Assessment for Surgery for Aortic Valve Replacement

JOHN QUINN, M.D.

Assistant Professor of Medicine and Cardiology, Department of Medicine, Medical College of Virginia, Health Sciences Division of Virginia Commonwealth University, Richmond, Virginia

Surgical management of symptomatic aortic valve disease has been successful in improving both survival and function. Data reported from the literature suggest an operative mortality of from 5% to 8%, with a lower risk for elective replacement, and a 65% to 75%[1-3] five-year survival rate as opposed to 50% to 60%[4] in non-surgical patients with symptomatic aortic valve disease.

Aortic Stenosis

Valve replacement for aortic stenosis is usually indicated when angina pectoris, exertional syncope or congestive heart failure occur. Previous statistics suggest that with angina, which is the most common symptom, 50% of patients are dead within five years and with congestive heart failure, 50% are dead within two years,[5] usually as a result of sudden death. In the adult, coronary disease is present in approximately half the cases of aortic stenosis. The etiology of angina may thus be multifactoral which makes this the most difficult of the major symptoms to assess. Obviously the severity of coronary disease influences subsequent survival; coronary bypass surgery at the time of valve replacement does not substantially alter perioperative mortality. At the present time a congenitally malformed bicuspid valve is the most common cause of aortic stenosis in the adult. Rheumatic aortic stenosis is now less common and occasionally calcific tricuspid stenosis ("aortic stenosis of the elderly") is seen.

Accurate clinical assessment of the severity of aortic stenosis may be difficult, especially in the elderly and in the patient with congestive heart failure. As cardiac output falls, the flow over the aortic valve diminishes and therefore the intensity of the murmur decreases. The usual physical findings with severe aortic stenosis include a narrow pulse pressure primarily because of decreased systolic blood pressure; a diminished carotid pulse volume with a slow rise to peak upstroke; a thrill often palpable in the second right interspace; and a murmur, most intense in the aortic area, typically of a crescendo-decrescendo type which peaks late in systole. An S4 gallop rhythm secondary to decreased ventricular compliance is audible. In the adult, the presence of an ejection click usually occurs only with pliable valves; the second sound may be diminished and becomes single as left ventricular ejection time increases. The electrocardiogram usually shows left ventricular hypertrophy. In the absence of congestive heart failure or concomitant multivalve disease, the chest X-ray reveals a normal heart size. The ascending aorta may be dilated secondary to post-stenotic dilatation, but the aortic knob is usually small as opposed to patients with generalized tortuosity of the aorta. Ordinarily the adult has valvular calcification which is seen better by fluoroscopy. The greater the calcification, the more likely the stenosis will be severe; thus fluoroscopy may provide useful information in selecting patients for catheterization. Single dimensional echocardiography reveals dense

Correspondence and reprint requests to Dr. John Quinn, Box 36, Medical College of Virginia, Richmond, VA 23298.

echoes in the aortic root but does not yield information on the severity of the valvular stenosis. The newer two-dimensional studies frequently allow one to determine a cross-sectional area, and show promise in the future of effectively selecting patients for catheterization by a non-invasive method when the clinical findings do not suggest severe stenosis.

Catheterization is the single best method to assess severity. With normal cardiac output, a large systolic gradient of greater than 50 to 70 mm Hg is usually seen with symptomatic stenosis. As cardiac output falls for a given fixed-valve area, the gradient falls; thus the valve area calculation is of major importance when the output is diminished. The usual aortic valve area is approximately 3 cm^2; the calculated valve area determined by hydraulic formula, using the observed gradient and measured cardiac output, should be less than 1 cm^2 with symptomatic aortic stenosis and is less than 0.7 cm^2 with severe aortic stenosis.

The decision regarding replacement in the asymptomatic patient is not clear. Studies in children ages 2 to 21[6] with gradients greater than 80 mm Hg reveal that over a period of time the group operated upon was functionally better than the medical group; however, survival data were similar. These findings may not be similar in the adult; however, if the gradient is severe or if progressive electrocardiogram or chest x-ray changes occur then one might consider surgery.

Aortic Regurgitation

Chronic aortic regurgitation has a more varied etiology and, to a large extent, progression of disease is dependent on the cause. In general the murmur is detected years before symptoms occur, and because progression, even with a large regurgitant volume, is often slow, the decision regarding valvular replacement is often more difficult than with aortic stenosis. Years may pass before development of fatigability and then several years more until frank congestive heart failure occurs, resulting in an average survival of approximately two years after the onset of heart failure.

Acute severe aortic regurgitation secondary to endocarditis, aortic dissection or trauma usually leads to immediate surgery; thus the decision as to valve replacement is not as difficult as with chronic aortic regurgitation.

In brief, several features on clinical examination that suggest substantial regurgitation are widened pulse pressure with a low diastolic valve, bisferins carotid pulsation, rapidly collapsing peripheral pulses, and a displaced apical impulse. The absence of a diastolic pressure of less than 70 mm or a widened pulse pressure with the pulse pressure greater than 50% of the peak systolic in patients without congestive heart failure usually excludes severe aortic regurgitation. The rapidly collapsing pulses seen with chronic aortic regurgitation are also seen in high-output states and are not specific. The typical murmur is a decrescendo diastolic type which lengthens as the regurgitant fraction increases, that is, the longer the murmur, the more severe the regurgitation. The other auscultatory sign of significant aortic regurgitation is the presence of an Austin Flint murmur, that is, a middiastolic apical rumble which may be produced by turbulence because of closure of the mitral valve in diastole by the aortic regurgitant jet, with antegrade flow through the mitral valve, as 75% of the stroke volume may return to the left ventricle. A systolic flow murmur is often heard but is not helpful in assessing severity. An S3 gallop rhythm is common.

The electrocardiogram in severe aortic regurgitation should show left ventricular hypertrophy. The chest x-ray reveals a dilated left ventricle.

The echocardiogram usually shows a dilated left ventricle with shuddering of the anterior mitral leaflet because of the regurgitant jet but is generally not helpful in assessing severity. If the mitral valve closes prematurely because of very high left ventricular end diastolic pressure then this is a sign of severe aortic regurgitation; however, clinically there is little difficulty in establishing severity at that point. One study suggests that if the end systolic dimension is greater than 5.5 cm[7] then the incidence of subsequent complications is high and surgery may be indicated. These data have not yet been fully substantiated, and concomitant coronary disease or a myopathic process limits the value of this one finding.

Cardiac catheterization will yield information regarding hemodynamics and the degree of regurgitation.

The symptomatic patient is usually helped by valve replacement, but the decision to operate in an asymptomatic, or minimally symptomatic, patient with clinically severe regurgitation

is a difficult one. Ideally one would want a battery of noninvasive predictors such as an electrocardiogram, a chest x-ray, and an echocardiogram to predict the subsequent course and therefore to select the ideal time for surgery. Hirshfeld[8] could establish no relationship between postoperative improvement or survival and preoperative heart size by x-ray, ECG, or New York Heart Association functional class. Spagnuolo[9] found that in young persons with rheumatic aortic regurgitation 65% developed angina, heart failure, or died within three years if they had the combination of 1) diastolic pressure of less than 40, 2) cardiomegaly, and 3) left ventricular hypertrophy on electrocardiogram. With this triad of findings aortic valve replacement has been suggested.

Another consideration as to valve replacement for aortic regurgitation is that of preservation of myocardial function as in severe prolonged aortic regurgitation; irreversible myocardial changes may occur which cannot be corrected by surgery. The regurgitant volume will be eliminated by surgery and thus the patient may have symptomatic improvement, but myocardial function will remain poor; in addition the long-term survival rate with depressed left ventricular function is worse.[8] In the future one would hope to replace valves at the point just prior to irreversible myocardial changes, but at present no tests are available to assess early irreversible changes accurately. Currently, exercise nuclear gated scans are being evaluated for exercise-induced wall motion abnormalities which may indicate early irreversible changes[10]; the future of aortic regurgitation evaluation lies in assessing left ventricular function for timely valve replacement.

In summary, valve replacement is indicated for both symptomatic aortic stenosis and insufficiency. The prognostically important symptoms associated with aortic stenosis are angina, syncope or presyncope, or presyncope and congestive heart failure. The asymptomatic patient with aortic stenosis may be considered for valve replacement, but the long-term advantages of surgery in this group are not clear for adults. The symptomatic patient with severe aortic regurgitation is also a surgical candidate. The asymptomatic patient with clinically severe aortic insufficiency who has, or subsequently develops, left ventricular hypertrophy on electrocardiogram, with cardiomegaly and low diastolic blood pressure, may also be considered for valve replacement; however, the indications are not as clear-cut as with the symptomatic patient. In the future more emphasis may be placed on left ventricular function to determine early irreversible changes so that ventricular function may be preserved postoperatively. Arguments favoring earlier valve replacement are decreasing perioperative mortality, modification in prosthesis design to reduce long term complications, and higher perioperative mortality in the severely ill patients. The major argument against earlier valve replacement is that there is not yet an ideal valve; thus the potential for such complications as endocarditis, thromboembolism, and valve malfunction still exists.

REFERENCES

1. Barnhorst DA, Oxman HA, Connolly DC, et al: Isolated replacement of the aortic valve with the Starr-Edwards prosthesis. *J Thorac Cardiovasc Surg* 70:113–118, 1975.

2. Behrendt DM, Austen WG: Current Status of prosthetics for heart valve replacement *Prog Cardiovasc Dis* 15:369–401, 1973.

3. Copeland JG, Griepp RB, Stinson EB, et al: Long-term-follow up after isolated aortic valve replacement. *J Thorac Cardiovasc Surg* 74:875–889, 1977.

4. Rapaport E: Natural history of aortic and mitral valve disease. *Am J Cardiol* 35:221–227, 1975.

5. Ross J Jr, Baunwald E: Aortic stenosis. *Circulation* 37–38 (Suppl V): 61–67, 1968.

6. Wagner HR, Ellison RC, et al: Clinical course in aortic stenosis. *Circulation* 56 (Suppl 1):47–56, 1977.

7. Bonow RO, Henry WL, et al: Echocardiographic changes in asymptomatic patients with aortic regurgitation: Observations on the optimum time to operate, abstract. *Circulation* 58:II–23, 1978.

8. Hirshfeld JW, Epstein SE, Roberts AJ, et al: Indices predicting long-term survival after valve replacement in patients with aortic regurgitation and patients with aortic stenosis. *Circulation* 50:1190–1199, 1974.

9. Spagnuolo M, Kloth, Taranta A, et al: Natural history of rheumatic aortic regurgitation. *Circulation* 44:368–380, 1971.

10. Borer JS, Bacharach SL, Green MV, et al: Exercise induced left ventricular dysfunction in symptomatic and asymptomatic patients with aortic regurgitation. Assessment with radionuclide cineangiography. *Am J Cardiol* 42:351–357, 1978.