Clinical outcome and echocardiographic findings of native and prosthetic valve endocarditis in the 1990s

R. Schulz, G. S. Werner, J. B. Fuchs, S. Andreas, H. Prange*, W. Ruschewskif and H. Kreuzer

Department of Cardiology, Centre for Internal Medicine, *Department of Neurology, †Department of Thoracic and Cardiovascular Surgery, Georg-August-University, Göttingen, Germany

Prosthetic valve endocarditis is considered to be associated with a more severe prognosis than native valve endocarditis. Among other factors, inappropriate visualization of vegetations in prosthetic valve endocarditis by transthoracic echocardiography is responsible for this observation. Since the introduction of transoesophageal echocardiography into clinical practice the diagnostic sensitivity and specificity of the detection of vegetations located on prosthetic valves have been enhanced. Therefore we aimed to determine and compare the prognosis of prosthetic valve endocarditis and native valve endocarditis in the era of this improved diagnostic approach.

One hundred and six episodes of infective endocarditis in 104 patients were seen at our institution between 1989 and 1993. Eighty patients (77%) had native valve endocarditis and 24 (23%) had late prosthetic valve endocarditis. In the latter group two patients had recurrent infective endocarditis. Patients with prosthetic valve endocarditis were older (mean age 64 vs 54 years in native valve endocarditis; \( P<0.001 \)) and the majority was female (62% vs 38% in native valve endocarditis; \( P<0.05 \)). In prosthetic valve endocarditis, infection of a valve in the mitral position predominated (65% vs 30% in native valve endocarditis; \( P<0.001 \)), whereas in native valve endocarditis more than half the cases had isolated aortic valve endocarditis (51% vs 27% in prosthetic valve endocarditis; \( P<0.01 \)). In prosthetic valve endocarditis more cases were caused by Staphylococcus aureus (31% vs 14% in native valve endocarditis; \( P=0.08 \)), whereas in native valve endocarditis the most frequent organisms were streptococci (29% vs 19% in prosthetic valve endocarditis; \( P=0.12 \)). Differences in the clinical features of native valve endocarditis and prosthetic valve endocarditis could not be found except for a higher rate of embolism in native valve endocarditis (40% vs 19% in prosthetic valve endocarditis; \( P<0.05 \)). Vegetations could be detected by transthoracic echocardiography more frequently in native valve endocarditis (71% vs 15% in prosthetic valve endocarditis; \( P<0.0001 \)). Transoesophageal echocardiography visualized vegetations in 95% of the episodes of native valve endocarditis and in 80% of the episodes of prosthetic valve endocarditis (\( P=0.09 \)). Thus, the diagnostic gain by transoesophageal echocardiography was greatest in prosthetic valve endocarditis. Patients with native valve endocarditis had significantly larger vegetations than patients with prosthetic valve endocarditis (\( P<0.05 \) for length, \( P<0.001 \) for width). The median time to diagnosis was similar in native valve endocarditis and prosthetic valve endocarditis (31 vs 28 days).

Surgery was performed in 74% of patients with native valve endocarditis and in 58% of those with prosthetic valve endocarditis; the median time delay between the diagnosis of infective endocarditis and surgery tended to be shorter in prosthetic valve endocarditis than in native valve endocarditis (45 vs 60 days). The in-hospital mortality and the mortality during a follow-up of 22±10 months did not significantly differ between native valve endocarditis and prosthetic valve endocarditis (21% vs 17%; 28% vs 25%).

In summary in the era of transoesophageal echocardiography, late prosthetic valve endocarditis does not seem to carry a worse prognosis than native valve endocarditis. This can be attributed in part to the improved diagnostic accuracy achieved by transoesophageal echocardiography leading to comparable diagnostic latency periods in both patient groups. Finally, better characterization of vegetations on prosthetic valves by transoesophageal echocardiography allows early lifesaving surgery in patients with prosthetic valve endocarditis.

(Eur Heart J 1996; 17: 281–288)

Key Words: Infective endocarditis, prosthetic valves, transoesophageal echocardiography, prognosis.
Introduction

The prognosis of prosthetic valve endocarditis is believed to be more severe than native valve endocarditis\(^{[1-9]}\). One factor possibly contributing to higher mortality rates in prosthetic valve endocarditis is the difficulty in diagnosing the disease with certainty. Early diagnosis is especially important for the clinical outcome of infective endocarditis, since mortality increases with a longer diagnostic delay\(^{[10,11]}\). The diagnosis of infective endocarditis is mainly based on clinical features and blood culture findings. If these are non-specific or absent the diagnosis relies on the demonstration of vegetations on echocardiography. Therefore the advent of transthoracic echocardiography was considered as a major diagnostic aid in infective endocarditis\(^{[12,13]}\). Unfortunately transthoracic echocardiography has some limitations in its ability to visualize vegetations.

The application of transoesophageal echocardiography to patients with infective endocarditis led to higher diagnostic specificity and sensitivity in detecting vegetations than transthoracic echocardiography\(^{[14-18]}\). Because of the close anatomical relationship of the oesophagus to the heart, transoesophageal echocardiography is able to visualize vegetations with a diameter as small as 1–2 mm. The acoustic shadowing of prosthetic valves can be overcome with the help of transoesophageal echocardiography, thus markedly improving imaging of vegetations in prosthetic valve endocarditis\(^{[19]}\).

However, it had not been established if this overall improvement in diagnostic imaging also influenced the clinical outcome of infective endocarditis. Therefore the aim of the present study was to determine the prognosis of infective endocarditis in the era of transoesophageal echocardiography with special reference to patients presenting with prosthetic valve endocarditis.

Methods

We searched the database of the University Hospital of Göttingen, Germany, for all patients admitted with a diagnosis of infective endocarditis between June 1989 and December 1993.

Patients were included in the study only if they had positive histopathological evidence of infective endocarditis by autopsy or cardiac surgery and/or a clinically well-defined diagnosis according to recently established diagnostic criteria (positive blood cultures, fever, new or changing heart murmurs, characteristic echocardiographic findings)\(^{[20-23]}\). Patients who did not demonstrate vegetations on echocardiography were judged to have infective endocarditis only if they had positive blood cultures and clinical features compatible with infective endocarditis. Alternatively, histopathological proof of infective endocarditis was required.

After having applied the above mentioned criteria to all available patient charts, 104 patients with 106 episodes of infective endocarditis were found to have a definite diagnosis of infective endocarditis. Sixty-four patients (61.5\%) had been admitted from other hospitals to our institution which serves as a tertiary referral centre for an area with approximately 1 million inhabitants. The remaining 30 patients (28.5\%) were seen throughout their treatment at our hospital. We compared 80 patients with native valve endocarditis and 24 with late prosthetic valve endocarditis. Patients with early prosthetic valve endocarditis (i.e. endocarditis occurring within the first 2 months after valve insertion) were excluded. Patient data were retrospectively analysed from the medical records.

Clinical data

We recorded the age and gender of the patients, predisposing factors (e.g. pre-existing heart disease), possible sources of infection (dental or other surgery) and the distribution of infected valves.

The infective organism for each episode of infective endocarditis was obtained from blood culture findings. Blood cultures were judged as positive if the same organism was isolated from more than three consecutive blood samples. The laboratory data representative for infection (white blood cell count and erythrocyte sedimentation rate) were analysed. Leukocytosis was defined as a white blood cell count above 10 000 \(\mu L^{-1}\), the erythrocyte sedimentation rate was considered elevated if it was greater than 20 mm in the first hour. The presence or absence of fever and splenomegaly were assessed. Fever was defined as temperatures greater than 38.0°C, splenomegaly as a longitudinal sonographic diameter of the spleen of more than 11 cm. The time between the onset of clinical symptoms (fever, weight loss, embolic events etc.) and the diagnosis of infective endocarditis (positive echocardiographic and blood culture findings) was established for each patient. The individual therapeutic regimen (antibiotic therapy, surgery) and the clinical course (death, occurrence of complications such as abscess formation, embolism, persistent fever or overt heart failure) were evaluated. Major embolic events were defined by both clinical symptoms (sudden neurologic deficits, ischaemia of a limb) and definitive findings on diagnostic imaging procedures (computed tomography of the brain, ultrasound and computed tomography of the abdomen and kidney). Heart failure was staged according to NYHA classes I–IV. If available, histopathological proof for infective endocarditis was taken from reports at surgery or autopsy. Finally, mortality during follow-up after hospital discharge was examined, which was done by contacting the patients or their physicians via phone calls in May 1994.

Echocardiographic data

Echocardiographic examinations were performed with a Hewlett Packard Sonos 1000 device. For transthoracic echocardiography we used a 2.5 MHz probe and for
transoesophageal echocardiography a 5 MHz monoplane probe. If vegetations could not be viewed by transthoracic echocardiography despite a clinical suspicion of infective endocarditis or if transthoracic echocardiography findings were inconclusive transoesophageal echocardiography was performed. Furthermore, transoesophageal echocardiography was obtained if it was thought that it would aid in therapeutic decision making by better characterizing vegetations already detected by transthoracic echocardiography. All examinations had been recorded on VHS video tapes and were reviewed independently by two experienced echocardiographers blinded to the clinical course of the individual patient. The detection rate of vegetations was determined for transthoracic echocardiography and transoesophageal echocardiography. Vegetations were classified by size (length and width in mm), mobility (mobile or immobile) and echodensity (low, medium or high with respect to the surrounding myocardial tissue). An abscess was diagnosed if a circumscribed area of low echodensity could be detected within the tissue adjacent to an infected valve.

**Statistical analysis**

The data are shown as mean ± standard deviation. When there was no normal distribution of individual values, the median of each group was given. Comparison of the parameters between the two patient groups was tested by the Student’s t-test; comparison of discrete parameters was made by the Chi-square test. Multivariate risk analysis using the Cox Harzards ratio model was carried out to define the influence of various factors on the clinical outcome of infective endocarditis. Life table analysis was performed by the Kaplan-Meier method. Differences of survival curves were evaluated by a multigroup non-parametric test. All data were analysed on a personal computer by using the statistical software package NCSS (Number Cruncher Statistical System, Version 5.03 by Jerry L. Hintze, Kaysville, Utah, U.S.A.). Statistical significance was defined as P<0.05.

**Results**

**Clinical characteristics**

Of the 104 patients with infective endocarditis, 80 patients (77%) had native valve endocarditis and 24 (23%) had prosthetic valve endocarditis. Two female patients with prosthetic valve endocarditis had recurrent episodes of infective endocarditis. The mean age was higher for prosthetic valve endocarditis (64 ± 8 years, range: 43–80 years) as compared with native valve endocarditis (54 ± 16 years, range: 20–82 years; P<0.001), (Fig. 1). The ratio of female to male patients was 0:6 in native valve endocarditis (30 females, 50 males) and 1:4 in prosthetic valve endocarditis (14 females, 10 males; P<0.05).

Predisposing factors for the development of infective endocarditis were present in 38 patients with native valve endocarditis (48%). Possible sources of infection, such as preceding dental or other surgery, could be found in 40% of episodes in each group (32 episodes of native valve endocarditis, 10 episodes of prosthetic valve endocarditis).

The distribution of infected valves is shown in Table 1. More than half of the native valve endocarditis episodes had isolated aortic valve endocarditis, whereas in prosthetic valve endocarditis isolated infection of a prosthesis in the mitral position clearly predominated. The majority of episodes of prosthetic valve endocarditis involved mechanical valves (24 episodes, 92%), only two episodes (8%) occurred in patients with a bioprosthetic valve. The blood culture findings are shown in Fig. 2. In native valve endocarditis the most frequent organisms were streptococci, in prosthetic valve endocarditis there was a prevalence of staphylococci. Culture-negative infective endocarditis was observed in 13 episodes of native valve endocarditis and three episodes of prosthetic valve endocarditis (16% vs 11.5%).

The laboratory data and clinical features of native valve endocarditis and prosthetic valve endocarditis are summarized in Table 2. Patients with prosthetic valve endocarditis tended to present more often without fever and without splenomegaly when compared to patients with native valve endocarditis, but these differences did not reach statistical significance. With regard to laboratory parameters, none of the patients...
with native valve endocarditis had a normal erythrocyte sedimentation rate as opposed to three patients with prosthetic valve endocarditis.

Leukocytosis was detected with a relatively low frequency in both patient groups (35% in native valve endocarditis, 42% in prosthetic valve endocarditis). The latency period between the onset of clinical symptoms and the time of diagnosis was similar in native valve endocarditis (median 31 days, range: 0–183 days) and prosthetic valve endocarditis (median 28 days, range: 0–182 days).

### Echocardiographic data

Transcatheter echocardiography was performed in all episodes of infective endocarditis. In 58 episodes of native valve endocarditis (73%) and in all but one episode of prosthetic valve endocarditis (96%) transesophageal echocardiography was carried out. The results of transcatheter echocardiography and transesophageal echocardiography are given in Table 3. Vegetations could be detected by transcatheter echocardiography in 71% of native valve endocarditis, but in only 15% of prosthetic valve endocarditis ($P<0.0001$). Transesophageal echocardiography was able to visualize vegetations in 95% of native valve endocarditis and in 80% of prosthetic valve endocarditis ($P=0.09$).

Thus, the detection rate for vegetations could be enhanced through by 24% the additional application of transesophageal echocardiography in native valve endocarditis and 65% in prosthetic valve endocarditis.

In Table 4, the characteristics of the vegetations as observed by transesophageal echocardiography are shown. Patients with native valve endocarditis had larger vegetations than patients with prosthetic valve endocarditis. The percentage of mobile vegetations was similar between prosthetic valve endocarditis and native valve endocarditis, as was the distribution of echodensity among the vegetations. In prosthetic valve endocarditis the patients who underwent surgery had a trend toward larger and more mobile vegetations on transesophageal echocardiography than the patients who were only treated by antibiotic therapy (length $12 \pm 7$ mm vs $8 \pm 5$ mm, $P=ns$; width $5 \pm 2$ mm vs $4 \pm 3$ mm, $P=ns$; mobility 79% vs 42%, $P=0.05$). In

### Table 2 Laboratory values and clinical features in 106 episodes of infective endocarditis

<table>
<thead>
<tr>
<th></th>
<th>Native valve endocarditis (n=80)</th>
<th>Prosthetic valve endocarditis (n=26)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fever</td>
<td>63 (79%)</td>
<td>16 (62%)</td>
</tr>
<tr>
<td>Mean maximal temperature (Celsius)</td>
<td>39-3 ±0-7</td>
<td>39-2 ±0-6</td>
</tr>
<tr>
<td>Mean white blood cell count (number of cells . ul)</td>
<td>10-000 ±5-200</td>
<td>12-200 ±7-800</td>
</tr>
<tr>
<td>Leukocytosis</td>
<td>28 (35%)</td>
<td>11 (42%)</td>
</tr>
<tr>
<td>Mean ESR (mm . 1 h)</td>
<td>64 ±28</td>
<td>62 ±30</td>
</tr>
<tr>
<td>ESR not elevated</td>
<td>0</td>
<td>3 (17%)</td>
</tr>
<tr>
<td>Splenomegaly</td>
<td>24 (30%)</td>
<td>4 (15%)</td>
</tr>
<tr>
<td>Predisposing factors</td>
<td>38 (48%)</td>
<td>3 (17%)</td>
</tr>
<tr>
<td>Possible source of infection</td>
<td>32 (40%)</td>
<td>10 (40%)</td>
</tr>
<tr>
<td>Congestive heart failure (NYHA classes III and IV)</td>
<td>37 (46%)</td>
<td>11 (42%)</td>
</tr>
<tr>
<td>Embolism</td>
<td>32 (40%)</td>
<td>5 (19%)*</td>
</tr>
</tbody>
</table>

* $P<0.05$ for comparison of native valve endocarditis vs prosthetic valve endocarditis.

Fever $>38^\circ$ C; leukocytosis $>10\ 000\ \mu l^{-1}$; ESR (erythrocyte sedimentation rate) = normal $<20\ mm\ .\ h$; splenomegaly = longitudinal sonographic diameter $>11\ cm$.

### Table 3 Detection of vegetations by transthoracic (TTE) and transesophageal echocardiography (TEE)

<table>
<thead>
<tr>
<th></th>
<th>TTE</th>
<th>TEE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Examined</td>
<td>Positive</td>
</tr>
<tr>
<td>Native valve endocarditis (n=80)</td>
<td>80 (100%)</td>
<td>57 (71%)</td>
</tr>
<tr>
<td>Prosthetic valve endocarditis (n=26)</td>
<td>26 (100%)</td>
<td>26 (100%)</td>
</tr>
</tbody>
</table>

* $P<0.0001$, ** $P = 0.09$ for comparisons of native valve endocarditis vs prosthetic valve endocarditis.

Eur Heart J, Vol. 17, February 1996
Table 4 Characteristics of vegetations on transoesophageal echocardiography (TEE)

<table>
<thead>
<tr>
<th></th>
<th>Length (mm)</th>
<th>Width (mm)</th>
<th>Echodensity (%)</th>
<th>Mobility (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Native valve endocarditis</td>
<td>13 ± 6</td>
<td>8 ± 5</td>
<td>10</td>
<td>62</td>
</tr>
<tr>
<td>(n=58)</td>
<td></td>
<td></td>
<td>62</td>
<td>28</td>
</tr>
<tr>
<td>Prosthetic valve endocarditis</td>
<td>10 ± 6*</td>
<td>5 ± 3**</td>
<td>5</td>
<td>77</td>
</tr>
<tr>
<td>(n=25)</td>
<td></td>
<td></td>
<td>77</td>
<td>18</td>
</tr>
</tbody>
</table>

*P<0.05, **P<0.001 for comparisons of native valve endocarditis vs prosthetic valve endocarditis.

Native valve endocarditis, vegetation characteristics did not significantly differ between surgically and non-surgically treated patients (length 13 ± 7 mm vs 13 ± 5 mm; width 8 ± 5 mm vs 8 ± 4 mm; mobility 64% vs 70%). Abscess formation was seen in four episodes of prosthetic valve endocarditis (17%) and in six episodes of native valve endocarditis (8%).

**Clinical outcome**

All patients were treated by antibiotic therapy. Fifty-nine patients (74%) with native valve endocarditis and 14 patients (58%) with prosthetic valve endocarditis underwent surgery. Early surgery (i.e. during the initial hospital stay) was performed in all but one patient with native valve endocarditis who had late surgery. The median time delay between the diagnosis of infective endocarditis and surgery tended to be shorter in prosthetic valve endocarditis than in native valve endocarditis (45 vs 60 days), but this difference was not significant. The main indication for surgery in both groups was heart failure resistant to medical therapy (68% in native valve endocarditis vs 55% in prosthetic valve endocarditis, followed by persistent fever (23% vs 36%) and recurrent embolism (9% vs 7%). All cases with abscess formation were transferred to immediate surgery due to destruction of the infected valve. The median duration of hospitalization tended to be longer in prosthetic valve endocarditis than in native valve endocarditis (86 vs 52 days, P=0.14).

Overt heart failure (NYHA class III and IV) complicated the clinical course in similar proportions in native valve endocarditis and prosthetic valve endocarditis (46% vs 42%). A higher rate of embolism occurred in native valve endocarditis (40% vs 19% in prosthetic valve endocarditis; P<0.05). Seventeen patients (21%) with native valve endocarditis and four (17%) with prosthetic valve endocarditis died during their initial hospital stay. In prosthetic valve endocarditis the in-hospital mortality rate was similar in surgically and non-surgically treated patients (21% vs 17%). In native valve endocarditis the patients who had surgery had a lower overall death rate than the patients who were managed without surgery (15% vs 38%). Factors associated with a high mortality of infective endocarditis were embolism (P<0.005), congestive heart failure (P<0.01) and mitral valve involvement (P<0.05). Age and gender did not influence survival. Patients with negative blood cultures did not have a worse prognosis than patients with positive blood cultures. The nature of the infective organism also did not significantly determine the clinical outcome of infective endocarditis.

After a median follow-up of 22 ± 10 months, mortality rates were 28% for native valve endocarditis and 25% for prosthetic valve endocarditis. After hospital discharge, five patients died of causes unrelated to infective endocarditis (four patients with native valve endocarditis, one patient with prosthetic valve endocarditis). Two patients died due to late sequelae of their infective endocarditis (one patient with native valve endocarditis and prosthetic valve endocarditis, respectively). One death occurred in a patient with recurrent prosthetic valve endocarditis. The survival curves for the two patient groups are given in Fig. 3.

**Discussion**

Prosthetic valve endocarditis can be divided into early and late prosthetic valve endocarditis. Early prosthetic valve endocarditis develops within the first 2 months after valve replacement and is most often caused by
staphylococcal contamination in the peri-operative period. In contrast, late prosthetic valve endocarditis is defined as occurring more than 2 months after surgery and is contracted in a similar manner as native valve endocarditis. Consequently, the pattern of infection in late prosthetic valve endocarditis resembles that of native valve endocarditis. In earlier reports, mortality of prosthetic valve endocarditis ranged between 46% and 64%[9,24] with the highest fatality rates occurring in early prosthetic valve endocarditis. Patients with late prosthetic valve endocarditis are supposed to carry a prognosis intermediate between early prosthetic valve endocarditis and native valve endocarditis5.

Some studies performed during the 1980s had already shown a steady improvement of the prognosis of late prosthetic valve endocarditis[25,26]. These observations were mainly ascribed to the success of early surgical intervention in these patients[27,28]. In our retrospective analysis of 106 episodes of infective endocarditis, the in-hospital mortality in the group of patients with late prosthetic valve endocarditis was very low (17%). Mortality in prosthetic valve endocarditis was not significantly different from mortality in native valve endocarditis during the initial hospital stay and after a median follow-up period of about 2 years. With regard to the above mentioned data this shows further improved clinical outcome for patients with prosthetic valve endocarditis in the 1990s.

One major setback for the diagnosis of prosthetic valve endocarditis is the low diagnostic yield of transthoracic echocardiography in these patients. This may postpone the initiation of appropriate therapy and contribute to the high mortality rates formerly observed in prosthetic valve endocarditis. By the combined echocardiographic approach used in this study (transthoracic echocardiography supplemented by transoesophageal echocardiography as needed) we could detect vegetations in 95% of the episodes of native valve endocarditis and 80% of the episodes of prosthetic valve endocarditis. A considerable diagnostic gain through the additional application of transoesophageal echocardiography was achieved in patients with prosthetic valve endocarditis in accordance with recent studies[17,29]. The main reason is that the shadowing effect of prosthetic valves is reduced when performing echocardiography via the transoesophageal route[19]. At a time when transoesophageal echocardiography was not available, Nihoyannopoulos found an average duration of symptoms before the diagnosis of infective endocarditis of more than 9 weeks[11]. In our study this time interval was considerably shorter and it was similar between native valve endocarditis and prosthetic valve endocarditis. Thus, our data reflect shortening of the diagnostic latency period by the use of transoesophageal echocardiography. Since the mortality of infective endocarditis increases with a longer diagnostic delay, we postulate that transoesophageal echocardiography had a beneficial effect on the clinical outcome of infective endocarditis.

During the study period we routinely used monoplane transoesophageal echocardiography for the evaluation of our patients. In the meantime, transducer technology has improved with widespread availability of bi- and multiplex transoesophageal echocardiography. One possible advantage of bi- and multiplex transoesophageal echocardiography over monoplane transoesophageal echocardiography might be their superior delineation of prosthetic valves in the aortic position and a better recognition of abscess formation, but the high diagnostic sensitivity achieved with monoplane transoesophageal echocardiography in our study would have been only marginally enhanced.

In our study population, the surgery rate was relatively high when compared with data from the literature. The main cause was that about 60% of our patients were admitted from other hospitals because of associated multimorbidity and a complicated clinical course of infective endocarditis. In the pre-transoesophageal echocardiography era, the choice of therapy was mainly made on the basis of clinical considerations. With the advent of transoesophageal echocardiography, therapeutic decision making may be influenced by the possibility of a more precise characterization of vegetations by this technique. Because larger and mobile vegetations are thought to be associated with a more complicated course of infective endocarditis[29], patients who demonstrate such findings on transoesophageal echocardiography are likely to undergo surgery. On the other hand if small and immobile vegetations are found, conservative management may be applied. Accordingly, in the group with prosthetic valve endocarditis, patients who had surgery had larger and more mobile vegetations on transoesophageal echocardiography than those who could be managed by conservative therapy alone. The patients with prosthetic valve endocarditis had a slightly lower overall surgery rate and a shorter interval between the diagnosis of infective endocarditis and surgery than the patients with native valve endocarditis. The in-hospital death rate of prosthetic valve endocarditis was similar in surgically and non-surgically treated patients. In our opinion, these data illustrate the value of transoesophageal echocardiography in guiding therapy in patients with prosthetic valve endocarditis. With the help of transoesophageal echocardiography, surgery could be avoided in some of these patients but if clearly indicated it could be performed as early as possible.

The percentage of negative blood cultures was similar among patients with native valve endocarditis and prosthetic valve endocarditis. In prosthetic valve endocarditis, more episodes were caused by Staphylococcus aureus infection. Previous studies suggested a more severe clinical course for patients with culture-negative infective endocarditis and for infective endocarditis caused by certain infective organisms such as Staphylococcus aureus[2,30]. However, we could not detect a difference in clinical outcome of infective endocarditis with positive or negative blood cultures. Furthermore infective endocarditis caused by Staphylococcus aureus was not associated with a higher mortality rate than infective endocarditis caused by other organisms.
There were no apparent differences in laboratory data and clinical features among the patient groups except for major embolic events which occurred more often in patients with native valve endocarditis than prosthetic valve endocarditis. Since the risk of embolism is thought to increase with vegetation size, the higher rate of embolism in native valve endocarditis might be explained by the echocardiographic finding of larger vegetations in these patients. The preceding oral anticoagulation of the patients with prosthetic valves could have led to smaller vegetations and hence to a lower thromboembolic risk in prosthetic valve endocarditis. The higher embolic risk in native valve endocarditis was an adverse prognostic factor and certainly contributed to the higher mortality rate in non-surgically vs surgically treated patients with native valve endocarditis. Many patients with native valve endocarditis were admitted with extensive cerebral embolism and severely impaired neurologic status which made them ineligible for early life-saving therapy. Congestive heart failure (NYHA class III and IV) was the second most important prognostic factor after embolism. As in earlier studies, it was the most prevalent complication of infective endocarditis and the leading cause of surgery. Mitral valve endocarditis also had an adverse effect on the prognosis of infective endocarditis and mitral valve infection has been reported to be associated with a higher rate of embolism; however, in our study population it could not be linked to a higher incidence of embolic events. The patients with prosthetic valve endocarditis were older than the patients with native valve endocarditis. Studies performed in the pre-transesophageal echocardiography era have found a worse prognosis for infective endocarditis with advancing age. Therefore one would have expected a higher mortality rate of the patients with prosthetic valve endocarditis, but we could not confirm an adverse effect of age on the prognosis of infective endocarditis. Perhaps this might be due to the ability of transesophageal echocardiography to improve the detection of vegetations in elderly patients.

In summary, the prognosis of native valve endocarditis and late prosthetic valve endocarditis in the era of transesophageal echocardiography is comparable. This improved prognosis of prosthetic valve endocarditis may be attributed in part to the introduction of transesophageal echocardiography into clinical practice which shortens diagnostic latency periods. The early detection of vegetations by transesophageal echocardiography leads to the timely institution of therapy. Finally, better characterization of vegetations by transesophageal echocardiography can adequately influence the therapeutic strategy in patients with infective endocarditis.

References


