

Aortic Stenosis: Pathophysiology, Diagnosis, and Medical Management of Nonsurgical Patients

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As the average lifespan continues to increase, nurses are managing more patients with aortic stenosis. When an asymptomatic patient begins to manifest signs and symptoms due to progressive narrowing and stiffening of the aortic valve, the only effective therapy is surgical replacement of the valve. But, some patients cannot undergo or do not opt for surgery. Nurses are challenged by the tenuous balance between the narrow range of preload and afterload to maintain forward blood flow and adequate cardiac output in patients with severe aortic stenosis. Understanding the complex normal anatomy and physiology of the aortic valve can help nurses appreciate the consequences of this type of stenosis. Nursing care for patients with aortic stenosis requires advanced skills in patient assessment and an appreciation of the hemodynamic responses to activities of daily living and to nursing interventions such as administration of medications. (*Critical Care Nurse*. 2013;33[2]:58-72)

Aortic stenosis is caused by narrowing of the orifice of the aortic valve and leads to obstruction of left ventricular outflow. This stenosis is rare in persons less than 50 years old.¹ Calcification of the aortic valve is the most common cause of aortic stenosis in adults in industrialized countries and affects more than 4% of North American and Europeans more than 75 years old.² In a study³ of 338 North American patients with severe asymptomatic aortic stenosis, the mean age was 71 (SD, 15) years. Aortic stenosis was also associated with higher morbidity and mortality rates than were diseases involving other cardiac valves.⁴ For example, in a study⁵ of 161 patients, patients with moderate and

CNE Continuing Nursing Education

This article has been designated for CNE credit. A closed-book, multiple-choice examination follows this article, which tests your knowledge of the following objectives:

1. Describe the pathophysiology of aortic stenosis
2. Identify clinical manifestations of aortic stenosis
3. Discuss medical and nursing management of nonsurgical patients with aortic stenosis

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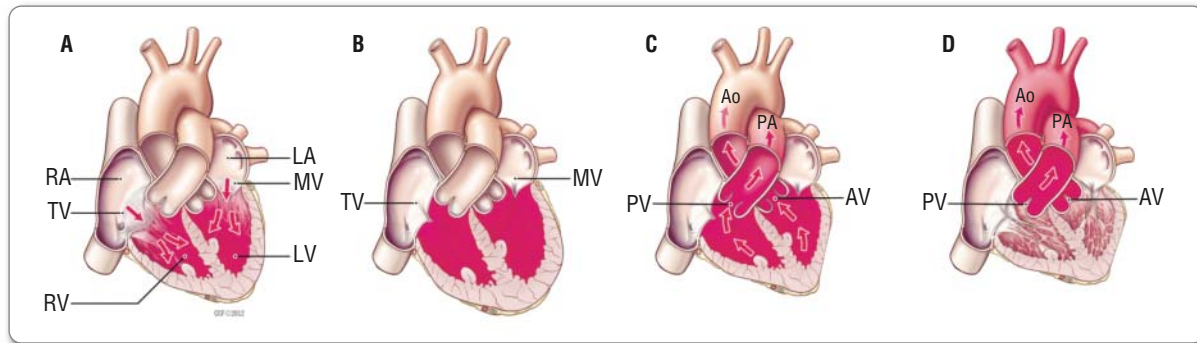


Figure 1 Normal heart valve function. All 4 valves open and close in response to pressure changes during diastole and systole to ensure forward progression of blood flow through the heart. A, Open tricuspid and mitral valves. In early and mid diastole, blood flows passively into the right and left ventricles through the tricuspid and mitral valves, respectively. In late diastole, the right and left atria contract. B, Closed tricuspid and mitral valves. In early systole, increasing ventricular pressures force the tricuspid and mitral valves to close. All 4 valves are closed briefly as the increase in ventricular pressure continues in response to ventricular contraction and twist (isovolumetric contraction). C, Open pulmonic and aortic valves. During mid systole, when ventricular pressures exceed pulmonic and aortic pressures, the pulmonic and aortic valves are forced to open, and blood is ejected into the pulmonary vasculature and aorta, respectively. D, Closed pulmonic and aortic valves. In late systole, ventricular muscle begins to relax and untwist. Back pressure against the pulmonic and aortic valves force the valves to close (isovolumetric relaxation).

Abbreviations: Ao, aorta; AV, aortic valve; LA, left atrium; LV, left ventricle; MV, mitral valve; PA, pulmonary artery; PV, pulmonic valve; RA, right atrium; RV, right ventricle; TV, tricuspid valve.

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severe aortic stenosis had 2-year mortality rates of 40.2% and 58.2%, respectively. In another study⁶ of 274 medically managed patients with severe aortic stenosis, 66.4% of whom had concomitant coronary artery disease, the cardiac related mortality rate in the median follow-up period of 377.5 days was 43.1%, including a sudden cardiac death rate of 3.9%.

Aortic stenosis is increasing in prevalence as the average lifespan continues to increase.^{7,8} In the prospective Cardiovascular Health Study⁹ of 5201 patients more than 65 years old, 26% had aortic sclerosis, a thickening or calcification of the valve without marked left ventricular obstruction, and 2% had aortic stenosis. By age 85, 48% had aortic sclerosis, and 4% had frank aortic stenosis.

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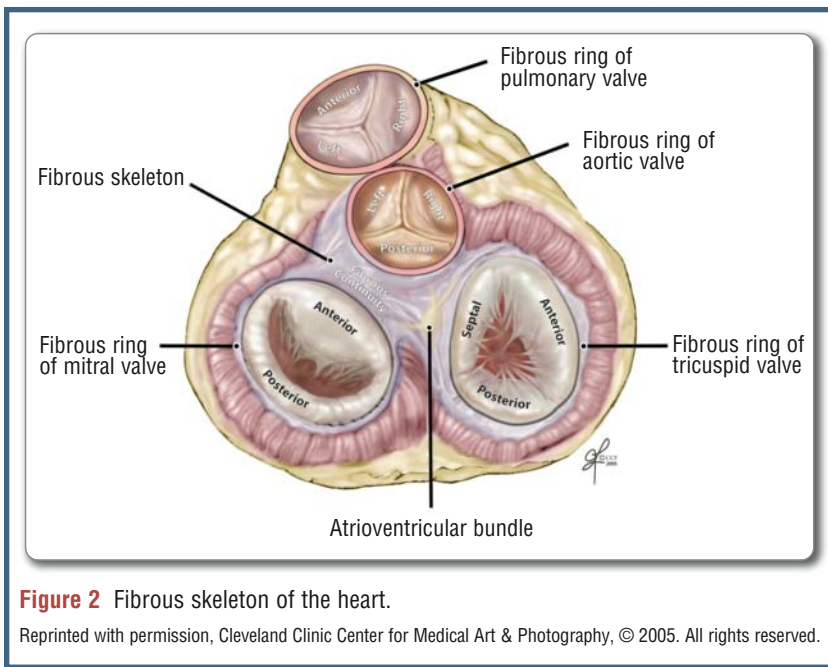
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In this article, we briefly review normal aortic valve anatomy and function and contrast normal function with the structural and functional changes associated with aortic stenosis. We also discuss the signs, symptoms, and physical examination findings associated with aortic stenosis; diagnosis and diagnostic studies; medical management of asymptomatic and symptomatic patients with aortic stenosis; and nursing considerations for patients with aortic stenosis.

Normal Heart and Valve Function

The aortic valve is 1 of 4 valves separating the 4 chambers of the heart. Each valve has leaflets that open easily and close fully in response to pressure changes produced during systole and diastole to ensure forward progression of blood through the heart. An increase in forward pressure across a valve forces the leaflets to open. An increase in backward pressure against a valve forces the leaflets to close¹⁰ (Figure 1). The valves are stabilized and supported by the fibrous skeleton, a sheetlike structure of dense fibrous connective tissue that separates the atria from the ventricles and encircles each valve, creating a ring or annulus¹¹ (Figure 2). The annulus acts as an anchor to the heart muscle.¹¹

Normal systole involves myocardial contraction and rotation or twist. A brief clockwise rotation of the apex



and a counterclockwise rotation of the base occur just before systole as left ventricular pressure increases (known as isovolumetric contraction). This movement is followed by a sustained counterclockwise rotation of the apex and a clockwise rotation of the base during the ventricular ejection phase to essentially wring blood content from the left ventricle^{2,12} (Figure 3). Ventricular twist augments ejection of blood through the aortic valve and into the aorta and reduces myocardial oxygen demand.¹² Diastole involves myocardial relaxation and progressive untwisting, producing a suction effect that pulls blood into the left ventricle.¹²

Closure of the mitral and tricuspid valves marks the onset of systole and produces a sound known as S_1 , best auscultated at the fifth intercostal space, left midclavicular line. Closure of the pulmonic and aortic valves marks the end of systole and produces a sound known as S_2 , best auscultated at the second intercostal space at the left or right sternal border.

Normal Anatomy and Physiology of the Aortic Valve

The aortic valve separates the left ventricle and the aorta. The valve is a complex structure with 3 relatively equal-sized leaflets and an annulus.¹¹ Each leaflet has a cup-shaped body with a top edge (free margin) and a base.¹¹ The leaflets open easily during systole to allow blood to eject from the left ventricle into the aorta and close fully

during diastole to prevent regurgitation of blood from the aorta back into the left ventricle (Figures 4 and 5). To enhance the integrity of the aortic valve when closed, the leaflets abut at a thickened area slightly below their free margins.^{10,11}

The aortic valve leaflets have 3 unique layers that synergistically contribute to valve function and competence.¹³ Each layer contains valvular interstitial cells that help maintain valve structure and function, inhibit angiogenesis in the leaflets, and repair cellular damage.^{13,14} The layer facing the aorta is the fibrosa, made primarily of collagen fibers that help evenly distribute the pressure load on the leaflet's surface.¹¹

Facing the left ventricle is the ventricularis, made primarily of elastic fibers that help maintain the leaflet's shape. The soft middle layer, the spongiosa, has glycosaminoglycans and proteoglycans that cushion and minimize

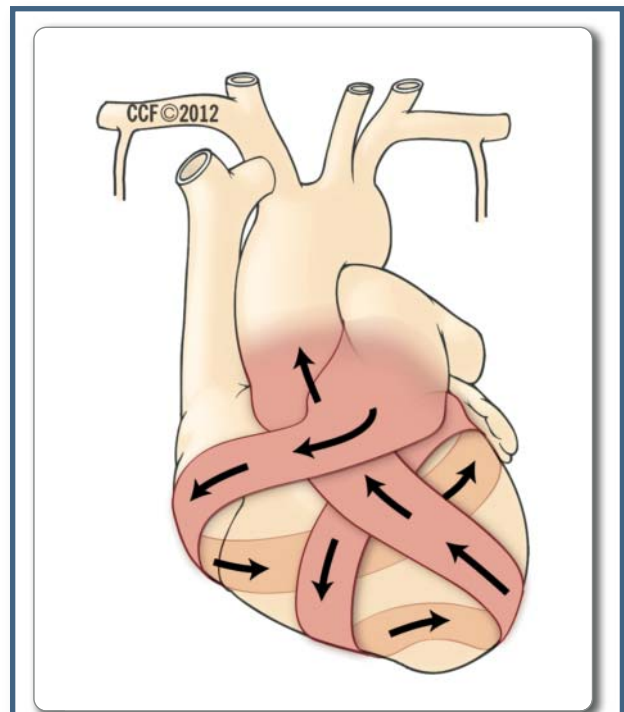


Figure 3 Twisting rotation of the heart during systole.

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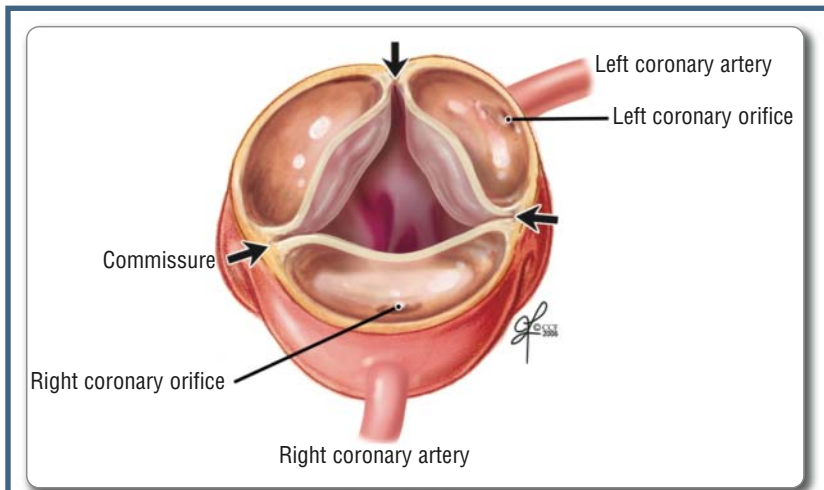


Figure 4 Normal aortic valve in the open position.

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bulging shape of the sinuses creates space behind the aortic valve leaflets during systole that prevents obstruction of blood flow into the coronary arteries. The space also provides a reservoir for pooling of blood during diastole for filling the coronary arteries.^{10,11} The base of each leaflet joins the fibrous skeleton of the heart to form an annulus that anchors the leaflet structure to the aortic wall at the level of the left ventricular outflow tract.¹¹

Aortic Stenosis

Aortic stenosis can be viewed on a continuum from aortic sclerosis to

severe aortic stenosis. Progression of stenosis is associated with increasing obstruction of blood flow through the left ventricular outflow tract and occurs over many years.^{1,8} Only 10% of patients with aortic sclerosis advance to hemodynamically important aortic stenosis.¹⁵ In aortic sclerosis, mild valve thickening or calcification affects normal leaflet motion.^{7,13} As the disease progresses, leaflets become thicker, calcium nodules form, and new blood

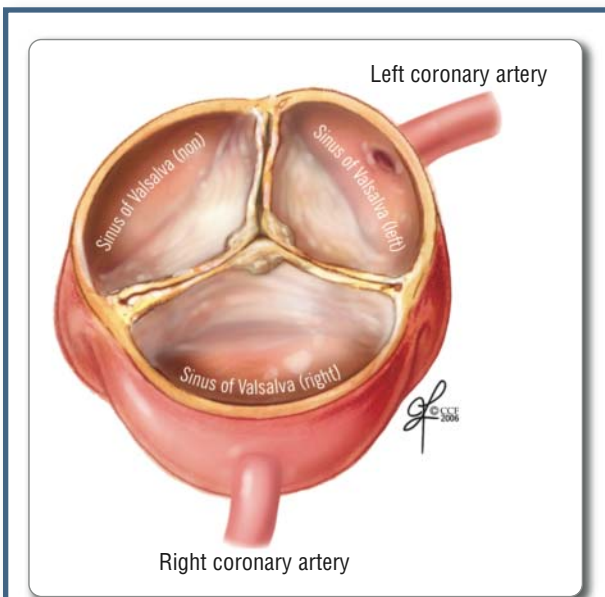


Figure 5 Normal aortic valve in the closed position.

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friction and stress-related damage between the fibrosa and the ventricularis^{10,11} (Figure 6).

The leaflets are joined, edge to edge, by dense collagen fibers called commissures (Figure 4). The commissures penetrate into the aortic wall, where they absorb some of the stresses of systole and diastole.¹¹ Behind each leaflet the aortic wall bulges outward to form the 3 sinuses of Valsalva (Figure 5). Two of the sinuses provide the points of origin for the right and left coronary arteries. The

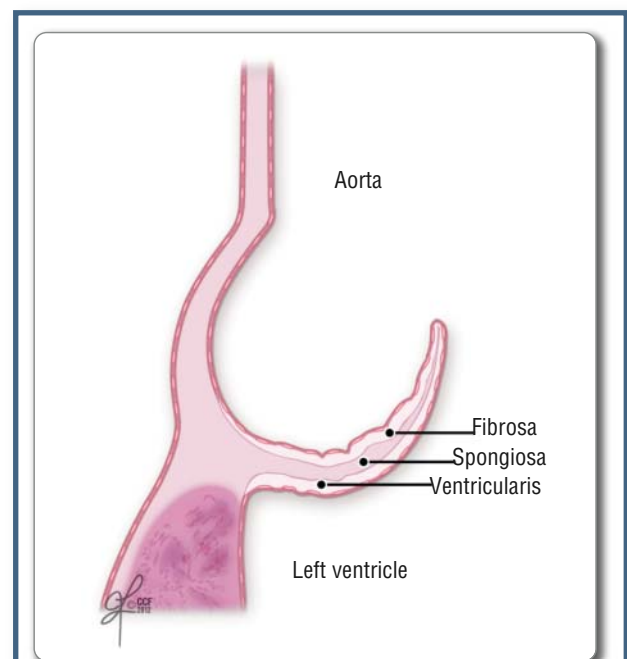


Figure 6 The 3 layers of the aortic valve leaflet.

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vessels appear.¹³ In aortic stenosis, calcium nodules located within the layers of the leaflet bulge outward toward the aorta and extend to the sinuses of Valsalva, causing restricted leaflet motion and obstruction of left ventricular outflow during systole^{1,13} (Figure 7). The 1% to 2% of adults born with 2 aortic valve leaflets, known as bicuspid aortic valve (Figure 8), account for about half of all occurrences of aortic stenosis.¹ Stenosis of a bicuspid aortic valve typically occurs at an earlier age (fifth to sixth decade) than does tricuspid valve stenosis (seventh to eighth decade) because 2 cusps, instead of 3, are forced to absorb the shearing stress of blood flow leaving the left ventricle.⁷

The most common cause of aortic stenosis is valve calcification, termed calcific aortic valve disease (CAVD), which was previously considered a normal consequence of aging.^{7,13} CAVD is an active cellular biological process characterized by alterations of the cells within the layers of the aortic valve. In one proposed mechanism, mechanical stress or disease causes valvular interstitial cells within the valve leaflets to transform from the usual state of maintenance and repair into an activated state in which cell proliferation is increased and myofibroblasts and osteoblasts develop, promoting calcification, osteogenesis, and bone formation.^{13,14,16} In 2 studies^{17,18} of 1524 stenotic aortic valves, bone formation was found in 10.9% to 13% of valve leaflets. In another proposed mechanism, mechanical stress associated with blood crossing the aortic valve damages the basement membrane of the leaflets, allowing entry and accumulation of T lymphocytes, monocytes, and low-density lipoprotein that then initiate inflammation and oxidation of the lipoprotein.^{13,16,19} Rheumatic heart disease, a consequence of untreated pharyngeal infections, rarely causes aortic stenosis in developed countries because of aggressive treatment of penicillin-sensitive streptococcal infections.¹⁹ The events that lead to the onset of aortic stenosis, although unclear, are similar to those associated with early atherosclerosis.

Pathophysiology of Aortic Stenosis

As the aortic valve progresses from sclerosis to stenosis, the left ventricle encounters chronic resistance to systolic ejection. The ventricle must generate a higher systolic pressure than the opposing pressure produced by the unyielding, calcified aortic valve. An increased resistance to systolic ejection is called afterload.⁸ To

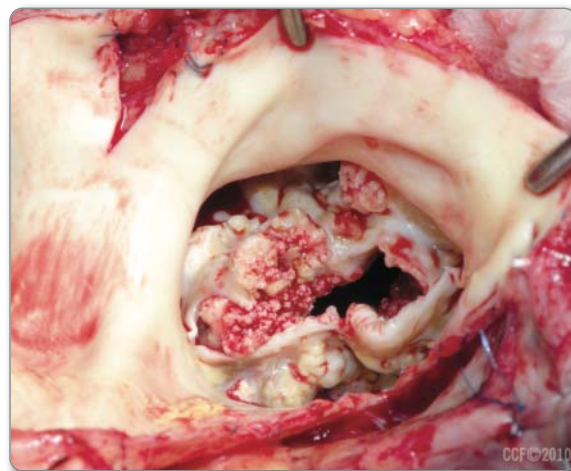


Figure 7 Calcified severely stenotic aortic valve.

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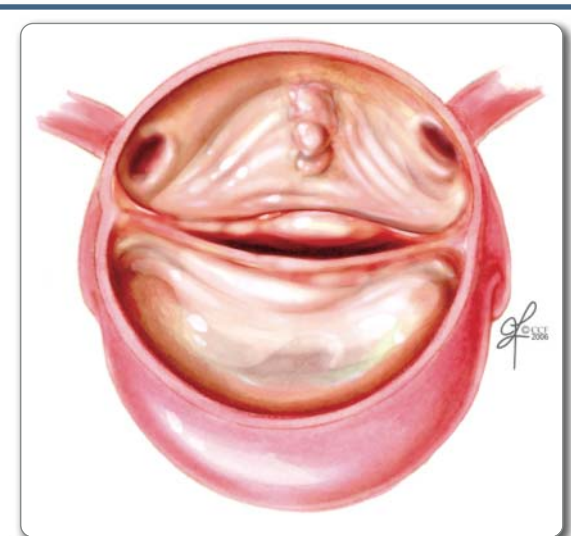


Figure 8 Bicuspid aortic valve.

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compensate for a high afterload, the left ventricular myocardial wall thickens; the diameter of the left ventricle maintains a normal size.⁷ Thickening of the left ventricular wall, known as concentric hypertrophy, strengthens left ventricular systolic contraction to maintain adequate stroke volume and cardiac output.⁷ Table 1 presents hemodynamic parameters and the effects of aortic stenosis.

Although left ventricular hypertrophy is a compensatory mechanism, the sequelae may be detrimental. Effects of high left ventricular afterload include decreased left

Table 1 Hemodynamic parameters and the effects of aortic stenosis^a

Parameter	Definition		
Stroke volume (SV)	Volume of blood ejected from the ventricle with each contraction		
Cardiac output (CO)	Volume of blood ejected from the heart per minute CO = heart rate (per minute) × SV		
Preload	Volume of blood in the ventricle at end diastole (producing a stretch of ventricular muscle cells)		
Afterload	Resistance the heart must overcome to eject blood from the ventricle		
Systemic vascular resistance (SVR)	Resistance to blood flow in all systemic vasculature		
	Reflects	Normal range	Effects of moderate to severe aortic stenosis
Right atrial pressure	Right ventricular preload	2-7 mm Hg	Increases
Pulmonary artery (PA) pressure	Pressures in the pulmonary vasculature	Systolic 15-30 mm Hg Diastolic 4-12 mm Hg	Increases when PA systolic pressure >60 mm Hg (severe pulmonary hypertension)
Pulmonary artery occlusion pressure	Mean left atrial pressure (indirect reflection of LV preload)	2-12 mm Hg	May increase
Left ventricular pressure (LVP)	LV afterload (systolic) LV preload (diastolic)	Systolic 90-140 mm Hg Diastolic 5-12 mm Hg	Increases
Aortic pressure (AP)	SVR and preload	Systolic 90-140 mm Hg Diastolic 60-90 mm Hg	Decreased preload causes decreases in LVP and AP, increased SVR Increased preload causes increased LVP to maintain AP
Systemic vascular resistance (SVR)	LV afterload	700-1600 dynes · sec · cm ⁻⁵	Increases
Pulmonary vascular resistance	Resistance to blood flow in pulmonary vasculature	20-130 dynes · sec · cm ⁻⁵	Increases
Cardiac output/resting	Volume of blood ejected from the heart per minute	5-8 L/min	Decreases

^a Based on information from Otto and Bonow.⁸

ventricular myocardial elasticity and coronary blood flow and increased myocardial workload, oxygen consumption, and mortality.^{2,7} Left ventricular hypertrophy increases diastolic pressure and delays left ventricular untwisting; thus, a forceful atrial contraction (commonly called atrial kick) is needed for optimal filling of the left ventricle to maintain stroke volume and cardiac output.^{4,7} Late manifestations of left ventricular hypertrophy include a smaller left ventricular chamber size, which decreases preload and worsens systolic dysfunction. The result is insufficient stroke volume, cardiac output, and ejection fraction.^{1,7,15} Finally, backward transmission of increased left ventricular pressure to the lungs may cause pulmonary venous hypertension and reactive vasoconstriction of the pulmonary vasculature.^{1,20}

As a result of the detrimental effects associated with left ventricular hypertrophy, patients with aortic stenosis become increasingly dependent on atrial kick to maintain stroke volume and cardiac output. Loss or

compromise of atrial kick as a result of atrial fibrillation, ventricular pacing, and/or intravascular fluid volume overload may precipitate pulmonary congestion, hypotension, and angina.^{7,21,22} Atrial arrhythmias may result from an extension of calcific infiltrates from the aortic valve into the conduction system.^{1,10,11} In one study,²² chronic atrial fibrillation was predictive of heart failure and stroke and new-onset atrial fibrillation was associated with cardiac decompensation (see Case Report).

Grading of Aortic Stenosis

Aortic stenosis is graded as mild, moderate, or severe. Grading is based on 3 hemodynamic parameters measured by using Doppler echocardiography: aortic jet velocity, mean aortic valve pressure gradient, and aortic valve area^{7,15} (Table 2). Aortic jet velocity is blood flow measured at the narrowest orifice of the aortic valve during systole.²³ Aortic jet velocity is a direct measurement of the severity of stenosis and is the strongest predictor

Case Report

M r S was 84 years old, 178 cm tall, and weighed 72 kg. He came to the emergency department because of increasing shortness of breath, intermittent chest pressure, and dyspnea on exertion for the past 3 weeks. He stated that he had slept in his recliner for the past 2 nights because of the increasing shortness of breath. Vital signs on admission were heart rate 148 beats per minute, respiratory rate 24 breaths per minute, oxygen saturation 93% on 6 L of oxygen via nasal cannula, and blood pressure 109/59 mm Hg. A 12-lead electrocardiogram revealed rapid atrial fibrillation. Mr S's medical history included hypertension, multivessel coronary artery disease, hypercholesterolemia, dilated cardiomyopathy, and aortic stenosis with an aortic valve area of 0.6 cm². He had been evaluated for aortic valve replacement 6 months earlier, but he refused to have surgery. Two attempts to cardiovert him from atrial fibrillation to sinus rhythm were unsuccessful. He was given aspirin 325 mg orally and amiodarone 150 mg intravenously followed by continuous infusion at 1 mg/min. A nitroglycerin infusion was started at 20 µg/min. He received furosemide 80 mg intravenously to promote diuresis and heparin 5000 IU subcutaneously. Admission laboratory studies included electrolyte levels, coagulation studies, and serum level of brain natriuretic peptide. The results were normal except for the level of brain natriuretic peptide, which was 2800 pg/mL (reference range, <130 pg/mL). Chest radiography revealed pulmonary congestion. During the preceding 30 minutes, his respiratory rate had increased to 32 breaths per minute, with worsening shortness of breath and oxygen saturation levels of 86% to 88% on 100% oxygen via a nonrebreather mask. He was intubated and admitted to the coronary intensive care unit. The diagnosis was decompensated heart failure, severe aortic stenosis, and uncontrolled atrial fibrillation. On arrival to the unit, his heart rate was 89 beats per minute and his blood pressure was 112/78 mm Hg. Ventilator settings were assist control at a respiratory rate of 12 breaths per minute and fraction of inspired oxygen 0.50. A pulmonary artery catheter was placed, and hemodynamic values were measured and calculated: pulmonary artery pressure 78/36 mm Hg, right atrial pressure 21 mm Hg, pulmonary artery occlusion pressure 29 mm Hg, cardiac output 2.89 L/min, and systemic vascular resistance 1882 dynes · sec · cm⁻⁵. An additional 40 mg of furosemide was given intravenously, and sodium nitroprusside 5 µg/min was started to decrease blood pressure and reduce the systemic vascular resistance. ■

Table 2 Grades of aortic valve stenosis^a

Grade	Aortic jet velocity, m/s	Mean aortic valve pressure gradient, mm Hg	Aortic valve area, cm ²
Mild	<3.0	<25	1.5
Moderate	3-4	25-40	1.0-1.5
Severe	>4	>40	<1.0

^a Based on information from Bonow et al.²¹

of clinical outcome.²⁴ The narrowed orifice produces a nozzle effect as blood jets through the valve opening; the narrower the orifice, the faster the speed (velocity).⁷ Mean aortic valve pressure gradient is the difference between the higher left ventricular pressure and the lower aortic pressure measured just above the aortic valve during systole. The gradient indicates the degree of valvular resistance to left ventricular ejection.⁷ The aortic valve area is based on measurements taken across

the aortic valve. This parameter is more susceptible to measurement error than are jet velocity and pressure gradient.^{1,23}

Although grading of aortic stenosis relies on validated measurements of aortic jet velocity, pressure gradient, and aortic valve area, no single value defines severity, and the rate of progression of aortic stenosis

is impossible to calculate.²¹ A normal aortic valve is 3.0 to 4.0 cm² in area (about the size of a nickel).^{4,7} Aortic stenosis is considered hemodynamically important when the valve area is less than 1.0 cm² (about the size of the head of a golf tee)⁴; however the degree of obstruction resulting in signs and symptoms is widely variable.^{8,24} For that reason, severe aortic stenosis is defined as “the degree of valve obstruction at which symptoms might be caused by valve obstruction.”²⁴

Table 3 Clinical manifestations of aortic stenosis^a

Clinical manifestation	Causes	Significance
Decreased exercise tolerance due to exertional dyspnea or fatigue	Diastolic dysfunction Decreased cardiac output with exercise	If early indications of aortic stenosis are not recognized, can delay diagnosis and treatment
Angina	Increased left ventricular workload and oxygen consumption	May occur with or without coexisting coronary artery disease Commonly precipitated by exertion and relieved with rest Mean survival after symptom onset 5 years if no surgical repair of aortic valve
Syncope	May be precipitated by high left ventricular pressures causing acute baroreceptor-activated vasodilation leading to decreased cardiac output or by an inability to increase stroke volume, when needed, through a narrow, stiff aortic valve	Usually occurs during exercise Mean survival after symptom onset 3 years if no surgical repair of aortic valve
Heart failure	Diastolic dysfunction resulting in pulmonary congestion and dyspnea	Most ominous symptom of aortic stenosis Mean survival after symptom onset 2 years if no surgical repair of aortic valve

^a Based on information from Carabello and Paulus.⁷

Clinical Manifestations

The classical clinical manifestations of angina, syncope, and heart failure do not occur until late in aortic stenosis.¹⁵ Because of the prolonged latency period of asymptomatic disease progression, patients are often unaware of their condition until a systolic murmur is detected during a physical examination, evaluation of new onset of atrial fibrillation, or cardiac catheterization for symptomatic coronary artery disease. Patients' typical initial descriptions include decreased exercise tolerance, dyspnea on exertion, exertional dizziness, and lightheadedness²⁴ (Table 3). Many patients do not recognize the initial manifestations of aortic stenosis because of the gradual change in hemodynamic status. Decreased exercise tolerance manifested as exertional dyspnea or fatigue has been attributed to cardiac ischemia, elevated left ventricular end-diastolic pressure, and decreased cardiac output.¹ Angina may occur in patients with CAVD as a consequence of coronary artery disease.¹ In patients without coronary artery disease, angina may be due to decreased subendocardial blood flow and/or increased myocardial oxygen demand associated with concentric hypertrophy.^{2,25} Blood flow to the myocardium may be limited by insufficient capillary density into the hypertrophied left ventricular muscle and/or by endocardial compression due to increased filling pressures.^{7,8}

Syncope occurs because of decreased cerebral perfusion associated with decreased cardiac output or during exercise and times of decreased preload, such as after

arising from a seated position; dehydration; and use of diuretics.^{1,7} Normally, exercise should cause blood pressure to increase and systemic vascular resistance to decrease, and because the increase in blood pressure is greater than the decrease in systemic vascular resistance, stroke volume and cardiac output increase.⁷ The normal response to exercise may not occur in patients with aortic stenosis because the narrowed aortic valve orifice may limit the augmented stroke volume necessary to counterbalance the decrease in systemic vascular resistance.⁷ Another possible explanation of syncope in patients with aortic stenosis is that high intraventricular pressure produced during exercise prompts an inappropriate left ventricular baroreceptor reflex, resulting in vasodilation leading to a decrease in cardiac output.¹

The gold standard for diagnosing aortic stenosis is noninvasive 2-dimensional Doppler echocardiography.

In aortic stenosis, signs and symptoms of heart failure include exertional dyspnea, paroxysmal nocturnal dyspnea, orthopnea, and pulmonary congestion. Symptoms can occur when forward blood flow from the pulmonary vasculature encounters high diastolic pressure in the left ventricle.^{7,26} Delayed active myocardial relaxation during early diastole decreases left ventricular filling time; thus, the blood volume required to provide adequate distending pressure required by the stiff left ventricular chamber is not met.⁷ Typical indications of congestion

Table 4 Diagnostic studies in aortic stenosis^a

Study	Purpose
Doppler echocardiography	Estimation of severity of aortic stenosis, left ventricular size, and ejection fraction Estimation of pulmonary pressures, aortic valve gradient, aortic valve area Assessment of thickening of aortic valve leaflet, reduced leaflet motion, reduced valve opening
Cardiac catheterization	Assessment of coronary arteries to determine need for simultaneous coronary artery bypass surgery and aortic valve replacement Direct measurement of left ventricular and ascending aortic pressures to determine aortic valve pressure gradient Determination of left ventricular systolic pump function quantified by measuring left ventricular end-diastolic and end-systolic volumes, and ejection fraction
12-Lead electrocardiography	Evidence of left ventricular hypertrophy: Increased R-wave amplitude of the QRS complex in lead V ₆ , increased S-wave amplitude in lead V ₁ ST-segment depression and T-wave inversion in leads facing the left ventricle: I, aVL, V ₅ , and V ₆
Chest radiography	Determination of heart size Detection of calcification in the aortic valve (lateral view) With heart failure, enlarged heart size from dilatation of left atrium and left ventricle, venous congestion, and pulmonary edema
Stress testing	Determination of the degree of exercise tolerance Distinguish between asymptomatic and symptomatic aortic stenosis
Brain natriuretic peptide	Determination of severity of increased left ventricular pressure and volume overload Distinction between cardiac and noncardiac dyspnea

^a Based on information from Kurtz and Otto,¹ Mookadam et al,²⁷ and Bergler-Klein.²⁸

in heart failure may include jugular vein distension and pulmonary rales.¹⁹

Palpation of the carotid artery and auscultation of heart sounds provide valuable insight in patients with aortic stenosis. Careful palpation of a carotid artery can reveal indications of the resistance of the calcified aortic valve to opening, subsequent delay in left ventricular ejection, and decreasing volume.¹ Gentle pressure to the right carotid artery slightly above the clavicle reveals a slowly increasing carotid upstroke that takes longer to reach peak pressure (pulsus tardus) and weaker pulse amplitude.⁷ In elderly patients, age-related changes in arterial compliance and stiffness can mask carotid changes associated with severe aortic stenosis, causing carotid artery upstroke and amplitude to appear normal.^{4,21}

Turbulent blood flow through the aortic valve can be heard as a systolic ejection murmur that peaks in early systole in mild aortic stenosis and progressively later as aortic stenosis becomes more severe.^{1,7} The crescendo-decrescendo late-peaking murmur is heard best at the upper right sternal boarder at the second intercostal space and may radiate to the carotid arteries.^{1,4} In older patients, the murmur may be less intense and may radiate to the apex of the heart rather than to the base.⁴

Diagnosis and Diagnostic Studies

The gold standard for diagnosing aortic stenosis is noninvasive 2-dimensional Doppler echocardiography¹ (Table 4). Findings on physical examination and 2-dimensional Doppler echocardiography can usually indicate the extent and severity of aortic stenosis. Cardiac catheterization provides an invasive, direct measurement of intracardiac and aortic pressures.⁷ Catheterization becomes necessary only when noninvasive data are inconclusive or do not support clinical findings and before surgical aortic valve repair in patients who are at risk for coronary artery disease.²¹ Tests that can provide support for a diagnosis of aortic stenosis include 12-lead electrocardiography and chest radiography.⁷

Exercise stress tests are contraindicated in patients with symptomatic aortic stenosis but may be considered in asymptomatic patients to assess for underlying signs and symptoms.^{1,7,21} Many patients who report they have no symptoms become symptomatic for the first time when subjected to a stress test.³ The stress test should be supervised by an experienced physician with close observation of the electrocardiographic tracings and blood pressure.²¹ During exercise, patients with aortic stenosis may experience signs or symptoms such as hypotension

or failure to develop the usual increase in blood pressure. Abnormal hemodynamic responses to exercise should prompt a change in a patient's status from asymptomatic to symptomatic.⁷ Brain natriuretic peptide (BNP) is a peptide hormone released from ventricles in response to increased ventricular pressure.⁷ Serum levels of BNP increase in patients with asymptomatic aortic stenosis shortly before the onset of signs and symptoms, and higher levels correlate with the severity of the signs and symptoms.^{7,28} Patients with a serum baseline BNP greater than 130 pg/mL are likely to become symptomatic within 6 months, and BNP greater than 550 pg/mL is predictive of a poor outcome.²⁸

Medical Management of Asymptomatic Patients

Currently no known medical therapy is available to prevent CAVD or delay the progression of aortic stenosis.^{21,24} Treatment focuses on reducing cardiovascular risk factors, including hypertension, diabetes mellitus, smoking tobacco, high cholesterol levels, overweight, and lack of exercise.²⁴ Periodic evaluation by a health care provider includes echocardiographic monitoring and education about progression of aortic stenosis, recognition of signs and symptoms of worsening aortic stenosis, and prompt reporting of the signs and symptoms at the onset.^{1,19,24} Having patients compare current activity level with past activity level may indicate if usual activity has been altered to avoid signs and symptoms.²⁴ Physical activity is not restricted in mild aortic stenosis, but competitive sports should be avoided by patients with moderate to severe aortic stenosis.²¹

Guidance for medication therapy is limited and is primarily based on expert consensus. Statin therapy has been evaluated as a means of retarding progression of valvular stenosis. In some studies,²⁹⁻³¹ statins were effective in slowing the progression of aortic stenosis, but the results of larger randomized controlled trials³²⁻³⁴ did not support those findings. Current guidelines recommend statin therapy for patients with aortic stenosis and hypercholesterolemia to reduce cardiovascular events.^{1,21} Antibiotic prophylaxis before dental and other invasive procedures was standard therapy for patients with aortic stenosis until recently. Currently, antibiotic prophylaxis is indicated solely for patients with rheumatic aortic stenosis, to prevent recurrent rheumatic fever.²¹ The changes in the guidelines were based on newer evidence that bacteremia from routine activities such as tooth

brushing, flossing, and chewing occurred more often than did bacteremia related to dental procedures.⁸ Thus, maintaining optimal oral health and hygiene and routine dental care convey the greatest risk reduction. Further, controlled studies indicating that endocarditis was prevented by short-term antibiotic therapy are lacking; the risk of antibiotic therapy outweighs potential benefit.⁸

The prevalence of patients with hypertension and aortic stenosis is high. In a study³⁵ of 1873 patients with asymptomatic aortic stenosis, 50.9% had hypertension. No clear management guidelines are available beyond starting antihypertensive medications at low doses and titrating up to the target doses used in randomized controlled trials, while monitoring blood pressure and signs and symptoms of the stenosis.^{7,21} Hypertension in patients with aortic stenosis contributes to the increased workload of the hypertrophied left ventricular during systole by increasing left ventricular afterload.⁴ Treatment must be expertly guided in patients sensitive to hemodynamic changes, because inappropriately high doses of antihypertensive medication can result in hypotension and exacerbation of heart failure.^{1,7}

Vasodilators are the preferred therapy for treatment of hypertension.¹ Angiotensin-converting enzyme inhibitors cause vasodilatation by inhibiting the formation of angiotensin II, a potent vasoconstrictor, and are well tolerated in patients with moderate aortic stenosis.^{7,19}

In a recent retrospective study,²⁵ patients with mild, moderate, and severe

Many patients do not recognize the initial manifestations of aortic stenosis—patients' typical initial descriptions include decreased exercise tolerance, dyspnea on exertion, exertional dizziness, and lightheadedness.

aortic stenosis who received angiotensin-converting enzyme inhibitors or angiotensin receptor blockers had lower all-cause mortality and cardiovascular event rates during a mean follow-up of 4.2 years than did patients who did not receive these medications.²⁵

β -Blockers are not routinely used in patients with aortic stenosis and have been considered unsafe because they depress myocardial function and can induce left ventricular failure.^{1,7} However, a retrospective study³ of the use of β -blockers in patients with asymptomatic severe aortic stenosis who were nonsurgically managed indicated that use of β -blockers was an independent predictor of improved survival. The investigators³ suggested

Table 5 Surgical interventions for aortic stenosis^a

Procedure	Indication	Description
Aortic valve replacement	Symptomatic severe aortic stenosis Severe aortic stenosis with ejection fraction <50% Severe aortic stenosis and a need for any other heart surgery	Aortic valve is removed and a new valve (mechanical or biological) is sewn to the annulus of the native valve
Balloon aortic valvuloplasty	Bridge to aortic valve replacement in patients in unstable condition Palliative to reduce symptoms when surgery is high risk	A balloon is placed across the stenotic valve and inflated and deflated several times per second to widen the valve annulus and reduce degree of stenosis
Transcatheter aortic valve implantation	Seriously ill patients who are not candidates for conventional aortic valve replacement surgery	Replacement valve inside a stent that is deployed over the diseased aortic valve annulus via a transapical or transcatheter approach

^a Based on information from Mookadam et al.²⁷

that β -blockers may prevent or attenuate atrial fibrillation and other poorly tolerated tachyarrhythmias. Patients with aortic stenosis who are taking antihypertensive medications may require periodic decreases in the dosage to prevent hypotension as the aortic valve progressively narrows.⁸ Prognosis is good for patients with moderate to severe aortic stenosis who remain asymptomatic, but once even mild signs and symptoms appear, life expectancy is limited to 2 to 5 years.¹⁹

Medical Management of Symptomatic Patients

Once severe aortic stenosis has been diagnosed, retrospective analyses²⁴ reveal that the onset of signs and symptoms can be anticipated within 5 to 10 years. After onset, without surgical intervention, the mean life expectancy is 2 to 3 years²¹ (Table 5). Surgical repair is the only effective treatment for symptomatic aortic

stenosis; however, some patients may not be consid-

For patients who do not have surgical repair, medical management of angina, exertional syncope, and signs and symptoms of heart failure becomes necessary.

ered surgical candidates or may require medical stabilization before surgery; other patients refuse surgical options altogether.^{6,25} For patients who do not have surgical repair, medical management of angina, exertional syncope, and signs and symptoms of heart failure becomes necessary.

Treating angina in patients with severe aortic stenosis is a challenge. Among patients with aortic stenosis, the 20% to 60% who experience angina also have coronary

disease, making it difficult to determine the cause of the angina.^{4,8} Although little information is available to guide therapy, treatment strategies and goals for angina relief in nonsurgical patients include bed rest, oxygen therapy, use of β -blockers to decrease oxygen consumption, and treatment with nitrates to enhance oxygen delivery via dilatation of the coronary arteries.²¹ β -Blockers can help restore balance to myocardial oxygen supply and demand by blocking the cardiac β_1 receptors responsible for increasing heart rate and contractility.¹⁹ β -Blockers and nitrates must be used cautiously because of the risk of decreasing preload and systemic blood pressure in patients who are preload dependent.²¹ Low-dose intravenous nitroglycerine or low-dose sublingual nitroglycerine tablets (200 μ g) may be preferred over the more commonly prescribed 400- μ g tablets.

Syncope usually occurs during exercise and is not specifically treated after the event ends, except to encourage rest, unless the syncope is due to an arrhythmia.⁷ If the syncope is associated with a tachyarrhythmic or bradyarrhythmic event, antiarrhythmic medications or implantation of a pacemaker and/or an internal cardiac defibrillator may be indicated. New-onset symptomatic atrial fibrillation is treated with prompt cardioversion.^{7,8}

Pulmonary congestion caused by heart failure is treated with digitalis, diuretics, and an angiotensin-converting inhibitor or angiotensin-receptor blocker, with careful avoidance of an excessive reduction in preload that could precipitate hypotension and decreased cardiac output.²¹ Diuretic therapy is used with the utmost of care because it can precipitate life-threatening hemodynamic compromise in patients with aortic stenosis, who are so dependent on preload.¹ This is particularly true

Ten hours after admission to intensive care, Mr S's blood pressure was 98/51 mm Hg; he was receiving sodium nitroprusside 50 µg/min. Cardiac output increased to 3.6 L/min and systemic vascular resistance decreased to 1180 dynes · sec · cm⁻⁵. Urine output was 1500 mL, respirations were 16 breaths per minute, and oxygen saturation was greater than 98%. A trial of continuous positive airway pressure was started. Mr S tolerated the intervention with no increase in respirations and no decrease in oxygen saturation. Echocardiography revealed an ejection fraction of 15% (normal 55%-70%). The left ventricle appeared concentric, with severely decreased left ventricular function. Mr S also had severe aortic stenosis, with a peak gradient of 80 mm Hg (mean gradient 48 mm Hg). The area of the aortic orifice was 0.6 cm². On auscultation, a harsh systolic ejection murmur was heard at the second intercostal space at the right sternal border. Eleven hours after admission to the cardiac unit, Mr S was extubated and started on 6 L of oxygen via nasal cannula. A multidisciplinary team that included the cardiologist, cardiothoracic surgeon, clinical nurse specialist, and bedside nurse met with Mr S and his family to discuss management options. Mr S insisted that he still did not want surgery or any other intervention and requested that a do-not-resuscitate order be placed in his record. Treatment was started with oral amiodarone 400 mg 3 times per day and warfarin 5 mg daily for atrial fibrillation. Nitroglycerin and sodium nitroprusside infusions were titrated off as oral medications (isosorbide dinitrate 10 mg 3 times per day and captopril 25 mg 3 times per day) were started. Mr S continued taking medications that he had been taking at home: digoxin 0.125 mg daily, furosemide 20 mg daily, and pravastatin 40 mg daily. The pulmonary artery catheter was removed. ■

in elderly women, who tend to have an especially small, hypertrophied ventricle.⁸

Decompensated heart failure caused by severe left ventricular systolic dysfunction and concomitant hypertension can be treated with sodium nitroprusside, a potent intravenous vasodilator, in an intensive care unit with invasive hemodynamic monitoring (pulmonary artery catheter) to guide treatment.^{21,36} Intra-aortic balloon pump therapy may enhance afterload reduction. Such strategies to decrease resistance to left ventricular emptying can improve cardiac output, optimize cardiac function before aortic valve surgery, and provide a bridge from intravenous vasodilators to oral vasodilators³⁶ (see Case Report, Update 1). Table 6 summarizes treatment strategies and their hemodynamic effects in aortic stenosis.

Nursing Considerations

Caring for medically managed patients with aortic stenosis requires knowledge and understanding of the tenuous balance between the narrow range of preload and afterload necessary to maintain forward blood flow and adequate cardiac output. In the intensive care unit, medication management is based on the desired hemodynamic parameters; a pulmonary artery catheter is used to calculate adequate preload, afterload, and cardiac

output. Hemodynamic considerations must always be weighed when nurses respond to signs and symptoms associated with aortic stenosis, such as when providing general nursing care and activities. Orthostatic hypotension may occur when a patient goes from a supine or seated position to a standing position or after administration of vasodilators such as nitrates or diuretics.

Goals in patients' daily plan of care include balancing rest and activity to maintain oxygen supply and demand and maintaining heart rate, blood pressure, temperature, and fluid volume status within reference ranges. Nurses should monitor patients for potential indications of hemodynamic decompensation associated with activity, such as hypoxia, arrhythmias, changes in blood pressure, shortness of breath, chest pain, and prolonged status of nothing by mouth. Medical tests or procedures that require patients to receive nothing by mouth beforehand should be scheduled early in the day to reduce the possibility of volume depletion that may lead to hemodynamic compromise.¹ Key nursing goals in acute care are resolution of acute signs and symptoms, prevention of deterioration in clinical status, and prevention of new signs and symptoms.

Assessment strategies are tailored to patients with aortic stenosis and include visualization for jugular vein

Table 6 Treatment strategies and hemodynamic effects^a

Treatment	Heart rate	Blood pressure	Preload	Afterload	Contractility	Cardiac output	Nursing considerations
Vasodilators (nitrates)		↓	↓				Low-dose intravenous or low-dose sublingual nitroglycerin; avoid hypotension
β-Blockers	↓	↓			↓	↓ Initially	Controversial in aortic stenosis; decrease in systolic contraction can induce heart failure
Diuretics		↓	↓				Management of pulmonary congestion; avoid hypotension
Angiotensin-converting enzyme inhibitor		↓	↓	↓			Decrease afterload; avoid hypotension
Positive inotrope (digoxin)	↓				↑		May precipitate ventricular arrhythmias
Intra-aortic balloon pump	↓		↓	↓	↑	↑	Patient needs anticoagulation therapy, frequent assessment of pulses/limb perfusion, supine positioning

Abbreviations: ↓, decreased; ↑, increased; blank cells, not applicable.

^aBased on information from Turkoski et al.²⁷ and Aksoy et al.²⁸

distension and auscultation of heart sounds such as S₄ and S₃. Inadequate response to therapy requires prompt attention and collaboration with the medical team to avoid deterioration in clinical status. For example, if afterload reduction strategies (vasodilatation) do not produce sufficient reduction in systemic vascular resistance or blood pressure, titration of vasodilator medications may be considered. Angina unrelieved with oxygen, rest, and nitrate therapy or associated with hypotension may prompt intra-aortic balloon pump therapy to augment coronary perfusion and decrease myocardial workload.

Patients and their families should receive education throughout the hospital stay, including information on

strategies to comply with modifying risk factors for coronary artery disease, as described previously.⁸ Patients with aortic stenosis must be educated to recognize worsening of signs and symptoms and to promptly report the changes to the appropriate health care provider. Symptomatic patients should be evaluated for surgical replacement of the aortic valve.^{8,21} For symptomatic patients who are non-surgical candidates (or who refuse surgery), education should include prevention of worsening symptoms or onset of new symptoms through balanced activity and rest and the impact of medication adherence on cardiac function and pathological changes associated with aortic stenosis. All nonsurgical patients should be assessed for appropriate coping mechanisms and psychosocial support systems and should be referred, as needed, for counseling and discussion of resuscitation status (see Case Report, Update 2).

Conclusion

Medical management of patients with asymptomatic aortic stenosis is challenging. Severe symptomatic aortic stenosis cannot be corrected with medical therapy, but some patients do not desire surgical intervention or meet criteria for surgical repair or replacement of the calcified aortic valve. Acute and critical care nursing care is difficult because of patients' tenuous hemody-

Case Report, Update 2

Mr S remained in stable condition with oral medications and was transferred to the cardiac step-down unit on day 3.

Education for him and his family was focused on management of signs and symptoms, medications, diet, activity, and coping strategies. On day 5, Mr S was discharged home with follow-up instructions to see his physician in 2 weeks and to immediately report any worsening signs and symptoms. ■

dynamic status. Understanding aortic stenosis, including the pattern of progression, hemodynamic challenges, and treatment modalities, will facilitate nurses' recognition of the consequences of underaggressive or overaggressive treatment of the signs or symptoms of angina, syncope, and heart failure that can result in hemodynamic collapse. **CCN**

Financial Disclosures
None reported.

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To learn more about caring for patients with aortic stenosis, read "A New Option for the Treatment of Aortic Stenosis: Percutaneous Aortic Valve Replacement" by Lauck et al in *Critical Care Nurse*, June 2008;28(3):40-51. Available at www.cconline.org.

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