Cartilage healing: A review with emphasis on the equine model

Marc R. Desjardins, Mark B. Hurtig

Abstract

Articular cartilage is a remarkably resilient tissue capable of withstanding considerable stress and repeated loading. Since this tissue has no blood vessels, nerve elements, or lymphatics, it is not surprising that it has a limited capacity for repair when damaged. In the horse, cartilage damage occurs as an occupational hazard. Furthermore, developmental defects such as osteochondrosis can lead to osteochondritis dissecans. Resultant cartilage flaps, fissures, and poorly organized subchondral bone produce disruption of joint surfaces.

Veterinarians are often called upon to intervene when damaged cartilage has healed incompletely. Basic understanding of the physiology and repair mechanisms of cartilage is paramount to successfully managing such injuries. This literature review gives a brief overview of recently published clinical and experimental studies on the healing of cartilage. The discussion centers on the equine model.

Résumé

Guérison du cartilage : revue de la littérature avec emphase sur l'espèce équine comme modèle

Le cartilage articulaire est un tissu très résistant capable de tolérer le stress et les forces biomécaniques. Ce tissu ne possède ni vaisseaux sanguins, ni éléments nerveux ou lymphatiques; il n'est donc pas surprenant que le cartilage ait une capacité limitée de régénération lorsqu'il est endommagé. Les dommages cartilagineux représentent, chez le cheval, un risque relié à ses activités. De plus, des problèmes de croissance tels l'ostéochondrose peuvent favoriser l'apparition de lésions d'ostéochondrite disséquante caractérisées par un os sous-chondral dont l'architecture est pauvrement organisée et par la formation de fissures et de charnières cartilagineuses, le tout résultant en une disparité des surfaces articulaires. Les vétérinaires sont souvent appelés à intervenir dans des situations où le cartilage endommagé n'est pas complètement guéri. Des connaissances de base sur la physiologie et sur les mécanismes de réparation du cartilage sont essentielles pour avoir du succès dans nos interventions sur de telles lésions. Cette revue de littérature fournit un aperçu des études cliniques et expérimentales récemment publiées sur la guérison du cartilage. La discussion est orientée vers le modèle équin. *(Traduit par Dr Thérèse Lanthier)*

Can Vet J 1990; 31: 565-572

Department of Clinical Studies, Ontario Veterinary College, University of Guelph, Guelph, Ontario N1G 2W1. Present address of Dr. Desjardins: Department of Health Management, Atlantic Veterinary College, University of Prince Edward Island, Charlottetown, Prince Edward Island C1A 4P3.

Repair of articular cartilage has been studied in several species including rabbits (1-5), dogs (6,7), calves (8), chicks (9), and horses (10-15). Among these studies, it is generally accepted that articular cartilage has very limited reparative powers (11,12,15).

Three mechanisms contribute to articular cartilage repair (16). Intrinsic healing is proliferation of injured or exposed chondrocytes and production of new matrix. Extrinsic healing is cartilaginous metaplasia of granulation tissue originating from subchondral bone. Third, matrix flow participates in healing of cartilage by producing a wave-like flow formation from the perimeter that extends into the defect (6,7,17). Cellular events involved in the repair of articular cartilage are influenced by a number of factors including the thickness, size, and anatomical location of the defect (10-15). Post-traumatic and post-operative management of joint injuries varies considerably and is poorly understood. However, the degree of weight-bearing allowed and the length of convalescence can affect the quality of the reparative tissue (2,18).

Morphological and biochemical studies have documented the inability of superficial or partial-thickness cartilaginous defects to undergo repair (2,7,8,11,18,20). This can be attributed to the fact that the injury does not breach the junction between the unmineralized and mineralized cartilage (tidemark), thereby preventing the underlying subchondral bone from supplying blood vessels to the area. Since the inflammatory response is absent, repair is limited to intrinsic healing which has very limited potential for complete repair (7,19,21). Matrix flow has been shown to reduce the size of the defect (3), but complete restoration of a normal hyaline cartilage surface is unlikely.

Full-thickness cartilage defects benefit from the production of granulation issue by subchondral mesenchymal cells. This tissue matures with time and undergoes metaplasia to fibrocartilage, or hyaline-like cartilage (2,7,10,22). Subchondral bone drilling (forage), spongialization (removal of the subchondral bone), and abrasion chondroplasty (shaving) of the articular surface have been performed in attempts to stimulate the (osteo)chondrogenic response and improve the quality of the reparative tissue (1,2,13,15,23-26). In these studies, breaching the subchondral bone plate allowed pluripotential mesenchymal cells from the subchondral bone and bone marrow to improve the quality of the reparative tissue. However, the architecture and proteoglycan concentration of the reparative tissue were inferior to uninjured cartilage.

Neochondrogenesis can be stimulated by several factors, namely loading by the opposing articular surface, decreased oxygen tension, and motion (16,29). Motion is the most easily controlled of these factors. Recent experimental work in rabbits has demonstrated the

beneficial effects of continuous passive motion (CPM) on the healing of full-thickness cartilage defects (18). Continuous passive motion has been shown to be an effective way to stimulate neochondrogenesis and improve joint function in experimental adolescent and adult rabbits (18) and in human patients (30,31). Furthermore, both intermittent active motion and CPM prevent the formation of intra-articular adhesions which are detrimental to cartilage healing. Nevertheless, Salter concluded that neither immobilization nor intermittent active motion provide an adequate stimulus for the healing of full-thickness defects (18). A recent study concluded that immobilizatin combined with 8 h/day of CPM was as effective as CPM 24 h/day in promoting cartilage repair (32).

Joint biology and the problems of cartilage healing remain relevant to our understanding of the basic processes of cartilage metabolism (22). Despite the large body of information on the healing of cartilage, little agreement exists on a recommended technique for promoting the formation of high quality articular cartilage in humans or any other animal species. Confusion and controversy remain regarding the quality and fate of reparative tissue of cartilage. Some of the contention that the quality of reparative tissue is poor may be explained by the variety of experimental models that have been used, the use of many different animal species, and a lack of standardization regarding postoperative management (such as physiotherapy and duration of convalescence). All these factors affect joint biology and therefore the reparative mechanisms of articular cartilage.

Human and equine orthopedists do not deal with the same clinical problems regarding articular cartilage. Similarly, their patients have very different requirements in the return to function of injured joints. Chondromalacia patellae in man has been the focus of attention of clinical orthopedists interested in healing of cartilage (23–25,33–37). The literature regarding cartilage removal as a treatment for chondromalacia patellae and other joint-related problems is confusing and contradictory, but abrasion chondroplasty (shaving) remains widely practised (22,37–44). Mitchell and Shepard (44) recently studied the effects of articular shaving in experimental rabbits and concluded that superficial cartilage defects do not undergo repair nor do they cause degenerative arthritis.

The majority of articular injuries in competitive horses are osteochondral fractures. The equine surgeon faces the challenge of restoring function to a horse so it is capable of racing, jumping, and other athletic pursuits. These fundamental requirements have prompted several researchers to carry out practical experiments designed to explore the equine osteochondrogenic response (10–15,45,46). In designing experimental joint models, investigators have attempted to address clinical orthopedic problems relevant to the athletic horse by concentrating on two major joints, the carpus and the stifle. Major differences in joint biomechanics between the carpus and the stifle warrant separate consideration of the specifics of cartilaginous healing.

Articular trauma to the carpal bones has long been established as a major cause of lameness in the racehorse (47–50). Recent work on cadaver specimens has shed some light on the etiology and pathogenesis of carpal fractures. Such fractures result from stress concentration due to linear compression (51), angular displacement, and sudden acceleration of the individual carpal bones (52). Fresh, localized chip fractures (type I) occurring at the margin (perimeter) of a carpal bone are the most common type of carpal fractures in racehorses (47,48). The area of the defect is usually small and not critical to joint function, so it is not crucial what type of tissue repairs the defect as long as the fragment is resorbed or removed (48). Removal of these fragments can help decrease physical and chemical irritation to the joint, and reduce degenerative changes (53).

When the lesions are more extensive, the limitations of cartilage healing become apparent. McIlwraith *et al* (48) reported that racehorses with large defects have a poor prognosis, and Hurtig *et al* (14) used an experimental model to demonstrate that such lesions were more likely to become nonhealing and expansive. Large defects may be associated with the development of subsequent degenerative joint disease (11,13,14).

Experimental models dealing with the carpus have concentrated mainly on the third carpal bone, with lesions created anywhere from the anterior margin of the bone to a more central location (10,12–15,54).

Riddle (10) was the first to investigate healing of articular cartilage in the horse after creating superficial and full-thickness defects on the radial and intermediate facets of the third carpal bone. He demonstrated that full-thickness defects were covered with granulation tissue at one month and this tissue matured to fibrocartilage by four months. At six months, imperfect hyaline cartilage had been produced within the full-thickness defects, but partial-thickness defects had not healed after eight months.

In another study, investigations were done on the healing process of 8 mm diameter full-thickness cartilage defects on the medial aspect of the distal radius and 4 mm full-thickness defects on the distal aspect of the radiocarpal bone and the proximal surface of the third carpal bone (radial facet) at the joint margins (12). None of the defects were completely repaired at 17 weeks or 67 weeks, and incomplete repair was more commonly associated with the 4 mm diameter lesions. Two types of repair tissues were often found in the defects. Immature hyaline cartilage was found in the deeper layer, closely associated with the subchondral bone, whereas the superficial layer was mostly fibrous or fibrocartilaginous in nature.

Vachon *et al* (13) recently reported that, following forage (subchondral bone drilling) of partial- and full-thickness cartilaginous defects of the radial facet of the third carpal bone, the reparative tissue in drilled defects was of superior quality to the fibrous tissue in nondrilled defects. The fibrocartilage in the drilled full-thickness lesions was anchored better to the underlying subchondral bone, covered a greater surface of the defect, and was thicker than repair tissue in nondrilled defects. Forage of the partial-thickness car-

tilaginous defects did not result in complete resurfacing of the defects and anchorage of the repair tissue to the bed of the defect was merely through the drill holes or in areas of inadvertent subchondral bone perforation (13,15).

A research team recently investigated the effects of location and lesion size on the repair mechanisms of equine articular cartilage (14). Small weight-bearing lesions on the concave portions of the radial and third carpal bones healed better than nonweight-bearing lesions on the rounded anterior rim of these bones. Large lesions (15 mm diameter) healed poorly regardless of their location.

Stifle joint lesions are an important cause of hindlimb lameness. Manifestations of naturally occurring joint disease in the stifle include subchondral bone cysts and osteochondritis dissecans (OCD) (55–58). Management of these conditions remains controversial (57,59,60), but most authors agree that horses do not respond to conservative treatment (prolonged periods of rest) and generally remain lame after such treatment (58,59,61). Pascoe *et al* (58) reported on the necropsy findings of two cases with bilateral, lateral trochlear ridge osteochondrosis. These horses had undergone unilateral surgical curettage of large OCD defects. Fibrocartilaginous repair was evident in both cases at three and at 14 months after surgery. Degenerative changes involving the opposing lateral patellar facet were present in both operated and unoperated femurs. Severity of the degenerative changes appeared to be related primarily to the degree of alteration in the contour of the lateral trochlear ridge (58). Surgical curettage of the lesions appears to produce better clinical and radiographical results (58,59), but information on the nature of the repair tissue within the operated defects is limited (62).

Convery *et al* (11) studied the repair capacity of osteochondral defects of 9, 15 and 21 mm diameter, at the center of the weight-bearing surface of the medial femoral condyle of mature Shetland ponies. Defects of 3 mm diameter were created on the nonweight-bearing surface of the medial femoral condyle. After three months, the 3 mm diameter defects were completely repaired with a mixture of fibrous tissue and fibrocartilage. However, none of the 9, 15 or 21 mm diameter defects was completely repaired at any time up to nine months.

Using a model similar to that used by Convery *et al* (11), Kold *et al* (46) created full-thickness cartilaginous defects (linear or elliptical) and subchondral cavity defects on the weight-bearing surface of the medial femoral condyle. They reported the formation of subchondral bone cysts following the creation of full-thickness linear defects. Subchondral cavities of various size communicating with the joint via a 3 mm diameter cartilage defect did not heal. A mixture of fibrous and fibrocartilaginous tissue filled both the cavities and the cartilaginous defects. These repair tissues did not provide satisfactory healing.

Effects of lesion size and location on repair of articular weight-bearing and nonweight-bearing surfaces of the femoropatellar joint were studied recently (14). Small (5 mm) and large diameter (15 mm) defects were created at the junction of the lateral trochlea and trochlear groove in an area directly contacted by the patella (weight-bearing) or high on the axial side of the lateral trochlear ridge of the femur not contacted by the patella (nonweight-bearing). Matrix flow and fibrocartilage contributed to the repair of all defects. Better healing occurred in small weight-bearing lesions compared to large or nonweight-bearing lesions. In published clinical reports (58,62) authors have stated that the size of defects involving the femoral trochlear ridges was not a limiting factor in terms of sufficient healing for athletic function. Completeness of repair of curetted defects in a hinge-like gliding joint surface such as the femoropatellar joint has been viewed as less critical to athletic function than defects affecting a high loading surface such as the middle carpal joint (13,67,68).

When treating a diseased joint, the surgeon must keep two main objectives in mind: restoring function, and restoring form to the joint. What advantages are to be found in a technique that is successful in dealing with a particular problem but, in the process, creates a secondary problem that is equally or more detrimental to the joint? This particular dilemma becomes apparent when dealing with the repair of cartilaginous defects in clinical and experimental cases.

Secondary or "kissing" lesions on the articular surface opposite full-thickness and partial-thickness defects are consistent findings in clinical and experimental carpal joint injuries (10,12,13,63,64). These lesions are characterized by hypertrophy and thickening of articular cartilage with superficial loss of metachromasia (12), reduction in hexosamine content (63), hypercellularity or hypocellularity in the tangential layer with superficial chondrocytic clusters and focal thinning of the cartilage (13), or ulcerative depressed areas (15). Their cause remains unclear, but kissing lesions may occur following a decrease in weight bearing, thereby reducing the diffusion of nutrients to the cartilage (65,66). Kissing lesions secondary to partial-thickness cartilaginous defects are less extensive than kissing lesions opposite full-thickness defects (15,63). According to Riddle (10), kissing lesions appear two to four months after the full-thickness defects have been created. In contrast, other studies have shown that lesions were not present at 17 weeks following surgery but were evident between 28 and 54 weeks. It was observed, however, that some reversibility of the lesions occurred by 67 weeks postoperatively (12). The long-term implications of these lesions on joint function in the athletic horse are unknown. In clinical situations, most surgeons leave a kissing lesion undisturbed (63,67).

Postoperative management of the injured joint can be a determining point for repair of defects. Continuous passive motion in rabbits for a period of seven days was shown to improve the quality of the repair tissue by disrupting adhesions and optimizing cartilage nutrition (18). Nonweight-bearing CPM is not possible in the horse, but passive flexion and early postoperative ambulation have been shown to be beneficial to cartilage healing in full-thickness defects by disrupting synovial adhesions in nonweight-bearing locations,

particularly at the cranial rim of the middle carpal joint (14). The same authors hypothesized that synovial membrane adhesions interfered with cartilage nutrition. Other investigators, however, have associated synovial membrane adhesions with a fibrous type of repair (10,12). In recent studies on full- and partial-thickness defects, synovial adhesions were not found to be a source of repair tissue nor were they found to interfere with the repair process (13,15,54).

De Palma *et al* (7) found that the development of hyaline cartilage depended on functional stresses from early motion and weight bearing. Compression in the presence of a low oxygen concentration was found to facilitate cartilage formation (69). The healing process of cartilage in weight-bearing versus nonweight-bearing surfaces has been incompletely studied. Confusion remains as experimental investigations have produced conflicting results (4,7,70). The literature dealing with equine cartilage healing is replete with experimental models that have used empirical and subjective means to determine weight-bearing areas without ever quantifying the nature, the magnitude, or the location of the forces acting on articular surfaces. Among the studies examining cartilage healing in the carpus, lesion sites have varied from the anterior margin of the radial and third (intermediate and radial facets) carpal bones (10,12,14) to centrally located sites on radial or third carpal bones (13–15). Hurtig *et al* (14) hypothesized that lesions at the anterior margin of the carpal bones were nonweight-bearing whereas lesions centrally located on concave surfaces were weight-bearing. Only recently has a study located and quantified peak pressures along the dorsal rim of the carpus using pressure-sensitive films. The same study found a strong mathematical between stress concentration due to linear compression and the occurrence of carpal fractures (51). Objective correlations need to be made between the lesion site, the type of repair tissue, and the magnitude of forces acting on a particular articular surface. Perhaps such data would shed some light on noted discrepancies between clinical studies (58,62) and research (14) when considering the effects of lesion size on repair of cartilaginous defects in femoral trochlear ridges. Controversy has been fuelled by the lack of similarity between experimental models that compare the effects of a directly loaded surface (radiocarpal and middlecarpal joints) to those of a gliding surface (femoropatellar, femorotibial, and tarsocrural joints). Intra-articular contact area and stress distribution can be helpful measurements in understanding the biomechanical character of a joint. Although several human joints have been studied in that fashion (6,72), such data have only been collected in foals (73) and are lacking in adult horses.

Doubts regarding the quality and the durability of cartilage repair tissue have been expressed (2,13,14, 27,48,74). Vachon *et al* (13) hypothesized that the lack of repair in carpal bone injuries was related to the high density of the subchondral bone and the high loading forces the repair tissue had to withstand. Hurtig *et al* (14) further theorized that opposing articular surfaces were more likely to protrude into large defects and impinge on fragile repair tissues, resulting in their disruption. The physical properties of normal cartilage and repair tissue in full-thickness cartilaginous defects were studied using compression testing in rabbits (5,75). The repaired articular surfaces were shown to be less suited for repetitive loading than normal articular cartilage because the former deformed more easily (75). The same authors concluded that a repaired articular surface was inadequate as a replacement for normal cartilage in weight-bearing areas. Nevertheless, the presence of healed and nonhealed 6 mm diameter osteochondral defects in the weight-bearing portion of the femoral condyles of dogs did not affect the condyles' ability to distribute load (76). These authors concluded that knees of adult dogs could accommodate moderate incongruities without obvious morphological consequences.

Convalescence time has been the subject of considerable debate. Maturation of the repair tissue to hyaline-like cartilage was achieved between 17 and 28 weeks postoperatively for a 4 mm diameter defect (12) and 28 weeks postoperatively for a 15 mm diameter defect (10). In both studies, little correlation was found between prolonged healing time and the quality of the repair tissue.

In conclusion, disagreement and controversy regarding recommended surgical treatment, postoperative therapy, and convalescent time of horses with cartilaginous defects will likely remain until experimental protocols become standardized and meaningful experimental data can be extrapolated to clinical cases. One must also consider the fact that joints in which cartilage healing was studied experimentally were not subjected to a racehorse's level of exercise and therefore have not endured similar tissue trauma. In spite of recent experiments on the healing of equine cartilage, clinicians dealing with an athletic horse often choose a conservative approach to debridement of articular defects. The general approach includes removing the bone fragment(s), and debridement of the full-thickness cartilaginous defect to remove undermined cartilage and expose bleeding subchondral bone. Debridement of partial-thickness cartilaginous defects is not performed. The overall conservative approach to treating osteochondral lesions reflects the lack of confidence of clinicians in the quality and durability of the repair tissue and the fact that fibrous repair tissue is commonly dislodged during athletic exercise (48). CVJ

References

1. Meachim G, Roberts C. Repair of the joint surface from subarticular tissue in the rabbit knee. J Anat 1971; 109: 317–327.
2. Mitchell NS, Shepard N. The resurfacing of adult rabbit articular cartilage by multiple perforations through the subchondral bone. J Bone Joint Surg 1976; 58-A: 230–233.
3. Ghadially FN. Superficial (partial-thickness) defects and other injuries in articular cartilage. In: The Fine Structure of Synovial Joints. London: Butterworths, 1983: 261–279.
4. Ghadially FN. Deep (full-thickness) defects in articular cartilage. In: The Fine Structure of Synovial Joints. London: Butterworths, 1983: 280–306.
5. Suzuki Y. Studies on the repair tissue of injured articular cartilage. Biochemical and biomechanical properties. J Jpn Orthop Assoc 1983; 57: 741–752.

6. Calandruccio RA, Gilmer WS. Proliferation, regeneration and repair of articular cartilage of immature animals. J Bone Joint Surg 1962; 44-A: 431-455.
7. DePalma AF, McKeever CD, Subin DK. Process of repair of articular cartilage demonstrated by histology and autoradiography with tritiated thymidine. Clin Orthop Rel Res 1966; 48: 229-242.
8. Campbell CJ. The healing of cartilage defects. Clin Orthop Rel Res 1969; 64: 45-60.
9. Itay S, Abramovici A, Nevo Z. Use of cultured embryonal chick epiphyseal chondrocytes as grafts for defects in chick articular cartilage. Clin Orthop Rel Res 1987; 220: 284-303.
10. Riddle WE. Healing of articular cartilage in the horse. J Am Vet Med Assoc 1970; 157: 1471-1479.
11. Convery FR, Akeson WH, Keown GH. The repair of large osteochondral defects. An experimental study in horses. Clin Orthop Rel Res 1972; 82: 253-262.
12. Grant BD. Repair mechanisms of osteochondral defects in Equidae: a comparative study of untreated and X-irradiated defects. Proc Am Assoc Equine Pract 1975; 21: 95-114.
13. Vachon A, Bramlage LR, Gabel AA, Weisbrode S. Evaluation of the repair process of cartilage defects of the equine third carpal bone with and without subchondral bone perforation. Am J Vet Res 1986; 47: 2637-2645.
14. Hurtig MB, Fretz PB, Doige CE, Schnurr DL. Effects of lesion size and location on equine articular cartilage repair. Can J Vet Res 1988; 52: 137-146.
15. Shamis DS, Bramlage LR, Gabel AA, Weisbrode S. Effect of subchondral bone drilling on repair of partial-thickness cartilage defects on third carpal bones in horses. Am J Vet Res 1989; 50: 290-295.
16. Stockwell RA, Meachim G. The chondrocytes. In: Freeman MAR, ed. Adult Articular Cartilage. Tunbridge Wells, England: Pittman Medical Publishing Co, Ltd., 1979: 69-114.
17. Ghadially FN, Ailsby RL, Oryschak AF. Scanning electron microscopy of superficial defects in articular cartilage. Ann Rheumatol 1974; 33: 327-332.
18. Salter RB, Simmonds DF, Malcolm BW, Rumble EJ, MacMichael D, Clements ND. The biological effect of continuous passive motion on the healing of full-thickness defects in articular cartilage. J. Bone Joint Surg 1980; 62-A: 1232-1251.
19. Mankin HJ. The reaction of articular cartilage to injury and osteoarthritis. (second of two parts). New Engl J Med 1974; 291: 1335-1340.
20. Mitchell NS, Shepard N. The healing of articular cartilage in intra-articular fractures in rabbits. J Bone Joint Surg 1980; 62-A: 628-634.
21. Fuller JA, Ghadially FN. Ultrastructural observations on surgically produced partial-thickess defects in articular cartilage. Clin Orthop Rel Res 1972; 86: 193-205.
22. Mankin HJ. The response of articular cartilage to mechanical injury. J Bone Joint Surg 1982; 64-A: 460-466.
23. Bentley G. Chondromalacia patellae. J Bone Joint Surg 1970; 52-A: 221-232.
24. Childers JC, Ellwood SC. Partial chondrectomy and subchondral bone drilling for chondromalacia. Clin Orthop Rel Res 1979; 144: 114-120.
25. Ficat RP, Ficat C, Gedeon P, Toussaint JB. Spongialization: a new treatment for diseased patellae. Clin Orthop Rel Res 1979; 144: 74-83.
26. Pridie RH. A method of resurfacing osteoarthritic knee joints. Proc Br Orthop Assoc. J Bone Joint Surg 1959; 41-B: 618-619.
27. Whipple RR, Gibb MC, Lai WM, Mow VC, Mak AF, Wirth CR. Biphasic properties of repaired cartilage at the articular surface. Trans Orthop Res Soc 1986; 10: 340.
28. Furukawa T, Eyre DR, Koide S, Glimcher MJ. Biochemical studies on repair cartilage resurfacing experimental defects in the rabbit knee. J Bone Joint Surg 1980; 62-A: 79-89.
29. Ham AW. Joints. In: Ham AW, ed. Histology. Philadelphia: JB Lippincott Company, 1979: 463-482.
30. Salter RB, Hamilton HW, Wedge JH, *et al.* The clinical application of basic research on continuous passive motion for disorders and injuries of synovial joints. J Orthop Res 1984; 1: 325-342.
31. Salter RB. The biologic concept of continuous passive motion of synovial joints. The first 18 years of basic research and its clinical application. Clin Orthop Rel Res 1989; 242: 12-25.
32. Shimizu T, Videman T, Shimazaki K, Mooney V. Experimental study on the repair of full thickness articular cartilage defects: effects of varying periods of continuous passive motion, cage activity, and immobilization. J Orthop Res 1987; 5: 187-197.
33. Chakraverty RN. Excision of patella for chondromalacia. J Bone Joint Surg 1972; 54-B: 760-761.
34. Goodfellow J, Hungerford DS, Woods C. Patello-femoral joint mechanics and pathology. 2. Chondromalacia patellae. J Bone Joint Surg 1976; 58-B: 291-299.
35. Insall J. The Pridie debridement operation for osteoarthritis of the knee. Clin Orthop Rel Res 1974; 101: 61-67.
36. Wiles P, Andrews PS, Devas MB. Chondromalacia of the patella. J Bone Joint Surg 1956; 38-B: 95-113.
37. Milgram JW. Injury to articular cartilage joint surfaces. I. Chondral injury produced by patellar shaving: a histopathologic study of human tissue specimens. Clin Orthop Rel Res 1985; 192: 168-173.
38. Hotchkiss RN, Tew WP, Hungerford DS. Cartilaginous debris in the injured human knee. Correlation with arthroscopic findings. Clin Orthop Rel Res 1982; 168: 144-156.
39. Friedman MJ, Berasi CC, Fox JM, Del Pizzo W, Snyder SJ, Ferkel RD. Preliminary results with abrasion arthroplasty in the osteoarthritic knee. Clin Orthop Rel Res 1984; 182: 200-205.
40. Johnson LL. Pathology of the knee. In: Johnson LL, ed. Arthroscopic Surgery: Principles and Practice. 3rd ed. St-Louis: CW Mosby Company, 1986: 498-667.
41. Hawkins RB. Arthroscopic treatment of sports-related anterior osteophytes in the ankle. Foot Ankle 1988; 9: 87-90.
42. Kozinn SC, Scott RD. Surgical treatment of unicompartmental degenerative arthritis of the knee. Rheum Dis Clin North Am 1988; 14: 545-564.
43. Merchant AC. Patellofemoral disorders. In: Chapman MW, Madison M, eds. Operative Orthopaedics. Philadelphia: JB Lippincott Co., 1988: 1699-1707.
44. Mitchell NS, Shepard N. Effect of patellar shaving in the rabbit. J Orthop Res 1987; 5: 388-392.
45. Fisher AT, Stover SM, Pool RR. Healing of full thickness articular cartilage defects in the horse: a comparison of weightbearing to nonweightbearing areas. (Abstract) Vet Surg 1986; 15: 120.
46. Kold SE, Hickman J, Melson F. An experimental study of the healing process of equine chondral and osteochondral defects. Equine Vet J 1986; 18: 18-24.
47. Bramlage LR. Surgical diseases of the carpus. Vet Clin North Am [Large Anim Pract] 1983; 5: 261-274.
48. McIlwraith CW, Yovich JV, Martin GS. Arthroscopic surgery for the treatment of osteochondral chip fractures in the equine carpus. J Am Vet Med Assoc 1987; 191: 531-537.
49. Palmer S. Prevalence of carpal fractures in Thoroughbred and Standardbred racehorses. J Am Vet Med Assoc 1986; 188: 1171-1173.
50. Raker CW, Baker RH, Wheat JD. Pathophysiology of equine degenerative joint disease and lameness. Proc Am Assoc Equine Pract 1966; 12: 229-241.
51. Turner TA, Colahan PT. Evaluation of carpal contact and carpal compression to determine areas of stress concentration predisposing to carpal fractures in the horse. (Abstract) Vet Surg 1987; 16: 104.
52. Palmer S, Barlow D, Chun JI. Kinematics of the equine carpus. In: Gillespie JR, Robinson NE, eds. Equine Exercise Physiology 2. Davis. ICEEP Publications, 1987: 599-606.
53. Martin GS, McIlwraith CW. Arthroscopic anatomy of the intercarpal and radiocarpal joints of the horse. Equine Vet J 1985; 17: 373-376.
54. French DA, Barber SM, Leach DH, Doige CE. The effect of exercise on the healing of articular cartilage defects in the equine carpus. Vet Surg 1989; 18: 312-321.
55. Wyburn RS. A degenerative joint disease in the horse. NZ Vet J 1977; 25: 321-322,335.
56. Trotter GW, McIlwraith CW. Osteochondritis dissecans and subchondral cystic lesions and their relationship to osteochondrosis in the horse. J Equine Vet Sci 1981; 5: 157-162.
57. Jeffcott LB, Kold SE, Melsen F. Aspects of the pathology of stifle bone cysts in the horse. Equine Vet J 1983; 15: 309-311.
58. Pascoe JR, Pool RR, Wheat JD, O'Brien TR. Osteochondral defects of the lateral trochlear ridge of the distal femur of the horse: clinical, radiographic, and pathological examination and results of surgical treatment. Vet Surg 1984; 13: 99-110.

59. McIlwraith CW, Martin GS. Arthroscopic surgery for the treatment of osteochondritis dissecans in the equine femoropatellar joint. Vet Surg 1985; 14: 105–116.
60. White NA, McIlwraith CW, Allan D. Curettage of subchondral bone cysts in medial femoral condyles of the horse. Equine Vet J 1989; (Suppl 6): 120–124.
61. Stromberg B, Rejno S. Osteochondrosis in the horse. I. A clinical and radiologic investigation of osteochondritis dissecans in the knee and hock joint. Acta Radiol 1978; 358 (Suppl): 139–152.
62. McIlwraith CW. Diagnostic and Surgical Arthroscopy in the Horse. Philadelphia: Lea & Febiger, 1990: 113–159.
63. Richardson DW, Clark CC. Biochemical changes in articular cartilage opposing full-and partial-thickness cartilage lesions in horses. Am J Vet Res 1990; 51: 118–122.
64. Trotter GW, Yovich JV, McIlwraith CW, Norrdin RW. Effects of intramuscular polysulfated glycosaminoglycan on chemical and physical defects in equine articular cartilage. Can J Vet Res 1989; 53: 224–230.
65. Radin EL, Igor LP. A consolidated concept of joint lubrication. J Bone Joint Surg 1972; 54-A: 607–612.
66. Kincaid SA, Van Sickle DC. Regional histochemical and thickness variations of adult canine articular cartilage. Am J Vet Res 1981; 42: 428–432.
67. McIlwraith CW. Diagnostic and Surgical Arthroscopy in the Horse. Philadelphia: Lea & Febiger, 1990: 33–83.
68. McIlwraith CW, Vachon A. Review of pathogenesis and treatment of degenerative joint disease. Equine Vet J 1989; (Suppl 6): 3–11.
69. Basset CAL, Herrmann I. Influence of oxygen concentration and mechanical factors on differentiation of connective tissues in vitro. Nature 1961; 190: 460–461.
70. Stover SS, Pool RR, Fisher AT. Healing in osteochondral defects: a comparison of articulating and nonarticulating locations. Trans Orthop Res Soc 1987; 12: 275.
71. Kimizuka M, Kurosawa H, Fukubayashi T. Load-bearing pattern of the ankle joint. Contact area and pressure distribution. Arch Orthop Traum Surg 1980; 96: 45–49.
72. Brown TD, Shaw DT. In vitro contact stress distribution on the femoral condyles. J Orthop Res 1984; 2: 190–199.
73. Firth EC, Hartman W. An in vivo study on joint fitting and cartilage thickness in the radiocarpal joint of foals. Res Vet Sci 1983; 34: 320–326.
74. Hjertquist SO, Lemperg R. Histological, autoradiographic and microchemical studies of spontaneously healing osteochondral articular defects in adult rabbits. Calcif Tissue Res 1971; 8: 54–72.
75. Coletti JM, Akeson WH, Woo SLY. A comparison of the physical behavior of normal articular cartilage and the arthroplasty surface. J Bone Joint Surg 1972; 54-A: 147–160.
76. Nelson BH, Anderson DD, Brand RA, Brown TD. Effect of osteochondral defects on articular cartilage. Contact pressures studied in dog knees. Acta Orthop Scand 1988; 59: 574–579.

Conseil de l'ACV 1991–1993
Appel de mise en candidature — Conseillers pour le Québec et l'Ontario

Nous vous invitons à soumettre votre candidature au poste de conseiller de l'ACV pour la province du Québec et de l'Ontario, pour un mandat de trois (3) ans débutant le 1er janvier 1991 et se terminant le 31 décembre 1993.

Le règlement de l'ACV fut récemment modifié pour permettre l'élection d'un conseiller de l'ACV dans ces provinces (Québec et Ontario) où la cotisation à l'ACV n'est pas perçue par l'association provinciale.

Un(e) membre de l'ACV peut se porter candidat(e) au conseil de l'association si il ou elle: i) **demeure** dans la province où il ou elle se présente; ii) est **proposé(e)** par deux (2) membres en règle de l'ACV qui demeurent dans cette province; iii) **accepte** cette nomination; et iv) est **en règle** avec l'ACV.

Les mises en candidature doivent se faire sur le formulaire ci-dessous, et doivent parvenir au bureau du secrétariat de l'ACV, 339, rue Booth, Ottawa (Ontario) K1R 7K1, **timbrées au plus tard le 8 septembre 1990**. Chaque mise en candidature doit être signée par **deux** membres en règle de l'ACV et par le (la) **candidat(e)**. S'il y a plus d'un(e) candidat(e), des bulletins de vote seront postés à tous les membres de l'ACV de la province en question le 24 septembre 1990.

Nous demandons aux candidat(e)s de nous faire parvenir une photographie récente ainsi qu'une **courte** biographie.

FORMULAIRE DE MISE EN CANDIDATURE

Conseiller du/de la candidat(e) pour: _____ Québec _____ Ontario

Information sur le (la) candidat(e)

NOM: _____ **ADRESSE:** _____

_____

Mis(e) en candidature par les membres en règle de l'ACV suivants :

1) _____ _____
(Nom au complet, lettres moulées) (Signature)

2) _____ _____
(Nom au complet, lettres moulées) (Signature)

Je suis un membre en règle et, si élu(e), j'accepte de siéger au conseil de l'ACV pour un mandat de un an. _____
(Signature du/de la candidat(e))

CVMA 1991–1993 Council
Call for Nominations — Ontario and Quebec Councillors

Nominations are hereby invited for the position of CVMA Councillor for the provinces of Ontario and Quebec, for a term of three (3) years starting on January 1, 1991, and ending on December 31, 1993.

The CVMA By-Laws have been amended to provide for the election of a CVMA Councillor in provinces (Ontario and Quebec) where CVMA dues are not collected by the provincial association.

A member of the CVMA is eligible for election to the Council of the Association if he or she: i) is a **resident** in the province for which he or she is nominated; ii) is **nominated** by two (2) CVMA members in good standing who reside in that province; iii) **consents** to the nomination; and, iv) is in **good standing** in the CVMA.

Nominations must be submitted on the form below, and must be sent to the CVMA Secretariat, 339 Booth Street, Ottawa, Ontario K1R 7K1, **postmarked on or before September 8, 1990**. Each nomination must be signed by **two** CVMA members in good standing and countersigned by the **nominee**. If there is more than one candidate, ballots will be mailed to all CVMA members on September 24, 1990.

We would ask nominees to send us a recent photograph as well as a **short** biography.

NOMINATION FORM

Councillor nominee for: _____ Ontario _____ Quebec

Nominee Information

NAME: _____ **ADDRESS:** _____

_____

Nominated by the following active CVMA members:

1) _____ _____
(Print full name) (Signature)

2) _____ _____
(Print full name) (Signature)

I am an active member and, if elected, agree to serve as a councillor for a one-year term. _____
(Signature of nominee)