Annotation

ARTICULAR CARTILAGE – TO REPAIR OR NOT TO REPAIR

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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”. There has been complacent acceptance of the common and random degeneration of joints with ageing. 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. The long-term efficacy of these treatments remains unpredictable and controversial.

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. Fetal articular cartilage, however, has an excellent potential to heal spontaneously. Namba and his associates 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 al 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

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contain all the necessary ingredients, namely hyaline cartilage, an intact tidemark, and a firm base of bone. The main problem, however, is the availability of grafts from less ‘important areas’ and the uncertain fate of these multiple donor sites. The dead spaces between circular grafts, the lack of integration between donor and recipient hyaline cartilage, and the differences in thickness of the transposed cartilage are all further sources of clinical concern. Noble and Alexander have shown that in the knee there is a precise mathematical relationship between the thickness and structure of bone, articular cartilage and the meniscus.

None of the experimental methods for repair in osteoarthritic joints has been shown to stimulate the formation of a surface durable enough to function as articular cartilage for a long time. This uncertainty must be made clear if such methods are to be recommended for young patients as a means of repairing chondral defects and preventing further arthritis. So far, current methods of repair have been based on unproven assumptions. We currently lack standardised methods of assessment. Precise arthroscopic evaluation will initiate the plan for treatment. Reliable information on the state of the articular cartilage will be available from cartilage sequence MRI, articographic digital imaging and accurate mapping of the location and morphology of the defects.

The emergence of tissue engineering indicates that techniques such as autologous chondrocyte implantation and the use of transforming growth factors have a significant potential for treatment at the cellular level. None of these methods, however, has yet been shown to stimulate the formation of a surface durable enough to function as articular cartilage for a long period of time. Caution must be exercised in recommending such treatment to young patients.

The challenge to restore the damaged articular surface is multidisciplinary and has provoked great interest among research scientists, clinicians and patients. Reasonable progress has been made but further advances in the management of this problem can be expected over the next decade. It is important to realise the problems which we already face because of unrealistic expectations fostered by the media. Strict criteria are needed for the selection and assessment of patients. Such activities should be confined to a few centres where proper appraisal of the value of various techniques can be undertaken.

When we think about repair of articular cartilage, we should remember the view of Henry Mankin: “It should be clear that cartilage does not yield its secrets easily and that inducing cartilage to heal is not simple. The tissue is difficult to work with, injuries to joint surface, whether traumatic or degenerative are unforgiving and the progression to osteoarthritis is sometimes so slow that we delude ourselves into thinking we are doing better than we are. It is important, however, to keep trying.”

References


