Assessment of mitral Björk-Shiley prosthetic dysfunction using digitised M mode echocardiography

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SUMMARY Digitised M mode echocardiograms were analysed in 22 patients with possible Björk-Shiley mitral prosthetic dysfunction. Patients with paraprosthetic mitral regurgitation had a significantly greater shortening fraction, an increased peak rate of dimension change during systole, and an increased peak velocity of circumferential fibre shortening than those with poor left ventricular function. Patients with a clotted prosthesis had lower values for shortening fraction and peak rate of dimension change during systole than patients with paraprosthetic regurgitation. In this latter group, the peak rate of dimension change during diastole and peak lengthening rate were greater than in either those patients with poor left ventricular function or those with a clotted prosthesis. In addition, the peak lengthening rate was greater in those with a clotted prosthesis than in those with poor left ventricular function.

Thus M mode echocardiography is a useful method of assessing mitral prosthetic dysfunction and allows patients with paraprosthetic regurgitation to be distinguished from those with either poor left ventricular function or a clotted prosthesis.

Patients whose condition deteriorates after valve replacement may present a difficult problem in management, as it may not be possible to distinguish clinically between those with poor left ventricular function and those with a surgically remediable complication such as paraprosthetic regurgitation or thrombosis of the prosthesis. Phonocardiography and echocardiography have been used to assist the diagnosis in such patients, particular attention having been given to the duration of time intervals and abnormal movement of echoes from the prostheses themselves. The disordered left ventricular wall motion that may accompany these disturbances has, however, received little attention. Using a simple digitising technique, wall motion during systole and diastole was analysed in 22 patients with a Björk-Shiley tilting disc prosthesis who presented with clinical deterioration.

Patients and methods

All 22 patients studied presented with possible prosthetic mitral valve dysfunction, one on two occasions (case 8a and 8b). Table 1 summarises the clinical details. The original mitral valve disease was rheumatic in origin in all but three patients, in whom it was due to ruptured chordae in two (cases 2 and 19), and to a floppy mitral valve in one (case 5). All the patients presented with breathlessness as the main cause of limitation of exercise tolerance. Peripheral emboli had occurred in three patients: to the retina in case 9, the brachial artery in case 14, and to the cerebral circulation on three occasions in case 16. One patient (case 7) had had a cardiac arrest and was undergoing assisted mechanical ventilation at the time of study. Auscultation showed clear valve clicks in all the patients who were subsequently proved to have a paraprosthetic leak or primary impairment of left ventricular function, but mitral clicks were absent in all those who had a thrombosed prosthesis. In one of these last patients (case 15) mitral systolic and diastolic murmurs were also present. Chest x ray films showed evidence of pulmonary congestion in all patients and cardiomegaly in all but one (case 4). In addition, pleural effusions were frequently present. Table 2 shows the results of cardiac catheterisation in

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Table 1 Clinical details

Case No	Age (yr)	Sex	Operations	Cardiac rhythm	JVP (cm)	Findings/operative procedure†
<i>Mitral paraprosthetic regurgitation</i>						
1	38	F	Valvotomy (twice), MVR (Björk-Shiley, 29 mm)	AF	10	1 cm dehiscence, fibrin and platelet thrombus. Porcine heterograft inserted
2	50	M	MVR (Björk-Shiley, 31 mm)	AF	2	3 areas of dehiscence. No thrombus seen. Fresh Björk-Shiley valve inserted
3	50	F	MVR (Björk-Shiley, 29 mm)	SR	3	Dehiscence of ½ of ring valve; grew <i>Staphylococcus epidermidis</i> . Fresh Björk-Shiley valve inserted
4	61	F	MVR (Björk-Shiley, 29 mm), CABG (twice)	SR	0	¼ of ring detached. Resutured
5	64	M	MVR (Björk-Shiley, 29 mm)	AF	10	1 cm dehiscence, small clot
6	66	F	Valvotomy (once), mitral repair (once), MVR (Björk-Shiley, 29 mm)	AF	6	No 4 Starr-Edwards valve inserted 1 cm dehiscence. Resutured
7	71	F	Tricuspid and mitral valve repair	AF	16	2 cm dehiscence. Starr-Edwards valve inserted
8a*	43	F	Tricuspid and mitral repair, MVR (Björk-Shiley, 29 mm)	AF	20	2 cm dehiscence. Fresh Björk-Shiley valve inserted
8b*	44	F	Mitral repair, MVR (Björk-Shiley, 29 mm)	AF	20	Two areas of 1 cm dehiscence. No 4 Starr-Edwards valve inserted
9	57	M	MVR (Björk-Shiley, 29 mm)	AF	4	1 cm dehiscence. Resutured
10	58	F	Valvotomy (once), MVR (Björk-Shiley, 29 mm)	AF	10	Large dehiscence, ? infected. Fresh Björk-Shiley valve inserted
11	63	F	Valvotomy (once), MVR (Björk-Shiley, 29 mm)	AF	10	Large leak. No 4 Starr-Edwards valve inserted
12	48	F	Valvotomy (once), MVR (Björk-Shiley, 29 mm)	AF	0	½ of ring detached. Fresh Björk-Shiley valve inserted
<i>Clotted mitral prosthesis</i>						
13	66	F	Valvotomy (once), MVR (Björk-Shiley, 29 mm)	AF	3	Old thrombus in Strut area interfering with disc movement. Porcine heterograft inserted
14	31	M	Valvotomy (once), MVR (Björk-Shiley, 31 mm)	SR	2	Thrombus on ventricular side interfering with disc movement. Starr-Edwards valve No 4 inserted
15	47	F	Valvotomy (once), MVR (Björk-Shiley, 29 mm)	AF	2	Thrombus. Immobile disc in half open position at necropsy
16	65	F	MVR (Björk-Shiley, 29 mm), AVR (Björk-Shiley, 27 mm)	AF	0	Massive thrombus mainly on ventricular side of MVR. Small strands of fibrin on AVR. Both valves replaced with porcine heterograft
17	58	M	Valvotomy (once), MVR (Björk-Shiley, 29 mm)	AF	4	Thrombus encasing ventricular aspect of valve. Porcine heterograft
<i>Poor left ventricular function</i>						
18	51	M	MVR homograft (Björk-Shiley, 29 mm)	SR	20	
19	56	M	MVR (Björk-Shiley, 31 mm)	AF	3	
20	34	M	MVR (Björk-Shiley, 31 mm)	AF	20	
21	61	M	MVR (29 mm) (Björk-Shiley, 29 mm)	AF	10	
22	55	M	MVR (Björk-Shiley, 29 mm)	AF	20	

*Case 8a and 8b are the same patient.

†Necropsy findings in case 15, otherwise operative findings.

MVR, mitral valve replacement; JVP, jugular venous pressure; AF, atrial fibrillation; SR, sinus rhythm; AVR, aortic valve replacement; CABG, coronary artery bypass graft.

11 patients. The remaining 11 patients underwent surgery without cardiac catheterisation, case 8 on two occasions.

ECHOCARDIOGRAPHY

Records were obtained using an Ekoline 20 or a Cambridge multichannel or Honeywell 1856 strip chart recorder, at a paper speed of 100 mm/s, with a simultaneous electrocardiogram. All patients were studied

recumbent, unless severely orthopnoeic, and in the left semilateral position. Echocardiograms were recorded just below the level of the mitral prosthesis.

Echocardiograms were digitised¹ to determine instantaneous left ventricular transverse dimension and its peak rate of change during systole and diastole, expressed both in absolute terms and also normalised to instantaneous dimension, the latter corresponding to peak velocity of circumferential fibre shortening during systole. The shortening fraction was

Table 2 Cardiac catheterisation data

Case No	Mean RA pressure (mm Hg)	Pressures in mm Hg				Cardiac index (l/min/m ²)	Left ventricular angiography
		PA (mean)	LA	LV	AO		
Mitral paraprosthetic regurgitation							
1*	8	60/20	v=40 mean 22	140/20	130/75	3.8	Good LV. Severe paraprosthetic leak
4*	4	60/30	v=60 mean 24	130/20	130/80	NA	Good LV. Severe paraprosthetic leak
10	15	75/45	NA	110/12-18	110/90	1.3	Good LV. Severe paraprosthetic leak
12*	12	60/33	v=45 mean 22	130/18	130/80	NA	Good LV. Severe paraprosthetic leak
Clotted mitral prosthesis							
13	17	100/50	PAW v=40 mean 32	100/1-8	100/70	1.4	Moderate LV dysfunction. 1/3 mitral regurgitation
15	NA	110/60(80)	NA	NA	70/50	NA	NA
Poor left ventricular function							
18	16	85/45(55)	PAW v=40 mean 28	170/10-25	170/90	1.2	Large LV with generalised hypokinesia. No mitral regurgitation
19	6	35/10(23)	PAW v=27 mean 21	130/6-20	130/80	1.7	Dilated LV with generalised hypokinesia. No mitral regurgitation
20	28	54/35(45)	NA	100/20-34	100/70	1.5	Large dilated LV with generalised hypokinesia. No mitral regurgitation
21	9	76/30(45)	PAW v=35 mean 25	100/10-24	100/55	1.6	Dilated LV. Poor basal movements with apical aneurysm. No mitral regurgitation
22	21	94/40(55)	PAW v=35 mean 30	115/6-25	115/70	1.8	Enlarged LV with generalised severe dyskinesia. No mitral regurgitation

*Performed at referring hospital.

RA, right atrium; PA, pulmonary artery; LA, left atrium; LV, left ventricle; AO, aorta; PAW, pulmonary capillary wedge pressure; NA, not available.

calculated as $(Dd-Ds)/Dd$, where Dd and Ds represent maximum and minimum dimension respectively, and expressed as a percentage. Measurements were made as recommended by the working group in echocardiography of the European Society of Cardiology.² The dominant movement of the interventricular septum during ejection was assessed and described as anterior, indeterminate, or posterior. The terms normal and paradoxical were deliberately avoided, since normal—that is, posterior motion of the septum—is unusual with a normally functioning mitral prosthesis and may indicate dysfunction.

Results

LEFT VENTRICULAR DIMENSIONS

End diastolic left ventricular dimension was 5.1 ± 0.66 cm in patients with paraprosthetic regurgitation, 4.5 ± 1.1 cm in those with an obstructed prosthesis, and 5.9 ± 0.74 cm in those with left ventricular disease, the difference between the latter two being significant at the 5% level. End systolic dimen-

Fig. 1 Percentage fractional shortening in group 1 (mitral paraprosthetic regurgitation), group 2 (clotted mitral prosthesis), and group 3 (poor left ventricular function). Values are means $\pm$ SD.

Fig. 2 (a) Peak rate of dimension change during systole and (b) during diastole; and (c) peak circumferential fibre shortening rate and (d) peak lengthening rate in group 1 (mitral paraprosthetic regurgitation), group 2 (clotted mitral prosthesis), and group 3 (poor left ventricular function). Values are means $\pm$ SD.

sion in patients with paraprosthetic regurgitation (2.8 ± 0.84 cm) was significantly less than that in patients with left ventricular disease (5.0 ± 0.94 cm, $p < 0.001$) although similar to that in patients with prosthetic obstruction (3.3 ± 1.5 cm).

SYSTOLIC FUNCTION

Fractional shortening (Fig. 1) in patients with paraprosthetic regurgitation ($45 \pm 9.9\%$) was much greater than that in those with poor left ventricular function ($17.2 \pm 7.9\%$, $p < 0.001$), or an obstructed prosthesis ($28 \pm 9.3\%$, $p < 0.01$). Similarly, the increase in the peak rate of dimension change in systole (Fig. 2a) was much greater in those with paraprosthetic leak (14.1 ± 4.8 cm/s) than in those with poor left ventricular function (6.6 ± 2.7 cm/s, $p < 0.05$). Peak circumferential fibre shortening rate (Fig. 2c) was also significantly greater in the group with paraprosthetic regurgitation ($3.9 \pm 1.8/s$) than in that with poor left ventricular function ($1.3 \pm 0.52/s$, $p < 0.01$). In the patients with a clotted prosthesis, the mean value was $2.3 \pm 1.6/s$. There was no significant difference in any measurement of systolic function between the group with clotted mitral prostheses and that with poor left ventricular function, both of which were significantly depressed in comparison with normal.

DIASTOLIC FUNCTION

The rate of dimension change during diastole (Fig. 2b) was greatly increased to 25.3 ± 7.4 cm/s in patients with paraprosthetic regurgitation. In this group the value was much greater than that either in patients with clotted prostheses (11.8 ± 1.1 cm/s, $p < 0.001$) or in those with poor left ventricular function (8.8 ± 3.3 cm/s, $p < 0.001$), with almost no overlap. As in systole, there was no significant difference between the two latter groups. Peak normalised rate of dimension increase was also significantly greater in the patients with paraprosthetic regurgitation ($6.2 \pm 2.6/s$) than that in those with thrombosed valves ($3.4 \pm 1.6/s$, $p < 0.001$) or poor left ventricular function ($1.8 \pm 0.78/s$, $p < 0.001$). These latter two values were also significantly different ($p < 0.01$).

SEPTAL MOTION

Septal motion was in a posterior direction during ejection in all patients with paraprosthetic regurgitation but one (case 2), in whom it was indeterminate. By contrast, in those with clotted prostheses it was anterior in all but one (case 14), in whom the dominant direction was posterior, while in those with left ventricular disease it was posterior in three (cases 18, 20, and 22), indeterminate in one (case 19), and anterior in one (case 22).

Discussion

Complications related to prosthetic valves are a major cause of death after valve replacement, accounting for up to half of late deaths.³⁻⁶ Most reoperations are for paraprosthetic regurgitation with or without infective endocarditis, while those for clotted or obstructive valves are notably less common. Recognition of these complications may be difficult since in either the clinical picture may resemble that of severe left ventricular disease with limitation of exercise tolerance, evidence of pulmonary congestion, and fluid retention. The presence or absence of a pansystolic murmur does not help in the diagnosis since severe paraprosthetic leak may be unaccompanied by a murmur, while secondary tricuspid regurgitation may result from an increase in left atrial pressure from any cause. Prosthetic valvular dysfunction must, therefore, be positively excluded in any patient whose condition deteriorates after valve replacement regardless of the physical signs.

Several approaches to the non-invasive diagnosis of these complications have been investigated. A major dehiscence may cause strikingly abnormal motion of the prosthesis that can be recognised on fluoroscopy. This finding is diagnostic when present, but severe paraprosthetic regurgitation may occur with a normal angle of tilt if enough strategically placed sutures remain in place.⁷ In patients with mitral Starr-Edwards paraprosthetic regurgitation, shortening of the interval between A2 and the opening click of the prosthesis has been reported. The corresponding interval can be measured for the Björk-Shiley valve as that between A2 and the time of complete opening (OC) of the disc assessed by M mode echocardiography. Although a short A2-OC interval is usually present in association with prosthetic valve dysfunction,⁸⁻¹⁴ it is not invariably found and nor does it allow the condition to be distinguished from left ventricular disease. It may simply be a sign of a raised left atrial pressure. An alternative approach has been to observe the prosthesis itself. Yuste *et al* noted an abnormal anterior motion of the disc just after full opening,¹⁵ whose presence correlated with paraprosthetic regurgitation^{13 14 16 17}; the specificity and sensitivity of this finding are unknown, and it may persist after successful surgery.¹³ The same lack of specificity applies to measurements of the left atrial dimension. Failure of this dimension to decrease after operation implies a persistent increase in left atrial pressure, but neither measurements of dimension nor of aortic root motion¹⁸ give specific information about the underlying mechanism.

An alternative approach has been to examine left ventricular wall motion directly, with the aim of looking for the physiological consequences of re-

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gurgitation. Early studies showed that septal motion was appreciably affected by a paraprosthetic leak, the reversed or indeterminate motion seen with a normally functioning prosthesis being replaced by a strikingly normal motion, with posterior displacement during ejection and the reverse during filling. This finding proved to be both specific and sensitive in detecting such patients. There are, however, disadvantages in considering septal motion in isolation. The normal reversal of the direction of septal motion after open heart surgery is not well understood. Motion of the whole heart within the thorax may make interpretation of septal motion relative to a fixed transducer on the chest wall difficult to interpret. Additional tricuspid regurgitation may lead to right ventricular overload with an independent effect on septal motion. For these reasons, we have assessed changes in left ventricular dimension measured from digitised M mode echocardiograms. In a previous study from this hospital, 14 patients with mitral Starr-Edwards paraprosthetic regurgitation were shown to have peak rates of dimension increase appreciably higher than normal.¹⁹ The present series deals exclusively with Björk-Shiley prostheses, and again values were higher than those seen in patients whose prosthetic function was normal. End diastolic dimension was normal, although end systolic dimension was reduced. Both this, and the increased shortening fraction, presumably reflect the increased stroke volume and reduced impedance to ejection which are likely to have been present in these patients.

In situ thrombosis is also an important complication of the Björk-Shiley prosthesis in the mitral position. It may occur many months or years after operation in patients whose anticoagulant control appears to have been excellent. When present, it progresses rapidly and the patient presents in a low output state, requiring urgent cardiac surgery. The diagnosis is best made from loss of the closing click of the valve and from a characteristic reduction in the velocity and amplitude of disc motion. Left ventricular function was also modified in a characteristic way, with a reduction in shortening fraction and peak rate of dimension decrease during systole. During diastole, peak normalised rate of dimension increase was also low. This picture closely resembled that in patients with primary left ventricular disease, the only difference being a lower rate of dimension increase in the former patients. The reason for this remarkable similarity in physiological response is not clear. It may well reflect, in part, a low stroke volume and a high peripheral impedance to ejection common to both conditions. A similar apparent depression of left ventricular function also occurs in mitral stenosis, which is reversible with successful surgery.²⁰

These results have greatly affected our management of patients in whom evidence of cardiac enlargement and pulmonary congestion persists or develops after mitral valve replacement. The first priority is to establish the underlying cause, frequently a surgically correctable lesion. If M mode echocardiography shows evidence of normal systolic function associated with a rapid rate of increase in diastolic dimension then a paraprosthetic leak is almost certainly present. Furthermore, when notable clinical deterioration is evident we refer such patients for cardiac surgery without further investigation. Thrombosis of the prosthesis is a medical emergency. Deterioration may be rapid, and the incidence of major peripheral embolism or low output state leading to cardiac arrest is high. Its presence is diagnosed from loss of the normal clicks and from impaired motion of the disc, established either by echocardiography or by screening. When these features are present, we recommend urgent surgery, again without further investigation. The pattern of left ventricular wall motion in these patients is less specific than that in those with a paraprosthetic leak. It is essential that its similarity to that in left ventricular disease is recognised so that this additional diagnosis is not made in error and the patient denied operation on account of any presumed increase in risk. Severe primary left ventricular disease excludes further surgery and makes the ultimate prognosis poor. In such cases we prefer to perform cardiac catheterisation in order to establish the diagnosis beyond all reasonable doubt, although it is nearly always apparent from non-invasive investigations. This plan of management has great advantages in that the delay and clinical deterioration associated with cardiac catheterisation and injection of angiographic contrast medium in seriously ill patients with surgical complications are usually avoided. As far as possible, a definite diagnosis is established in all patients, either at operation or from the combined results of invasive and non-invasive investigations.

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