Late strut fracture in a Björk-Shiley valve prosthesis
(current series)

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SUMMARY Fracture of the short strut of the latest series of Björk-Shiley mitral valve prostheses occurred 12 months after implantation. The patient died despite emergency surgery to excise and replace the prosthesis. Björk-Shiley 29–33 mm valves with a 60° opening angle manufactured between February 1981 and March 1982 have a predicted strut fracture rate of 0.3% (three times greater than that of valves produced outside these dates). Unimplanted valves from this batch have now been recalled by the manufacturers.

One of the most dramatic and tragic events associated with prosthetic heart valve replacement is sudden mechanical failure due to blockage or detachment from the surrounding tissues. The resulting heart failure is almost always fatal unless urgent replacement of the heart valve is undertaken. Equally dramatic, but less well recognised, is fracture or dislocation of the prosthetic valve disc.

We report a fatal case of sudden mechanical failure of a current series Björk-Shiley mitral valve prosthesis due to fracture of the minor disc support strut one year after the valve was inserted.

Case report

A 52 year old woman with rheumatic heart disease underwent replacement of the mitral valve with a Björk-Shiley MBRC 29 mm prosthesis and of the aortic valve with an Omniscience 23 mm prosthesis in July 1982. She was well during the next 12 months and was taking warfarin, dipyridamole, and digoxin. In July 1983 she suddenly became short of breath, which was associated with faintness and tightness in the chest. On physical examination she was agitated, restless, cyanosed, clammy, and severely dyspnœic and was coughing up white frothy sputum. The pulse rate was irregular at 120 beats/min and the blood pressure unrecordable. Her jugular veins were distended up to the ear lobes. Examination of the chest showed coarse bilateral basal crepitations. Prosthetic sounds from at least one valve were heard, with a moderately loud pansystolic murmur at the lower left sternal edge. The electrocardiogram indicated atrial fibrillation with salvos of ventricular extrasystoles. A chest radiograph confirmed severe pulmonary oedema and showed that the minor strut and disc of the Björk-Shiley prosthesis were in the apex of the left ventricle (Fig. 1).

Despite vigorous medical treatment the patient lost consciousness and because her haemodynamic state rapidly deteriorated she was intubated and ventilated, and external cardiac massage was started.

Cardiopulmonary bypass through femoral artery and vein cannulae was instituted three and a half hours after admission. After median sternotomy the 29 mm Björk-Shiley valve ring (Fig. 2) was excised through a left atrial incision and the carbon disc and fractured strut retrieved from the left ventricle. The aortic valve functioned well on examination, and a new valve of similar size and type to that removed was sutured into the mitral orifice. After she had been weaned from bypass, her right ventricular function was excellent, but even with a filling pressure of 25 mm Hg the left ventricle only transiently maintained an aortic pressure of 40 mm Hg despite intensive positive inotropic support. No improvement was noted after several periods of prolonged circulatory assistance, and arrest occurred five minutes after the bypass procedure had been stopped.

At necropsy the pleural cavities were filled with fluid and the lungs severely oedematous. Both prosthetic valves were well positioned and mechanically satisfactory, there being no other significant abnormality.

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In 1969 the Björk-Shiley disc valve prosthesis was introduced for replacement of diseased heart valves. This was modified in 1971, when the disc material was changed to pyrolitic carbon. Sixty eight thousand 60° convex-concave valves have been distributed since 1976, and approximately 50 strut fractures of this valve have been reported to the manufacturers. The current series was introduced in 1978 after modifications to the orifice ring struts which support the disc. These consisted of machining the valve ring and major strut from one piece, although the minor strut was still welded on afterwards.

Strut fractures in valves produced since these modifications were made have been reported previously on only two occasions. The manufacturers, however, predict a strut fracture rate of 0-3% in the 8000 29–33 mm valves with a 60° opening angle manufactured between 1 February 1981 and 1 March 1982 (a rate three times greater than that of valves produced outside these dates) and have therefore recalled all unimplanted valves from this batch. Valves manufactured after 1 March 1982 have been subject to further modification and testing, and to date no strut fractures have been reported in 16 000 patients after a mean follow up of 100 000 implant months.

These three reports are all of short strut fracture, liberating the disc. In one report 1 metallurgical analysis suggested a stress fracture at the weld. Metallurgical analysis was also undertaken in our case but was not contributory. Strut fracture occurred at three and eight months after implantation 1 2 in the earlier reports and 12 months after implantation in our case. Data from the manufacturers indicate that more than 90% of strut fractures have occurred within two years of implant.

There have been several reports of mechanical failure in the earlier series of Björk-Shiley valves: major strut fracture in the 1971 model 3; fracture of the pyrolitic carbon disc of the same model 4; and dislocation of all the earlier type of disc in the 1969 model 5. In all previous reports of Björk-Shiley strut fracture the valve has been in the mitral position, presumably reflecting the more stressful flow dynamics across the valve orifice compared with those in aortic prostheses.

In two reports prosthetic heart sounds and mitral regurgitant murmurs were absent. 2 3 In our patient clattering prosthetic sounds were heard, with no consistent temporal relation to the cardiac cycle, presumably because the strut or disc of the mitral prosthesis was colliding with the aortic Omniscience valve prosthesis. There was also a pansystolic murmur internal to the cardiac apex, although this disappeared with haemodynamic deterioration.

All the reported cases have presented acutely so that screening of any asymptomatic patients with suspect valves in situ is unlikely to be of value. In the five reported cases of valve disc dislocation or fracture already cited only two patients survived. It is clear that the only hope of survival is urgent valve replacement and that there is no place in such cases for cardiac catheterisation or other investigations which might delay surgery.

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References