

Valvular heart disease, part 1

Diagnosis and surgical management of aortic valve disease in older adults

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Aortic valve disease is a common cause of morbidity and mortality in older patients. The etiology, physical findings, and natural history of aortic stenosis and aortic regurgitation may differ in older patients compared with younger patients. In addition, symptoms of aortic valve disease may be masked or exacerbated by co-existent coronary artery disease, hypertension, pulmonary diseases, and other systemic disorders that commonly occur in the geriatric population. Clinical assessment, along with various non-invasive cardiac techniques including ECG, chest x-ray, and echocardiogram are important in identifying aortic valve disease as the cause of abnormal signs and symptoms in these patients. Recognition of aortic valve abnormalities has important therapeutic implications because aortic valve replacement is usually associated with favorable short- and long-term results, even in patients over age 65.

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Key words: valve disease • aortic stenosis • aortic regurgitation

Age-related changes to the aortic and mitral valves that occur in older adults influence the development of symptoms and subsequent complications of valvular heart disease. This article (part 1 of a two-part CME article on valvular heart disease)

will discuss the diagnosis and management of aortic stenosis and aortic regurgitation in older adults. Next month, in part 2 of the article, the valvular heart disease discussion will continue with a focus on the diagnosis and management of mitral valve disease.

Aortic stenosis

Aortic stenosis (AS) related to degenerative changes of the aortic valve can cause cardiac disability in older patients. Although thickening and calcification of the aortic valve associated with a systolic murmur has been observed in up to 50% of patients over age 65, severe AS is relatively infre-

quent. The exact incidence of severe AS in these patients is unknown.¹⁻⁵

Etiology. Calcification and degeneration of an intrinsically normal trileaflet aortic valve typically becomes apparent after age 65. These changes appear to be an exaggeration of the normal aging process. In most cases, the degree of distortion and resulting calcific degeneration of the aortic valve is mild, and clinical effects are limited to a systolic murmur. Extensive calcification resulting in immobilization of the aortic cusps and severe senile or calcific AS is infrequent; nonetheless, it represents the most common cause of isolated AS in patients over age 75. Isolated AS in patients less than age 65 is often associated with a bicuspid aortic valve; between ages 65 and 75 either bicuspid or tricuspid aortic valves may be present. Rheumatic fever is seldom, if ever, the cause of isolated AS in older patients.

The cause of calcific degeneration of the aortic valve in older adults is not clear. The location of the earliest changes suggests that mechanical factors (wear and tear phenomenon) are involved. The reason why the normal aging changes of the aortic valve are exaggerated in some older patients is also unknown. A number of risk factors are associated with progression of AS, including smoking, hypercholes-

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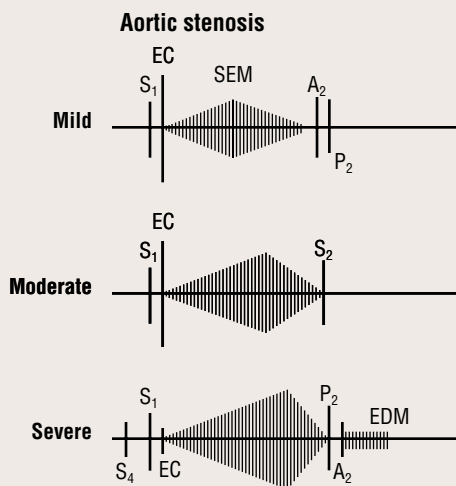


Figure. Schematic diagram of cardiac auscultation findings in mild, moderate, and severe aortic stenosis (AS). With progression of the disease, the systolic ejection murmur (SEM) becomes longer and louder and peaks later in systole; the ejection click (EC) diminishes as the valve becomes less mobile; a fourth heart sound (S_4) is heard in severe cases, reflecting decreased left ventricular compliance; A_2 progressively diminishes and paradoxical split of S_2 is present in severe AS; an early diastolic murmur (EDM) may be elicited in patients with coexistent aortic regurgitation.

S_1 =first heart sound; A_2 =aortic component of the second heart sound; P_2 =pulmonary component of the second heart sound.

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terolemia, chronic renal disease, diabetes mellitus, and hypercalcemia.

Pathophysiology. The anatomic severity of valve obstruction and the impedance of left ventricular (LV) outflow gradually increases with age. The left ventricle initially responds to this abnormal after-load by the widespread parallel reduplication of sarcomeres, which leads to macroscopic concentric left ventricular hypertrophy (LVH), thus maintaining a normal systolic wall tension. Left ventricular compliance is decreased because of hypertrophy. Pulmonary congestive symptoms are often delayed until some degree of systolic dysfunction occurs, ordinarily late in the natural course of the disease. Coexistent coronary artery disease and hypertension, which are more prevalent in the older population, may exacerbate the pathophysiologic conse-

quences of AS and mask or worsen its clinical manifestations. In contrast, angina pectoris is the most common symptom in young or middle-aged patients with AS. Angina pectoris in older patients with severe AS has clinical characteristics similar to the typical effort angina of coronary artery disease. In the older patient with AS, coronary heart disease is often present. Cardiac catheterization is required to identify associated coronary artery disease.²

The 5-year survival rate is poor when congestive heart failure, angina, or syncope develops in unoperated patients with AS. Heart failure carries the worst prognosis; survival is less than 2 years in most of these patients if surgery is not performed. Syncope, which usually occurs during or immediately after exercise, most likely results from an abrupt decrease in cardiac output. Possible

mechanisms include ventricular arrhythmias, bradyarrhythmias, or conduction disturbances related to calcific involvement of the conduction system, especially when mitral annular calcification is also present.

As the aortic orifice size decreases, LV systolic pressure increases to sustain cardiac output, and a systolic gradient develops across the valve. A mean gradient less than 25 mm Hg is generally associated with an aortic valve area greater than 1.5 cm², which is considered mild AS. Gradients greater than 50 mm Hg determined by echocardiography and cardiac catheterization suggest that the aortic valve area is less than 1 cm², indicating severe AS.^{2,6}

Symptoms. The clinical picture of AS in older patients may differ in several aspects from that seen in younger patients. Symptoms attributed to congestive heart

failure are the most common complaints among older adults. In contrast, angina pectoris is the most common symptom in young or middle-aged patients with AS. Angina pectoris in older patients with severe AS has clinical characteristics similar to the typical effort angina of coronary artery disease. In the older patient with AS, coronary heart disease is often present. Cardiac catheterization is required to identify associated coronary artery disease.²

Physical examination. Early or mid-systolic low-intensity, medium-pitch murmurs can be heard in 30 to 55% of patients over age 60. In most of these patients, the systolic murmur is hemodynamically unimportant and is caused by mild thickening and calcification of one or more of the aortic valve leaflets with no significant gradient (ie, aortic sclerosis) or by an aortic flow phenomenon caused by moderate aortic dilatation.^{7,8} This systolic ejection murmur is usually less intense and terminates earlier in systole than the murmur associated with significant aortic valve obstruction (figure). Aortic sclerosis is a soft, grade 1, early systolic murmur with normal first and second heart sounds and no ejection click (EC).

In patients with mild AS, an EC precedes the soft systolic murmur at the aortic area with preserved aortic valve closure. As AS progresses in severity, the EC disappears. The fourth heart sound (S_4) may be heard preceding the first sound, and the murmur is longer, peaking late in systole. The second heart sound (A_2) is reduced in intensity or is absent. A soft early diastolic murmur of mild aortic regurgitation may be heard.

Several other typical features of AS may not be present in older patients. For example, the small (parvus) and slow rising (tardus) carotid pulse with a palpable anacrotic shoulder, which is characteristic of severe AS, may not always be detected in older patients. Decreased vascular compliance associated with advanced age can cause widening

of the pulse pressure and can interfere with perception of the slow rise of the pulse and the prolonged ejection time. In addition, low cardiac output and associated aortic regurgitation can preclude adequate assessment of the rate of rise of the pulse wave. The cardiac impulse may be sustained and forceful, with lateral displacement, but palpation of the precordium is frequently unremarkable because 50% of older patients with AS do not exhibit cardiomegaly on clinical examination.

It is not unusual for an older patient with a systolic murmur and with symptoms of heart failure, angina, or syncope to have associated coronary or hypertensive heart disease. Because clinical symptoms and signs are not specific, the detection of severe AS in this setting may be difficult. Various non-invasive techniques may aid in the diagnosis of AS in the older patient.

ECG. The ECG is typically normal in patients with mild or moderate AS. Depending on the ECG criteria used, 60 to 90% of patients with severe AS may manifest evidence of LVH. Occasionally LVH is absent even in severe AS. On the other hand, LVH may be caused by longstanding associated systemic hypertension. Conduction disturbances, most commonly left anterior hemiblock; complete bundle branch block; usually left, first-degree atrial ventricular block; or complete heart block are encountered in approximately one-third of patients and presumably are related to calcific involvement of the conduction system. Atrial fibrillation is more common in older than in younger patients with AS and may result in hemodynamic deterioration.

Chest x-ray. The chest x-ray in AS may reveal left ventricular prominence; cardiomegaly in solitary AS is usually absent, but it may be present in patients with combined AS and aortic regurgitation or left ventricular failure. Dilatation of the ascending aorta and calcification at the area of the aortic valve on the lateral chest film are detected in most patients with severe AS.

Echocardiography. Two-dimensional

and doppler echocardiography are the imaging modalities of choice for diagnosing AS and estimating its severity. Although the presence or absence of AS is readily diagnosed on two-dimensional echo, the severity of AS cannot be judged on the basis of the echo image alone. Doppler echo is required to non-invasively assess the severity of AS. Again, if the mean gradient across the aortic valve is greater than 50 mm Hg, severe AS can be diagnosed. The systolic gradient depends not only on the severity of obstruction, but also on flow. In patients with severe heart failure and low cardiac output, the AS may still be severe even if the mean gradient is less than 50 mm Hg. The calculation of aortic valve area is also highly dependent on accurate measurements of the diameter of the LV outflow tract.⁹ Doppler echo is important in distinguishing older patients with aortic sclerosis from those with AS.^{10,11}

Cardiac catheterization. Cardiac catheterization is performed in the older patient to exclude associated coronary heart disease, other valvular lesions, hemodynamic findings of elevated LV end-diastolic pressure, and pulmonary hypertension as well as to measure the systolic gradient across the aortic valve and to determine LV function by LV gram.¹²

Pharmacologic management. Medical management for chronic AS is not effective. Digitalis and diuretics may improve heart failure in patients who are not considered surgical candidates because of the high risk of surgery. Nitrates may be used cautiously to treat angina pectoris. Vasodilators are contraindicated in patients with AS because of their potential to cause dangerous hypotension.

Chronic aortic regurgitation

Chronic aortic regurgitation (AR) can be caused by rheumatic heart disease, but more often it is caused by prolapse of the aortic leaflet associated with myxomatous changes, aortic valve ring ectasia associated with aortic root dilatation, or aortic aneurysm. Infective endocarditis, possibly on a bicuspid

aortic valve, or dissection of the ascending aorta will also cause AR. Age-related progressive dilatation of the thoracic aorta is probably a normal aging process involving the media and usually causes only mild AR. Approximately 10% of patients with systemic hypertension may have evidence of mild AR due to aortic valve ring dilatation. Degenerative calcific AS is usually associated with mild rather than severe AR.¹³⁻¹⁵

Pathophysiology. Aortic regurgitation results in volume overload of the left ventricle. With compensated AR, there is a proportional increase in LV radius and wall thickness, thus maintaining a normal chamber radius-to-wall thickness ratio and allowing the LV to eject an augmented stroke volume against normal or reduced afterload. The constant relation between pressure and the chamber radius-to-wall thickness ratio is maintained by the addition of new sarcomeres in series, fiber elongation, and LV enlargement.¹

Symptoms. Patients with isolated AR typically remain asymptomatic for a longer time than patients with isolated AS. The first objective manifestation of AR may be an awareness of pulsation in the neck or at the precordium, especially in the recumbent position. Palpitations, pounding head, and fatigue are other symptoms experienced by these patients. These complaints may be present for many years before symptoms of LV dysfunction or myocardial ischemia develop. Exertional dyspnea, orthopnea, and paroxysmal nocturnal dyspnea are the principal complaints associated with myocardial dysfunction. Syncope is rare and angina pectoris is less frequent than in patients with AS; however, nocturnal angina associated with hot-flashes, sweating, and palpitations may be disturbing.^{1,2}

Physical examination. The pulse pressure is usually increased in patients with AR. The carotid pulse has a brisk quality and a bisferiens pattern (double hump) on palpation. Cardiac impulse may be displaced laterally and inferiorly and may be sustained. Determin-

ing the severity of AR in the older patient can be difficult because of the frequent occurrence of a wide pulse pressure resulting from a stiff atherosclerotic aorta rather than from AR. On auscultation, the first heart sound is usually soft. Aortic valve closure is also soft or even absent. An aortic ejection sound is often heard at the left sternal border and a third sound suggests increased LV end-systolic volume.

The hallmark of AR is the early diastolic murmur starting with aortic valve closure of the second sound usually heard at the aortic area and transmitted better along the left, rather than the right, parasternal border. When aortic root dilatation is the cause of AR, the murmur may be transmitted down the right parasternal border.¹ This high-pitch early diastolic murmur is better detected with the diaphragm of the stethoscope firmly applied to the chest wall with the patient sitting up or leaning forward after deep expiration.

ECG. Left axis deviation and a pattern of LV diastolic volume overload with prominent Q-waves in leads I, V1 through V3, and V6 and relatively small R-wave in V1 are usually observed in patients with chronic AR.

Chest x-ray. Mild cardiac enlargement is a common finding in severe chronic AR. Calcification of the aortic valve is often present in patients with combined AS and AR. Dilatation of the ascending aorta is more marked than in AS and may involve the aortic arch. Aneurysmal dilatation of the proximal ascending aorta should suggest the presence of aortic root disease. For example, aneurysmal dilatation of the proximal ascending aorta may be present in patients with Marfan syndrome, cystic medial necrosis, dissection of the aorta, or annuloaortic ectasia.

Echocardiography. Signs of LV volume overload, including dilated left atrium and left ventricle and hyperdynamic wall motion, are usually found in patients with severe chronic AR. The addition of two-dimensional echo can be helpful in the differential diagnosis of AR. Aortic valve disease can be dif-

ferentiated from aortic root causes of AR. Doppler echo is probably more sensitive than auscultation in detecting even mild degrees of AR; often, the murmur of mild AR is not heard.^{10,11}

Cardiac catheterization. Invasive procedures are important for assessing coronary arteries and determining the severity of aortic regurgitation with aortography.¹⁶ Hemodynamic changes involving the left and right heart are important in assessing LV function and the degree of pulmonary hypertension.

Acute aortic regurgitation

The most common causes of acute AR include infective endocarditis and dissecting aneurysm of the ascending aorta. Acute AR can also be secondary to trauma, due to spontaneous rupture or prolapse of myxomatous leaflets.¹⁷ In addition, post-operative AR can occur as the result of faulty surgical incisions. All of these conditions are not unusual in older patients.

Pathophysiology. The response of the LV to acute AR is a function of the rate at which the regurgitation develops. In acute AR, the stroke volume remains relatively small, and tachycardia becomes an important mechanism in maintaining cardiac output. Tachycardia is one of the cardinal signs of acute AR. Despite this tachycardia, the effective cardiac output remains diminished.

Symptoms. Once a diagnosis of acute AR has been made, prompt action is required as these patients remain at high risk for sudden deterioration because of severe and progressive congestive failure. Auscultation will typically reveal a diminished or absent first heart sound because of premature closure of the mitral valve. The diastolic murmur of AR usually begins immediately with aortic valve closure and is characteristically associated with a high pitch and decrescendo. With the presence of a torn aortic cusp, the murmur may have a musical high-frequency quality. With worsening of heart failure, the diastolic murmur tends to diminish in intensity and duration.

ECG. The ECG generally reveals a per-

sistent sinus tachycardia and, as LVH is the usual finding in chronic AR, there is insufficient time for the development of increased voltage of LVH in acute AR.

Chest x-ray. Chest x-ray may be normal. The aortic root may be enlarged, particularly when primary aortic root disease such as aortic aneurysm is present. Pulmonary congestion is usually conspicuous.

Echocardiography. Echocardiography is indispensable for confirming the presence and severity of acute AR. Transesophageal echo is indicated when aortic dissection is suspected. Other diagnostic imaging methods such as CT imaging and MRI may be required if echo does not provide sufficient information.

Pharmacologic management of chronic and acute AR. In patients with chronic AR, vasodilators may be used to reduce the degree of aortic regurgitation. Hydralazine, nifedipine, and angiotensin-converting enzyme inhibitors have been used.¹⁸⁻²⁰ In the patient with acute AR due to endocarditis, blood cultures should be drawn and broad-spectrum antibiotics begun until blood cultures identify the particular organism.²¹ Acute AR caused by aortic root dissection is a surgical emergency.


Surgical management of aortic valve disease

Aortic valve replacement is recommended in older patients with severe aortic valve disease who are symptomatic. Before surgery, the cardiologist will assess the patient's need for endocarditis prophylaxis. A discussion of the decision to institute prophylaxis is beyond the scope of this article; however, physicians interested in the recommendations can review the American College of Cardiology/American Heart Association guidelines for the management of patients with valvular heart disease.²² Because one-third of older patients with AS have associated coronary disease, it is often necessary to perform coronary artery bypass of the diseased coronary arteries at the time of aortic valve replacement. To be a candidate for surgery,

the patient's operative mortality rate should be less than 5%, depending on a variety of medical and surgical factors. Operative morbidity and mortality is increased in the older or debilitated patient and in the presence of associated problems including severe LV dysfunction; extensive coronary heart disease; other valvular disease; infective endocarditis; chronic renal, pulmonary, hepatic, and cerebral disease; and diabetes mellitus. Long-term survival is dependent upon the patient's age and comorbidities. Survival is approximately 80% at 5 years and 60 to 70% at 10 years after surgery in the low-risk older patient.²³

Tissue or biological valves are prone to gradual and progressive deterioration due to leaflet calcification. Mechanical valves are generally free of deterioration in the aortic position. Pericardial valve prostheses carry a better long-term prognosis than porcine valves. After 10 years, approximately 20% of these valves deteriorate and must be replaced. After 15 years, approximately 55% require reoperation. Long term, follow-up studies of homograft replacement are not available.²⁴ The selection of the valve to be used is determined by the patient's age, anatomy, LV function, lifestyle, and possible contraindications to anticoagulation therapy. The incidence of systemic thromboembolism from mechanical aortic valve replacement ranges from 1 to 2% per year even with adequate anticoagulation. The incidence appears to be less in patients with bioprosthetic valves even though these patients do not generally require anticoagulation.

Coming next

This article will conclude next month with a discussion of mitral valve disease. 

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1. Which of these symptoms in the older patient with aortic stenosis carries the worst prognosis?
 - a. palpitations
 - b. angina pectoris
 - c. heart failure
 - d. reduced exercise tolerance
 - e. none of the above
2. The older patient who presents with a systolic murmur at the aortic area with a late peaking crescendo/decrescendo murmur, absent ejection click, and markedly reduced or absent second heart sound most likely has:
 - a. chronic aortic regurgitation
 - b. acute aortic regurgitation
 - c. mild aortic stenosis
 - d. severe aortic stenosis
 - e. none of the above
3. Doppler echocardiogram in a patient with aortic stenosis with a mean gradient greater than 50 mm Hg is consistent with severe aortic stenosis.
 - a. True
 - b. False
4. The early decrescendo high-pitched diastolic murmur of aortic regurgitation is better heard with the bell of the stethoscope in the recumbent position.
 - a. True
 - b. False
5. The patient with acute aortic regurgitation caused by aortic root dissection may be considered a surgical emergency.
 - a. True
 - b. False
6. Angina pectoris is the most common symptom in older patients with aortic stenosis.
 - a. True
 - b. False
7. The first objective manifestation of isolated aortic regurgitation may be an awareness of pulsation in the neck or at the precordium, especially in the recumbent position.
 - a. True
 - b. False
8. An early diastolic murmur starting with aortic valve closure of the second sound usually heard at the aortic area and transmitted better along the left, rather than the right, parasternal border is the hallmark characteristic of:
 - a. chronic aortic regurgitation
 - b. aortic stenosis
 - c. aortic sclerosis
 - d. acute aortic regurgitation

In addition to the exam questions, answer the following evaluation questions: (1=strongly agree, 6=strongly disagree)

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