Heart Failure in Patients with Valve Disease: The Timing of Valve Replacement

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Timing of valve replacement has traditionally been linked with the development of heart failure or symptoms resulting from pathophysiologic sequences resembling heart failure [1,2]. Almost 30 years after the first valve replacement, patients with aortic, mitral, or tricuspid valve stenosis receive prosthetic valves primarily to relieve symptoms associated with pulmonary and/or systemic venous congestion. Only patients with aortic stenosis have actual left ventricular dysfunction as the cause of these symptoms. Individuals with mitral and tricuspid stenosis usually develop pulmonary and systemic venous congestion, respectively, despite normal left and right ventricular function.

Thus, the end-points for recommending mitral, aortic, or tricuspid valve stenosis are usually straightforward and clearcut; these indications often involve symptoms of pulmonary or systemic venous congestions. Unfortunately, indications for valve replacement are often not as obvious in patients with valvular regurgitation.

Aortic Regurgitation

Two distinct clinical syndromes of aortic regurgitation exist, i.e., acute and chronic. Acute aortic regurgitation (AAR) is often the result of endocarditis, dissection, or traumatic injury to the valve itself. Patients with AAR often present with symptoms of severe left ventricular failure: marked dyspnea and even pulmonary edema. These symptoms develop rapidly over hours or days. The diagnosis may not be suspected initially because of the absence of the usual peripheral manifestations of chronic aortic regurgitation (CAR), i.e., bounding arterial pulses with rapid upstroke and fall-off. Often the murmur of AAR is short and early diastolic in location. It may be overlooked if the patient is tachypneic or suffering from pulmonary edema. Echocardiography with Doppler often characterizes the lesion with considerable accuracy.

Management of these patients usually occurs in an intensive care unit since they are often quite unstable. Initially, individuals are managed medically with diuretics, supplemental inspiratory oxygen, vasodilation, and positive inotropes, e.g., nitroprusside and dobutamine or amrinone [3–5]. Hemodynamic monitoring may be required. If the patient is exceedingly unstable, urgent aortic valve replacement may be necessary [6,7]. A minority of patients become stable during
intravenous vasodilator and positive inotropic therapy. A trial of oral medications is warranted in these individuals, e.g., an ACE inhibitor such as captopril or enalapril combined with digoxin. Patients with AAR who respond to oral medication should be followed closely with careful attention to clinical and echocardiographic signs of left ventricular decompensation. Elective but urgent aortic valve replacement may be required in these individuals [8].

Patients with CAR present a different challenge for the clinician compared with the hectic, often stormy course of AAR. Individuals with CAR remain stable for decades without developing signs and symptoms of left ventricular failure. The diagnosis is usually made quite easily even by inexperienced clinicians who recognize the bounding peripheral pulses, the enlarged left ventricle, and the easily audible diastolic murmur. The difficulty in managing patients with CAR involves the decision concerning aortic valve replacement. Indeed, the appropriate timing of aortic valve replacement in patients with CAR is one of the most difficult clinical problems of modern cardiology [9].

Objective manifestations of left ventricular failure can often be demonstrated at cardiac catheterization in patients who are asymptomatic. Indeed, such patients may remain clinically stable for years despite having a severe hemodynamic abnormality [10]. This paradoxical situation has resulted in controversy among cardiologists concerning the timing of surgical therapy (aortic valve replacement) in patients with aortic regurgitation. On the one hand, irreversible left ventricular dysfunction can develop that persists despite successful aortic valve replacement [11]. Indeed, patients whose heart size, measured by roentgenography, fails to decrease during the first 6 months after operation face a 57% 6-year mortality, as opposed to a 15% 6-year mortality in those whose heart size decreases [12]. This implies that irreversible left ventricular dysfunction adversely affects survival after successful aortic valve replacement. On the other hand, recent results of aortic valve replacement in patients with aortic regurgitation and severe left ventricular dysfunction are not as discouraging as they once were [13,14]. Prosthetic heart valves and cardiac surgery with cardiopulmonary bypass can, however, entail significant complications, mortality, or both.

The long-term prognosis is poor in patients with aortic regurgitation and electrocardiographic evidence of left ventricular hypertrophy with ST-T changes. Long-term prognosis is similarly impaired in patients with markedly dilated, hypcontractile left ventricles secondary to long-standing aortic regurgitation [15–20]. These individuals have left ventricles with markedly increased end-diastolic and end-systolic volumes, reduced regurgitant volume to end-diastolic volume ratios, and inappropriately elevated mean and end-systolic wall stress [16–20].

These findings have led a number of investigators to advise cardiac catheterization in these patients, with aortic valve replacement to follow for patients demonstrating left ventricular dysfunction even in the absence of symptoms [15–20]. Prospective studies to support such arguments are lacking although the reasoning and conclusions that have led to these recommendations seem quite reasonable. On the other side of this debate are a number of reports confirming excellent long-term results in patients who undergo aortic valve replacement