiac Catheterization. For the evaluation of aortic stenosis, echocardiography provides hemodynamic data that are as reliable as those obtained from cardiac catheterization. Echocardiography allows for a serial, noninvasive assessment of mean and peak pressure gradients, as well as the calculation of the actual AVA during the years of disease progression. In addition, 2-D echocardiography provides data on left ventricular function and other valvular lesions, if present. Transesophageal echocardiography provides excellent short-axis images of the

aortic valve, and thus allows for direct measure of the AVA by planimetry in many patients.³²

Cardiac catheterization was previously considered to be the gold standard for hemodynamic assessment of aortic stenosis severity. Since reliable hemodynamic data can now be obtained in nearly all patients noninvasively with echocardiography, cardiac catheterization is no longer necessary for the determination of transvalvular pressure gradients. It is now usually performed only in patients who are to undergo aortic valve replacement. Its principle goal is to assess the extent of concomitant coronary artery disease (which is common in the elderly) by angiography rather than to determine the hemodynamic severity of aortic stenosis.

Impact of Left Ventricular Function on Assessment of Aortic Stenosis Severity. In patients with normal systolic function of the left ventricle, determination of the transvalvular pressure gradient by either echocardiography or cardiac catheterization is sufficient for judging the severity of aortic stenosis. However, in patients with diminished left ventricular function, the actual valve area should be calculated.

Patients with diminished left ventricular function, seemingly severe aortic stenosis (a small valve area), and a low transvalvular pressure gradient at rest represent a special category. Their hemodynamic findings can be due either to severely reduced left ventricular function caused by severe aortic stenosis, or to a less-than-severe aortic stenosis in the setting of contractile dysfunction unrelated to aortic stenosis. In both conditions, transvalvular blood flow is diminished, which in combination with a low transvalvular gradient leads to a calculation of a small valve area (Equation 1). The distinction between the two groups is important, because aortic valve replacement will lead to postoperative improvement in left ventricular function only in those patients with genuinely severe aortic stenosis, not in those with contractile dysfunction due to other causes such as myocardial infarction.

Dobutamine infusion, in conjunction with either echocardiography or cardiac catheterization, can be used to distinguish between the two groups.³³⁻³⁵ Dobutamine augments the cardiac output, and thus increases the flow and blood velocity across the

aortic valve. In patients with genuinely severe aortic stenosis, dobutamine infusion will lead to a proportional increase in both the transvalvular flow and the transvalvular gradient. The calculated AVA will, therefore, remain in the severe range.

In patients who do not have a truly fixed aortic valve orifice and in whom contractile dysfunction is unrelated to aortic stenosis, the aortic valve will open further because of the higher transvalvular flow during dobutamine infusion. Consequently, the transvalvular pressure gradient will not rise. A combination of an increased transvalvular flow and a low transvalvular gradient will lead to a calculated valve area that is larger than the one obtained at rest, and it may no longer be in the severely reduced range.

However, the results of dobutamine testing should be interpreted with caution. Some patients with truly severe aortic stenosis can have a significant increase in the calculated AVA, even if the physical dimensions of the aortic valve do not change. The discrepancy is due to flow dependence of both the Gorlin formula and the continuity equation.^{32,35}

MANAGEMENT

There is no effective medical treatment for aortic stenosis. All symptomatic patients with severe aortic stenosis should be considered for surgical correction. Aortic valve replacement may also be considered in certain subsets of asymptomatic patients. All other patients with aortic stenosis should be followed medically until they become surgical candidates. All patients with aortic stenosis should receive standard antibiotic prophylaxis prior to dental, GI, genitourinary, or other procedures capable of causing bacteremia.³⁶

Asymptomatic Aortic Stenosis. Serial Echocardiography. Patients with severe

Table II. Frequency of Echocardiography in Patients with Aortic Stenosis

AORTIC STENOSIS GRADE	PERFORM ECHOCARDIOGRAM
Mild	Every 5 years
Moderate	Every 2 years
Severe	Annually
Based on data from Bonow et al. ³⁷	

asymptomatic aortic stenosis should undergo periodic echocardiographic assessment of left ventricular function. Aortic valve surgery may be considered even in asymptomatic patients if the left ventricle starts to fail. Patients with less-than-severe aortic stenosis should undergo periodic echocardiography to assess disease progression as judged by transvalvular gradients. Current national guidelines are summarized in Table II. The frequency of echocardiographic examination depends on the ACA, which diminishes on average by 0.12 cm² per year, and the peak pressure gradient, which rises by up to 10–15 mm Hg per year.³⁷

Stress Testing. Severe aortic stenosis is considered a contraindication for exercise stress testing.³⁸ Nonetheless, in patients with hemodynamically significant aortic stenosis and unclear symptoms, exercise stress testing may be useful and safe if performed by a skilled physician. The test may identify patients who need aortic valve replacement even in the absence of classic symptoms of aortic stenosis. Patients with hemodynamically significant aortic stenosis who develop an exercise-induced drop in blood pressure should be referred for aortic valve replacement, even if they are otherwise asymptomatic.³⁹ In addition, exercise stress testing, in conjunction with either radionuclide imaging or echocardiography, allows for the evaluation of left ventricular function and the assessment of concomitant coronary artery disease.

Symptomatic Aortic Stenosis. Aortic Valve Replacement. Any elderly patient with symptomatic hemodynamically significant aortic stenosis should be considered for aortic valve replacement. Surgery should also be considered in selected subsets of asymptomatic patients with severe aortic stenosis. These include: 1) patients who become hypotensive during stress testing; 2) patients with diminished left ventricular function; and 3) patients who need other cardiac surgery, such as coronary artery bypass grafting.

A patient's age, even if advanced, is not a contraindication for surgery. However, the older the patient, the higher the surgical mortality and morbidity. For instance, in octogenarians, early surgical mortality for isolated aortic valve replacement is in the 3%–6% range, at least twice as high as in younger patients.^{17,18,40}

Surgical mortality in the elderly rises sharply if additional cardiac surgery is performed. Octogenarians who undergo both aortic valve replacement and coronary bypass grafting have a surgical mortality of 20%–28%.^{18,40}

Choice of Prosthetic Valve. Either a mechanical or a bioprosthetic (tissue) valve prosthesis can be used to replace the stenosed native aortic valve. Although bioprosthetic valves are less durable than mechanical valves, they are preferred in the elderly. Bioprosthetic valves, unlike mechanical ones, do not require long term anticoagulation, except for the first 3 months postoperatively.⁴¹ Moreover, the rate of structural failure of bioprostheses in the aortic position is relatively low. A major study revealed that after an 11-year follow up, structural failure developed in 15% of aortic valve bioprostheses; no structural failures were observed with mechanical valves in the same time period.⁴² An aortic bioprosthetic valve will probably last 15–20 years in most patients—often longer than the life expectancy of many elderly individuals.

Postoperative Course. Following aortic valve replacement, left ventricular hypertrophy gradually recedes, and left ventricular function eventually improves in a significant proportion of patients.⁴³ Whether left ventricular function will improve after surgery depends on whether the preoperative left ventricular dysfunction was due to aortic stenosis alone or due to a comorbid condition. For instance, patients with no prior myocardial infarction have a low surgical mortality and a significant improvement in left ventricular ejection fraction postoperatively. On the contrary, patients with aortic stenosis and a history of preoperative myocardial infarction have much poorer outcomes.⁴⁴

If coexistent mitral regurgitation was present preoperatively, its severity often decreases postoperatively, even in the absence of any concomitant mitral valve surgery.⁴⁵ This improvement in mitral regurgitation is the result of lower systolic left ventricular pressures postoperatively. Long term follow up studies have revealed that patients aged 65 years or more, who survive aortic valve surgery for pure aortic stenosis, assume a life expectancy of the general population within 2 years following surgery.⁴⁶

Aortic Balloon Valvuloplasty. Although this catheter-based intervention does not require open heart surgery, the rate of serious complications (such as death, stroke, aortic rupture, and aortic regurgitation) exceeds 10%.⁴⁷ Furthermore, its benefits are short lived.

At present, the procedure is reserved mainly for patients who are poor surgical candidates and/or whose survival is severely limited by a concomitant illness such as terminal cancer.⁴⁸ In addition, valvuloplasty can be used as a bridge to aortic valve replacement in very sick patients. For instance, balloon valvuloplasty can provide a temporary relief of aortic stenosis symptoms in an elderly patient with a broken hip who needs orthopedic surgery.

CONCLUSIONS

Because Western populations are aging, physicians in the industrialized world will increasingly encounter aortic stenosis. Aortic valve surgery remains the only effective form of treatment, and should be performed once symptoms develop, even in the very elderly, with an acceptable risk of surgical mortality and morbidity.

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