Echophonocardiography in diagnosis of mitral paravalvular regurgitation with Björk-Shiley prosthetic valve

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SUMMARY The echophonocardiographic features in three patients with a mitral Björk-Shiley prosthesis and paravalvular regurgitation are presented. The characteristic features are an early diastolic humping of the Björk-Shiley disc echo, associated with normal rather than paradoxical septal motion, and a reduced A2-MVO interval. The diagnosis was confirmed at operation in one patient, at cardiac catheterisation and operation in the second, and at necropsy in the third. These features were abolished by surgical correction of the leak in both operated cases.

Haemodynamic deterioration in patients with prosthetic heart valves may be rapid and severe but its precise cause may be obscure without resort to full cardiac catheterisation. Since many of these patients may be acutely ill, it is clearly desirable to be able to make a non-invasive assessment of the cause. The echophonocardiographic abnormalities caused by mechanical dysfunction of a variety of ball and disc valves have been described.^{1 2} With the increasing use of the Björk-Shiley prosthetic valve, the echocardiographic appearances of the normally functioning valve have been described,^{3 4} but its assessment is more difficult because of the spatial asymmetry of the disc and the variable orientation of the valve according to the method of insertion. Nevertheless, echocardiographic abnormalities caused by dysfunction of the Björk-Shiley valve in the aortic⁵ and mitral⁶ positions have been reported, the latter authors describing a study of two patients with valve obstruction and two patients with paravalvular regurgitation.

We present a combined echophonocardiographic study of three patients with paravalvular regurgitation, which confirms the echocardiographic findings of Bernal-Ramirez and Phillips⁶ and shows also that they are associated with a reduced interval between the aortic second sound and mitral valve opening (A2-MVO) as previously described for other prosthetic valves.

Subjects and methods

The echocardiograms were obtained with a Smith Kline ultrasonoscope using a 2.25 MHz transducer
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(1.5 cm diameter). Permanent recordings were obtained at paper speeds of 50 and 200 mm/s, using a Cambridge strip chart recorder. The echocardiograms were obtained with the patients in a slightly left lateral position, with the head and thorax raised at 20°. The transducer was positioned in the third or fourth left intercostal spaces and directed towards the prosthetic mitral valve to record maximal excursion of the tilting disc. Further echocardiograms were obtained of the aorta and left atrium and of the body of the left ventricle below the prosthetic mitral valve. Echophonocardiograms recorded at 200 mm/s were used to measure the interval between the first high-frequency component of the second heart sound and peak opening of the prosthetic valve (A2-MVO interval), and the opening and closing velocities of the Björk-Shiley valve echo. Values greater than 180 and 300 mm/s were accepted as normal for opening and closing velocities, respectively⁶; the normal A2-MVO interval for a tilting disc prosthesis (Lillehei-Kaster) was taken as 90 ± 10 ms.²

Case reports

CASE 1

A 40-year-old woman with rheumatic heart disease underwent mitral valve replacement with a 29 mm Björk-Shiley prosthesis in January 1978. Her immediate postoperative course was complicated by an embolic occlusion of the right ilio-femoral artery necessitating emergency embolectomy; an organised clot, sterile on culture, was removed from

Fig. 1 (Case 1) (a) Echophonocardiogram showing a reduced A2-MVO interval of 35 ms and normal opening and closing rates of 500 and 700 mm/s, respectively. The abnormal early diastolic hump is indicated by the arrows. The dotted lines in this and subsequent figures indicate the aortic second sound and the point of maximal prosthetic disc opening. The time lines indicate an interval of 40 ms. All recordings shown were made at a paper speed of 50 mm/s but measurement of A2-MVO interval and disc velocity were made from recordings at 200 mm/s (not illustrated); normal values: opening velocity > 180 mm/s, closing velocity > 300 mm/s; A2-MVO interval 90 ± 10 ms. BS, Björk-Shiley mitral prosthesis. (b) During shorter RR intervals the whole Björk-Shiley valve diastolic slope is rounded anteriorly rather than having the normal variable downward slope. Septal motion is normal rather than paradoxical. (c) Postoperative echophonocardiogram. A2-MVO interval 100 ms. Loss of early diastolic hump and return of normal diastolic slope of the Björk-Shiley valve echo. Return of paradoxical septal motion.

the common iliac artery. On the twentieth post-operative day, she developed pulmonary oedema and was re-admitted to hospital from convalescence. She was critically ill, grossly orthopnoeic, and febrile but with no stigmata of bacterial endocarditis. Clear opening and closing clicks of the Björk-Shiley valve were heard and there was a faint mid-diastolic murmur at the cardiac apex, but there was no audible pansystolic murmur.

The electrocardiogram showed uncontrolled atrial fibrillation and the chest radiograph confirmed pulmonary oedema. The echophonocardiogram is shown in Fig. 1a and 1b. The Björk-Shiley valve has normal opening and closing velocities, normal amplitude of movement, but an intermittent rounded early diastolic humping of the Björk-Shiley valve is seen, together with an abnormal diastolic slope in the shorter RR intervals. The phonocardiogram shows an A2-MVO interval of 35 ms and septal motion is normal rather than paradoxical. At emergency operation the next day the mitral prosthesis was found to be detached anteriorly by about 1.5 cm, and several granulations were seen on the valve ring. The valve was excised and a new 29 mm Björk-Shiley valve was sutured in its place.

Staphylococcus albus was cultured from the excised valve and a six-week course of streptomycin and benzylpenicillin resulted in a full clinical and radiological recovery.

The postoperative echophonocardiogram is shown in Fig. 1c. This shows loss of the early diastolic humping, restoration to normal of the Björk-Shiley valve diastolic slope and of the A2-MVO interval (100 ms), and return of paradoxical septal movement.

CASE 2

A 44-year-old woman underwent closed mitral valvotomy for rheumatic mitral stenosis in January 1978. Unfortunately, she developed significant mitral regurgitation and in February 1978 the diseased mitral valve was replaced with a 31 mm Björk-Shiley prosthetic mitral valve.

Her immediate postoperative period was complicated by the development of a left hemiparesis, but there was rapid and almost total recovery of function within a few days. By the time of her discharge she was well, with a clinically competent prosthetic mitral valve. Three months after operation she was re-admitted with a seven-day history of nausea, vomiting, dyspnoea, and orthopnoea. On admission, she was clearly ill with congestive failure and tricuspid regurgitation. The pulse was irregular, 100/min with runs of bigeminal rhythm, and jugular venous pressure was raised to 10 cm, with dominant *cv* waves and rapid *y* descent. A

loud pansystolic murmur was loudest at the tricuspid area but was heard faintly at the mitral area and in the axilla, and normal opening and closing clicks of the prosthetic mitral valve were present. There were widespread basal crepitations and the liver was enlarged and pulsatile.

The electrocardiogram on admission suggested digoxin toxicity and the chest *x*-ray film confirmed pulmonary oedema. The serum digoxin level was 4.3 nmol/l. Her condition rapidly improved with increasing diuretic therapy and temporary discontinuance of digoxin, with disappearance of the signs of tricuspid regurgitation and clearing of the chest *x*-ray film. However, a soft pansystolic murmur remained at the axilla.

The echophonocardiogram shortly after admission is shown in Fig. 2a. The essential findings are the normal opening and closing velocities and amplitude of excursion of the Björk-Shiley valve, but a short A2-MVO interval of 55 ms, normal rather than paradoxical septal movement, and early diastolic humping of the Björk-Shiley mitral valve. The other echocardiographic views in this case show a dilated left ventricular cavity with a vigorously contracting posterior left ventricular wall (Fig. 2b) and a dilated left atrium with systolic expansion of the left atrial wall in the region of the aortic valve (Fig. 2c).

In view of these findings, cardiac catheterisation was performed. The mean left atrial pressure was 27 mmHg, with a *v* wave of 50 mmHg rising to 70 mmHg after angiocardiography. Left ventriculography confirmed severe regurgitation around the Björk-Shiley prosthesis into an enlarged left atrium. At operation, the Björk-Shiley prosthesis looked normal. There was no cloth wear and the disc functioned normally. There was a tear 0.5 cm lateral to the valve corresponding to about one-sixth of the circumference of the sewing ring. All the suture material was intact and there was a rim of tissue attached to the sewing ring in this area.

The paraprosthetic leak was thus a result of tissue failure and tearing. This defect was closed using a series of interrupted and continuous sutures through the cardiac tissue approximating it to the valve ring, and the heart was defibrillated to sinus rhythm. The postoperative echophonocardiogram is shown in Fig. 2d. The diastolic slope of the Björk-Shiley valve is now normal, with loss of the early diastolic hump, and the A2-MVO interval is 120 ms. A mid-cavity view confirmed the return of paradoxical septal motion.

CASE 3

A 68-year-old woman underwent tricuspid valvotomy and mitral valve replacement with a

Fig. 2 (Case 2) (a) Early diastolic hump of the Björk-Shiley valve echo (arrowed). A2-MVO interval 55 ms. Opening velocity 440 mm/s; closing velocity 890 mm/s. (b) Echocardiogram from body of left ventricle (LV) below Björk-Shiley valve. The cavity is dilated ($LVID_d = 7.0$ cm). The interventricular septum (IVS) shows normal motion. The posterior left ventricular wall (PLVW) amplitude is increased at 1.75 cm (normal 0.9 to 1.4 cm). $LVID_d$, left ventricular internal diameter in diastole. (c) Increased diameter of left atrium at 5.2 cm (normal 1.9 to 4.0 cm) with systolic expansion (arrowed). The aortic leaflets approach each other during systole. These features are consistent with mitral regurgitation and reduced forward flow of a significant degree. (d) Postoperative echophonocardiogram. A2-MVO interval 110 ms. Loss of early diastolic hump on Björk-Shiley valve echo. LV cavity record showed return of paradoxical septal motion.

31 mm Björk-Shiley valve in September 1978. Tricuspid stenosis (orifice 2 cm) was fully relieved by incision of the commissures and during the first two postoperative weeks there was no evidence of tricuspid regurgitation. After the third week, however, there was gradually increasing pulmonary and peripheral oedema with hepatomegaly and the jugular venous pulse showed *cv* waves 6 cm above the sternal angle. The prosthetic valve clicks were clear but a pansystolic murmur varying with respiration appeared at the lower end of the sternum together with a distinct low-pitched diastolic murmur at the apex. A soft pansystolic murmur at the apex was considered to be radiating from the tricuspid area while the apical diastolic murmur was thought to signify obstruction of the Björk-Shiley valve. Cardiac catheterisation was planned, but the patient's condition deteriorated rapidly and she died in the sixth postoperative week. An echophonocardiogram recorded 48 hours before death when the patient was critically ill is shown in Fig. 3. There is a short A2-MVO interval, normal opening and closing velocities of the Björk-Shiley valve, and early diastolic humping of the valve echo; the interventricular septum moved posteriorly in systole.

At necropsy, examination of the prosthesis showed that the sutures along 25 per cent of its circumference adjacent to the aortic valve had cut out (Fig. 4); the remaining sutures were intact and there was no thrombosis. The pyrolite disc was freely mobile. The tricuspid valve was widely

patent and the cusps appeared to be functionally normal.

Discussion

Echophonocardiographic findings in prosthetic mitral valve dysfunction have previously been described. Reported non-invasive evidence of paravalvular mitral regurgitation was limited to a reduced interval between A2 and opening click (OC) and return to normal of septal motion with Starr-Edwards and Lillehei-Kaster prostheses^{1,2} and the presence of a pansystolic murmur. These findings, however, are not entirely reliable; a shortened A2-OC interval may be found with an obstructed prosthetic mitral valve as well as with paravalvular regurgitation. A reduced A2-OC interval may also be found in the presence of left ventricular dysfunction.² Return of normal septal motion after open-heart operations may be a normal finding, partly related to the time after operation, and has been observed with clot obstruction of Lillehei-Kaster prostheses.² The presence, absence, and loudness of the pansystolic murmur associated with paravalvular regurgitation is unreliable as the murmur may diminish or disappear with increasing severity of regurgitation⁷ and this feature is seen in the above cases. The first specific non-invasive sign associated with Björk-Shiley paravalvular regurgitation was described in two patients by Bernal-Ramirez and Phillips.⁶ They noted the presence of an unusual 'hump' during the opening phase of the

Fig. 3 (Case 3) Echophonocardiogram showing a reduced A2-MVO interval (40 ms) and normal Björk-Shiley valve opening and closing velocities (400 and 530 mm/s, respectively). The Björk-Shiley valve echo shows an early diastolic hump.

Fig. 4 (Case 3) Björk-Shiley valve showing extensive detachment of sewing ring adjacent to aortic valve.

valve movement together with a return of normal septal motion with this particular prosthetic valve. No phonocardiographic data were presented in this paper.

Our study confirms the echocardiographic finding of an early diastolic 'hump' and its reversibility by operation. The return to normal of septal motion is also seen. In addition, the demonstration of a reduced A2-MVO interval is in agreement with the reduced A2-OC interval found with a variety of other malfunctioning prosthetic mitral valves, compared with the normal interval of 90 ± 10 ms.²

Previous studies with the Starr-Edwards ball valve have shown that the echocardiographic features are the result of both ball and ring motion, and the echocardiographic findings with the Björk-Shiley valve probably represent echoes from the disc and ring also. All the patients presented had severe paravalvular regurgitation and the early diastolic hump of the Björk-Shiley valve may be associated with abnormal rocking of the partially detached mitral valve ring and disc. The sign may be confined to patients with a severe degree of regurgitation, and its prominence may depend upon the plane of abnormal rocking in relation to the ultrasound beam.

The expected echocardiographic findings of left ventricular diastolic overload seen in mitral regurgitation apply in the patient with paravalvular regurgitation (Fig. 2b). These findings, together with a reduced A2-MVO interval in the presence of good left ventricular function, normal rather than paradoxical septal motion, and an early diastolic humping of the Björk-Shiley valve appear to be useful in making a specific non-invasive diagnosis of paravalvular regurgitation associated with the

Björk-Shiley prosthetic mitral valve.

In all three cases described in this paper, the echophonocardiograms were highly suggestive of this diagnosis though on admission the clinical diagnosis was obscure and the signs of severe paravalvular regurgitation unimpressive. In the first patient, the previous embolic episode, absence of a pansystolic murmur, and possible faint diastolic murmur suggested that clot was obstructing the mitral valve. In the second patient, the improvement after temporary discontinuation of digoxin therapy suggested that digitalis toxicity had played an important factor in the symptomatology, but subsequent echophonocardiography suggested the correct diagnosis. The clinical signs in the third patient, as in the first, were more suggestive of clot obstruction of the mitral prosthesis than paravalvular regurgitation.

The non-invasive investigations described may provide information with relative ease and speed, which is valuable in the diagnosis and management of surgical emergencies related to the prosthetic mitral valve. Because of individual variation, particularly in the echocardiographic appearance of the normally functioning prosthesis, their value is much enhanced by possession of good-quality postoperative reference tracings on all patients after mitral valve replacement.

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