The basic problem with the fixation of a prosthesis, cemented or cementless, is biomechanical. The forces transmitted between it and bone will depend on the design and the surface geometry of the implant, the mechanical properties of its surface, and the operative technique. Biological problems may arise from the relative biocompatibility of the implant surface, its biochemistry, the quality of the bone bed, and the degree and duration of early fixation. The aim of cementless implantation is to ensure osseo-integration: to achieve permanent fixation of the prosthesis in its bony surroundings with no interposed connective tissue. The focal point for research is therefore the interface.

At first, forces will be transmitted mainly in compression. This means that cementless implants must be designed to achieve primary stability and to transmit forces as orthogonal stress components wherever possible. Apart from its geometrical design, the second major factor determining success or failure is the surface of the implant. Roughening of the surface can increase the area interfacing with bone. Plane, smooth metallic surfaces transmit compression forces only, but bony ingrowth into a porous surface permits the transmission of some tensile and shear loads. Roughening of the surface, therefore, not only enlarges the available area, but also increases resistance to shear forces. However, an increase in surface area makes the biocompatibility of any coating material of even greater significance. With the exception of titanium, metal surfaces can corrode and this may release ions into the surrounding tissues. The larger the surface, the more ions are released. The most biocompatible materials now in clinical use are titanium and its alloys, and hydroxyapatite.

The use of hydroxyapatite ceramic as a coating for femoral components of hip arthroplasties is the subject of two papers in this issue of the Journal (pages 732 and 741). The material has been shown to have a stimulating effect on bone formation, known as osseo-induction. It enhances osseo-integration and there are indications that chemical bonding may occur between hydroxyapatite and bone. There is, therefore, a possibility that tensile forces might be transmitted as well. As hydroxyapatite is applied to metal implants by a plasma-spray process a full coating cannot be achieved in the depths of the cavities of a porous surface. There are other basic questions which require consideration.

The load-bearing capacity of a coated implant depends on the adhesive strength of the coating to the implant, the cohesive strength within the coating itself, and the strength of the bone interlock. An important prerequisite for any long-lasting coating, therefore, is its firm attachment to the implant. Although the bonding of bone to hydroxyapatite appears to be stronger than bone to titanium, the long-term strength of the bond between hydroxyapatite and the various implant surfaces is still unknown. This bond is certainly weak in the case of polyethylene implants and may also prove to be a problem when the coating is applied to metallic surfaces. Hydroxyapatite is a very brittle material with a high modulus of elasticity, so there is a risk of its separation from more elastic materials when the composite is subjected to bending.

Chemical and physical disintegration of hydroxyapatite in the course of time has been reported and granules may be transported away from the implant surface by macrophages. Should these extremely hard granules enter a joint space, extensive abrasion of polyethylene and metal must be expected. Such a disaster has not yet been seen in clinical practice, but must remain a matter of concern. The development of excessive pararticular ossification is another theoretical possibility, but this has never been reported.

The secondary fixation of an implant can be compared to the healing of a fracture. The decisive step is the transition of osteoprogenitor mesenchyme into bony trabeculae. In normal long-bone healing this transition occurs through intermediate fibrocartilaginous
tissue. But, as has been observed in fracture healing, direct bone formation ('primary bone healing') is possible under ideal conditions: when there is absolute mechanical stability at the interface and excellent biocompatibility. Mechanical stability and biocompatibility are provided by both rough-surfaced titanium implants, and by hydroxyapatite coatings. The authors of the two articles in this issue have shown convincing histological evidence of primary bone healing to hydroxyapatite. No 'callus formation' of fibrous or fibrocartilaginous tissue was detectable around any of the eight implants obtained at post-mortem. Furthermore, the combination of centrifugal and centripetal bone formation not only produces faster osseo-integration, but also allows the bridging of larger gaps between the bone bed and the implant.

The long-term clinical outcome of hydroxyapatite-coated implants will depend on a variety of factors. These include the thickness of the coating, the chemical purity of the hydroxyapatite ceramic, its bonding strength, and its porosity. Thicker coatings are more brittle and more porous; thinner coatings are more likely to be resorbed. However, it can be assumed that even where hydroxyapatite eventually disappears from the surface, osseo-integration would be maintained by the bio-inert properties of the underlying titanium. The great advantages of early osseo-integration have to be balanced against the potential problems of a 'weak interface'. The problem has apparently shifted from the bone–coating junction to the coating–metal interface.

The findings reported in the two papers in this issue are consistent with our own histological, radiological and clinical observations on hydroxyapatite-coated polyethylene cups. Bonding at a molecular level is evident but the use of this material in clinical practice requires further examination and research. Success would mark the transition of orthopaedics from the age of mechanics to that of molecular biology.

ERWIN W. MORSCHER

Bone grafts

Even excluding the case described in Genesis I, bone grafting has a very ancient lineage. It is used to promote osteogenesis between adjacent bones (for nonunion or arthrodesis), to fill cavities in bone (as after curettage of cysts), and to bridge gaps or defects (tumour resection). We are grateful that it works, though we are not quite sure how it does. The graft is dead, yet it induces the growth of new bone which slowly envelops and ultimately replaces the implant, although this is not always the case. Some grafts are rapidly incorporated, others are mysteriously resorbed; some provoke a violent inflammatory reaction and are rejected, others seem totally inert and persist as passive sequestra.

Most of what we know about graft behaviour comes from experiments in lower mammals. A cancellous autograft placed in a well-vascularised bed is rapidly incorporated: blood vessels grow into the graft, new bone appears on the trabecular surfaces and dead bone is slowly resorbed. Cortical autografts are incorporated more laboriously: the impenetrable matrix is attacked by osteoclasts and then, as resorption surfaces are exposed, osteoblastic activity increases and new bone is laid down. Osteogenesis in these conditions derives from the few surviving bone cells on the graft surface and osteoprogenitor cells in the graft marrow and the vascular host bed. There is also evidence that the graft matrix contains a morphogenetic protein which stimulates bone induction in mesenchymal tissues (Urist 1980).

Allografts, which are to a greater or lesser extent immunogenic and always dead, behave somewhat differently. With fresh allografts, the initial osteogenic response is followed by a vigorous inflammatory reaction, and often, rejection of the implant. Immunogenicity can be reduced by 'treating' the graft in various ways. Deep frozen and freeze-dried allografts are incorporated in much the same way as autografts – at least in lower mammals – though the process is slower and less thorough (Heiple, Chase and Herndon 1963). Demineralisation is equally effective, and may even enhance osteogenesis (Urist and Strates 1970). Since these grafts are truly dead and devoid of viable marrow cells, osteo-induction is believed to be due – at least in some measure – to Urist's bone morphogenetic protein.

However, the argument about bone induction is by no means cut and dried. Two reports in this issue of the Journal illustrate the complexity of the matter. Guo et al (page 791), working with rodents, found that allogeneic implants readily induced ectopic bone formation and, moreover, that this reaction was not affected by varying the mineral content of the graft. On the other hand the paper by Schwarz et al (page 787) suggests that frozen allogeneic grafts have little or no effect on bone repair in higher mammals.

This calls into question the entire concept of 'bone induction' and the role of bone morphogenetic protein. Factors such as immunogenic responsiveness, size of defect, structure of the graft and the nature of the host bed are all important. It is also well known that foreign grafts of all kinds are effectively incorporated if autolo-