INTRODUCTION

Surgical insertion of prosthetic devices into the human body has become a common practice worldwide. Prosthetic hip and knee replacements, prosthetic cardiac valve insertion, mesh repair of hernias, insertion of breast implants, to name but a few, are carried out in enormous numbers all over the world at astronomical health care costs in terms of equipment, personnel, hospital stay, and management of complications \(^1\). Infection of prosthetic devices is one of the most common complications and it has huge implications as measured by patient morbidity, mortality, and cost. It has been estimated that the cost of managing an infected hip joint prosthesis, including removal, exceeds the cost of insertion by a factor of six \(^2\).

Tissue injury is inevitable with surgery. The combination of tissue injury and bacteria introduced at the time of surgery triggers a cascade of events. If the bacteria are cleared, the injured tissue is repaired but the presence of a foreign body affects the cellular and humoral immunity responsible for this cascade and also provides direct protection to bacteria, helping them to grow and colonize. As a consequence, a much smaller bacterial inoculum is required to produce a wound infection if an implanted prosthesis is present than in a prosthesis-free environment \(^1\).

It has been found that the extent to which such colonization is favored depends also on several properties of prostheses that influence tissue reactivity. Some of these are chemical composition (protein-based materials are more reactive than nonprotein-based materials), shape (sharp edges are more reactive than smooth contours), surface characteristics (abraded surfaces are more reactive than smooth surfaces), particle size (particulate material is more reactive than solid material), and so on \(^1\).

In the presence of a prosthesis, the normal inflammatory process is exaggerated and persistent, thereby producing more tissue damage. The phagocytic and bactericidal properties of granulocytes are decreased in the presence of a foreign body, probably because direct contact between the two results in a premature lysosomal discharge, thereby exhausting the leucocyte metabolically and influencing its phagocytic ability \(^2\).

Foreign bodies embedded in biological systems also exert adverse effects on humoral immunity, activating the complement and clotting cascades, among others. The resultant microvascular thrombosis decreases tissue perfusion, causing more tissue damage \(^3\).
Prosthetic devices also promote bacterial growth by providing a sanctuary from the host defense mechanisms, and there seems to be a crucial period of time before graft healing, hence the increased susceptibility of recently implanted prostheses to infection in comparison to established implants. An implanted prosthesis gets covered by a layer of fibrin clot as early as 30 minutes after surgery, and this layer adsorbs bacteria and promotes their growth, safe from the immune mechanisms (1).

Some bacteria have the ability to adhere to prostheses without the benefit of a fibrin layer. The classic example is *Staphylococcus epidermidis*, which produces a glycocalyx layer (“slime”) that helps it to adhere to the prosthetic surface and also offers protection against antibodies and, more importantly, against antibiotics. However, slime is also produced by a large number of other bacteria implicated in prosthesis infection, notably *S. aureus* and *Pseudomonas*. Finding a means for drugs to penetrate slime would presumably be useful in decreasing the rate of infections in prostheses (3).

THE ROLE OF PROPHYLACTIC ANTIBIOTICS

It may be useful to distinguish between acute, often postoperative, hospital-acquired infections commonly associated with temporary prosthetic devices like urinary catheters, peripheral venous access lines, and the like, and the subacute and chronic infections associated with permanent devices that arise months to years after implantation. The former are usually caused by the patient’s indigenous bacterial flora and are controlled relatively easily by removing the device. The majority of deep, late-appearing infections, on the other hand, result from low-grade pathogenic bacteria that may originate from the patient but are also present in the ambient air or on personnel involved in the surgical procedure. It is believed that these are almost always introduced at the time of surgery and contaminate surgical wounds even when the utmost care is taken to maintain a sterile field at the time of surgery (2–4). Peroperative use of polypropylene coveralls (5) and whole-body exhaust-ventilated suits in an ultraclean system (6) can effectively reduce the bacterial contamination rate by 50%; unfortunately, contamination cannot be fully avoided. The administration of prophylactic antibiotics can prevent wound infection if an appropriate antimicrobial is present in the tissues in sufficient concentration during surgical exposure (4). Several experimental and clinical studies undertaken during the last half century have proven the efficacy of prophylactic systemic antibiotics in preventing infection (7–9) but there is a shortage of such data when dealing with surgical implants (10).

It is impossible and probably risky to categorically declare certain antibiotics as the gold standard for specific procedures as bacterial resistance can occur after continuous use of a particular antibiotic for any length of time. Every center will have its own unique spectrum of bacterial resistance and antibiotic protocols. However, it is important to highlight certain key features of prophylactic antibiotic use in order to make the best choice. Therapy should be instituted within 15 to 60 minutes of the initial surgical incision (11). Starting sooner (6 to 24 hours before surgery) may result in the selection of resistant bacteria at the time of incision. Starting antibiotics too soon may also result in intraoperative antibiotic blood and tissue levels that are too low. Starting antibiotic therapy later (more than 2 hours) diminishes the extent of infection but not the incidence. The continued administration of antibiotics for more than 6 to 48 hours after surgery has not been proven advantageous, with the exception of intravascular devices.