Thrombotic obstruction of the Björk-Shiley valve: the Glasgow experience

SJ RYDER, HEATHER BRADLEY, JJ BRANNAN, MA TURNER, WH BAIN

From the Department of Cardiac Surgery, Western Infirmary, Glasgow

ABSTRACT Thrombotic obstruction is the most feared complication of the Björk-Shiley tilting disc prosthesis. From 1971 to 1982 1186 Björk-Shiley valves were implanted in 900 patients. There were 93 deaths in hospital. Eight hundred and seven patients have been followed for a total of 4146 patient years; 14 patients were lost to follow up. Nineteen cases of thrombotic obstruction were identified at necropsy or at a repeat operation in the study group. The mitral prosthesis was thrombosed in 16 patients, the aortic in two, and the tricuspid in one. The incidence for this complication has been calculated to be 0.46 per 100 patient years for all valve positions, 0.79 for single mitral valve replacements, 0.18 for aortic replacements, and 0.63/100 for the tricuspid position. The maximum possible incidence of this complication in this population has been calculated to be 1.4 per 100 patient years. The mortality rate was 41.7% for reoperation and 63% for the development of the complication. Risk factors that have been identified are inadequate anticoagulant control, poor preoperative exercise capacity, and possibly also the implantation of small prostheses.

The Björk-Shiley tilting disc prosthesis is an extensively used mechanical valve of proved long term functional durability. It is, however, vulnerable to rapidly progressive malfunction if thrombus forms on the valve and restricts the movement of the disc. The incidence of this complication has been reported in several series. The figures are generally based on those patients who presented to hospital or who were operated on for the complication and may therefore represent only the "tip of the iceberg" of the true incidence of thrombus formation.

We are fortunate in the West of Scotland in that demography and the centralisation of the cardiac surgical service enable us to follow our patients relatively closely. Follow up data have been analysed and we have used them, firstly, to estimate a possible range for the incidence of this complication within the population and, secondly, to elucidate the aetiology of the condition.

Patients and methods

In Glasgow during the years 1971–82, 1186 Björk-Shiley valves were implanted in the 900 patients comprising this series. After operation a standard postoperative management and follow up regimen were used for all patients. Anticoagulant control (maintaining the thrombotest response at 5–10%) was achieved by the use of warfarin, which was started after the removal of the pericardial and mediastinal drains. In most cases this was by the evening of the first postoperative day. Heparin was not used routinely in the postoperative period. Anticoagulant control was achieved thereafter by regular attendance at an outpatient clinic. The patients also attended an outpatient clinic six weeks after operation for examination by a member of the surgical staff. They were then reviewed by medical and surgical cardiologists at least annually. Review data are stored on floppy discs and were available for microprocessor analysis. Details of the study group are shown in table 1.

Ninety three of the 900 patients operated on died in hospital. The remaining 807 patients form the population which is "at risk." This group has been used as the basis from which to calculate the incidence of thrombotic complications. During the follow up period (1971–83) 81 patients have died (late
death group). The causes of death in 50 of these patients have been ascertained by a search of case records, necropsy reports, and (where the patient did not die in hospital) questioning of their general practitioners. Seven patients are known to have emigrated, and a further seven are lost to this series, giving a “lost to follow up” group of 14 patients.

Results

DETAILS OF GROUP WITH THROMBOSED VALVES

During the follow up period 19 patients presented with pulmonary oedema and hypotension resembling acute left heart failure. One of these patients also had signs of an aortic “saddle embolus.” In these patients the diagnosis of prosthetic malfunction was suspected from the clinical presentation. In addition, the prosthetic valvular clicks were absent or reduced in all patients. The disc movement was assessed by simple radiological screening of the valve (fig. 1) Valves manufactured before 1975, however, do not contain a radiopaque marker, and the disc cannot be seen by x ray screening. Echocardiography and cardiac catheterisation were performed as required. In all patients the diagnosis of thrombotic obstruction was confirmed either at a repeat operation or at postmortem examination.

The mean interval between operation and the development of thrombotic obstruction was 32 months (range one month to seven years (fig. 2). The mean duration of symptoms of prosthetic malfunction was 15 days (range 24 hours to 10 weeks).

Six of the patients had a history of poor anticoagulant control requiring hospital admission for supervised management. Four of these had previously undergone psychiatric treatment for depression. In eight patients, however, anticoagulant control appeared to be satisfactory on the basis of their anticoagulant records and the results of the thrombotests performed on admission. Details of anticoagulant control were not available for five patients, but they had been taking their warfarin regularly.

Eighteen of the patients had a history of rheumatic fever; one patient had congenital aortic stenosis and two patients had had ischaemic heart disease requiring coronary artery surgery.

OUTCOME

Seven patients died without surgery and 12 patients underwent emergency surgery for repeat replacement of the thrombosed valve. Five of these patients

Table 1  Details of the group studied

<table>
<thead>
<tr>
<th></th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients operated on</td>
<td>900</td>
</tr>
<tr>
<td>Valves implanted</td>
<td>1186</td>
</tr>
<tr>
<td>Hospital deaths</td>
<td>93</td>
</tr>
<tr>
<td>Patients alive and followed for at least 1 year</td>
<td>807</td>
</tr>
<tr>
<td>Late deaths</td>
<td>81</td>
</tr>
<tr>
<td>Patients lost to follow up</td>
<td>14</td>
</tr>
</tbody>
</table>

Fig 1  Radiograph of mitral and tricuspid prostheses showing the radiopaque marker. The mitral prosthesis (right) was thrombosed.
died. Thus the operative mortality for reoperation was 41.7% while the total mortality for the development of thrombotic obstruction is 63%. There have been no late deaths among those surviving reoperation. Table 2 provides a breakdown of the numbers of patients at risk of thrombosis, the position of the valves they received, and the number of valves in each position which thrombosed. The 807 at risk patients were followed for a total of 4146 patient years. The 19 prostheses which clotted give a mean incidence of thrombotic obstruction of 0.46 per 100 patient years (2.35% of the patients and 1.8% of the prostheses being affected). These figures compare favourably with the incidence of 1.17 per 100 patient years reported recently by Daen et al.16

We considered several possible aetiological factors and compared their incidence in the group with thrombosed group valves and in the total group of patients who were followed up.

**Possible causal factors**

**Age**
The mean age at operation of the group with thrombosed valves was 46 years, compared with 48 years for the whole series.

**Sex**
The male to female ratio in the thrombosed group was 1:18 and in the whole series was 1:1.9. This difference is significant at the 1% level. Seventy-four per cent of the valves, however, became thrombosed after single mitral valve replacements, a group in which the female to male ratio is 3:1. When this is taken into account the sex ratio in the thrombosed group is not significantly different from that in the whole series.

**Preoperative exercise tolerance**
95% of the patients with thrombosed prostheses were in New York Heart Association grades III or IV before operation while only 69% of the patients in the whole series were similarly distributed. This difference is significant at the 2% level.

Table 2. Incidence of thrombotic obstruction 1971–83: 807 patients followed for 4146 patient years

<table>
<thead>
<tr>
<th>Valve replaced</th>
<th>No of patients followed</th>
<th>No (%) of prostheses thrombosed</th>
<th>Patient years</th>
<th>Incidence/100 patient years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitral</td>
<td>383</td>
<td>14 (3-7)</td>
<td>1765</td>
<td>0.79</td>
</tr>
<tr>
<td>Aortic</td>
<td>196</td>
<td>2 (1-0)</td>
<td>1103</td>
<td>0.18</td>
</tr>
<tr>
<td>Tricuspid</td>
<td>6</td>
<td>0</td>
<td>45</td>
<td>0</td>
</tr>
<tr>
<td>Mitral and aortic</td>
<td>162</td>
<td>2 (1-2) (mitral)</td>
<td>851</td>
<td>0.23</td>
</tr>
<tr>
<td>Mitral and tricuspid</td>
<td>35</td>
<td>0</td>
<td>224</td>
<td>0</td>
</tr>
<tr>
<td>Mitral, aortic, and tricuspid</td>
<td>25</td>
<td>1 (4) (tricuspid)</td>
<td>158</td>
<td>0.63</td>
</tr>
<tr>
<td>Total</td>
<td>807</td>
<td>19 (2-4)</td>
<td>4146</td>
<td>0.46</td>
</tr>
</tbody>
</table>

Table 3. Size of thrombosed valves

<table>
<thead>
<tr>
<th>Size of valve (mm)</th>
<th>25</th>
<th>27</th>
<th>29</th>
<th>31</th>
<th>33</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number implanted</td>
<td>23</td>
<td>65</td>
<td>262</td>
<td>254</td>
<td>40</td>
</tr>
<tr>
<td>No (%) thrombosed</td>
<td>2 (8-7)</td>
<td>2 (3-1)</td>
<td>6 (2-3)</td>
<td>6 (2-4)</td>
<td>1 (2-5)</td>
</tr>
</tbody>
</table>

**Previous valve surgery**
Ten of the patients with the complication (52.6%) had had a previous mitral valvotomy while 280 (34.7%) patients in the whole series had undergone previous valve surgery. The difference is not significant.

**Annular calcification**
Twelve of the patients with thrombosed valves (63.2%) had annular calcification compared with 56.6% (457 cases) of the whole series. Again there was no significant difference.

**Valve size**
Table 3 shows the distribution of thrombotic obstructions in the mitral and tricuspid positions. Despite the apparent relationship between small prostheses and thrombotic obstruction, the relatively small numbers make this inference statistically invalid.

**Cardiac rhythm**
Perhaps surprisingly, four of the patients with thrombosed prostheses were in sinus rhythm at the time of presentation. These were both the patients with aortic prostheses and two of the patients with mitral prostheses. The other patients were in atrial fibrillation, but clotting of the atrium was not found in association with the thrombosed prosthesis in this group.

**Discussion**
Thrombosis of the Björk-Shiley valve was first reported simultaneously by Cokkinos et al13 and Messmer et al in 1972. Later reports have linked valve thrombosis with inadequate anticoagulation12,17 and this has been thought to be the prime
cause of the complication. Valve thrombosis has been observed in patients whose anticoagulant treatment had seemed adequate but this has been thought to be a rare occurrence. From studies of the postoperative anticoagulation records and of thrombotest measurements on readmission there was nothing to suggest that in eight (42%) of our patients anticoagulation had been other than satisfactory. This implies that the valvular thrombosis in patients whose anticoagulant treatment is well controlled is not uncommon. There must therefore be other factors in the aetiology of the condition, and we believe that the following are relevant: 1 pannus ingrowth; 2 blood flow through the valve; 3 size of implanted valve; 4 quality of anticoagulation.

The pathological examination of thrombatically occluded valves removed at a repeat operation or at necropsy yielded very similar findings in our series and in others that have been reported. The thrombus progressively limits disc movement by extending across the atrial aspect of the lesser orifice until it fixes the disc at an angle of between 45° and 60°, producing acute valvular stenosis and incompetence (fig 3). The macroscopic appearance of the thrombus was often described as laminar, which implies a chronic process of thrombus deposition, until movement of the disc occluder is eventually acutely prevented. Wright et al contended that minor thrombosis leading to pannus formation may produce small limitations of disc motion before thrombosis of the valve. This view would be supported by the fact that in our series the duration of symptoms ranged from 24 hours to 10 weeks. The initial symptoms were often described by our patients as resembling a “flu like” illness. Possibly a subclinical form of endocarditis may be an initiating factor in the formation of thrombus on the valve and sewing ring. We could find no firm evidence, however, to support this idea from studies of white cell counts, blood cultures, body temperatures, and erythrocyte sedimentation rates at the time of presentation with thrombotic obstruction. We consider it more likely that the illness caused the patient to stay in bed, thereby reducing transvalvar flow, and predisposing towards thrombus build up. In support of this hypothesis, we have noted a correlation between poor exercise tolerance and incidence of thrombotic obstruction (see below). Annular calcification was thought by our group to provide a possible nidus for the formation of thrombus, but our figures do not support this hypothesis.

Severely impaired exercise tolerance appears to be a significant predisposing factor. Many of these patients have impaired myocardial contractility secondary to rheumatic carditis and as a result have chronically low cardiac outputs. The resulting low transvalvar flow predisposes to platelet aggregation.

Despite the apparent relationship between small prostheses and thrombotic obstruction, the relatively small numbers make the association nonsignificant. Interestingly, however, the incidence of thrombosis in the 29, 31, and 33 mm sizes are very similar—2.3%, 2.4%, and 2.5%. The orifices of these valves are in fact identical, and the alteration in implantation size is brought about by changes in the dimensions of the sewing ring.

Björk and Henze showed that the incidence of thrombosis of aortic prosthetic valves was 8.1 episodes per 100 patient years in the absence of anticoagulation and 0 per 100 patient years with adequate systemic anticoagulation with warfarin. Thus adequate anticoagulation is the most important factor in preventing the development of thrombotic obstruction. Six of our patients had a history of poor compliance with anticoagulant treatment, four of them having been previously treated for depressive illness. It would therefore be sensible to avoid the implantation of valves requiring permanent anticoagulation in patients who are not likely to follow their anticoagulation regimen in a controlled manner.

With regard to diagnosis, we have found radiological screening to be the most useful diagnostic procedure. All Björk-Shiley prostheses made since 1975 have a radiopaque marker in the perimeter of the disc. We have rarely found it necessary to proceed to more invasive studies in this seriously ill group of patients. It is our experience that

Fig 3  Thrombosed Björk-Shiley aortic prosthesis showing thrombus on the aortic surface of the disc, mainly affecting the lesser orifice.
Thrombotic obstruction of the Björk-Shiley valve: the Glasgow experience

Table 4  Maximum possible incidence of thrombotic obstruction

<table>
<thead>
<tr>
<th>No of thrombosed valves</th>
<th>19</th>
</tr>
</thead>
<tbody>
<tr>
<td>No lost to follow up</td>
<td>14</td>
</tr>
<tr>
<td>No of deaths, unknown cause</td>
<td>26</td>
</tr>
<tr>
<td>Incidence of thrombotic obstruction per 100 patient years</td>
<td>1.4</td>
</tr>
<tr>
<td>Maximum possible</td>
<td>1.4</td>
</tr>
<tr>
<td>Range</td>
<td>0.46–1.4</td>
</tr>
</tbody>
</table>

Fig 4  Actuarial survival curve of all patients receiving Björk-Shiley prostheses from 1970 to 1982.

patients are often aware of an alteration in the sounds produced by the valve’s opening and closing mechanism, and their comments should be taken seriously. We have no experience of phonocardiography, suggested by other workers, in the quantification of the alterations of prosthetic valvular sounds produced as a result of thrombotic obstruction. Echocardiography was carried out before operation in two of the patients presenting with thrombosed prostheses, but we did not find this investigation helpful in confirming the diagnosis.

All the patients who presented to our unit and in whom the diagnosis of thrombotic obstruction was established underwent emergency operation for replacement of their prostheses. It has not been our practice to remove the thrombus either mechanically or by the use of thrombolytic agents as others have done. We are concerned about the risk of damaging the valve occluder and retaining struts and the possibility of producing systemic embolism or haemorrhage during thrombolytic treatment. What is the replacement of choice at the repeat operation? In four of our patients Björk-Shiley valves were reimplanted and in one other patient a St Jude prosthesis was used. In the remainder bioprosthetic porcine xenografts were used. Among those patients who had Björk-Shiley valves reimplanted we have not so far had a further episode of thrombotic obstruction. Despite this we are of the opinion that in those patients who have shown poor compliance with anticoagulant treatment a bioprosthesis is the better choice.

The incidence for this complication in our series was 0.46% patient years for all prostheses (0.18% patient years for the aortic replacements and 0.79% patient years for mitral valve replacements), which compares very favourably with the results in other published series. To quote Albert Starr in a recent manuscript reviewer’s comment, “The patients presented in this review all lived long enough to undergo reoperation. These patients represent only the tip of the iceberg with regard to the natural history of this complication, since ‘approximately 50% of the patients in whom thrombosis of the tilting disc valve has been reported have died rapidly in the hospital before reoperation’. A further percentage of patients, possibly a significant number, die even prior to hospitalisation.” We have identified those patients dying in hospital without operation. With regard to those patients dying before hospital admission, we have analysed the records of all the patients who died after leaving hospital to determine the cause of their death. If this could not be determined, in the absence of necropsy or detailed clinical information, then these patients were regarded as having died from thrombotic valvular obstruction. Using these criteria and including patients lost to follow up, we have calculated a figure of 1.4 per 100 patient years as the maximum possible incidence of this complication in our population (table 4).

All the valves which thrombosed in this series were of the original spherical flat disc design. The series includes 151 patients, in whom altogether 184 concavo-convex modified Björk-Shiley valves were implanted. These patients have been followed for a total of 452 patient years, up to the end of 1983. So far, we have not identified a case of thrombotic obstruction in this group. Figure 3 is an actuarial survival curve of all patients in the series.

In summary, thrombotic obstruction of the Björk-Shiley valve is a serious complication with a uniformly fatal outcome unless promptly diagnosed and replaced at an emergency repeat operation. Risk factors which we have identified are inadequate anticoagulation control, poor preoperative exercise capacity, and possibly also the use of small prostheses.

References


Ryder, Bradley, Brannan, Turner, Bain
Thrombotic obstruction of the Björk-Shiley valve: the Glasgow experience.
S J Ryder, H Bradley, J J Brannan, M A Turner and W H Bain

Thorax 1984 39: 487-492
doi: 10.1136/thx.39.7.487

Updated information and services can be found at:
http://thorax.bmj.com/content/39/7/487

These include:

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/