Promoting Articular Cartilage Repair

Joseph A. Buckwalter and James A. Martin

INTRODUCTION

For more than 250 years, physicians and scientists have been seeking ways to repair or regenerate synovial-joint articular surfaces following articular-cartilage loss or degeneration (1-3). (Repair refers to restoring a damaged articular surface with new tissue that resembles but does not duplicate the structure, composition, and function of articular cartilage; regeneration refers to forming new tissue indistinguishable from normal articular cartilage [4-6].) They made little progress for the majority of these 250 years, but in the last three decades clinical and basic scientific investigations have shown that implantation of artificial matrices, growth factors, perichondrium, periosteum and transplanted chondrocytes, and mesenchymal stem cells can stimulate formation of cartilaginous tissue in synovial-joint osteochondral defects (6-10). Other work has demonstrated that joint loading and motion can influence articular cartilage and joint healing (11-13), and that mechanical-loading influences the repair process in all of the tissues that form parts of synovial joints (5,14,15). In addition, review of several operative procedures used to treat osteoarthrosis (OA), including osteotomies, penetration of subchondral bone, and joint distraction and motion, has shown that these procedures can stimulate formation of new articular surfaces (9). The apparent potential of these multiple methods for stimulating formation of cartilaginous-articular surfaces has created great interest on the part of patients, physicians, and scientists, however the wide variety of methods and approaches to assessing their results have made it
difficult to evaluate their success in restoring joint function and to define their most appropriate current clinical applications.

ARTICULAR-CARTILAGE LESIONS

Better understanding of articular-cartilage lesions and degeneration has also contributed to the recent interest in cartilage repair and regeneration (1,9,16–18). Advances in synovial-joint imaging and arthroscopic techniques have increased understanding of the frequency and types of chondral defects and made it possible to diagnose and evaluate these lesions with greater accuracy (19). Age-related superficial cartilage fibrillation and focal lesions of the articular surface must be distinguished from cartilage degeneration occurring as part of the clinical syndrome of OA (2,18,20). Superficial articular-cartilage fibrillation occurs in many joints with increasing age and does not appear to cause symptoms or adversely affect joint function. Isolated articular-cartilage and osteochondral defects appear to result from trauma that often leaves the majority of the articular surface intact (17,19). They commonly occur in adolescents and young adults who wish to maintain a high level of activity and in some of these individuals cause joint pain, effusions, and mechanical dysfunction. Although the natural history of isolated chondral and osteochondral defects has not been well-defined (10,21,22). However, clinical experience shows that, in skeletally mature individuals, when these lesions are left untreated they fail to heal, and that defects that involve a significant portion of the articular surface may progress to symptomatic joint degeneration. For this reason treatment of selected isolated chondral and osteochondral defects may help delay or prevent the development of OA. Because treatment by debridement alone produces variable results (9,19), investigators have sought better methods of treating these focal defects.

PENETRATION OF SUBCHONDRAL BONE

Penetration of subchondral bone was the first method developed to stimulate formation of a new articular surface and is still the most commonly used (2,9,23). In regions with full thickness loss or advanced degeneration of articular cartilage, penetration of the exposed subchondral bone disrupts subchondral blood vessels leading to formation of a fibrin clot over the bone surface (2,5,9). If the surface is protected from excessive loading, undifferentiated mesenchymal cells migrate into the clot, proliferate, and differentiate into cells with the morphologic features of chondrocytes (24). In some instances they form a fibrocartilagenous articular surface (Fig. 1), but in others they fail to restore an articular surface (25,26).

Surgeons first debrided degenerated articular cartilage and drilled into the subchondral bone through arthrotomies and found that many patients reported a decrease in symptoms following recovery from the procedure (27–30). One group advocated treating patellar articular-surface degeneration by excising damaged cartilage along with underlying subchondral bone, a procedure they referred to as “spongialization.” They found good or excellent results in a high percentage of their patients (31). Surgeons have developed a variety of other methods of penetrating subchondral bone to stimulate formation of a new cartilaginous surface including arthroscopic abrasion of the articular surface and making multiple small-diameter defects or fractures with an awl or similar instrument (9,19,23,25,26,32).