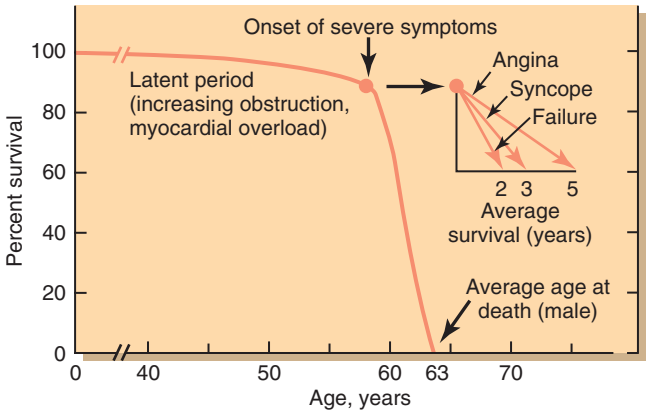

FIGURE 7-1 Natural history of severe aortic stenosis without surgery once symptoms develop. (From Ross J Jr, Braunwald E: Aortic stenosis, Circulation 38:61, 1968)

identified as increased voltages on the electrocardiogram (ECG) and a sustained but nondisplaced point of maximal impulse on palpation. The resistance to blood flow in systole results in the classic harsh crescendo-decrescendo systolic murmur. Although it is typically heard best in the aortic position, the murmur may radiate to the apical region (known as Gallivardin's phenomenon). Because the stiff, calcified, and restricted aortic valve leaflets make little excursion in systole, their closing no longer produces a sound; this results in an inaudible aortic component of S_2. The ventricle cannot quickly eject blood through the small aortic orifice, and the carotid pulses may have a resulting low amplitude (described in Latin as *pulsus parvus*) and delay in reaching their peak (*et tardus*). See Table 7-2 for a summary of physical examination, ECG, and chest radiography findings for chronic valvular heart lesions.

Transthoracic echocardiography (TTE) has become the "gold standard" for confirming the presence of severe aortic valve stenosis. It offers the ability to visualize the valve as well as the use of Doppler imaging to estimate the peak instantaneous and mean valve gradients. Importantly, an estimated valve area can be derived for a more reliable measure of stenosis severity. Criteria for differentiating mild, moderate, and severe stenosis have been published (Table 7-3). In most cases, echocardiography is sufficiently accurate for clinical decision making regarding the valve, but patients may still require invasive coronary or computed tomographic (CT) angiography to exclude obstructive coronary disease before valve replacement. If doubt remains regarding stenosis severity, hemodynamic measurements at the time of cardiac catheterization can confirm the degree of stenosis.

Treatment

For decades, surgical replacement of the aortic valve was the only treatment proven to prolong life in symptomatic severe aortic valve stenosis. Aortic valve replacement (AVR) is a class I indication (level B evidence) for symptomatic patients with severe aortic stenosis. AVR is also a class I indication in patients with asymptomatic aortic stenosis who have LV systolic dysfunction that is believed to be the result of the stenosis (level C evidence). AVR can restore survival rates in these patients almost to normal.

For patients who are deemed to be at acceptable surgical risk, there are two prosthetic options that may be considered for AVR. Mechanical prosthetic valves (see example in Fig. 7-2) have the advantage of excellent flow characteristics; they typically last for the patient's lifetime but require anticoagulation. Bioprosthetic valves (Fig. 7-3), which are made from either porcine or bovine material, have the advantage of not requiring long-term anticoagulation. Because the leaflets are made of biologic tissue, the lifespan and durability of bioprosthetic valves are finite, and valve re-replacement is invariably required 10 to 20 years after implantation.

In those for whom open surgical replacement of their aortic valve would pose an inappropriately high risk, a third option became available in the United States in November of 2012 when the U.S. Food and Drug Administration (FDA) approved the use of a percutaneously placed bioprosthetic valve (Fig. 7-4). In appropriately selected patients, this valve may be delivered via a catheter through the femoral artery to the aortic valve, and then expanded into position by a balloon, effectively squeezing the native valve against the aortic wall. It can also be placed through a small apical incision in the chest wall, entering the heart through the LV apex. Complications may include regurgitation around the prosthesis, stroke, and damage to the peripheral vessels during catheter insertion. Currently, these techniques are approved in the United States only for patients with symptomatic severe stenosis who have been declared ineligible for open surgical valve replacement due to excessive risk.

Prognosis

The mortality risk of asymptomatic severe aortic valve stenosis appears to be very low based on available studies, probably less than 1% per year. However, in sedentary individuals, absence of symptoms can be deceiving. On occasion, it is reasonable to perform carefully monitored exercise stress testing in patients professing to be asymptomatic. Once symptoms have appeared, the survival rate with severe stenosis, as demonstrated by Ross and Braunwald, is abysmal, with about half of those patients who develop heart failure dying within 2 years after symptom onset.

AORTIC REGURGITATION

Definition

When aortic valve leaflets fail to adequately coapt in diastole, blood regurgitates into the left ventricle. As with other compensatory mechanisms of the body, the left ventricle can tolerate large volumes if the progression to severe regurgitation occurs slowly enough. When severe regurgitation develops rapidly, hemodynamic collapse and death can occur. Therefore, the causes, clinical presentation, and management of acute versus chronic severe aortic regurgitation should be considered separately.

Pathology

Aortic regurgitation may occur as a result of leaflet abnormalities, aortic root disease, or a combination of these factors. Infectious endocarditis and aortic dissection are the two most