

It is well known that the capacity for repair of articular cartilage is limited. Hunter in 1743, stated that: “from Hippocrates to the present age, it is universally allowed that ulcerated cartilage is a troublesome thing and that when destroyed, it is not recovered”.¹ There has been complacent acceptance of the common and random degeneration of joints with ageing.^{2,3} Notions that degeneration may be arrested, reversed or repaired have often been disregarded or viewed with cynicism. After all, if it gets bad enough, there is always metal, acrylic and high-density polyethylene!

Partial-thickness defects of articular cartilage do not heal spontaneously and usually progress to more widespread degeneration. Injuries which penetrate the subchondral bone undergo repair with fibrocartilage, a principle upon which techniques such as drilling, abrasion chondroplasty, microfracture, or those involving carbon fibre, have been based. Although fibrocartilage fills and covers the defect, with a period of relief of symptoms, unlike hyaline cartilage, it will resist tension but not compression which is needed to withstand long-term variable cyclic loading and shearing forces and allow smooth articulation. This is also helped by the low coefficient of friction of hyaline cartilage.⁴ The long-term efficacy of these treatments remains unpredictable and controversial.⁵

When selecting methods to restore an articular surface, it is important to distinguish repair from regeneration. Repair involves the replacement of lost tissues by cell proliferation and the synthesis of new extracellular matrix. Unfortunately, repaired articular cartilage generally fails to replicate the structure, composition or function of normal articular cartilage. Regeneration describes the formation of an entirely new joint surface, to duplicate the original articular cartilage. This has proved impossible so far, leaving us with repair, with variable results and an unknown

prognosis, as the only option.⁴ Fetal articular cartilage, however, has an excellent potential to heal spontaneously. Namba and his associates⁶ have developed a model in the fetal lamb to investigate the capacity of articular cartilage to heal after the creation of a superficial defect. An orderly sequence of repair was seen after the creation of partial-thickness defects in the distal femur of the fetus at mid-gestation. This model may be useful for the investigation of the interactions between chondrocytes and extracellular matrices, after mechanical stimulation. Fundamental knowledge of the metabolism of fetal articular cartilage may provide an insight into the latent reparative ability of mature cartilage. Even more exciting is the recent work by Isogai et al⁷ from Harvard, which suggests that the formation of phalanges and small joints is possible with the selective placement of periosteum, chondrocytes and tenocytes, into a biodegradable synthetic scaffold. Clearly, there is potentially a brilliant future for such methods of tissue engineering.

Meanwhile, the two most significant techniques which have gained widespread use and interest during the last decade have been autologous chondrocyte transplantation and mosaicplasty. Chondrocytes hold the key to the restoration of the articular surface. The main problems with chondrocyte transfer, as popularised by Genzyme, have been the expense and logistics, the need for two operations, and the containment of the cultured chondrocytes within a restricted space. Once chondrocytes mature or differentiate, their capacity to reproduce slows down, although their metabolic activity continues. Although embedded in lacunae within the extracellular matrix which they produce, they can respond to growth factors, cytokines and exogenous mechanical stimuli. Changes in these substances have been shown to have a notable affect on the degeneration and synthesis of articular cartilage. Transforming growth factors, however, can have a profound influence on the metabolism of chondrocytes and chondrogenesis, and are likely to revolutionise the treatment of defects of articular cartilage, allowing the replacement and regeneration of osteochondral defects.

At present, osteochondral autograft transplantation, or ‘mosaicplasty’, seems to be the only surgical technique capable of restoring the height and shape of an articulating surface in focal osteochondral defects. The mosaic grafts

V. Bobic, MD, Consultant Orthopaedic Knee Surgeon
Royal Liverpool University Hospitals, Prescot Street, Liverpool L7 8XP,
UK.

J. Noble, ChM, FRCS, FRCS E, Consultant Orthopaedic Surgeon
Hope Hospital, Stott Lane, Salford M6 8HD, UK.

©2000 British Editorial Society of Bone and Joint Surgery
0301-620X/00/20707 \$2.00

J Bone Joint Surg [Br] 2000;82-B:165-6.