Editorial:
The Changing Role of Surgery in the Management of Infective Endocarditis

ELLIO T RAPAPORT, M.D.

IN THE PAST THREE DECADES a striking change has occurred in the microbiological spectra, clinical features and natural history of infective endocarditis. Undoubtedly, the development of a host of potent antimicrobial agents and their widespread availability and, at times, indiscriminate use, have played a key role. Other important factors include the use of prosthetic heart valves, the increasing use of immunosuppressant agents and the epidemic of intravenous drug abuse in urban centers.

It is ironic that cardiac surgery in the uninfected heart has created the environment for a significant increase in the number of cases of infections of the heart. As improvements in the design of both mechanical and tissue valves have occurred, providing enhanced long-term survival, and as the immediate operative risk has decreased, a large population of patients has been spawned who currently have one or more prosthetic valves. These valves, however, may be the source of various morbid events, among which is infective endocarditis. At one center, approximately one-third of those found at autopsy to have infective endocarditis had a prosthetic heart valve.1 The irony lies in the fact that prosthetic valve replacement is increasingly being used therapeutically to manage prosthetic valve infection.

The changing clinical picture of infective endocarditis is startling. The classic pattern of subacute bacterial endocarditis characterized by a protracted history of constitutional symptoms including low-grade fever, night sweats, weight loss, general malaise, easy fatigability, arthralgia and pallor, coupled with changing cardiac murmurs and evidence of small, repeated peripheral embolizations in a patient with known pre-existing heart disease, is infrequently seen. Today, in contrast, the patient is likely to be acutely toxic and far more febrile. He tends to be ill for less time before he seeks medical help, frequently has either a prosthetic heart valve or no known previous cardiac abnormality, often is an intravenous drug abuser (particularly in an urban setting), may present without a murmur but with septic pulmonary emboli reflecting tricuspid valve involvement or a major peripheral embolus (particularly if the microorganism is a fungus), and, finally, may develop acute pulmonary edema and/or peripheral vascular collapse reflecting sudden hemodynamic deterioration.

The difference in the clinical picture of infective endocarditis primarily reflects the change in microbiology. Whereas oral strains of Streptococcus viridans were responsible for over 70% of cases of infective endocarditis in the pre-antibiotic era, they now constitute well under half of such cases.2 Many more cases of staphylococcal, gram-negative bacteria and fungal infections are seen today. The exact percentage breakdown of various microorganisms will differ materially from center to center, depending on the population served.

In the preceding article appearing in this issue of Circulation, Richardson, Karp, Kirkin and Dismukes describe their experience at the University of Alabama Medical Center with the treatment of infective endocarditis over the past decade. In 43% of their patients, a Streptococcus was involved, in 36% a Staphylococcus, but only 8% were due to gram-negative organisms and 4% to fungus infections. As they point out, their small experience with gram-negative and fungal endocarditis presumably reflects the infrequency with which they are exposed to the problem of drug addiction; there were only three known intravenous drug abusers in their series. In contrast, over almost the same period of time, we have seen 97 episodes of infective endocarditis among 79 known drug addicts at the three teaching hospitals of the University of California, San Francisco. Although we observed a comparable incidence of Staphylococcus infections, 21% of our cases were due to fungi, either isolated or mixed, and just one specific gram-negative organism, Serratia marcescens, was the offending pathogen in 8% of the cases. This disparity in the incidence of various pathogens causing infective endocarditis is also apparent in the experience of others.3-7

The offending organism and the valve on which it is lodged have a profound bearing on management. In their compilation of surgical experience at the University of Alabama Medical Center, Richardson et al. reported only a 14% early mortality among 81 patients with native valve endocarditis. The longer term survival compares reasonably with their experience in other types of patients requiring prosthetic replacement. However, it should be noted that only two of their patients had a gram-negative infection (one died) and there were no fungal infections among their group. Our perioperative mortality of 16% among 27 cases managed surgically is comparable. However, 15 of these patients had either pure or mixed fungal infections (Candida in all but one) and half were dead at a mean 16 months postoperatively because of persistent infection, complications related to continued drug abuse, including reinfection, or complications from the original surgery.

From the Section of Cardiology, Department of Medicine, San Francisco General Hospital, and the University of California, San Francisco.

Address for reprints: Elliot Rapaport, M.D., Professor of Medicine, University of California Service, San Francisco General Hospital, San Francisco, California 94110.

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Sterilization of valves infected with organisms like *Serratia* or *Candida* is rarely accomplished medically. Consequently, early surgical extirpation is indicated in view of the almost universal lethal outcome with antibiotic treatment alone, and before serious complications ensue. Thus, although no patient in the series reported in this issue by Richardson et al. was treated surgically for the primary indication of a resistant organism, this should not be extrapolated to infections resulting from these specific organisms. Tricuspid valve endocarditis was also seen infrequently by Richardson and his associates, despite the relatively high incidence of staphylococcal infections. Only 3% of their native infective endocarditis group had a recognized pure tricuspid valve infection and another 2% had multivalvular infections which included the tricuspid valve. Even if one looks only at narcotic addicts with infective endocarditis, a great variability is reported in the incidence of tricuspid involvement. Cherubin et al. found tricuspid regurgitation in only 18%, although 48% had *Staphylococcus aureus* infections. In contrast, Banks found an 84% incidence of tricuspid valve involvement in 50 heroin addicts in whom 70% with positive blood cultures grew out of *Staphylococcus.* Part of these differences probably reflects differing criteria used to establish tricuspid valve infection. Some investigators require that the murmur of tricuspid insufficiency be present, while others accept pulmonary infiltrates consistent with pulmonary emboli together with positive blood cultures as adequate criteria for the diagnosis of tricuspid valve endocarditis.

It is important to point out that the conclusions reached by Richardson et al. regarding the indications for surgery in staphylococcal native valve endocarditis are not applicable to tricuspid valve involvement. Tricuspid valve endocarditis due to *Staphylococcus* has a surprisingly good prognosis when managed medically. In our experience at the University of California, San Francisco, among 28 I.V. drug abusers with *Staphylococcus aureus* tricuspid valve endocarditis and in two additional patients who had combined aortic and tricuspid valve infection, there was only one death within the first two weeks of hospitalization with medical management; a patient with combined aortic and tricuspid endocarditis died from CNS septic emboli on the twelfth day. There were two late deaths; one died 49 days after admission from pneumonia and congestive heart failure and the other had an unrelated death due to an automobile accident 96 days after his initial admission. It is thus clear to us that most Staphylococcal infections can be brought under control with appropriate antibiotic therapy when the infection is confined to the tricuspid valve, despite the fact that the patient may experience bouts of septic pulmonary emboli. Surgery was unnecessary in any of our patients with tricuspid valve *Staphylococcus* infective endocarditis either because of hemodynamic impairment or for irradiation of the infection.

Richardson et al. have presented impressive results for the surgical management of the acute phase of infective endocarditis. Their early mortality of 14% contrasts favorably not only with the collected surgical experience of 31% reported in a recent interview but also with the results of medical management in their own institution. It is clear that a more aggressive role for surgical management of infective endocarditis is warranted. There is increasing recognition that patients with native valve endocarditis who develop significant hemodynamic impairment manifested by moderate to severe cardiac decompensation are best managed surgically with immediate operation in view of the 50–89% mortality rate under these circumstances otherwise. As described elsewhere in this issue by Wilson et al., surgery can be accomplished successfully even when the infection is still rampant and blood cultures are still positive.

Nevertheless, one should cautiously interpret retrospective statistical comparisons of the outcome of patients with infective endocarditis treated medically with those managed surgically, even in the same institution. In the absence of a randomized, controlled prospective study, compilations of surgical and medical mortality are fraught with hidden bias and selection. This is particularly important in light of the recommendation of the University of Alabama surgical group that patients with *Staphylococcal* endocarditis, regardless of hemodynamic state, should undergo early valve replacement. This conclusion is premature, in my judgment, and should await a controlled randomized study of patients with staphylococcal endocarditis of the mitral and aortic valves, in which patients without significant hemodynamic impairment are managed either medically or surgically. In the absence of such a study, it would seem more prudent to recommend that all patients with native valve endocarditis due to *Staphylococcus aureus* who do not have evidence of moderate or severe cardiac failure should be initially managed medically with appropriate antibiotics. This may prove to be adequate. Of course, such patients should be followed extremely closely and if evidence of hemodynamic deterioration appears, they should then be considered for urgent or emergency valve replacement.

References

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