Acute thrombotic obstruction with Björk-Shiley valves

Diagnostic and surgical considerations

Eight patients have had thrombotic obstruction of a prosthetic valve since 1971, six mitral valves and two aortic. All eight patients had a Björk-Shiley valve. During the same period 159 Björk-Shiley valves were placed, 85 in the mitral and 74 in the aortic area. This represents a valve thrombotic occlusive incidence of 4.4 percent in our series, 5.9 percent of mitral and 2.7 percent of aortic prostheses. Among the six patients with mitral prostheses only one survived. The two patients with occluded aortic valves survived. The onset of symptoms was very abrupt in most patients and progressed very rapidly. Acute pulmonary edema was observed in five patients. Anticoagulation was considered inadequate in all patients. Aspirin or dipyridamole was being used in seven patients at the time of thrombosis. The data indicate a high frequency of thrombotic occlusion of Björk-Shiley valves in the absence of full anticoagulation with warfarin derivatives and emphasizes the urgent need for surgery once valve thrombosis is suspected.

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Many improvements have been made in the last decade in the quality, durability, and hemodynamic characteristics of prosthetic heart valves. The Björk-Shiley valve is an example of an improved model and is presently in wide use around the world. An unsolved problem, however, is the ever-present risk of thromboembolism and the concomitant need for continuous anticoagulation. This report describes our experience with eight patients who had acute thrombotic occlusion of a Björk-Shiley valve associated with inadequate anticoagulation.

Patients in the study

Between Jan. 1, 1971, and March 15, 1977, we implanted 159 Björk-Shiley valves: Of these, 85 were mitral and 74 aortic. During the same period we have observed thrombosis of the Björk-Shiley valves in seven of our own series (4.4 percent), two aortic (2.7 percent) and five mitral (5.9 percent), and in one other
Fig. 1. Case 6. a, Valve after removal of soft clots. Note the well-organized thrombi between the small struts and the disc on the ventricular side, producing impairment of disc closure. b, Same valve in an open position. Note that the presence of thrombi between the small struts and disc does not interfere with valve opening, suggesting that valve incompetence and disappearance of closing click may precede valve stenosis.

### Table 1

<table>
<thead>
<tr>
<th>Case No. (sex, age)</th>
<th>Primary diagnosis and valve size</th>
<th>Complications of anticoagulation and other contributing factors</th>
<th>Anticoagulation at time of thrombosis (mg./day)</th>
<th>Interval between discontinuation of coumarin and valve thrombosis (mo.)</th>
<th>Interval between valve replacement and thrombosis (mo.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>MR (F, 59) MVR (2/11/74), No. 27</td>
<td>Gastrointestinal bleeding × 2</td>
<td>ASA 900, dipyridamole 100</td>
<td>4</td>
<td>10</td>
</tr>
<tr>
<td>2</td>
<td>MS (F, 36) MVR (10/17/74), No. 27</td>
<td>Subdural hematoma</td>
<td>Dipyridamole 400</td>
<td>6</td>
<td>21</td>
</tr>
<tr>
<td>3</td>
<td>MS, MR (M, 34) MVR (5/21/72), No. 31</td>
<td>Atrial fibrillation</td>
<td>ASA 1,200, dipyridamole 100</td>
<td>No coumarin</td>
<td>53</td>
</tr>
<tr>
<td>4</td>
<td>MI (F, 35) MVR (3/27/74), No. 31</td>
<td>Aortic insufficiency</td>
<td>ASA 900; dipyridamole 100</td>
<td>14</td>
<td>29</td>
</tr>
<tr>
<td>5</td>
<td>MS (F, 31) MVR (7/9/74), No. 27</td>
<td>Metrorrhagia, pregnancy, and abortion</td>
<td>ASA 1,200</td>
<td>19</td>
<td>29</td>
</tr>
<tr>
<td>6</td>
<td>MI (M, 42) MVR (3/15/73), No. 27</td>
<td>Late tamponade, hematuria, two subdural hematomas</td>
<td>ASA 900, dipyridamole 100</td>
<td>3</td>
<td>47</td>
</tr>
<tr>
<td>7</td>
<td>AS (F, 59) AVR (11/19/73), No. 23</td>
<td>None</td>
<td>No coumarin</td>
<td>16</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>AI, MR (F, 31) AVR, MVR (5/23/74), No. 25 mitral, No. 21 aortic</td>
<td>None</td>
<td>ASA 900, dipyridamole 100</td>
<td>No coumarin</td>
<td>21</td>
</tr>
</tbody>
</table>

**Legend:** MR, Mitral regurgitation. MS, Mitral stenosis. AS, Aortic stenosis. AI, Aortic insufficiency. MVR, Mitral valve replacement. AVR, Aortic valve replacement. ASA, Acetylsalicylic acid.

The patient with a mitral valve implanted at another institution. The ages ranged from 31 to 59 years (mean 40); six were women and two men. Thrombosis occurred between 10 and 53 months after valve insertion (mean 28 months). In one instance no anticoagulants were used at all. In five patients coumarin derivatives had to be discontinued because of hemorrhagic complications and were replaced by acetylsalicylic acid (ASA) and dipyridamole. In two patients ASA and dipyridamole were used exclusively from the start (Table I).

### Clinical presentation

**Mitral valve thrombosis.** The clinical presentation of these six patients is summarized in Table II. Five patients had recent onset (2 to 12 days) of dyspnea. One patient had had an episode of congestive heart failure (CHF) a month previously, which resolved with medical treatment. The presenting problem was a femoral embolus in one other patient. Of significance, however, was the rapid development of acute pulmonary edema, which was observed in five of these six patients. In the
Fig. 2. Case 4. a, Preoperative chest x-ray film showing pulmonary edema. This patient had hemoptysis and a pulmonary wedge pressure of 40 mm. Hg. b, Postoperative x-ray film following removal of the thrombosed prosthesis and insertion of a porcine xenograft valve.

one patient without clinical evidence of pulmonary edema, a pulmonary wedge pressure of 32 mm. Hg was observed. New murmurs of mitral regurgitation were present in four patients and a murmur of mitral stenosis in one patient. A constant finding was the absence of a prosthetic closing click in all patients. Systemic hypotension (<88 mm. Hg systolic) was recorded in five patients and cardiac arrest occurred in three patients. Elevated central venous pressure (>20 cm. H₂O) or pulmonary wedge pressure (≥32 mm. Hg) was observed in all patients. Chest roentgenograms consistently showed evidence of pulmonary plethora or edema. The lactic dehydrogenase (LDH) level was elevated in the four patients in whom it was measured. Cardiac catheterization showed a mitral valve gradient of more than 25 mm. Hg in the two patients studied and cineangiograms clearly revealed prosthetic valve incompetence and stenosis with marked delay of left atrial emptying. Filling defects around the prostheses suggestive of thrombotic material were also observed. An echocardiogram was obtained in only two patients and was diagnostic, demonstrating minimal or absent disc motion in both.

Aortic valve thrombosis. The clinical findings are summarized in Table II. Both patients had a 2 week history of increasing dyspnea. In addition, one had CHF and several syncopal episodes. On physical examination both patients had absent prosthetic clicks, mur- murs of aortic stenosis and insufficiency, and low systemic pressure. Laboratory studies revealed only mild LDH elevation, and cineangiograms showed marked aortic insufficiency in both patients. A gradient of 112 mm. Hg across the aortic prosthesis was demonstrated in one patient.

Clinical course

The diagnosis was not suspected in one patient and she died 12 hours after the onset of pulmonary edema on the medical service. Autopsy showed extensive thromboses completely covering both sides of the mitral prosthesis.

Five patients underwent mitral valve replacement within 12 hours of the onset of acute pulmonary edema. Two of these patients had a cardiac arrest prior to operation and three were in profound shock with unobtainable blood pressure at the time of transfer to the operating room.

Before clinical deterioration occurred, cardiac catheterization was carried out in two patients and echocardiography in two others. Once pulmonary edema occurred, deterioration was very rapid. In one patient the onset of pulmonary edema was fulminant, heralded only by a femoral embolus 12 hours previously.

At operation fresh and old thrombi in both sides of the valve were evident in all patients. The origin of the thrombi appeared to be mainly at the struts close to the
Table II

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Thrombosed valve</th>
<th>Clinical presentation</th>
<th>Preoperative clinical findings</th>
<th>Abnormal laboratory data</th>
<th>CVP (cm. H₂O)</th>
<th>PWP (mm. Hg)</th>
<th>Catheterization and other studies</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Mitral</td>
<td>CHF, pulmonary edema</td>
<td>Absent clicks; MR murmur; BP 80/0; arrest</td>
<td>LDH 3,300; PT 41%</td>
<td>39</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Mitral</td>
<td>CHF x 2, pulmonary edema</td>
<td>Absent clicks; MR, MS, TR murmurs; BP 72/0 → 40/0; arrest</td>
<td>LDH 502; bilirubin 2.2; PT 54%</td>
<td>30</td>
<td></td>
<td>MV gradient 25 mm. Hg; PA saturation 33%; cineangiogram: MS MR, immobile disc</td>
</tr>
<tr>
<td>3</td>
<td>Mitral</td>
<td>Pulmonary edema</td>
<td>Absent clicks; BP 110/70</td>
<td>LDH 443; bilirubin 4.0; PT 71%</td>
<td>21</td>
<td>40</td>
<td>MV gradient 28 mm Hg; cineangiogram: minimal MR</td>
</tr>
<tr>
<td>4</td>
<td>Mitral</td>
<td>Femoral embolus, pulmonary edema</td>
<td>Absent clicks; MR murmur; BP 80/0 → 0; arrest</td>
<td>LDH 312; PT 37%</td>
<td>32</td>
<td></td>
<td>Echo: no disc motion</td>
</tr>
<tr>
<td>5</td>
<td>Mitral</td>
<td>Pulmonary edema</td>
<td>Absent clicks; MR murmur; BP 84/58 → 0</td>
<td>PT 100%</td>
<td>32</td>
<td></td>
<td>Echo: minimal disc motion</td>
</tr>
<tr>
<td>6</td>
<td>Mitral</td>
<td>Dyspnea, low output syndrome</td>
<td>Absent clicks; BP 88/0</td>
<td>LDH 265; PT 100%</td>
<td>10</td>
<td></td>
<td>Cineangiogram: marked AI, disc open, immobile; phonocardiogram: absent clicks, AI, AS murmurs</td>
</tr>
<tr>
<td>7</td>
<td>Aortic</td>
<td>Dyspnea; new murmurs</td>
<td>Absent clicks; AI, AS murmur; BP 80/40</td>
<td>LDH 310; PT 100%</td>
<td>38</td>
<td></td>
<td>Aortic gradient 112; Cineangiogram: AI</td>
</tr>
</tbody>
</table>


small opening of the valve; the discs were immobile in a semiopen position (Fig. 1). In two cases thrombectomy was initially attempted but was abandoned owing to heavy thrombotic involvement on the left ventricular side, where actual fibrous tissue ingrowth appeared to encase the valve. The Björk-Shiley valve was replaced with a procline xenograft valve in all five patients. There was only one survivor. Three patients died of left ventricular failure—two of them in the operating room and one on the second postoperative day. The other patient died on the second postoperative day, 12 hours following an unexplained seizure and cardiac arrest. This patient had had two previous craniotomies for subdural hematomas related to anticoagulation. Autopsy was unrevealing.

The two patients with thrombosed aortic valves underwent thrombectomy within 3 days of admission and both survived.

Discussion

Thrombosis of a Björk-Shiley valve was first reported simultaneously by Cokkinos, Messmer, and their colleagues in 1971. Subsequent reports have shown association of valve thrombosis with inadequate anticoagulation. Valve thrombosis has been observed in patients whose anticoagulation programs had seemed adequate but this occurrence seems rare. The incidence of thrombosis in Björk's series of 1,250 cases was 8.1 per 100 patient-years after aortic valve replacement without anticoagulation treatment, and with anticoagulation the incidence was zero after aortic implants and 2.5 after mitral valve replacement. Lepley and associates reported a 2.5 percent incidence of thrombosis in 521 patients followed from 1½ to 6½ years.

Autopsy studies of patients who died late after valve replacement showed only one case in which thrombi in a patient with a Björk-Shiley valve who was receiving anticoagulation. 9

Thrombosis of the Björk-Shiley valve has been observed as early as 3 to 6 days postoperatively. In those three cases a hypercoagulable state was considered the cause of thrombosis. Two cases have been reported of thrombosis occurring twice in the same patients treated successfully. 12, 13

Thrombosis of the Björk-Shiley valve in the tricuspid
position has also been reported by Messmer's group and by Bourdillon and Sharratt. The experience of these last authors with one case led to early echocardiographic detection and elective tricuspid valve replacement in another patient.

Our experience is limited to valves in the mitral and aortic positions. In all of our patients, thrombosis was associated with inadequate anticoagulation including the use of ASA and dipyridamole. We have not seen thrombosis of a Björk-Shiley valve in a patient who has consistently been treated with coumarin derivatives for anticoagulation.

**Diagnostic consideration.** The clinical presentation and laboratory findings of the thrombosed Björk-Shiley aortic valve have been well described by others. Essentially, they consist of acute or subacute onset of aortic insufficiency and stenosis with new aortic murmurs and disappearance of the prosthetic clicks associated with a low output state and heart failure that may rapidly progress to pulmonary edema.

Thrombosis of the mitral Björk-Shiley prosthesis should be suspected in any patient who is not receiving consistent anticoagulation with coumarin derivatives or heparin. On physical examination the single most important sign is the absence of prosthetic sounds, especially the loss of the crisp closing click and the appearance of murmurs of mitral regurgitation and stenosis. Additional clinical findings are those of CHF and pulmonary edema. The decreased systemic pressure from low cardiac stroke volume is striking. Laboratory investigation has shown no remarkable findings other than moderate elevation of LDM, which was present in all six patients in whom the value was measured. LDH levels prior to valve dysfunction were lower in the four patients who had determinations between the time of valve insertion and dysfunction, which perhaps indicates that hemolysis is associated with valve thrombosis. LDH elevation was the only consistent chemical abnormality noted, and the value was very high in one of the patients (3,300 U.; normal, 50 to 225 U.). Severe hemolysis with hemoglobinuria as the only presenting symptom has been reported in a patient with a thrombosed Björk-Shiley valve. LDH levels usually have been found to be normal in patients with normally functioning Björk-Shiley valves.

Phonocardiographic studies confirm the ausculatory findings and allow exact measurement of the valve opening and closing intervals. Others have found this test to be useful for early detection of Björk-Shiley valve dysfunction.

The electrocardiographic findings are nonspecific and include evidence of acute cor pulmonale, right axis deviation, and left atrial enlargement. These findings were inconsistent in our series. Chest x-ray films show evidence of pulmonary plethora or edema (Fig. 2). Prosthetic valve sounds are relatively easy to record by echocardiography, and this technique often provides diagnostic information in the presence of valve dysfunction. Failure to identify poppet motion independent from the strut and sewing ring echoes is a specific finding when the recording quality is satisfactory (Fig. 3). Other echocardiographic findings, including changes in the time of valve opening and closure and chamber enlargement, are helpful when previous tests are available for comparison. For this reason the importance of obtaining base-line echocardiograms soon after prosthetic valve insertion must be emphasized.

Cardiac catheterization may show evidence of mitral stenosis and insufficiency with increased mitral valve gradient and marked venous oxygen desaturation. Angiograms may show mitral insufficiency with delayed emptying of dye from the left atrium and a negative shadow on the valve indicating a thrombus (Fig. 4).

None of the valves in this series had a radiopaque
Fig. 3. Case 5. a, Echocardiogram performed 4 months before final admission. Note the disc opening (VO) and closure (VC). CW, Chest wall. IVS, Interventricular septum. S, Strut. SR, Sewing ring. b, Echocardiogram performed during final admission. There is no detectable disc motion.

marking ring. With the new valves now in use which incorporate this radiopaque material in the disc, the diagnoses should be instantaneous by fluoroscopy or cineradiography alone. With the use of these new valves this should become the first diagnostic maneuver and probably will eliminate the need for cardiac catheterization and angiograms.

Surgical consideration. When thrombosis of a Björk-Shiley valve is suspected, definitive diagnosis should be pursued immediately, since this constitutes a surgical emergency. Two courses of action can be taken: If the patient has a valve with a radiopaque marker, the diagnosis should be easily confirmed by fluoroscopy or cineradiography and the patient should be immediately taken to the operating room for valve thrombectomy or replacement. If the valve is one of the older models and the patient’s condition permits, echocardiography is the preferred next specific, diagnostic, noninvasive test. Cardiac catheterization and cineangiography will confirm the diagnosis if necessary. If the patient’s condition is rapidly deteriorating, as occurred in six of our patients, catheterization should be omitted and operation performed without delay. In any hospital equipped for open-heart surgery the
Fig. 3. Cont'd. Case 5. c, Photographs of the valve showing almost complete encapsulation of the ventricular side with a miniscule opening (arrow). d, Atrial side and thrombotic material.

Fig. 4. a, Left ventriculogram (Case 3) showing a filling defect (arrows) suggestive of thrombotic material under the valve; also note dye in the left atrium indicating marked valve incompetence. b, Aortic root angiogram (Case 7) showing thrombus above the valve (arrow) and marked incompetence. c, Aortic root angiogram (Case 8) in diastole showing valve incompetence. d, Same study in systole showing a narrow jet of blood (arrows) indicating severe valve stenosis.
definitive diagnosis and treatment should be completed within hours.

We prefer simultaneous median sternotomy and institution of femoro-femoral bypass by a two team approach. This allows rapid oxygenation and safer dissection and release of pericardial adhesions for right atrial cannulation. A single large right atrial cannula is used; the mitral valve is exposed preferably through a superior approach. Prior to cross-clamping, local hypothermia with aortic root infusion of a cold electrolyte solution is instituted. The aortic valve is approached through a low oblique aortotomy. Thrombectomy may then be attempted; the feasibility of thrombectomy can be assessed on the ventricular surface of the valve with the aid of a laryngeal mirror. Valve competence can be tested by injecting saline through a left ventricular vent. If complete thrombectomy is not possible without damaging the prosthetic valve, no further attempt is made and the valve is replaced with a porcine xenograft valve.

We have preferred the tissue valve for replacement because of the frequent history of bleeding complications and the unreliability of many of our patients.

Postoperatively, the intra-aortic balloon pumping (IABP) was used in all but one of the patients who did not survive operation in this series; it was not necessary in the survivors. In our opinion IABP, if used, should not delay preparation for operation, since this is the only hope for survival and the condition of these patients tends to deteriorate very rapidly.

The excellent hemodynamic characteristics of the Björk-Shiley valve have encouraged its widespread use around the world; this valve and the porcine xenograft are currently our valves of choice. Unfortunately, there will continue to be many patients in whom anticoagulation will not be adequate, so that they will be at a high risk of valve thrombosis.

On the basis of our experience and that of others,1, 3, 5, 13, 14, 22, 23 we believe that full anticoagulation with coumarin derivatives is indicated for all patients with Björk-Shiley valves. Seven of our eight patients with valve thromboses were taking ASA and dipyridamole and therefore we no longer recommend these drugs. Contrary to Robicsek and Harbold,24 we consider inadvisability of anticoagulation an absolute contraindication to a Björk-Shiley prosthesis in any position. Furthermore, we support the use of heparin in the early postoperative period and then warfarin, as recommended by Lepley, 8 Björk, 25 and their co-workers.

If bleeding complications or difficulty maintaining anticoagulation occurs, these patients should be observed very closely and consideration given to elective valve replacement with a tissue valve. The disappearance of prosthetic clicks and echocardiographic or fluoroscopic evidence of impaired disc motion dictates prompt surgical intervention.

REFERENCES
8. Lepley D, Singh HM, Aris A, Flemma RJ, Mullen DC: Late evaluation of patients undergoing valve replacement with the Björk-Shiley prosthesis. Submitted for publication
Thrombotic obstruction with Björk-Shiley valves

Discussion

DR. LAWRENCE I. BONCHEK
Milwaukee, Wis.

I appreciate the opportunity to discuss this interesting paper, which Dr. Moreno-Cabral was kind enough to provide me in advance. This experience emphasizes the critical role of effective anticoagulation with Coumadin in the management of patients with mechanical prostheses of the tilting-disc design. In this series, as in others, virtually all reported cases of acute thrombotic obstruction of Björk-Shiley prostheses have been in patients who were not effectively anticoagulated with Coumadin, or who had Coumadin therapy interrupted, or who were maintained on antiplatelet drugs alone. Aspirin and dipyridamole do not provide adequate protection against major thromboembolic complications with Björk-Shiley prostheses. Several recent and separate reports by Björk, Stalpaert, Lepley, and Fernandez have each corroborated these assertions.

Experience with Starr-Edwards ball-valve prostheses has been similar. Both non-cloth-covered as well as cloth-covered ball valves have unacceptable thromboembolic rates without Coumadin anticoagulation. Most recently, the University of Oregon series of 29 patients with Model 2400 track valve aortic prostheses who did not receive Coumadin was reported in THE JOURNAL (71:680-684, 1976). Of 18 patients subjected to postoperative catheterization, 13 were found to have critically stenotic valve orifices. Of 11 others not restudied, two died suddenly, a third died after a stroke, and two others had emboli. In that series, as in the one reported today, very rapid clinical deterioration occurred in some patients without overt warning.

It is also pertinent that with stenotic ball valves, in contrast to tilting-disc valves, there are usually no abnormalities detectable by auscultation, cinefluoroscopy of ball motion, or echocardiography, because the restrictive pannus and thrombus are primarily in the valve orifice and do not interfere with poppet movement.

Clearly, now that tissue valves are available, patients who cannot or will not take Coumadin reliably should not receive mechanical prostheses. In the mitral location, where a large size prosthesis is readily accommodated, the Hancock porcine heterograft is preferred because of its proven record. In the aortic position, the Hancock sizes below 25 mm. may be accompanied by an important gradient, and the Ionescu-Shiley pericardial xenograft may offer some advantages in terms of hydraulic function. However, long-term follow-up with the latter valve is less comprehensive. In either case, for patients who cannot take anticoagulants, I personally prefer a stable transvalvular gradient to sudden death from thrombosis. In addition, the newer procedures for enlarging the aortic annulus should extend the applicability of tissue valves.

There are two questions I wish to ask. First, I would draw attention to the issue of early anticoagulation, since fibrin and platelet deposition in the first few postoperative days before Coumadin becomes effective may set the stage for late problems. In 1967, we reported in the Annals of Surgery the observation that a continuous infusion of Rheomacrodex markedly diminished the early deposition of white platelet and fibrin thrombus on prosthetic valves in calves. However, in man, we have observed that 10 percent Rheomacrodex at 25 c.c. per hour appears to be associated with increased intrathoracic bleeding and we do not use it any longer. In the manuscript, Dr. Moreno-Cabral advocates early systemic heparinization, and I would ask if he has data regarding the incidence of bleeding complications including delayed tamponade associated with this practice.

I would also ask about Dr. Moreno-Cabral’s preferred orientation of the mitral prosthesis. Dr. Björk has suggested that orientation of the larger orifice posteriorly provides better hydraulic function and fewer thromboembolic problems. My
experience does not support this assertion. In which direction were the mitral valves oriented in this series?

**DR. THOMAS FERGUSON**

*St. Louis, Mo.*

We have been strong proponents of the Björk-Shiley prosthesis in both the aortic and mitral positions at our institution for the past 4 years. The abstract by Dr. Moreno-Cabral prompted me to look up our experience with acute thrombotic obstruction with this device.

During the past 4 years, we have implanted 155 Björk-Shiley mitral valves, and there were four cases in which thrombosis of the valve occurred, an incidence of 2.6 percent. This first patient had complete obstruction of the valve orifice while on adequate anticoagulant therapy. This was the first time we had seen this entity, and so diagnosis was not made as soon as it should have been. She was operated upon but died postoperatively. The next two patients were on adequate Coumadin therapy for a number of months postoperatively, and in each anticoagulant therapy was stopped abruptly, in one patient for dental work and the other for removal of a ganglion on the wrist. In both cases the valves promptly became clotted, but both patients were operated upon and survived. The fourth patient died on the fifth postoperative day after valve replacement before full anticoagulation had been achieved. She died in pulmonary edema before an operation could be done.

In a similar time period, 113 aortic prostheses have been placed with only one patient having thrombotic occlusion, an incidence of 0.9 percent. This patient was the only patient in our aortic series who was not given Coumadin. The patient had had multiple episodes of severe epistaxis and refused to take Coumadin. He was placed on a regimen of Persantine and aspirin, and 1 year later he was admitted with an acutely clotted prosthesis. He survived reoperation for clot removal and on Coumadin therapy has done well.

In evaluating our statistics for thrombotic occlusion in patients on full anticoagulation, we find the incidence for mitral prosthesis is 1 in 155 or 0.6 percent and for the aortic prosthesis, none in 113 patients with aortic prosthesis. Our experience has led to several conclusions:

1. With the Björk-Shiley prosthesis in both the aortic and mitral positions, adequate, continuous, life-long anticoagulation is mandatory.
2. Anticoagulation must be with Coumadin rather than Persantine and Bufferin or any other combination. These two points have been emphasized by both Dr. Moreno-Cabral and Dr. Bonchek.
3. Stopping Coumadin therapy abruptly can be particularly disastrous, even after years of administration of the drug. This has been observed by many others.

4. The final point is that, based on our limited experience with reoperation in this group of patients, removal of the clot from the obstructed aortic prosthesis is relatively simple, whereas clot removal in the obstructed mitral prosthesis is very difficult. In the mitral position it is perhaps preferable to go directly to replacement of the prosthesis.

**DR. THOMAS BARTLEY**

*Gainesville, Fla.*

I concur in general with the remarks of Dr. Bonchek and Dr. Ferguson and have nothing to add, except that I seriously question the wisdom of simply debriding the thrombosed aortic tilting-disc prosthesis. I would like to hear Dr. Moreno-Cabral’s comments regarding this.

**DR. MORENO-CABRAL (Closing)**

I want to thank all the discussers for their kind comments.

In answer to Dr. Bonchek’s questions, our experience with early heparinization in these patients is very short. So far, we have had no complications related to its use, but the numbers are small and we cannot make any conclusions. One of our patients had tamponade 2 weeks postoperatively, but he was taking Coumadin, not heparin; for this reason, Coumadin was discontinued and ultimately a valve thrombosis developed.

Regarding the orientation of the mitral valve, in the five patients who were operated upon in our institution, the large orifice was oriented posteriorly. Prior to January of 1974, when Björk reported his hemodynamic studies on different prosthesis orientations, we were inserting valves in several different positions, but since then we have consistently oriented the large orifice of the valve posteriorly. As I said, in all of our thrombosed valves in this group, even the ones inserted prior to 1974, the large orifice was oriented posteriorly. I am not sure about the positioning of the valve inserted at another institution. It was not recorded in the operative report from that hospital, and I do not recall how it was oriented. I remember that we did that operation at 3:00 A.M., which may explain my hazy memory regarding this detail.

I appreciate Dr. Ferguson’s comments.

With regard to Dr. Bartley’s question about simple thrombectomy versus removing the valve, I do not have hard data to support my statement. I know that many successful thrombectomies have been reported around the world, and this can be quite an easy procedure to do. The two patients in whom we did simple thrombectomy survived and both are doing well. I would definitely change the valve in the patient who has had hemorrhagic complications from anticoagulation, and I would replace it with a tissue valve.