Systolic closure of aortic valve in patients with prosthetic mitral valves

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SUMMARY Systolic closure of the aortic valve was found in 10 of 36 patients who underwent mitral valve replacement. Eight patients had early systolic closure, and two had mid-systolic closure.

The left ventricular outflow tract dimension on M-mode and two dimensional echocardiograms, left ventricular posterior wall and septal thickness, left ventricular dimensions in systole and diastole, aortic valve opening, and mitral to aortic valve distance were not significantly different between patients with and without systolic closure of the aortic valve. Two of the 10 patients with systolic aortic valve closure were catheterised and in neither was there a gradient between the left ventricle and the aorta. The two patients with mid-systolic closure, however, were the patients who had the narrowest left ventricular outflow tract which could cause significant distortion of blood flow. Systolic closure of the aortic valve in patients with mitral valve replacement is probably not caused by left ventricular outflow tract obstruction, though abnormalities in laminar flow from the left ventricular outflow tract may be involved.

Mid-systolic partial closure of the aortic valve is one of the main echocardiographic features of hypertrophic cardiomyopathy,¹⁻³ and has been reported in cases of ruptured aneurysm of the right coronary sinus of Val-salva,⁴ mitral regurgitation,⁵ and ventricular septal defect.⁵ Early partial systolic closure of the aortic valve is one of the echocardiographic hallmarks of discrete subaortic stenosis,^{6,7} and has been described in a patient with parachute accessory mitral valve leaflet.⁸

We have noted the occurrence of early and mid-systolic closure of the aortic valve in patients with a mitral valve prosthesis. We report a study of this phenomenon aimed at finding its prevalence, clinical significance, and aetiology.

Subjects and methods

Thirty-six consecutive patients who underwent mitral valve replacement six months to 12 years (average 4.7 years) before the start of this study were studied prospectively. Twelve were male and 24 female; their ages ranged from 15 to 72 years (average 50.4 years). Twenty-three had a Starr-Edwards mitral prosthesis, and 13 had a Björk-Shiley prosthesis. The reasons for mitral valve replacement were pure mitral stenosis in

nine, pure mitral regurgitation in nine, and combined mitral valve disease in 18 patients. In addition, seven had aortic regurgitation, four tricuspid regurgitation (for which one had a tricuspid valve replacement), and three coronary artery disease, with aortocoronary bypass surgery in one patient.

ECHOCARDIOGRAPHY

M-mode and two dimensional echocardiograms were recorded using the Aloka model SSD-1105-E echocardiograph. M-mode echocardiograms were obtained with a 2.25 MHz transducer focused at 5 cm, and two dimensional echocardiograms with a handy scanner mechanical sector model ASV-25V, with a 90° sector with 5 to 30 c/s.

Left ventricular posterior wall and interventricular septal thickness, left ventricular systolic and diastolic dimensions, and the extent of aortic valve opening were recorded on the M-mode echocardiograms using standard methods.⁹ Two aortic valve cusps (right and non-coronary) were clearly recorded in all patients. Early systolic left ventricular outflow tract dimension was obtained on the M-mode by measuring the sagittal distance between the left side of the interventricular septum and the echo of the leading edge of the mitral sewing ring (defined as the first continuous line recorded from the prosthesis) at the onset of systole (Fig. 1). Early systolic left ventricular outflow tract

Fig. 1 A sweep from the aortic root to the left ventricular outflow tract. The whole dimension is depicted by the vertical broken line.

dimension was obtained from the two dimensional echocardiogram by measuring the minimal distance from the left side of the interventricular septum to the sewing ring of the prosthesis (Fig. 2). In addition, we measured the distance between the aortic valve and the leading edge of the prosthetic valve in mid-diastole (Fig. 3).

Early systolic closure of the aortic valve was defined as a sharp movement towards the centre of the aorta by one or both visible cusps (right and/or non-coronary) immediately after the maximal opening of the aortic valve. Mid-systolic closure was defined using the same criteria in mid-systole.

CARDIAC CATHETERISATION

Two patients had undergone left heart catheterisation. Resting pressures were recorded by slow withdrawal of the catheter from the left ventricular apex through the left ventricular cavity to the aorta. The angiographic studies included left ventriculography and coronary angiography.

STATISTICS

The probability value (P) was calculated by Student's t test and the Fisher two by two exact probability test.

Results

Systolic partial closure of the aortic valve was noted in 10 patients (group A), who could be divided into two subgroups. Group A1: two patients with mid-systolic closure of the aortic valve both had Starr-Edwards prosthetic valves (Fig. 4). Group A2: eight patients with early systolic closure of the aortic valve (Fig. 5) of whom four had Starr-Edwards and four had Björk-Shiley valves.

The remaining 26 patients had normal opening of the aortic valve (group B). There was no statistically significant difference between groups A and B regarding the interventricular septal thickness, left ventricular systolic and diastolic dimensions, interventricular septum/posterior wall thickness ratio, aortic valve opening, the dimensions of the left ventricular outflow tract on M-mode and two dimensional echocardiograms, and the aortic to mitral valve distance on the two dimensional echogram (Table).

It should be emphasised, however, that the two patients with mid-systolic closure of the aortic valve (A1) were distinct in having the narrowest left ventricular outflow tract dimensions on both M-mode (0.8 cm and 0.9 cm, respectively) and two dimensional echogram (0.5 cm each). In addition, both had a short mitral to aortic valve distance (0.5 and 0.9 cm).

Systolic fluttering of the aortic valve was found in nine patients in group A (90%) and 10 patients in group B (38.5%) (not significant on Fisher's test). Two patients, one in group A1 and the other in group A2, underwent cardiac catheterisation. No pressure gradients were found between the body of the left ventricle, left ventricular outflow tract, and the aorta. The patient in group A1 was catheterised because of a systolic ejection type murmur associated with mid-systolic closure of the aortic valve on echocardiography, raising the suspicion of left ventricular outflow tract obstruction. The patient in group A2 was catheterised because of angina.

Discussion

The present study disclosed a high prevalence of systolic partial closure of the aortic valve in patients with prosthetic mitral valves. To the best of our knowledge this phenomenon has not been previously reported. The prevalence of systolic closure in patients with the Starr-Edwards prosthesis (6/23, 26%) and those with the Björk-Shiley prosthesis (4/13, 29%) was not statistically different ($p < 0.05$).

Abnormal systolic closure of the aortic valve,

Fig. 2 Two dimensional echocardiogram showing the sewing ring of the prosthetic mitral valve (arrow), the interventricular septum (S), and aorta (A) in early systole. The left ventricular outflow tract dimension is depicted by the vertical broken line.

Fig. 3 Two dimensional echocardiogram showing the prosthetic mitral valve (arrow), and the aortic valve (A) in mid-diastole. The distance between the mitral and aortic valves is depicted by the horizontal broken line.

though recorded in different clinical situations,^{4 5 10 11} has been investigated only in the setting of discrete subaortic stenosis and hypertrophic obstructive cardiomyopathy.^{1 12} The incidence of this phenomenon in discrete subaortic stenosis is high,^{12 13} but its mechanism has not been clearly defined. It could be caused by the fixed left ventricular outflow tract obstruction creating a jet of turbulent blood that alters the normal motion of the aortic valve cusps.^{7 14} It should be emphasised that the aortic valve motion in our patients with early closure differs from that usu-

ally seen in discrete subaortic stenosis. In the latter situation the closure is generally abrupt and pronounced and the cusps remain in this semi-open position throughout systole.^{4 5} Contrary to this pattern, in our patients closure is less distinct and the aortic valve cusps reopen and remain in a fully open position until end-systole. This difference implies some diversity in mechanisms. Our patients differ from patients with discrete subaortic stenosis by the lack of left ventricular outflow tract obstruction. There were no significant differences between groups A and B

Fig. 4 *Mid-systolic closure of the aortic valve (arrow).*

Fig. 5 *Representative echocardiograms of early systolic closure of the aortic valve (arrows) in three patients.*

Table Comparison of echocardiographic variables in patients with and without systolic closure of aortic valve

	Systolic closure (group A)	Normal closure (group B)
Left ventricular posterior wall thickness (cm)	0.98±0.12	0.96±0.09
Interventricular septal wall thickness (cm)	0.91±0.06	0.97±0.09
Left ventricular systolic dimension (cm)	3.46±0.7	3.74±1.15
Left ventricular diastolic dimension (cm)	5.16±0.5	5.28±1.12
Aortic valve opening (cm)	1.82±0.27	1.73±0.32
Left ventricular outflow tract dimension (M-mode) (cm)	2.32±1.07	2.09±0.53
Left ventricular outflow tract dimension (2 D) (cm)	1.46±0.79	1.74±0.61
Distance between mitral and aortic valves (2 D) (cm)	0.95±0.59	1.02±0.32

regarding left ventricular outflow tract dimensions at the onset of systole on both M-mode and two dimensional echocardiograms (Table). Moreover, the lack of pressure gradient between the left ventricle, left ventricular outflow tract, and aorta in the patient in group A2 further supports our view. One possible explanation involves the presence of the prosthesis in the left ventricular outflow tract root which might cause some anatomical distortion and abnormality of the blood flow in this region. Another mechanism could be an abrupt tilt of the prosthesis shortly after the closure of the poppet, thereby changing the blood flow pattern across the aortic valve.

Two patients (group A1) had mid-systolic closure similar to that of hypertrophic obstructive cardiomyopathy. The early closure in hypertrophic obstructive cardiomyopathy is postulated to be caused by mid-systolic left ventricular outflow tract obstruction resulting from the protruding interventricular septum on the one hand and the anterior systolic motion of the mitral valve on the other.¹⁵⁻¹⁷ This finding in our patients gave rise to the suspicion of left ventricular outflow tract obstruction. Haemodynamic studies in one patient, however, disclosed no pressure gradient between the left ventricle, left ventricular outflow tract, and the aorta. Thus we must conclude that left ventricular outflow tract obstruction was not instrumental in causing mid-systolic closure in at least one of our patients. Nevertheless, the narrow left ventricular outflow tract combined with the distinctly short mitral to aortic valve distance could cause distortion of the blood flow in the left ventricular outflow tract and hence produce an abnormal motion pattern of the aortic valve. In addition, the high incidence of fluttering lends support to the hypothesis that

prosthetic mitral valves in some way alter the laminar left ventricular outflow tract flow pattern.

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