Bacterial Endocarditis and Prosthetic Valves

Among the major challenges to the physician and surgeon managing patients with valvular heart disease by the use of prosthetic valves is the possibility of postoperative bacterial endocarditis. Such infections occur in relation to the seat of the prosthesis and may occur either in early or late postoperative states. The challenges include both the phases of prevention and treatment.

In the area of prevention, one problem is that prophylactic antibiotic therapy does not necessarily prevent the occurrence of infection.1, 2 One reason is that among the organisms involved certain are resistant to the effects of the common prophylactic antibiotic programs employed. The organisms that have proved particularly troublesome are those in the category of penicillin-resistant Staphylococcus3 and various gram-negative organisms.4 Another reason is that organisms which are sensitive to the prophylactic antibiotics may survive at the operative site.

Assuming that one may come up with an antibiotic program appropriate to deal both with sensitive and resistant organisms, the evidence suggests that it is difficult to obtain sufficiently high concentrations of antibiotics for destruction of organisms in relation to sutures and in the tissues bordering on the seat of the prosthesis. These difficulties underlie an escape phenomenon in which organisms inadvertently inserted at operation lie dormant during the early postoperative period and later, weeks or months postoperatively, bloom so as to cause an evident and significant infection.2, 4

Assuming that certain late infections in relation to prosthetic valves represent infection some time after the operation, one must be aware of the various portals of entry of organisms to which the patient is subject.4-6 Respiratory or dental infections and infections relating to inserted intravenous needles or catheters are sources of bacterial endocarditis.

Nasogastric tubes and indwelling urinary catheters serve as potential causes of bloodstream contaminants. The physician mindful of the potential hazards of the foregoing may take appropriate steps to minimize these potentials for ultimate infection in relation to prostheses.

Existing infection, whether from organisms introduced at the time of operation or later, poses major problems to the patient. These take on two aspects, one being the various features of intracardiac infection, including embolism, and the other being malfunction of the prosthesis, either through the presence of vegetations or from partial separation of the prosthesis from its site of insertion.

In either case, the infection takes on the feature of being associated with devitalized tissue and abscess formation at the valve ring, each in relation to a foreign body, the latter including the prosthesis and its retaining sutures.

These features, coupled with the common presence of resistant organisms, make it more difficult to overcome the infection than is the case with bacterial endocarditis on natural valves.

Whether or not it becomes necessary to replace the prosthesis, either because of malfunction or continuation of infection, antibiotic therapy must not only be specifically geared to the infecting organism but must be intense and protracted. When bacteriologic “cure” is obtained it is important to realize that living organisms may still be present in a dormant state waiting for the day when antibiotic therapy is stopped for them to reactivate infection. This point underlies the logic of extending antibiotic therapy over a longer period than that employed in classic examples of bacterial endocarditis.

Considering the possible dormance of organisms with the highly dangerous nature of bacterial endocarditis complicating prosthetic valves, antibiotic therapy should properly be continued for longer periods than that practiced in the patient with ordinary bacterial endocarditis after a “bacteriologic cure” has been accomplished.

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References

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