Severe left ventricular dysfunction in critical aortic stenosis—reversal following aortic valve replacement

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Summary

A case of aortic valve stenosis is reported with progressive left ventricular dysfunction demonstrated by radionuclide angiography. A gradient of 120 mmHg was present across the aortic valve. At the time of surgery, the patient was in severe cardiac failure with a left ventricular ejection fraction of 22%. Two months postoperatively, the patient was asymptomatic, and repeat radionuclide angiography demonstrated an ejection fraction of 93%. The reversible nature of this patient's ventricular dysfunction suggests that the cardiac failure was related to 'afterload mismatch' caused by the stenotic valve, rather than due to depressed contractility. The encouraging result in this patient reinforces the view that surgical intervention is warranted in some patients with critical aortic stenosis and extreme impairment of left ventricular function, and that the close relation between ejection fraction and operative survival may not hold true in aortic stenosis.

KEY WORDS: aortic stenosis, radionuclide angiography, congestive cardiac failure.

Introduction

Patients with critical aortic stenosis and congestive cardiac failure who remain untreated by valve replacement have an average survival of approximately 2 years (Rapaport, 1975). Although some reports have indicated that many patients with aortic stenosis and cardiac failure do respond well to valve replacement (Croke *et al.*, 1977; Smith, Mcanulty and Rahimtoola, 1978), there remains some uncertainty about the role of surgery in these patients and also at

what level of ventricular dysfunction improvement may not be anticipated (Kennedy, Doces and Stewart, 1977). In this report, we describe a patient with progressive severe left ventricular dysfunction on the basis of tight aortic stenosis and the reversal of the dysfunction after surgery. The mechanisms for cardiac failure are discussed.

Case report

A 74-year-old male presented with a hydrocoele and symptoms of urinary retention. He was referred for cardiovascular assessment because of a long history of dyspnoea on exertion and infrequent episodes of nocturnal dyspnoea. There was no exertional chest pain or syncope, and the patient denied any previous hospital admissions or any serious illnesses.

Physical examination revealed a well-nourished man in no obvious distress. Blood pressure was 110/70 mmHg, and there was a delayed upstroke in the carotid pulses with a bilateral systolic thrill. The cardiac apex was in the mid-clavicular line and was of a diffuse, sustained nature. The first heart sound was soft, the second sound was soft and single, and a fourth heart sound was audible at the apex. A grade IV/VI harsh systolic ejection murmur was heard which radiated into the neck, and a soft early diastolic murmur was also present. Electrocardiogram showed left ventricular hypertrophy with a strain pattern, and a two-dimensional echocardiogram revealed a thick, poorly mobile aortic valve, and concentric, symmetric hypertrophy of the left ventricle. Chest X-ray showed a normal sized heart with clear lung fields. First-pass radionuclide angiography (Dymond *et al.*, 1979) in the right anterior oblique projection showed a reduced ejection fraction of 41%

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FIG. 1. Computer-derived end diastolic and end systolic perimeters from sequentially performed first pass radionuclide ventriculograms. Left image: initial presentation; Middle image: immediately preoperatively; Right image: 2 months post-aortic valve replacement. In all images the end diastolic perimeter is grey, the end systolic perimeter is black. EF = ejection fraction.

and moderate diffuse hypokinesis (Fig. 1). A diagnosis of severe aortic stenosis was made but the patient declined the offer of cardiac catheterization and subsequent valve replacement. Later that month, the patient underwent successful resection of his hydrocoele and transurethral prostatic resection under antibiotic cover.

He was not seen again until 5 months later when he presented to the casualty department with a 7-week history of nocturnal anginal chest pain and two attacks of paroxysmal nocturnal dyspnoea. On examination, he was tachypnoeic, his blood pressure was 90/70 mmHg and pulse was 90 beats per min. His cardiac apex was displaced two inches lateral to the midclavicular line. Auscultatory findings were unchanged apart from some accentuation of the pulmonary component of the second sound. Chest X-ray showed cardiomegaly with pulmonary oedema. Repeat radionuclide angiography showed further reduction in ejection fraction to 22% with severe diffuse hypokinesis (Fig. 1). A diagnosis of congestive cardiac failure complicating aortic stenosis was made. The patient was treated with bed rest and diuretics but continued to have nocturnal chest pain. Cardiac catheterization was carried out which revealed elevated right heart pressures as follows: pulmonary artery 55/25 mmHg; right ventricle 55/15 mmHg; mean pulmonary capillary wedge 22 mmHg. Aortic root pressure was 90/50 mmHg, and left ventricular pressure was 210/20 mmHg. Peak to peak gradient across a heavily calcified aortic valve was 120 mmHg. There was no mitral valve gradient. No left ventriculogram was performed as the aortic systolic pressure fell by 20 mmHg when the catheter entered the left ventricle, a sign consistent with severe aortic stenosis (Carabello, Barry and Grossman, 1979), and thus no valve area was calculated. Selective coronary arteriography revealed a 50% stenosis of the distal right coronary artery. The coronary circulation was balanced and the left coronary artery was normal.

Aortic valve replacement was carried out the next day. At surgery, the aortic valve was found to be bicuspid and heavily calcified. The valve was replaced with a #25 Carpentier porcine valve, and a saphenous vein bypass graft was placed from aorta to the distal right coronary artery. Postoperative course was uneventful apart from an episode of atrial fibrillation which terminated spontaneously. Two months postoperatively, the patient was asymptomatic: clinically the valve prosthesis was satisfactory, and chest X-ray showed a normal heart size with clear lung fields. Repeat radionuclide angiography revealed an ejection fraction of 93% with no wall motion abnormality (Fig. 1). At the time of writing the patient remains well.

Discussion

The patient described in this report presented with advanced, progressive left ventricular dysfunction on the basis of critical aortic stenosis, with dramatic improvement in ventricular function after aortic valve replacement. Previous studies have described improvement in ventricular function after valve replacement, both in patients with normal and reduced pre-operative ejection fractions (Croke *et al.*, 1977; Kennedy *et al.*, 1977; Smith *et al.*, 1978; Thompson *et al.*, 1979). The degree of improvement in ejection fraction has been found to be greater in patients with impairment of left ventricular function than in those with preserved function (Kennedy *et al.*, 1977; Thompson *et al.*, 1979).

It has been generally accepted that pre-operative ventricular function is an important prognostic guide to the outcome of valvular surgery (Cohn *et al.*, 1974), although the results of the aforementioned studies and of the patient in this paper suggest that this may not be true in all patients with aortic stenosis. A recent study by Forman, Firth and Bernard (1980) showed that pre-operative ejection fraction did not affect the 3-year postoperative

survival in patients with aortic stenosis, although it did in patients with aortic regurgitation. Thus it appears that the presence of a reduced ejection fraction and congestive cardiac failure in some patients with aortic stenosis represents a departure from the usual situation in valvular heart disease, inasmuch as ejection fraction and prognosis are not always intimately related.

It has been suggested by Gunther and Grossman (1979), that some patients with aortic stenosis develop left ventricular dysfunction on the basis of 'afterload mismatch' or excessive wall stress (Ross, 1976) rather than on the basis of depressed contractile state. This concept has been supported by the data of Carabello *et al.* (1980), who found that patients with aortic stenosis and congestive cardiac failure may be classified into two distinct groups. Patients who improved clinically and who showed improvements in ejection fraction postoperatively had significantly higher trans-valvar gradients than patients who died or who did not improve. Aortic valve area, left ventricular end diastolic volume or end diastolic pressure, cardiac output and ejection fraction did not reliably predict outcome. Wall stress was inversely and linearly related to ejection fraction in patients who improved following valve replacement, which, together with the high valve gradients, further supports the concept of afterload mismatch as the cause for the depressed ejection fraction and congestive failure. Patients who did not improve had higher wall stresses than would have been expected for any given ejection fraction and may have had irreversible myocardial damage.

The patient described in this report showed progressive deterioration in function with dramatic reversal following valve replacement. The deterioration that occurred between initial presentation and the time of surgery may have been due to the exhaustion of 'preload reserve', the mechanism by which stroke volume is held constant in the face of an increase in afterload (Ross, 1976).

Although the patient described had coronary artery disease as well as aortic stenosis, the left ventricular dysfunction and its reversal after surgery were probably not related to coronary artery status. Firstly, the coronary disease did not appear angiographically severe enough to cause ischaemia at rest; secondly, he denied anginal symptoms until late in his course, and thirdly, his left ventricular dysfunction was diffuse and not of the segmental nature usually associated with ischaemia.

In summary, the encouraging result in this individual, coupled with the results of other studies mentioned above, show that an aggressive approach is indicated in the management of congestive cardiac failure complicating aortic stenosis. This is especially so when high gradients exist across the diseased valves. Moreover, fears that patients with ejection fractions below 20–30% may have a level of dysfunction at which postoperative improvement cannot be anticipated (Kennedy *et al.*, 1977) appear to be unjustified.

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(Accepted 1 February 1983)