Prosthetic Aortic Valvular Endocarditis

By James Madison, M.D., Kyuhyun Wang, M.D., Fredrick L. Gobel, M.D., and Jesse E. Edwards, M.D.

SUMMARY

Infective endocarditis (IE) continues to be one of the most serious complications following cardiovascular surgery, particularly that for replacement of valves. In order to define more clearly the clinical course and the role of surgical therapy, clinical and necropsy data were reviewed in 16 adult patients with prosthetic aortic valvular endocarditis (PAVE) and compared with the experience cited in the literature. Positive blood cultures were obtained in each of the patients with bacterial endocarditis. Gram positive bacteria predominate and the onset of infection is usually later than 25 days postoperatively. In 11 of 16 patients, aortic insufficiency was recognized. Autopsy material demonstrated large perivalvular abscesses which loosened the attachment of the prosthetic valve in each case and which made successful operation unlikely. Aortic insufficiency appears to be of prognostic importance, since patients who developed aortic insufficiency early in the course of PAVE died. Survivors included patients who made an excellent response to medical therapy and who either did not develop aortic insufficiency or developed aortic insufficiency either late in the course or even after cure of PAVE. Poor response to medical therapy and progressive aortic insufficiency even in the absence of left ventricular failure appear to be indications for prompt surgical replacement of the prosthetic aortic valve.

Additional Indexing Words:
Bacterial endocarditis
Aortic insufficiency
Fungal endocarditis
Cardiac surgery

INFECTIVE ENDOCARDITIS (IE) following prosthetic valve placement continues to be one of the most serious complications of cardiac valvular surgery. The incidence of IE following valvular replacement is reported to vary from one to four percent, so that it is unusual for one institution to acquire a large experience with this complication. Recently, prompt surgical replacement of the infected prosthesis has been recommended when medical therapy appears to be failing.1-6 The unrelenting high mortality, the low incidence of cases and the need for clearer guidelines regarding the role of surgical therapy underline the need for additional information regarding this disease.

Accordingly, we reviewed our experience with prosthetic aortic valvular endocarditis (PAVE) and compared it with the experience in the literature. Our material consists of 16 patients studied during the period January 1963 through January 1973, in whom only the aortic valve had been replaced and who, at some time postoperatively, developed PAVE. Our infection rate following aortic valve replacement is about three percent.

In none of the cases was active endocarditis present at the time of replacement of the natural aortic valve. The criteria used for accepting cases as examples of PAVE were as follows: group I) diagnosis confirmed at autopsy (nine cases); group II) diagnosis confirmed at operation in cases which did not come to autopsy (three cases); group III) a clinical profile typical of IE in patients with an aortic valvular prosthesis and positive blood cultures (four cases).

Sixteen cases were found acceptable for inclusion in this study consisting of 13 men and three women. The ages ranged from 20 to 64 years. Autopsies were performed in ten of the 11 patients who died and specimens of heart were available for review in five of these cases (cases 4–8).

In each case, electrocardiograms were reviewed for conduction abnormalities. When the conduction abnormalities were present before the onset of IE, the
cases were not considered to be examples of conduction disturbance caused by IE. Some of the details of case 3 have been published previously.7

Observations

Criteria for Diagnosis

The clinical features of 16 patients are summarized in tables 1 and 2. All exhibited temperatures greater than 101° at some time during the course of PAVE.

Sande and associates* described their criteria for the clinical diagnosis of IE involving prosthetic valves. These include 1) bacteremia without obvious source occurring 25 or more days following prosthetic valve insertion, 2) bacteremia with gram positive organisms, and 3) new or changing murmur, especially that of aortic insufficiency. According to these authors, patients who exhibit bacteremia with gram negative organisms early in the postoperative period and without change in cardiac sounds are more likely to have bacteremia of noncardiac origin than IE.

Our data, in general, support these criteria, although there were exceptions. For example, contamination from the pump oxygenator during operation may lead to gram negative IE early in the postoperative period as illustrated by our cases 1 and 2. In three of nine autopsy-proven cases of PAVE, aor-

<table>
<thead>
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<th>Table 1</th>
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<tr>
<td><strong>Basic Data in 16 Cases of PAVE</strong></td>
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<th>Predominant presurgical lesion</th>
<th>Type of prosthesis</th>
<th>Organism</th>
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Abbreviations: AS = aortic stenosis; AI = aortic insufficiency; S-E = Starr-Edward.

<table>
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<tr>
<td><strong>Summary of Clinical Features in 16 Cases of PAVE</strong></td>
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<th>Positive blood cultures</th>
<th>Murmur of AI</th>
<th>Systemic embolism</th>
<th>Splenomegaly</th>
<th>Time of onset (postoperative)</th>
<th>Survival</th>
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<tr>
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<td>2‡</td>
<td>3</td>
<td>2</td>
<td>1</td>
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<tr>
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<td>12</td>
<td>2</td>
<td>11</td>
<td>13</td>
<td>6</td>
<td>5</td>
</tr>
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*In the two patients not listed (cases 6 and 8), Candida albicans was demonstrated at autopsy but negative blood cultures were present during life.
†Two patients developed AI, each after bacteriologic cure.
‡One died of myocardial infarction six months after bacteriologic cure and replacement of prosthetic aortic valve.

Abbreviations: AI = aortic insufficiency.
tic insufficiency had not been recognized clinically. Moreover, as in cases 13 and 16, aortic insufficiency may not be present at the time of active IE but develop later as mechanical stress is applied to tissues weakened by the infection. In these cases, aortic insufficiency developed late with regard to time of onset of infection and was progressive. This required reoperation for significant paravalvular leaks four years and five months, respectively, after bacteriologic cure of PAVE.

Signs and Symptoms

The distribution of signs and symptoms of IE in this study was similar to those found in IE affecting natural cardiac valves.

Splenomegaly was present in seven cases (44%), splinter hemorrhages in five (31%) and central nervous system symptoms were present in seven cases (44%), while Roth spots were found in two cases and an Osler’s node in one patient. Petechiae were present in nine patients (56%). A sedimentation rate greater than 100 mm/hour was found in four patients and night sweats were reported in six of the patients. Total leukocyte counts greater than 20,000/cu mm were present in only 19% of patients. Gross or microscopic hematuria (more than 5 RBC/HPF) was observed in 31% of patients. Only two of eight patients in this study in whom the onset of infection occurred more than 60 days after operation had gross or microscopic hematuria.

Bacteriology

With two exceptions, all patients exhibited positive blood cultures. The two exceptions were cases 6 and 8, in each of whom blood cultures before death had given negative results but Candida albicans was present in perivalvular tissues at autopsy. Gram positive bacteria were the infecting agents in 12 cases and gram negative bacteria in two cases. Of each of the latter two cases (cases 1 and 2), Pseudomonas maltophilia was the infecting organism and in one of these a similar organism was cultured from the pump oxygenator at the time of operation. Contamination of the pump oxygenator likely served as the source of infection in the other case, as the operations were performed two days apart in these two cases.

Unlike IE involving natural cardiac valves in which Streptococcal species are reported as the dominant organisms, Staphylococcus epidermidis was more commonly encountered in our series as noted by others. Contamination by cutaneous flora at the time of valve replacement has been implicated. Several reports have indicated that the incidence of postoperative IE was reduced from 4.2% to 1.2% with the prophylactic administration of antibiotics at the time of operation.

Prophylactic antibiotics should be tailored to the spectrum and sensitivity of prevailing cutaneous organisms in each institution. Late onset of IE due to Staphylococcus epidermidis without obvious source of infection may represent activation of dormant organisms introduced at the time of operation but suppressed by prophylactic antibiotics.

Previous Episodes of Bacterial Endocarditis

In five of our 16 patients, historical data either documented or suggested IE prior to the time of replacement of the aortic valve. In none of these was there clinical or anatomic evidence of active endocarditis at the time of removal of the natural valve. In case 8, pneumococcal endocarditis resulted in aortic insufficiency; two weeks following bacteriologic cure, the aortic valve was replaced. Six months later the patient was admitted with hemiparesis, fever and anemia. Evidence for minimal aortic insufficiency was present but repeated blood cultures gave negative results. During the two months of rehospitalization, pulmonary symptoms dominated for which five different antibiotics were given. After death, autopsy revealed a bronchiogenic carcinoma with cerebral metastases. The site of the aortic valve prosthesis was infected with numerous Candida organisms noted histologically. An embolus in the anterior descending coronary artery contained numerous organisms of this type.

In case 13, enterococcal endocarditis of the natural aortic valve resulted in severe aortic insufficiency necessitating surgical replacement of the aortic valve four years later. Thirty-one months following the insertion of a Starr-Edwards prosthetic valve, Staphylococcus faecalis endocarditis developed in this patient. The latter infection was successfully treated with antibiotics but surgical repair of a paravalvular leak was necessary four years after cure of the PAVE. In case 3, there was a history of IE following extraction of a tooth. Although blood cultures failed to grow organisms, aortic insufficiency resulted. At operation six months later, the noncoronary aortic cusp was noted to be perforated and the aortic valve was replaced by a Starr-Edwards prosthesis. One month postoperatively, PAVE due to Staphylococcus aureus developed. In case 15, the history was suggestive of IE five months prior to aortic valve replacement for aortic insufficiency. PAVE caused by Streptococcus viridans developed 36 days after insertion of the prosthesis. In case 14 when operation was done for aortic insufficiency, a perforation of the right aortic cusp suggestive of previous endocarditis was noted, although there was no history indicative of such an event.

Onset of Infection

Late onset of infection (longer than 25 days following operation) was observed in eight of 12 cases.
in groups I and II, and in three of four cases in group III. Early onset of infection (less than 25 days from time of operation) occurred in five of 16 cases among the three groups.

Disturbance of Atroventricular Conduction

There is a tendency for IE affecting natural aortic valves to cause disturbances of atrioventricular (A-V) conduction. This occurs either through direct invasion of the A-V conduction tissue or encroachment upon conduction tissue by the inflammatory process. In a report from our institutions on 55 cases of IE affecting the aortic valve only, first degree, second degree or third degree A-V block were described in 14 patients (25%). An A-V conduction disturbance was first noted in seven of 16 patients (44%) during the active course of PAVE, first degree in five patients, second degree in one, and complete heart block in one. Three of the seven patients developing an A-V conduction defect while suffering from PAVE died, one of cardiogenic shock and two suddenly. Murmurs of aortic insufficiency appeared during PAVE in four of seven patients (57%) with A-V block. The high incidence of conduction defects in PAVE may be due to extension of the infection along the suture lines, into the paravalvular area creating large paravalvular abscesses. These abscesses may include or disturb conduction tissue as well as create paravalvular leaks.

Aortic Insufficiency

Aortic insufficiency was noted in nine of 12 patients in groups I and II. In eight of these cases, aortic insufficiency developed during the course of recognized PAVE, while it preceded fever and positive blood cultures in one case. In this case (case 5), a prosthetic aortic valve had been inserted for aortic stenosis. Four weeks postoperatively, aortic insufficiency appeared. Three weeks later, blood cultures done for the first time yielded Staphylococcus aureus. No patient in group III had aortic insufficiency during the course of PAVE. Two patients (cases 12 in group II and 15 in group III) developed aortic insufficiency at intervals of one and two months after bacteriologic cure of PAVE. In both cases, aortic insufficiency was progressive and required reoperation four years and five months, respectively, from the time of bacteriologic cure of PAVE. Vegetations were absent at the time of operation in both patients.

Among patients with PAVE and aortic insufficiency, survival differed depending on whether the aortic prosthesis was originally placed for aortic insufficiency or for aortic stenosis. The prognosis appeared better if the aortic prosthesis were originally placed for predominant aortic insufficiency (four of nine survived; 44%) than for predominant aortic stenosis (two of seven survived; 29%). The differences, if valid, might be explained as follows: In patients with aortic stenosis, the left ventricle is hypertrophied and non-compliant secondary to a chronic, severe pressure overload. Because of a decrease in compliance, it may be less able to tolerate acute onset of aortic insufficiency secondary to a paravalvular leak. On the other hand, the left ventricle chronically volume overloaded from severe aortic insufficiency is dilated and compliant; it may therefore be better suited to handle the effects of a paravalvular leak, especially when this occurs early in the postoperative period.

Causes of Death

Causes of death in the ten patients with PAVE who died included sudden death in three cases (likely due to arrhythmias), congestive cardiac failure in two, surgical (reoperative) deaths in two, and bleeding into the brain and colon, one case each. One patient died of a bronchiogenic carcinoma metastatic to the brain but an embolus to the anterior descending coronary artery was also found at autopsy. In the two surgical deaths (cases 9 and 11), operation was undertaken as a last measure in the face of severe congestive cardiac failure secondary to paravalvular leaks. In each, the procedure was complicated by lack of adequate healthy tissue to which the prosthesis could be secured. Also, in one case (case 11), the infection had extended into the pulmonary trunk. This vessel ruptured during the course of the operation leading to massive hemorrhage. In one of the cases in which the patient died of persistent infection and cardiac failure (case 6), specific treatment had not been given for the Candida endocarditis which was first identified at autopsy (blood cultures had given negative results).

Pathology

The pathologic features of infectious endocarditis complicating aortic valvular prosthesis have peculiarities in the following categories: 1) the process at the valve seat, 2) infection extending through the aortic wall, and 3) distant effects of infectious endocarditis. The first of these is peculiar to infectious endocarditis complicating the presence of a prosthesis. The second is peculiar to endocarditis involving the aortic valve area, regardless as to whether a natural or prosthetic valve is involved, while the third is peculiar to bacterial endocarditis of either the natural or prosthetic mitral or aortic valve.

Infection at Valve Seat

At the valve seat, the infectious process tended to involve the entire circumference around the prosthesis. Abscess formation and destruction of tissue
Figure 1
Case 6. Candida PAVE. a) Base of antero-lateral wall of the left ventricle shows a mycotic aneurysm complicating infection at the prosthetic seat. b) Photomicrograph of the aneurysm shown in a. The anterior descending coronary artery appears in the right side of the illustration. Elastic tissue stain; ×1.5. c) Abscess formation at the base of the aneurysm portrayed in a and b. H & E; ×30. d) Spores and hyphae of Candida organisms in abscess shown in c. PAS; × 400.

Figure 2
Case 4. a) Aneurysm in antero-septal wall of the left ventricle complicating infection at prosthetic seat. Infecting organism was beta hemolytic Streptococcus. b) Case 5. Aortic valve prosthesis seen from above. Vegetations are sparse and partially cover the seat of the prosthesis. Infecting organism was Staphylococcus aureus.

with aneurysm formation was the hallmark of this process (figs. 1 and 2a). Consequences of the infectious process in this area included dehiscence of the seat of the prosthesis with resulting paravalvular leaks and formation of mycotic aneurysms of the structures bordering on the prosthetic seat. Vegetative formation was common (fig. 2b). When vegetations were found, these were not always extensive and it was not possi-
ble to distinguish between initially bland thrombi which became infected, and so represented the primary site of infection, or whether the infectious process started in the tissues bordering on the prosthesis and that the thrombi represented a process secondary to the infection.

**Figure 3**

Anatomic relations of the aortic valve. a) Horizontal section through base of heart, ascending aorta and pulmonary trunk to show varied anatomic relations of the root of the aorta. Tr. S. = transverse sinus of pericardium; P.A., L.A. and R.A. = posterior, left and right aortic valvular sinuses, respectively; R.P., L.P. and A.P. = right, left and anterior sinuses of pulmonary valve. b) Dissection has been carried through the wall of the infundibulum (R.V.) of the right ventricle and after exposure of the aortic wall its lateral wall has been removed to expose the aortic sinuses. R. = right; P. = posterior; L. = left. The posterior aortic sinus relates closely to the septal wall of the right atrium (R.A.), while the right aortic sinus is related to the infundibulum of the right ventricle. L.C. = ostium of left coronary artery; P.V. = pulmonary valve; C.S. = coronary sinus. c) Sagittal section through the commissure between the posterior aortic cusp (P.) and the left aortic cusp (not shown). The illustration shows continuity between the posterior aortic cusp and the anterior mitral leaflet (A.M.) through the intermediary of the aortic-mitral interventricular fibrosa (F.). At the confluence of the posterior mitral leaflet (P.M.), posterior wall of the left atrium (L.A.) and left ventricle there is incidental calcification of the mitral ring. Rectangle indicates plane of section used to prepare photomicrograph shown in d. B. = right aortic cusp. d) Photomicrograph of the aorta, posterior aortic cusp (P.), aortic-mitral interventricular fibrosa (F.) and the anterior mitral leaflet (A.M.). Interposed between the septal wall of the left atrium (L.A.) and the interventricular fibrosa is a wedge of epicardial tissue. Elastic tissue stain; × 3.
Infections Extending Through Aortic Wall

When infection of the tissues at the seat of an aortic valve prosthesis occurs, the process may extend through the wall of the aortic origin to involve contiguous structures in a manner similar to the potentials in this area when infection involves the natural aortic valve. The ultimate manifestation of these are dependent upon the degree of extension of the infectious process and the specific anatomic areas involved. The results may take the form of 1) an acquired fistula, 2) mitral insufficiency and 3) disturbance of A-V conduction.7

In order for the reader to gain a view as to the basis for these potentials, it is appropriate to review some of the important anatomic relationships of the aortic origin and the related aortic valve.

Except for that part of the aortic valve area from which the left coronary artery arises, the aortic valve is closely related to various intracardiac structures. The atrial septum abuts the posterior aspect in such a way that the posterior aortic sinus is closely related to the septal walls of the atria, particularly the right (fig. 3a). The wall of the right aortic sinus is formed by the septal wall of the right ventricular infundibulum (fig. 3b).

That part of the left aortic sinus from which the left coronary artery arises presents against the epicardium next to the pulmonary trunk, more anteriorly. That part of the left aortic sinus which is adjacent to the posterior sinus and part of the latter exhibit an intimate relationship with the anterior mitral leaflet.
This is portrayed by the fact that in these areas the aortic origin and related aortic cusps are in continuity with a fibrous membrane, the aortic-mitral intervalvular fibrosa (fig. 3c and d). The anterior mitral leaflet is in continuity with the lower aspect of the aortic-mitral intervalvular fibrosa. Along the left side of the intervalvular fibrosa lies a thin spur of epicardium and, in turn, along the left side of this spur is the septal wall of the left atrium.

The aforementioned summary of the anatomic relations of the aortic root will serve to explain the actual and potential complications of direct extension of infection through the wall of the aortic origin to adjacent structures. In the material at our disposal, several of the classical avenues of extension of infection were portrayed. Infection in relation to the posterior (non-coronary) cusp was associated with inflammatory processes involving the septal wall of the right atrium and underlying ventricular septum (fig. 4). In this region lies the membranous septum containing the bundle of His.

Extension of infection below the area at which the left and posterior cusps join led to infection of the epicardium between the aortic-mitral intervalvular fibrosa, on one hand, and the related septal wall of the left atrium, on the other (fig. 5). Though the underlying anterior mitral leaflet was spared in each case, it was potentially involved. Had the infection extended to the anterior mitral leaflet, destruction of this structure would have led to mitral insufficiency.

It will be recalled that the pulmonary valve and the origin of the pulmonary trunk lie at levels superior to that of the aortic valve. Yet infection extending through the aortic wall may proceed upward to cause secondary infection of the pulmonary trunk and its valve (fig. 6).

**Distant Lesions**

Infectious endocarditis complicating aortic valvular prostheses may, and frequently does, cause distant lesions no different from those complicating endocarditis of natural left-sided valves. These include embolism to and infarction of organs, metastatic abscesses and focal embolic glomerulonephritis (fig. 7).

**Treatment**

Antibiotic therapy was the sole treatment in ten cases and resulted in cure of four patients. Six other patients were treated surgically for prosthetic valve replacement in addition to receiving antibiotic therapy during the active phase of infection. This program resulted in cure of two patients. One died during the operation, while the remaining three died of complications of infection.

In considering reoperation for patients with PAVE, one may view three situations that may occur among such patients. The first is characterized by signs of infection but neither aortic insufficiency nor conduction defects. For these, reoperation is not indicated. At the other extreme are those patients with major degrees of aortic insufficiency or major conduction defects. These complications are signs of extensive destruction of tissue and frequently coexist. Reoperation for this...
group is indicated. This leaves a third group, intermediate between the two foregoing, in which aortic insufficiency and/or conduction defects are minimal. For this group it appears advisable to withhold operation unless aortic insufficiency or conduction defects progress in degree. Replacing a prosthetic valve in the face of bacteremia appears contrary to good medical judgment but successful cures of PAVE by early replacement of prosthetic valves during active infection have been reported.2, 4, 5, 17

Surgical intervention for PAVE caused by *Candida* remains a controversial issue as some feel reoperation is indicated simply in the presence of such an infection and in the absence of complicating aortic insufficiency or conduction defects.18

**Prevention**

The poor prognosis once PAVE develops underlines the need for effective preventive therapy among patients with prosthetic valves. Prompt treatment of any infection and prophylactic therapy during dental and surgical procedures are mandatory in such

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patients. In four of our patients (cases 4, 11, 13 and 14), PAVE was potentially preventable. Two cases developed bacteremia from peridontal infections and one from manipulation of the urinary tract. In the fourth patient (case 11), successful replacement of a prosthetic valve for severe aortic stenosis was followed by fatal PAVE 24 months later. The portal of entry was an untreated infected puncture wound of a finger. Thus, every patient with a prosthetic valve who develops infection in any part of the body, even when minor and localized, should receive immediate medical attention and appropriate antibiotic treatment for the potential blood stream contamination.

References
5. Walker SR, Shumway NE, Merigan TC: Management of infected cardiac valve prostheses. JAMA 208: 531, 1969
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J Madison, K Wang, F L Gobel and J E Edwards

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