Intravascular haemolysis after valve replacement: comparative study between Starr-Edwards (ball valve) and Björk-Shiley (disc valve) prosthesis

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ABSTRACT Seventy-four patients with single prosthetic valves (Björk-Shiley or Starr-Edwards) in the mitral or aortic position and 18 controls with rheumatic valvar heart disease were investigated for evidence of intravascular haemolysis. Serum lactate dehydrogenase (LDH) was used as the most sensitive indicator of haemolysis. Raised concentrations were found in a third of 39 patients with Björk-Shiley prostheses (mean 281 IU/l) and in all 35 patients with Starr-Edwards prostheses (mean 859 IU/l). Values were considerably higher in patients with Starr-Edwards prostheses and particularly in those with aortic prostheses (mean 927 IU/l). Eight out of 12 patients with haemosiderinuria had Starr-Edwards valves. Intravascular haemolysis was of little clinical significance in patients with Björk-Shiley prostheses, but some patients with Starr-Edwards prostheses became iron deficient as a result.

Although intravascular haemolysis in patients with prosthetic heart valves has been well recorded for ball valve prostheses (Walsh et al, 1969; Eyster et al, 1971), the newer disc prostheses have been the subject of fewer studies (Nitter-Hauge et al, 1974; Nitter-Hauge, 1976). So far as we know no direct comparison has been made between disc and ball valve prostheses and this study was undertaken to establish the incidence and severity of haemolysis in patients with either the Björk-Shiley (tilting disc) or the Starr-Edwards (ball) prosthesis in the mitral or aortic positions.

Patients

Seventy-four patients (32 male and 42 female), routinely attending the anticoagulant clinic, were studied. Of these, 37 patients had mitral valve replacements and 37 aortic, with either Björk-Shiley (39 patients) or Starr-Edwards (35 patients) prostheses with bare metal struts (table 1). All patients were satisfactorily controlled with warfarin. Two patients with aortic Starr-Edwards valves, who had haemolytic anaemia, were receiving iron supplements. Assessments were carried out at six months to six years after operation. Twelve comparable patients with mitral and six with aortic valve disease but without prosthetic valves were used as controls.

A full blood count (Coulter counter), with examination of the blood film and reticulocyte count was performed, and serum urea, electrolytes, bilirubin, aspartate aminotransferase, and the red cell isoenzymes of lactate dehydrogenase were measured. The normal value for our laboratory is less than 300 IU/l. Urine was examined for haemosiderin in 57 patients, and serum iron and total iron binding capacity (TIBC) were measured in 56.

Function of the prosthetic valve was assessed as accurately as possible by clinical examination and the absence of symptoms, the criteria used being the clarity of the prosthetic sounds, the presence or absence and quality of murmurs, and the absence of significant or increasing cardiomegaly. If a paraprosthetic leak or other malfunction was suspected, echocardiography, cardiac catheterisation, and angiocardiography were performed.
Intravascular haemolysis after valve replacement

Results (figs 1 and 2)

The mean serum LDH in patients with Starr-Edwards valves (859 IU/l) was significantly (P<0.001) higher than the mean value both for patients in the control group (217 IU/l) and those with Björk-Shiley valves (281 IU/l). Patients with Björk-Shiley valves had mean LDH values only slightly greater than controls, and the difference was not significant. The highest values were obtained in 20 patients with Starr-Edwards prostheses in the aortic position (mean 927 IU/l), in comparison to a mean LDH value of 272 IU/l in patients with aortic Björk-Shiley prostheses. Patients with mitral Starr-Edwards prostheses had a mean LDH concentration of 792 IU/l as compared to a mean concentration of 292 IU/l for those with Björk-Shiley valves. Four out of six patients with paravalvar leaks and Björk-Shiley prostheses had appreciably raised serum LDH (mean 1490 IU/l). The remaining two were normal. These patients were excluded when calculating the mean values because it was thought that a leaking valve by causing haemolysis would bias the results. A comparison of prosthesis size and LDH values showed that with Starr-Edwards prostheses LDH values tended to be higher as prosthesis size decreased, and this was particularly so with smaller prostheses in the aortic position. The differences, however, were not statistically significant. With Björk-Shiley prostheses haemolysis was so slight with all valves that size appeared to make no difference.

Haemoglobin concentrations (table 2) were definitely but not significantly lower in patients with Starr-Edwards prostheses in the aortic position than in the other groups.

Mean corpuscular volume, mean corpuscular haemoglobin concentration, and serum iron showed only small insignificant differences between groups, although serum iron concentrations tended to be lower in patients with Starr-Edwards valves in the aortic position. These estimations did not reliably indicate intravascular haemolysis, and reticulocyte counts were raised only if haemolysis was severe.

Twelve patients, eight of whom had Starr-Edwards valves, had haemosiderinuria. Correlation of urinary haemosiderin with serum LDH showed that haemosiderinuria occurred with a serum LDH greater than 900 IU/l. There was no relation between LDH and the length of time that the prosthesis had been in situ. Serum urea and electrolyte measurements were within normal limits in all patients. Bilirubin was normal in all except three patients, who showed marginally raised concentrations. Eight patients with Starr-Edwards aortic prostheses had slightly raised serum aspartate

Table 2 Haemoglobin concentrations with different type and position of prosthesis compared with controls

<table>
<thead>
<tr>
<th>Prosthesis</th>
<th>Position</th>
<th>Haemoglobin (g/dl)</th>
<th>Range</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Björk-Shiley</td>
<td>Mitral</td>
<td>11.5-16.8</td>
<td>14.1</td>
<td></td>
</tr>
<tr>
<td>Björk-Shiley</td>
<td>Aortic</td>
<td>12.0-15.3</td>
<td>14.2</td>
<td></td>
</tr>
<tr>
<td>Starr-Edwards</td>
<td>Mitral</td>
<td>11.5-16.5</td>
<td>13.6</td>
<td></td>
</tr>
<tr>
<td>Starr-Edwards</td>
<td>Aortic</td>
<td>10.7-16.6</td>
<td>12.9</td>
<td></td>
</tr>
<tr>
<td>Controls</td>
<td></td>
<td>13.1-16.0</td>
<td>14.3</td>
<td></td>
</tr>
</tbody>
</table>
aminotransferase and three with mitral Björk-Shiley valves had similar values. The remainder were all normal.

**Discussion**

Measurement of serum LDH (specific red cell isoenzyme) is a sensitive method of estimating intravascular haemolysis (Myhre et al, 1970). It was raised in all our patients with Starr-Edwards prostheses but in only a third of patients with Björk-Shiley valves. The aortic Starr-Edwards valve caused more haemolysis than the aortic Björk-Shiley valve, and a similar but smaller difference was found between the mitral prostheses. As there was no relation between haemolysis and the length of time the prostheses were in situ, haemolysis appears to be due to the physical characteristics of the prosthesis rather than to deterioration of the valve surface with time. Small-sized Starr-Edwards prostheses, particularly in the aortic position, appeared to cause more haemolysis than the larger valves, but haemolysis with the Björk-Shiley prostheses is so slight that size did not appear to make any difference.

There has been controversy as to whether prostheses of the same type cause different degrees of haemolysis in the aortic and mitral position (Crexells et al, 1972). We found that Starr-Edwards prostheses in the aortic position caused greater haemolysis than the same valve in the mitral position. Moreover, patients with Starr-Edwards prostheses are more likely to develop iron deficiency, particularly if the serum LDH is greater than three times the normal level and haemosiderinuria is present. The latter will lead to continuous urinary iron loss and eventual depletion of iron stores and correctable iron deficiency anaemia (Eyster et al, 1965).

We would therefore recommend that oral iron supplements are given to patients with high serum LDH and haemosiderinuria. This may apply even more to patients with multiple Starr-Edwards prostheses.

There is conflicting evidence as to the value of a raised serum LDH as an indicator of paravalvar leaks (Crexells et al, 1972; Nitter-Hauge et al, 1974). While raised levels in patients with Starr-Edwards valves make LDH an insensitive indicator of a paravalvar leak, all patients with disc prostheses and a serum LDH above twice normal had clinical and angiographic evidence of malfunctioning valves. The severity of the leak, however, judged by angiography and clinical assessment, did not appear to be related to the LDH level. Thus a high serum LDH in asymptomatic patients with disc prostheses indicates the need for careful follow-up, as it may be a precursor of more serious haemodynamic disorders. Not all malfunctioning valves, however, caused a raised serum LDH. It is possible that while an acute, massive leak will not cause much haemolysis, a small leak leads to greater haemolysis because of mechanical distortion of the red blood cells.

All six patients with paravalvar leaks had Björk-Shiley valves but despite this the Björk-Shiley prosthesis, with its central flow pattern and little turbulence, has haemodynamic advantages over ball valve prostheses (Björk, 1971). Since intravascular haemolysis is also reduced, it has considerable advantages over the Starr-Edwards prosthesis.

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**References**


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