Carpal Disease in Racing Horses

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Apart from the assistance stated in the acknowledgements, this thesis represents the original work of the author. Segments of the work have been published or are in press, under the author's name, as shown in the Publication list.

Signed: .....

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SUMMARY

Thirteen Standardbred horses were trained on a treadmill for 31 weeks as part of a larger study into the effects of overtraining. Synovial fluid was collected from the midcarpal joint at the start, and at seven, 15, 21, 26 and 30 weeks of training. Low grade signs of midcarpal joint disease developed in all horses during the last 16 weeks of the program. Synovial fluid leukocyte counts remained unchanged throughout the study, whereas total protein concentration and lactate dehydrogenase activity increased significantly with training. Sulfated glycosaminoglycan (GAGs) levels increased initially, but then decreased. Correlations between the clinical signs of joint disease and sulfated GAG levels were weak.

Synovial fluid sulfated GAGs were compared with other diagnostic variables for predicting the degree of articular cartilage damage in horses with midcarpal joint disease. Interpretation of radiographs was found to be the most accurate for the prediction of articular damage. Synovial fluid analysis was found to be of little value. There was no correlation between sulfated GAG concentration and articular cartilage damage, and no significant difference between sulfated GAG concentrations from horses with clinical evidence of joint disease and horses with no signs of joint disease trained on a treadmill.

Anatomical dissections of the midcarpal joint were performed on ten cadavers. The medial palmar intercarpal ligament (MPICL) was found to consist of four fibre bundles. The predominant orientation of these was proximodorsal to distopalmar. The lateral palmar intercarpal (LPICL) and dorsomedial intercarpal (DMICL) ligaments had a similar orientation but were simpler in structure. The alignment of these ligaments suggested that they resisted

transverse forces across the midcarpal joint. Using a dorsal transverse displacement of 1.5 mm of the proximal row of carpal bones relative to the distal row of carpal bones, it was demonstrated that the palmar intercarpal ligaments provided 22.7% of the restraining force while only contributing 9% of the ligamentous cross sectional area.

A study of 32 racing horses presented with midcarpal joint disease confirmed the high frequency of MPICL tearing (51%). Enlargement of the DMICL was also common (33%). There was no correlation between the severity of signs of midcarpal joint disease and the severity of MPICL tearing. An inverse relationship was demonstrated between subchondral bone damage within the midcarpal joint, and MPICL tearing (R=-0.55). There was no association between DMICL enlargement and osteochondral damage. A postmortem study of 142 joints of horses with no history of midcarpal joint disease demonstrated that the frequency of MPICL tearing in racing horses was 91%. Severity of tearing of the MPICL increased significantly with age. Histopathological evidence of degeneration (loss of organisation of collagen fibres) was consistently observed in MPICLs of adult horses. These changes were not observed in unborn term foals, but were present from one month of age. Enlarged DMICLs had regular collagen arrangement, but discrete areas of fibrovascular infiltration were consistently observed.

The race records of 42 horses undergoing midcarpal joint carpal arthroscopy were examined. Using multiple regression the extent of subchondral bone damage was the best predictor of postoperative performance. The addition of the grade of MPICL tearing significantly improved the prediction of postoperative performance, whereas the inclusion of the extent of articular cartilage damage had no effect.

PUBLICATION LIST

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CONTENTS

CHAPTER 1

LITERATURE REVIEW

Introduction

Much has been written about the pathogenesis of joint disease both in humans and horses. Most attention has focused on the degeneration of articular cartilage due to its importance in joint function and its limited ability to repair. However cartilage degeneration is the end result of a number of disease processes. The study of joint biomechanics has improved the understanding of some of the initiating events in joint disease in humans and there are a growing number of biomechanical studies in equine joints including the carpus. Other joint components which are involved in joint disease include the subchondral bone, intra-articular and periarticular ligaments, joint capsule and synovial membrane.

Ultimately though osteoarthritis results in the irreversible loss of articular cartilage. Conventional diagnostic techniques such as radiography are unable to detect cartilage damage until the degeneration is severe and there is associated bone pathology (Dyson 1987). Therefore the ability of newer diagnostic techniques to detect early changes in osteoarthritis needs to be evaluated. Markers of cartilage metabolism in synovial fluid have shown some promise as early detectors of cartilage breakdown (Messner *et al* 1993), but they have not been found useful in the clinical situation (Silverman *et al* 1990).

This review covers the anatomy and biomechanics of the normal carpus, the pathogenesis and diagnosis of equine carpal disease, and its effect on performance in racing horses. There is particular emphasis on the role of biomechanics in the development of carpal disease and the use of proteoglycan concentrations in synovial fluid as a diagnostic tool.

Anatomy of the Carpus

The equine carpus is a complex anatomical structure consisting of three major joints and multiple smaller articulations, supported by an array of ligaments and the fibrous joint capsule.

Articulations of the carpus

The antebrachiocarpal joint is bordered by the distal radial joint surface and the proximal radial carpal bone (CR), intermediate carpal bone (CI) and ulnar carpal bone (CU). The joint also includes the articulation with the accessory carpal bone (CA). The distal radial joint surface is divided into radial, intermediate and ulnar facets by a medial and a lateral parasaggital ridge (Hurtig and Fretz 1986). The synovial sac extends between the carpal bones of the proximal row as far as the intercarpal ligaments and encompasses the articulation with CA (Kainer 1987). A palmarolateral pouch of the antebrachiocarpal joint capsule protrudes between the long tendon of the ulnaris lateralis and the lateral styloid process of the radius (Kainer 1987).

The midcarpal joint is bordered by the distal surfaces of CR, CI and CU and the proximal surfaces of the second carpal bone (C2), third carpal bone (C3) and fourth carpal bone (C4). The radial carpal bone articulates with C2 and the large concave radial facet of C3. CI articulates with the intermediate facet of C3 and the proximal surface of C4 (Bramlage *et al* 1988, Getty 1975). A divisional ridge of CI interdigitates with the articulation between C3

and C4. CU articulates with C4 (Hurtig and Fretz 1986). The midcarpal synovial sac communicates with the carpometacarpal sac between C3 and C4 (Kainer 1987, Sisson 1975). The carpometacarpal sac is very limited in extent and closely applied to the bones (Sisson 1975), however distal outpouchings on the palmar aspect interdigitate with the origin of the suspensory ligament (Ford *et al* 1988).

Ligaments of the carpus

The bones of the carpus are held together by collateral and intercarpal ligaments along with the palmarcarpal ligament and the joint capsule (Kainer 1987, Bramlage *et al* 1988). The fibrous joint capsule is common to all three joints. Its dorsal part, the extensor retinaculum, is loose except during flexion (Sisson 1975). It attaches to the dorsal intercarpal and dorsal carpometacarpal ligaments, the distal radius, the carpal bones and the third metacarpal bone (Kainer 1987).

Kainer (1987) describes the palmar carpal ligament as forming the dorsal wall of the carpal canal while its deep face serves as the common fibrous joint capsule of the carpal joints. It attaches to the three palmar radiocarpal ligaments, three palmar intercarpal ligaments, four palmar carpometacarpal ligaments and the palmar surface of the carpal bones. Distally the palmar carpal ligament gives origin to the accessory ligament of the deep digital flexor tendon (Sisson 1975).

Of particular interest are two intra-articular intercarpal ligaments that have been described by

McIlwraith (1992) and Kannegieter and Colgan (1993). These probably correspond to the two short ligaments described by both Sisson (1975) and Kadletz (1932); the lateral ligament passing from CR to C2 and C3 and the medial ligament passing from CU to C3 and C4. McIlwraith (1992) describes the medial ligament as consisting of two parts. Both originate on the palmar aspect of CR. The more dorsal part is thinner and attaches to C3 while a thicker more tubular part attaches to C2. The lateral ligament runs from the distal palmar aspect of CU to the proximal palmar aspect of C3 and C4 (McIlwraith 1992, Sisson 1975). A further intercarpal ligament on the dorsomedial aspect of the joint, closely associated with the joint capsule has been described (Martin and McIlwraith 1985, Selway 1991).

Sisson (1975) describes three ligaments connecting CA to adjacent bones while Kainer (1987) describes four named according to their attachments; accessorioulnar, accessoriocarpoulnar, accessorioquartal and accessoriometacarpal. These ligaments transmit the action of the muscles inserting on CA (Sisson 1975). Both rows of carpal bones are connected by transverse dorsal and interosseous ligaments (Sisson 1975).

The lateral collateral ligament runs from the styloid process of the radius to the fourth and third metacarpal bones. A deep part attaches to CU (Kainer 1987). The medial collateral ligament arises on the medial styloid process of the radius and widens distally to attach to MC3 and MC2 (Sisson 1975) with deep attachments to CR, C2 and C3 (Kainer 1987). The first carpal bone when present is usually embedded in the palmar part of the distal end of the medial collateral ligament (Sisson 1975).

Biomechanics of the Carpus

The multiple articulations of the equine carpus result in an extremely complex functional unit. Previously much of the understanding of the biomechanics of the carpus has come from clinical observations of horses with carpal disease (Rooney 1968, Bramlage *et al* 1988). Biomechanical studies have confirmed some of these observations and are gradually improving our understanding of carpal joint function (Fredricson and Drevemo 1972, Palmer *et al* 1987, Colahan *et al* 1987, Leach and Dyson 1988, Hjertén and Drevemo 1994, Palmer *et al* 1994, Young *et al* 1994, Johnston *et al* 1995, Johnston and Roepstorff 1996). Complexity arises from the dual role of the carpus to dissipate forces passing proximally during the stance phase while allowing for flexion of the limb during the swing phase.

Joint movement

It has been stated that gross movement of the composite joint is restricted to flexion and extension in the sagittal plane (Palmer *et al* 1987), while multiple intercarpal articulations act to attenuate loads during weight bearing (Bramlage *et al* 1988, Young *et al* 1994). However, recent kinematic studies have demonstrated shearing movements across the joint (Hjertén and Drevemo 1994), and Kadletz (1932) suggested that rotation of the joint occurs during weight bearing based on the spiral arrangement of the carpal ligaments. The antebrachiocarpal joint is a rotating joint while the midcarpal joint functions as a hinged joint. The carpometacarpal joint is capable of minimal movement (Bramlage *et al* 1988) although Palmer *et al* (1987) describe it as a gliding joint. Also, Leach and Dyson (1988), from studies of instant centres of

rotation, concluded that there were two or more independent kinematic events occurring within the joint complex. During extension, the midcarpal joint closes before the antebrachiocarpal joint (Palmer *et al* 1987, Leach and Dyson 1988). Also CI and CU function as a unit, whereas CR moves independently of these two bones (Palmer *et al* 1987).

Mechanisms for force dissipation within the carpus

Axial weight-bearing is primarily directed through CR, CI and C3. These bones, together with the distal radius, are also the most often injured (Colahan *et al* 1987). Two mechanisms for dissipation of axial weight-bearing load have been described. Fredricson and Drevemo (1972), in a kinematic study of four horses, consistently observed overextension and concluded that this was physiological and important for shock absorption. The consistent observation of hyperextension in kinematic studies of both trotting and galloping horses supports this (Firth *et al* 1991, Johnston *et al* 1995). Bramlage *et al* (1988) concluded from clinical observations that medial to lateral displacement of the carpal bones enabled intercarpal ligaments to accept load. This protects the joint surfaces but necessitates adaptation of these ligaments to exercise (Bramlage *et al* 1988). Hjertén and Drevemo (1993) using high speed cinematography measured 10 mm of axial compression in the carpus in a 640 kg horse trotting at 3.7 m/s which, because of its magnitude, was assumed unlikely to be due to compression of bone and cartilage alone, and it has been more recently confirmed that the intercarpal ligaments dissipate axial force in an *in vitro* biomechanical study (Young *et al* 1994).

Carpal joint forces

The potential forces acting on the carpal joint include axial force, transverse force, an overextension moment and a rotational moment in an axial plane. Both external axial and transverse forces acting in the limb can be estimated from ground reaction forces (Hjertén and Drevemo 1987). Based on this, axial forces reach a maximum immediately after midstance, whereas the predominant transverse force is a braking force which also reaches a maximum immediately following midstance.

More detailed information on carpal forces has been gained from analysis of kinematic data. This work has demonstrated that there are two phases of overextension in the fast trotting Standardbred. An early rapid phase, associated with rapid braking of the foot on ground contact, and a later slow phase (Johnston and Roepstorff 1996). In all, the carpus in overextended for approximately 80% of the stance phase (Johnston and Roepstorff 1996). This work has also demonstrated that increasing speed shifts the rapid phase of overextension more towards midstance when higher axial loading occurs. The combination of rapid overextension and high axial loading may result in injury to the carpus. Also transverse oscillations have been observed at the proximal metacarpus immediately after heel strike (Hjertén and Drevemo 1994, Johnston and Roepstorff 1996). These are also initiated by rapid braking of the hoof. The magnitude of these transverse accelerations is greatest in the early phase of the stride prior to the stabilizing effect of high axial loading (Hjertén and Drevemo 1994), so that the soft tissue structures which span the carpal joints must attenuate much of this force.

Forces at the articular surfaces of the midcarpal and antebrachiocarpal joint

Colahan *et al* (1987), with the use of pressure sensitive film inserted in the antebrachiocarpal and midcarpal joints, found several distinct areas of stress concentration with loading of the leg which correlated with areas of high carpal fracture incidence. These were the distal dorsomedial aspect of CR and the dorsal midline of the antebrachiocarpal joint. Bramlage *et al* (1988) suggested that this occurred due to direct transmission of weight-bearing stress to bones without the ability to dissipate stress to soft tissue structures. The radial facet of the third carpal bone receives a large proportion of the load transmitted through CR. The hinged nature of the midcarpal joint is also thought to contribute to its susceptibility to trauma as overextension is not possible (Bramlage *et al* 1988).

Distribution of pressure has also been shown to change with increasing extension, although there have been conflicting results. Colahan *et al* (1987) in a cadaver model of carpal loading found that pressure in the midcarpal joint becomes more evenly distributed as the forces of extension and axial loading increase, while in the antebrachiocarpal joint the pressure becomes more concentrated at the dorsal midline of the joint. Because carpal extension increases with fatigue, Colahan *et al* (1987) concluded that fatigue was a more important cause of pathology in the antebrachiocarpal joint than the midcarpal joint. However, Palmer *et al* (1994) using a very similar model showed that as axial load on the leg increased there was a significant increase in pressure on the dorsomedial and dorsolateral aspects of C3, but not palmarly in the midcarpal joint, so the effects of extension and fatigue may also be involved in the pathogenesis of midcarpal joint disease.

Role of biomechanics in carpal disease

Although overextension appears to be physiological, it has also been implicated in the pathogenesis of carpal disease (Kainer 1987, Palmer *et al* 1986, Colahan *et al* 1987). Various suggestions have been made regarding the mechanisms for preventing overextension. Bramlage *et al* (1988) stated that the midcarpal joint was not capable of overextension due to the palmar location of the collateral ligaments and the shape of the articular surface. The finding of Colahan *et al* (1987) that increased extension produced a more even distribution supports this. In contrast, Firth *et al* (1991) suggested that the palmar carpal ligament and the accessorioquatral ligament are the major ligamentous structures limiting overextension.

Poor conformation has often been implicated as a cause of carpal disease despite a lack of objective evidence to support it (Beeman 1973, Auer 1980). Beeman (1973) stated that overextension of the knee was the most serious defect of the front limb. Magnusson and Thafvelin (1985) and more recently Barr (1994a) demonstrated that there was no significant correlation between the degree of overextension at rest and carpal disease. It is unknown whether the degree of overextension at rest correlates well with the degree of overextension during exercise, however how the leg lands is probably more important in the development of joint disease than how it appears at rest. Humans with very early signs of joint pain in the knee have been shown to have higher impulsive loads at heel strike (Radin *et al* 1991). This microincoordination was proposed as a cause of osteoarthritis because impulsive loading consistently produces osteoarthritis in animal models (Radin *et al* 1984). Bench knee or lateral deviation of the metacarpus has been shown to predispose to carpal joint problems

(Magnusson and Thafvelin 1985) probably because this conformation will remain during exercise.

Pathophysiology of Carpal Disease

The end result of joint disease is irreversible loss of articular cartilage, and sometimes subchondral bone, by the process of degenerative joint disease or osteoarthritis. A variety of joint insults can ultimately lead to osteoarthritis. Both supra physiological loads, and excessive cycles of repetitive physiological loads can lead to failure of various joint elements. Cartilage damage may be direct, but more commonly is secondary to subchondral bone changes or damage to soft tissue structures.

Soft Tissue Damage

Damage to the soft tissue structures of a joint may be detrimental in two ways. Traumatically induced synovitis and capsulitis result in the release of cartilage degrading enzymes (McIlwraith and Vachon 1988, Clyne 1987, Brown *et al* 1987), and damage to the soft tissue supporting structures of the joint can result in instability.

Synovial inflammation- McIlwraith and Vachon (1988) stated that synovitis is almost always present in cases of equine osteoarthritis. Lysosomal enzymes, prostaglandins, and cytokines are released from inflamed synovial membrane (McIlwraith and Vachon 1988, Clyne 1987,

Brown *et al* 1987, May *et al* 1988, Shinmei *et al* 1989), particularly from synovial cells and neutrophils (Spiers *et al* 1994). Certainly severe synovial inflammation plays a major role in cartilage degeneration, as is the case in septic arthritis (Hardy *et al* 1994) and experimentally induced synovitis (McIlwraith and Van Sickle 1981). However there is no definitive proof that synovitis is a primary cause of cartilage matrix degradation in the majority of joints with osteoarthritis. In fact Radin *et al* (1984) were unable to demonstrate synovial membrane inflammation in rabbits despite the production of cartilage degeneration with mechanical loading. Studies of proteoglycan concentration in synovial fluid in human knees with inflammatory joint disease (Dahlberg *et al* 1992) found no correlation between nucleated cell counts and proteoglycans and Little *et al* (1990) in equine carpi with osteoarthritis found no correlation between nucleated cell counts, total protein and joint swelling and pain, and proteoglycan concentrations. These authors concluded that synovitis probably does not play a direct role in cartilage matrix degradation. In contrast Pool (1995) observed a gradient of decreasing severity of articular cartilage damage, running away from congested and hyperplastic synovium, in association with chip fractures in the carpus, and concluded that this was due to the activity of inflammatory mediators released from the synovium. However, this finding could also be explained by a gradient of subchondral bone changes resulting in secondary overlying cartilage damage.

Joint effusion may be another detrimental effect of synovial inflammation. Induced effusions in rabbit stifles has been shown to increase subchondral intraosseous pressure, decrease bone blood flow, and cause subchondral hypoxia and hypercapnia (Kofoed 1986). Increased subchondral bone pressure is associated with joint pain in humans (Arnoldi *et al* 1975).

Damage to intra-articular soft tissue structures may also contribute to articular cartilage degeneration by intra-synovial haemorrhage. It has been shown that a single episode of haemarthrosis in rats causes proteoglycan degradation for up to three days (Niibayashi *et al* 1995). Haemarthrosis has been observed in the midcarpal joint in association with palmar intercarpal ligament tearing (Kannegieter and Colgan 1993).

Ligament damage- Damage to ligamentous structures is a common cause of joint instability and consequent osteoarthritis (Brandt 1991). Ligamentous damage in the carpus has only recently been described (Kannegieter and Burbridge 1990). With greater use of diagnostic ultrasound to examine the carpus (Denoix and Audigie 1993) a better understanding of the role of ligamentous damage in carpal disease may emerge.

Damage to the medial palmar intercarpal ligament of the midcarpal joint has been documented by McIlwraith (1992), Kannegieter and Colgan (1993) and Phillips and Wright (1994). Although the correlation between ligament damage and articular cartilage damage is not clear, clinical signs of joint compromise do appear to be consistent and horses with complete rupture have a poor prognosis (McIlwraith 1992). Kannegieter and Colgan (1993) also found that horses with more severe articular cartilage lesions had a higher incidence and greater severity of ligament lesions suggesting that although it may not be a primary cause of articular cartilage degeneration, instability may exacerbate lesions. In contrast McIlwraith (1992) and Phillips and Wright (1994) found no correlation between the degree of tearing and the degree of articular cartilage damage.

The cause of intercarpal ligament injury is unknown. Kannegieter and Colgan (1993) observed that palmar intercarpal ligament damage often occurred bilaterally and when this occurred the damage was more severe than unilateral damage. This implies that predisposing factors affecting both limbs such as conformation, gait or increased stress may be involved in ligament tearing. McIlwraith (1992) suggested that some form of hyperextension injury may produce these ligament lesions and therefore concomitant damage to the palmar carpal ligament may be involved in the clinical syndrome. Desmitis of the palmar carpal ligament has been reported by Denoix and Audigie (1993). Phillips and Wright (1994) proposed that tearing of the MPICL was due to fatigue failure caused by cyclical tensing of the ligament during exercise. Intra-articular ligaments in dogs have been shown to undergo degeneration before physical injury occurs (Vasseur *et al* 1985). Inadequate blood supply was suggested as a possible cause because degeneration was not evident in collateral ligaments whose extra-articular location provides sufficient blood supply from overlying soft tissues. Chronic synovial inflammation has also been incriminated in anterior cruciate ligament rupture in dogs (Niebauer and Menzel 1982).

An interesting soft tissue anomaly has been described by Selway (1991). Congenital enlargement of a normal ligament (Martin and McIlwraith 1985), or synovial fold (McIlwraith 1990) running from the distal radial carpal bone to the proximal second carpal bone was suggested as a cause of osteochondral damage by impingement on the articular surfaces. The incidence of the lesion was 74% in joints examined by arthroscopy. McIlwraith (1992) argued that changes in this ligament were probably secondary to osteochondral fragmentation.

Subchondral bone changes

Repetitive cyclical loading can produce two important changes in subchondral bone; increased density and stiffness of subchondral bone (Radin *et al* 1984) and fatigue failure (Bramlage *et al* 1988).

Subchondral bone sclerosis- Along with the soft tissues of a joint, subchondral bone plays a major role in force attenuation during rapidly applied load (Radin *et al* 1970). Articular cartilage is too thin to act alone as an effective shock absorber. The transition from compliant articular cartilage to relatively stiff subchondral bone creates a stiffness gradient and subsequent shear forces at the bone/cartilage interface. Any increase in subchondral bone stiffness increases this gradient and therefore increases the resultant shear stresses (Radin and Rose 1986). Increased subchondral bone stiffness has been shown to occur in rabbit stifles in response to repetitive impulsive loads (Radin *et al* 1984) and in the midcarpal joint in horses in training (Young *et al* 1991).

Radin and co-workers have shown in humans with osteoarthritis and a mechanical model in rabbits, that stiffening of subchondral bone is a common finding in osteoarthritis (Radin *et al* 1970, Radin *et al* 1972, Pugh *et al* 1974, Radin *et al* 1984). They have also shown that changes in subchondral bone precede cartilage changes and may therefore be the primary cause of osteoarthritis (Radin *et al* 1984). As subchondral bone sclerosis is observed in horses in training it is reasonable to assume that subsequent cartilage damage occurs. Young *et al* (1988) observed cartilage lesions in association with increased thickness and compacting of

the subchondral bony plate in five racehorses.

The significance of sclerosis of C3 is unclear. O'Brien *et al* (1985) found sclerosis of C3 was a common radiographic finding in race horses. Thus increased bone density is probably a normal response to training. However, Young *et al* (1991) also showed that horses with pathology of C3 (slab fractures or degenerative joint disease) had higher bone density than normal horses in training and that this corresponded with the area where most slab fractures occurred. To be clinically significant there must be resulting cartilage degeneration, or perhaps an increase in intraosseous pressure causing pain. Increased intraosseous pressure in subchondral bone has been demonstrated in osteoarthritis in man (Kiær *et al* 1988) and as stated previously is associated with pain (Arnoldi *et al* 1975).

Fatigue failure of bone- Most carpal fractures in racing horses are a result of fatigue failure of bone from repetitive loading rather than acute injuries. Pool (1995), based on histopathological examination of a large number of chip fractures, demonstrated that nearly all had evidence of chronic injury and repair. Fatigue failure occurs due to accumulation of damage from cyclical loading at physiological loads. The number of cycles of loading that lead to failure is dependant on the deformation or strain to which the bone is subjected at each cycle (Caler and Carter 1989). Increasing strain reduces the number of cycles to failure. Microcracks are normally present in bone and are thought to be fatigue cracks because their numbers increase following repetitive loading (Burr *et al* 1985). To prevent failure, damaged bone must be replaced by osteonal remodelling, or new bone added to the surface by modelling. Modelling to the extent seen in MC3 in response to loading (Nunamaker *et al*

1989) does not occur in the cuboidal bones of the carpus or the distal radius. These bones appear to rely on increased density of trabecular bone (Young *et al* 1988). This has been shown to result in an increase in the gradient of bone density in the dorsal aspect of C3 (Young *et al* 1988). This increase in gradient causes an increase in the shear forces within the bone on the dorsal aspect, therefore increasing the strains on the bone and reducing the number of loading cycles to failure.

Osteonal remodelling is performed by multicellular units which appear to be attracted to areas of microdamage (Mori and Burr 1993). Because of the space between the cone of osteoclasts and the following osteoblasts, these multicellular units act as pores in the bone. So in the initial stage of remodelling there is an increase in porosity of the bone and therefore weakening. The combination of microdamage and increased porosity decreases the elastic modulus and elevates the strains produced by the applied loads, accelerating damage (Martin 1995). Remodelling can therefore contribute to fatigue. Fatigue fracture is most common in young animals and humans for two reasons. Younger horses have been show to subject their bones to greater strains and therefore will fail with fewer cycles (Nunamaker *et al* 1990). Also bone turnover is naturally at its highest in the young so that there are a greater number of multicellular units available to be stimulated to remove damaged bone (Morris 1980).

Pool (1995) has observed deposition of woven bone rather than lamellar bone on subchondral trabeculae in third carpal bones which appeared to be developing slab fractures. Woven bone is mechanically weaker than lamellar bone but can be laid down more rapidly (Nunamaker 1996). A similar response occurs on the dorsal surface of the third metacarpal bone in

response to decreased stiffness due to cyclic fatigue (Nunamaker 1996). Therefore this finding in C3 suggests that the remodelling process is not keeping pace with the rate of accumulation of damaged bone.

It has been suggested that the type of fracture depends on the type of loading (Bramlage *et al* 1988). High loading in Thoroughbred carpi tends to produce small chip fractures. When slab fractures occur they are usually complete (Schneider *et al* 1988). Standardbreds subject their carpi to lower loading but over longer periods, producing deeper chip fractures and a higher frequency of incomplete slab fractures of C3 (Schneider *et al* 1988, Bramlage *et al* 1988).

Fatigue failure of bone in the carpus may manifest as chip or slab fractures, but also as softening of subchondral bone (Ross *et al* 1989, Dabareiner *et al* 1991) or, as is most common, a combination of the two (Dabareiner *et al* 1996). The most common sites are the distal CR, proximal C3 and distal radius (Thrall *et al* 1971, Larsen and Dixon 1970, Lindsay and Horney 1981, Mizuno 1996, Raidal and Wright 1996). McIlwraith *et al* (1987) found that proximal CI was also a common site. Bramlage *et al* (1988) describe bone loss from distal CR and lysis of C3 as early changes in midcarpal joint disease. There is evidence that subchondral bone lysis precedes chip fracture formation on distal CR (Barr 1994b) and slab fractures of C3 (Pool 1995).

Therefore the role of subchondral bone in the development of carpal disease is both primary, in the formation of fractures, and secondary, due to its effect on articular cartilage.

Articular cartilage degeneration

Osteoarthritis is the end result of a variety of joint insults which overwhelm the reparative potential of the chondrocyte. Direct trauma to the cartilage (Donohue *et al* 1983), intra-articular fractures (Huber *et al* 1992), subchondral bone sclerosis (Radin *et al* 1984), synovitis (Wittenburg *et al* 1993), instability from soft tissue injury (Kannegieter and Colgan 1993) and central nervous system disorders (Radin *et al* 1991a) have all been implicated in cartilage degeneration.

The limited ability of articular cartilage to repair is well documented (Mankin 1982, Hannie *et al* 1991, Shapiro *et al* 1993, Vachon *et al* 1986). Increased cellularity and high synthetic activity have been demonstrated in the equine carpus following articular cartilage injury (Richardson and Clark 1991). However, the repair of both partial thickness and full thickness defects is disappointing (Hanie *et al* 1991, Vachon *et al* 1986). Early detection of cartilage loss therefore is imperative in the management of horses with carpal osteoarthritis, yet no reliable method exists (Lohmander *et al* 1992).

Early changes in degeneration of articular cartilage- Controversy exists over the initial changes in articular cartilage undergoing degeneration whatever the inciting cause. The most widely held view is that depletion of proteoglycan from the cartilage matrix is the primary event (Richardson and Clark 1991, Lohmander *et al* 1992, Howell 1986, Hasty *et* al 1990, Ferguson 1987, Buckwalter *et al* 1988a, Vachon and McIlwraith 1990). Chondrocytes respond to trauma by increased production of proteolytic enzymes and reduced production of

enzyme inhibitors, with subsequent cleavage of proteoglycan aggregates and loss into synovial fluid (Howell 1986, Lohmander *et al* 1992, Hasty *et al* 1990). However, Radin *et al* (1991) and Freeman (1975) have suggested that changes in chondrocyte function and depletion of proteoglycan are secondary to mechanical disruption of matrix interactions. Mechanical loads on cartilage generate shear stresses that can break interfibrillar cross-links within the articular cartilage collagenous matrix. This allows increased hydration and swelling of the cartilage with loss of proteoglycans, as demonstrated when canine femoropatellar joints were impacted with single subfracture loads (Donohue *et al* 1983). An increase in cartilage proteoglycan content was found at four and six weeks after injury, but there was a decrease in proteoglycan associated with collagen fibres. Radin *et al* (1991) hypothesised that this substructural disorganisation of matrix precedes chondrocytic enzymatic production, and may allow entry of catabolic enzymes into the cartilage matrix. Freeman (1975) proposed fatigue failure rather than simple impacts as the cause of disruption of collagen fibres or crosslinks. Further evidence that structural change may be more important than biochemical change was reported by Guilak *et al* (1994) who demonstrated that there was no correlation between tensile stiffness and biochemical composition of articular cartilage in a canine model of early osteoarthritis.

Role of biomechanics in cartilage degeneration- The type of loads to which cartilage is subjected is thought to be important in the pathogenesis of cartilage degeneration. Cartilage is viscoelastic and under high rates of load becomes stiffer (Radin *et al* 1991). Purely increasing loading on cartilage does not necessarily lead to degeneration. Panush *et al* (1986) found no increase in the incidence of osteoarthritis in human runners compared with non runners.

Kiviranta *et al* (1988) in studies of exercising dogs found that moderate exercise actually increased proteoglycan content of articular cartilage in high load bearing regions, while more strenuous exercise, although resulting in decreased proteoglycan content, did not produce any gross cartilage degeneration (Kiviranta *et al* 1992, Arokoski *et al* 1993). Impulsive loading, load applied at high rate, is more damaging because it prevents stress relaxation from the outflow of water, as well as minimising contact area due to lack of deformation (Radin *et al* 1988). The high stresses at the cartilage bone interface produce deep horizontal splits in the cartilage (Radin *et al* 1991b). Radin *et al* (1991a) went further to show that humans with activity related knee joint pain exhibited subtle gait differences compared with pain free individuals. In particular they displayed a more rapid rise in ground reaction force which they concluded was a form of repetitive impulse loading. The differences in gait were attributed to minor neuromuscular incoordination. Repetitive impulse loading has been demonstrated in galloping horses by Ratzlaff *et al* (1987). They found a sharp rise in vertical force occurred immediately following hoof contact. It is possible that the rapidity of this rise varies between horses, as in humans, rendering some horses more susceptible to joint disease than others.

Articular cartilage proteoglycan depletion- Whatever the inciting cause, cartilage proteoglycan depletion is a central event in the development of osteoarthritis (Mankin *et al* 1982, Lohmander *et al* 1992, Maroudas *et al* 1973, Hasty *et al* 1990). Proteoglycans determine the compressive stiffness of articular cartilage (Kempson *et al* 1970, Freeman 1975, Buckwalter *et al* 1988b) and, with their loss, cartilage becomes more vulnerable to excessive forces and fibrillation develops (Clyne 1987, Fergusson 1987, McIlwraith and Vachon 1988).

Many enzymes have been incriminated in proteoglycan breakdown. Lysosomal enzymes such as cathepsin D, B and F (McIlwraith and Vachon 1988, Clyne 1987, Clark 1991, Fosang *et al* 1992) and neutral and acid metalloproteases such as collagenase and stromelysin (Pelletier and Martel-Pelletier 1985, Pelletier *et al* 1988, Hasty *et al* 1990, Fosang *et al* 1991) have been shown to degrade proteoglycans and have been isolated from joints with osteoarthritis. These enzymes are released from chondrocytes in response to cytokines such as interleukin-1, interleukin-6 and tissue necrosis factor alpha, produced by inflammatory cells and chondrocytes (Brown *et al* 1987, Arner and Pratta 1989, Shinmei *et al* 1989). However, examination of synovial fluid in humans with osteoarthritis has shown that aggrecan, the major proteoglycan in cartilage, was consistently cleaved at a single site within the interglobular domain of its core protein (Sandy *et al* 1992, Lohmander *et al* 1993). None of the proteolytic enzymes so far identified in osteoarthritic joints acts at this site (Fosang *et al* 1991, Fosang *et al* 1992). The isolation of this enzyme ("aggrecanase") is a major goal in osteoarthritis research as it has important implications in therapeutic protection of cartilage in diseased joints.

Whether proteoglycan breakdown is accompanied by reduced or increased proteoglycan synthesis is an area of dispute. Mankin *et al* (1981) demonstrated increased proteoglycan synthesis in human osteoarthritic cartilage while Moskowitz *et al* (1981) found no difference in synthesis between rabbits with meniscectomy and controls. Bulstra (1989) and Thompson and Oegema (1979) found that proteoglycan synthesis increased with the histological grade of cartilage degeneration in early stages, but was decreased with the more severe degenerative changes associated with late osteoarthritis. The complexity of chondrocyte synthetic response

in osteoarthritis was further demonstrated in a mice monoiodoacetate model by van Osch *et al* (1993). Various areas of the joint responded differently, with synthesis inhibited in some and stimulated in others. Within the normal equine carpus there is a similar large variation in proteoglycan synthesis, with the radial facet of the third carpal bone, an area of high loading, undergoing higher turnover than other sites (Richardson and Clarke 1991).

Factors involved in the perpetuation of degeneration- Once cartilage begins to degrade, a cycle of events perpetuates the process. A number of studies have shown that cartilage and bone fragments in synovial fluid are detrimental to the joint environment, producing both physical cartilage damage (Hurtig 1988, Huber *et al* 1992) and synovitis (Hurtig 1988, May *et al* 1988, Huber *et al* 1992, Myers *et al* 1992). Tew and Hackett (1981) demonstrated cartilage fragments in synovial fluid from equine joints with articular lesions, while osteochondral fragments are commonly produced in the carpus of racing horses (Bramlage *et al* 1988). Anticollagen antibodies and immune complexes have been identified in equine joints with secondary osteoarthritis and may be one of the mechanisms by which cartilage and bone particles perpetuate joint damage (Niebauer *et al* 1988).

Role of the nervous system in degeneration- Neurotransmitters are released into joints in response to mechanical or chemical activation of articular nerves (Caron *et al* 1992a). One of these, substance P, has been isolated from the synovial fluid of the mid carpal joint in horses and elevated concentrations have been demonstrated in diseased joints (Caron *et al* 1992b). These neurotransmitters induce inflammation both directly or through the action of other mediators (Caron *et al* 1992a).

Therefore both osteochondral debris and pain generated in the diseased carpus fuel the ongoing degenerative process.

Clinical evaluation of carpal disease

History

The history of horses with carpal disease is dependant on the type and the severity of the injury. Lameness is often bilateral (Bramlage *et al* 1988) so may not be noticed by owners or trainers. Early signs reported by owners or trainers may be poor performance, bearing in or out, particularly around turns, or filling and heat in the joint (Richardson 1990, Ross 1992). Larsen and Dixon (1970) reported that more than 50% of horses with carpal fractures had a history of gradual shortening or shifting of stride while Wyburn and Goulden (1974) found that a gradual onset of lameness was reported in 23%.

Clinical examination

Synovial distension of the antebrachiocarpal joint or the midcarpal joint is the hallmark of carpal disease (Stashak 1987). It is important though to examine the horse in the acute phase of the disease because heat, pain and swelling may disappear with rest (Auer 1980, Larsen and Dixon 1970). In a study of C3 fractures Schneider *et al* (1988) found enlargement of the midcarpal joint in 86% of cases. As the disease process becomes more chronic the fibrous

capsule becomes thickened, especially craniomedially (Bramlage *et al* 1988). This will produce a reduced range of motion which is helpful in evaluating the chronicity of the problem (Auer 1980). Schneider *et al* (1988) found that in 78% of horses with C3 fractures there was resistance to flexion.

The degree of lameness is proportional to the extent and duration of the damage (Lindsay and Horney 1981). Horses with small, acute articular fractures mostly exhibit minimal signs of lameness (Stashak 1987), while those with acute, large chips or slab fractures may stand with the joint partially flexed. Severe bilateral carpal disease can cause bizarre gaits which may be diagnosed as neurological disease (Richardson 1990). Gait analysis of horses with induced carpal lameness has shown that head and withers excursions are the most consistent variables for assessing lameness (Peloso *et al* 1993).

Careful palpation of the carpus is important (Ross 1992), although Richardson 1990 states that visual inspection is usually just as helpful. With the carpus flexed, the dorsal articular margins of all carpal bones should be palpated together with the dorsomedial proximal aspect of MC3 (Ross 1992). With chronicity, palpation for focal pain becomes less rewarding (Richardson 1990), although Larsen and Dixon (1970) reported that pain on deep palpation was always present at a fracture site. Auer (1980) and Stashak (1987) suggested that the carpal flexion test was a valuable tool. The increase in lameness following carpal flexion probably arises from stretching of the joint capsule and compromise to blood flow causing an increase in subchondral pressure (Dyson 1987).

Local anaesthesia

While carpal lameness can be confirmed by intrasynovial anaesthesia (Stashak 1987), this may only be the case if pain arises predominantly from the synovial membrane and joint capsule. Lameness arising from subchondral bone pain or a mechanical lameness produced by periarticular fibrosis will not be eliminated by intra-articular anaesthesia (Dyson 1987). Shepherd and Pilsworth (1993) described four cases of distal radius chip fractures that failed to respond to intra-articular anaesthesia. Two of the horses underwent arthroscopic surgery and were found to have intact articular cartilage overlying the chip fractures. The authors concluded that this prevented access of the anaesthetic agent to the subchondral bone.

The clinician must also be aware of all the structures which are desensitized with intra-articular anaesthesia. The distopalmar outpouchings of the carpometacarpal joint lie in close proximity to the palmar metacarpal nerves and interdigitate extensively with the fibres of the proximal aspect of the suspensory ligament (Ford *et al* 1988). Because of the communication between the midcarpal and the carpometacarpal joints, proximal suspensory lesions (Ross 1992) and stress fractures of the palmar proximal third metacarpal bone (Pleasant *et al* 1992) may block out with an intra-articular midcarpal joint block. Ross (1992) recommends evaluating the block within 10 to 15 minutes to avoid this problem. Dorsomedial articular fractures of MC3 also can be blocked with a midcarpal joint intra-articular block (Ross 1992)

If a major intra-articular fracture is suspected, radiographs should be taken first to avoid turning an incomplete fracture into a complete fracture (Stashak 1987, Fischer and Stover

1987). Varying volumes of anaesthetic agent are recommended. Stashak (1987) recommends five to 10 ml while Gabel (1983) uses 13 ml of mepivicaine. Auer (1980) recommends adding 50-100 mg of gentamicin to the local anaesthetic to reduce the risk of iatrogenic sepsis.

When carpal lameness is suspected and there is no response to intra-articular blocks, regional anaesthesia of the carpus can be performed. This involves blocking the ulnar and median nerves. It is best performed on a different occasion from lower limb nerve blocks to allow testing for loss of cutaneous sensation (Dyson 1984).

Ultrasound

The use of ultrasound in the diagnosis of equine carpal disease has only recently been reported. Technically, examination of the carpus with ultrasound is more difficult than the metacarpus, due to the bony projections of the carpus and the varied orientation and number of anatomical structures (Denoix and Audigie 1993). The normal ultrasonic appearance of the dorsal and lateral aspects of the carpus have been described by Tnibar *et al* (1993).

Denoix and Audigie (1993) described the ultrasonic findings in 45 horses with carpal abnormalities. Ultrasound allowed evaluation of the soft tissue structures of the carpus as well as bone surfaces. Soft tissue lesions that could be detected included desmitis and tearing of the extensor retinaculum, effusion in the dorsal recesses of the antebrachiocarpal and midcarpal joints, and desmitis of the palmar carpal ligament. Osteophytes, enthesiophytes and some fractures can also be visualised with ultrasound, although the relationship between bone

fragments and ligamentous structures is a more useful application.

Radiography

Stashak (1987) describes five standard radiographic views for the diagnosis of carpal disease. A dorsal palmar (DP), lateral medial (LM), dorsolateral palmaromedial oblique (DLPMO), dorsomedial palmarolateral oblique (DMPLO) and a flexed lateral medial (FLM). The skyline view of the third carpal bone is only taken in selected cases to assess the depth, length, and the exact location of a third carpal bone slab fracture (Stashak 1987). Schneider *et al* (1988) in a study of 371 third carpal bone fractures found that 10.8% were visible only on the skyline view. They concluded that this view was essential. O'Brien *et al* (1985) stated that a skyline projection is necessary to identify comminution, incomplete slab fractures or sclerosis of C3. Fischer and Stover (1987) described 12 sagittal C3 fractures all of which were only visible on a skyline view. Ross *et al* (1989) stated that the skyline view was mandatory for assessing carpal lameness in Standardbreds.

For these reasons Richardson (1990) and Ross (1992) recommend a DP, flexed LM, DL-PMO, DM-PLO, flexed skyline of the third carpal bone as a standard set of views. Obliques should be taken at 60 degrees off the dorso-palmar plane (O'Brien *et al* 1971). Skylines of the proximal row of carpal bones and distal radius help define position and shape of lesions identified on other views (Richardson 1990). The skyline needs to be well positioned to evaluate the radial fossa. Lateral deviation of the lower leg during positioning can obscure the radial fossa. The third metacarpal bone needs to be directly underneath the radius or slightly

medial (Ross 1992). Radiographs of the opposite carpus should always be taken as many carpal problems are bilateral (McIlwraith *et al* 1987, Stashak 1987) and followup radiographs two to three weeks later are important in horses with negative initial findings (Ross 1992).

McIlwraith *et al* (1987) and Kannegieter and Burbridge (1990) assessed radiographic diagnosis of carpal chip fractures by comparing findings with lesions observed during arthroscopic surgery. McIlwraith *et al* (1987) found radiography accurate in detecting distal CI fractures, proximal C3 fractures, and fractures of the distal radius whereas Kannegieter and Burbridge (1990) found proximal C3 and distal CI had a greater incidence of fractures not detected on radiographs. Radiography was less useful in predicting the degree of damage to distal CR, proximal CI and to a lesser extent proximal CR. On the distal CR there was often multiple fragments, soft degenerative bone, dorsolateral corner fragments and severe cartilage loss that was not evident on radiographs (McIlwraith *et al* 1987). In contrast Kannegieter and Burbridge (1990) reported a similar finding in only four joints out of 150. This discrepancy may be due to differing interpretation of radiographic changes. Richardson (1990) states that extensive articular damage is evidenced by decreased subchondral bone density extending away from the fracture, while McIlwraith (1992) admits that subtle radiographic changes on distal CR indicates major osteochondral damage. More recent studies have stressed the need for careful interpretation of subtle radiographic changes in the carpus. Dabareiner *et al* (1996) showed where subchondral lucency of distal CR was the only radiographic abnormality, 69% of horses had osteochondral fragments, while a further 26% had fibrillated cartilage and softening of subchondral bone.

The more frequent use of skyline views of C3 has highlighted a number of conditions. Sclerosis of the C3 is a common finding, but there is controversy over its significance. O'Brien *et al* (1985) found that sclerosis of C3 was common in horses with a fracture of C3, but also in horses in training with no evidence of carpal problems. Young *et al* (1988) found sclerosis in only one out of fifteen non-racing horses while 70% of racing horses had some degree of sclerosis. They concluded that a degree of sclerosis was normal in racing horses. Subchondral lucency (Ross 1992) is another condition best assessed on the skyline view. Often multiple lucent areas, with an intact dorsal margin, are found in Standardbreds, usually associated with sclerosis of the radial fossa.

McIlwraith (1991) has described the diseases of the carpus which cannot be detected radiographically. These include acute synovitis, synovial impingement, ligamentous sprain and desmitis, ligamentous and fibrous capsule tearing, articular cartilage tearing and early osteoarthritis. Moore and Schneider (1995) reported the arthroscopic findings in 41 cases with minimal or no radiographic changes. The most common finding was a crush fracture, incomplete slab fracture of C3, or damage to the articular cartilage of C3. Again, more careful interpretation of subtle radiographic changes may help diagnose these subchondral bone lesions, but because a 40% change in bone density is required before such lesions can be detected radiographically (Dyson 1987) many bony changes may go undetected, particularly early in disease processes.

The significance of an abnormal radiographic finding is a constant question. Jeffcott (1983) radiographed 45 working Swedish warmblood horses with minimal clinical signs of joint

disease. The most common finding was minor proliferative changes at the caudal aspect of the distal radial epiphysis (65%) and roughening of the cranial surface of the radius (39%) and CR (21%). Minor spurring was present on the cranial edges of antebrachiocarpal, midcarpal and carpometacarpal joints in 19% of the horses. Radiolucency was evident in the caudal aspect of C2 (13%) and the head of the second metacarpal bone (11%). Whether these signs are within the normal range of radiographic findings or early signs of joint problems before clinical signs develop remains to be determined.

Scintigraphy

Scintigraphy is a more sensitive indicator of bony pathology than radiography (Barbee and Allen 1990, Snow 1992), but Dyson (1987) claimed that it can demonstrate only an active, nonspecific joint involvement, so may not readily detect chronic changes such as osteoarthritis. In particular its ability to identify subchondral cystic lesions has been shown to be poor (Steckel 1991, Ray *et al* 1996). However it appears that scintigraphy is more useful for detecting more active joint pathology. Steckel (1991) claimed that stress induced sclerosis of C3 could only be detected by scintigraphy and that the increased blood flow associated with synovitis was also detectable.

Scintigraphy is useful in horses that respond to diagnostic anaesthesia but have negative radiographic findings (Snow 1992), and in early lesions before radiographic changes become apparent, usually by 10 to 14 days (Ross 1992). Scintigraphy is also valuable when local anaesthetic blocks are negative or confusing. Shepherd and Pilsworth (1993) demonstrated

this in three horses with negative intra-articular antebrachiocarpal joint blocks. All had increased technetium uptake in the carpus, two in the antebrachiocarpal joint and one in the midcarpal joint. These horses were subsequently shown to have distal radius chip fractures.

Computed tomography and magnetic resonance imaging

Computed tomography has some advantages over conventional radiography for imaging the carpal region (Martens 1996). Superimposition of adjacent structures is avoided and accurate measurement of bone density and size and position of lesions is possible. Good delineation between cortex and medulla is achieved (Kaser-Hotz *et al* 1994). This makes it particularly useful for planning surgical approaches as well as assessing subchondral sclerosis and osteolysis (Kaser-Hotz *et al* 1994). The major disadvantage is the requirement for general anaesthesia. Improved imaging of soft tissue structures over conventional radiography is possible but detail of ligamentous structures is poor (Kaser-Hotz *et al* 1994).

In contrast magnetic resonance imaging gives high soft tissue contrast and has been used to diagnose intra-articular ligament injuries (Liu *et al* 1994) and to evaluate cartilage thickness and degenerative changes (Tyler *et al* 1995). Application in the horse has so far been limited to cadaver specimens due to the difficulty of positioning even anaesthetised horses legs in the coils (Denoix *et al* 1993, Kaser-Hotz *et al* 1994).

Synovial fluid analysis

The use of synovial fluid analysis in the diagnosis of joint disease in horses is well documented (Van Pelt 1974, Moyer 1982, Tew 1982). The chemical, physical and cytological characteristics of synovial fluid change with disease (Moyer 1982). Moyer (1982) identified four situations where synovial fluid analysis was particularly useful. Firstly, in diagnosis of infectious or drug-induced arthropathies and subsequent monitoring of the response to treatment. Secondly, where intra-articular damage is not evident radiographically such as synovitis or undisplaced fractures. Synovial fluid analysis can also aid in establishing the clinical significance of radiographic changes, and in the selection of treatment. Tew (1982) suggested that synovial fluid analysis was most valuable for measuring the efficacy of therapy designed to resolve intra-articular inflammation. Normal values for synovial fluid have been shown to vary considerably from one laboratory to another (Shumacher *et al* 1986) making interpretation difficult.

Except in the case of septic arthritis, synovial fluid analysis rarely gives a specific diagnosis. Changes are mainly due to the degree of synovitis (Stashak 1987). Nilsson and Persson (1973) compared synovial fluid from joints with cartilage lesions and joints with changes predominantly in the synovial membrane and found little difference. Moyer (1982) divided joints into five groups according to synovial fluid findings. Joints with only a slight to moderate decrease in viscosity were classed as a traumatic effusion. Mild synovitis was characterised by a mild decrease in viscosity and mucin quality, while a marked decrease in viscosity and mucin quality with a mild increase in white cell count was interpreted as a

severe synovitis. End stage osteoarthritis was characterised by low protein and viscosity with very poor mucin quality and all layers of cartilage present in cartilage particle analysis. Both bone and cartilage debris indicated osteochondral damage. Due to extensive overlap between these groups, specific diagnosis by synovial fluid analysis is difficult, but an indication of the disease process can be obtained (Stashak 1987). Although reported as a method of determining the degree of articular cartilage damage, the examination of cartilage particles in synovial fluid has not been widely used (Tew and Hackett 1981).

Markers of matrix metabolism in synovial fluid

Due to the difficulty in detecting the degree of cartilage damage in a joint without surgical intervention, there has been much interest in markers of cartilage metabolism in synovial fluid (Lohmander *et al* 1992). Markers that have been studied include cytokines, proteinases, matrix components, serum antibodies to collagen and chondrocyte membrane proteins (Lohmander *et al* 1992). In particular, cartilage proteoglycans have attracted a lot of attention both in human (Dahlberg *et al* 1992, Messner *et al* 1993, Silverman *et al* 1990, Ratcliffe *et al* 1988, Lohmander *et al* 1989) and equine (Alwan *et al* 1991, Little *et al* 1990, Yovich *et al* 1991) osteoarthritis research.

Heimer *et al* (1992) showed that normal human synovial fluid contained similar intact proteoglycans to those found in articular cartilage. They concluded that the proteoglycans found in synovial fluid of normal joints probably derived exclusively from articular cartilage and underwent little proteolytic degradation. In diseased joints proteoglycans may be released

from the synovial membrane as well as articular cartilage, although those from the synovial membrane are generally of lower molecular weight (Heingård *et al* 1985). Both Carroll (1989) and Ratcliffe *et al* (1996) showed that there was little difference between the total proteoglycan concentration and the concentration of proteoglycans of cartilage origin. The concentration of proteoglycans in synovial fluid is determined by the amount of cartilage matrix in the joint, the metabolic activity of chondrocytes (Dahlberg *et al* 1992), the rate of clearance from the joint cavity (Page-Thomas *et al* 1987), and the activity of degradative enzymes (Lohmander *et al* 1992). The clearance half life of proteoglycans has been shown to be about 12 hours in rabbits, and was unchanged by synovitis (Page-Thomas *et al* 1987). A similar value has been found in horses (Linblad G, Lindholm S and Heingård D unpublished observations).

Proteoglycan concentrations in diseased joints were first reported by Seppala *et al* (1972). Interest in their use as a marker for osteoarthritis was stimulated by Heingård *et al* (1985) who developed an enzyme linked immunosorbent assay (ELISA) for proteoglycan. This assay was found to be both specific and sensitive for cartilage proteoglycans. Carroll (1987) compared a modified dimethylene blue (DMB) assay for sulfated glycosaminoglycan (GAG) and a radioimmune assay for proteoglycan. The DMB assay was shown to be suitable for measuring sulfated GAG levels in synovial fluid. The alcian blue method has also been reported by Messner *et al* (1993) and was shown to be less prone to interference than the DMB assay.

High concentrations of proteoglycans have been reported in acute inflammatory arthritis in

humans (Hascall and Glant 1987, Saxne *et al* 1987, Ratcliffe *et al* 1988), but more chronic joint disease is characterised by levels similar to controls (Ratcliffe *et al* 1988). The relationship between proteoglycan levels and various stages of osteoarthritis was further defined by Dahlberg *et al* (1992) and Mankin (1982). Proteoglycan concentration decreased with increasing cartilage degradation as graded by arthroscopy or radiographs. Lohmander *et al* (1989) showed that proteoglycan concentration in human knee joints increased dramatically in the first three to four weeks following joint injury. In a study of rabbits with anterior cruciate ligament transection, meniscectomy and meniscal substitution, concentrations of proteoglycans were found to increase with the severity of arthrosis in the first three months (Messner *et al* 1993). Thus, high proteoglycan concentrations in synovial fluid occur early in the disease process at a time when diagnosis by other means is difficult. Unfortunately the wide range of values makes it difficult to interpret a single sample. It is also impossible to determine if proteoglycan loss, as determined by a high synovial fluid concentration, is reversible or irreversible.

Studies in horses have shown variable results. Alwan *et al* (1990) showed a significant increase in sulfated GAG and keratan sulphate concentrations in joints that clinically showed evidence of osteoarthritis, but Yovich *et al* (1991) found no significant difference between proteoglycan levels in midcarpal joints with effusion and lameness and joints from horses free of lameness, and Little *et al* (1990) found no correlation between sulfated GAG concentration and the degree of cartilage erosion determined at post mortem in 13 normal joints and 11 joints with osteoarthritis. This variability in results is not surprising due to the heterogenous nature of the disease groups studied. More meaningful results have been

obtained in human studies when the patient groups have been stratified according to disease stage (Lohmander *et al* 1992). The effect of exercise on proteoglycan concentration also has not been taken into account in these studies. This may be important as proteoglycan concentration has been shown to increase immediately following strenuous exercise in horses (Yovich 1993). Also more recently it has been shown that there is substantial variation in both keratan sulfate and sulfated GAG concentrations between different joints within individual horses (Fuller *et al* 1996).

Despite their potential the use of these markers in the clinical situation has been questioned. Silverman *et al* (1990) found no correlation between proteoglycan levels and any clinical measurements in both acute and chronic joint disease in humans. They also found that the measurement of proteoglycan in sequential synovial fluids gave no indication of the state of the cartilage or the overall rate at which it was eroding. As previously described, similar findings have been reported in horses (Little *et al* 1990).

Arthroscopic examination

The arthroscope is used both as a diagnostic tool and in the treatment of various joint problems (McIlwraith 1983). The normal arthroscopic anatomy of the antebrachiocarpal joint and midcarpal joint has been described by Hurtig and Fretz (1986) and McIlwraith (1990). Arthroscopy provides a direct assessment of the articular cartilage and some fractures not visible on radiographs can be detected (McIlwraith 1990, Moore and Schneider 1995). Also, soft tissue structures which are difficult or impossible to image with ultrasound can be

assessed by athroscopy. Selway (1991) used arthroscopy to assess hypertrophy of a structure on the medial aspect of the midcarpal joint capsule described as a synovial plica by McIlwraith (1990). Arthroscopic surgery is the only technique available to detect damage to the intercarpal ligaments (Kannegieter and Colgan 1993, McIlwraith 1992).

When examining the carpal joints arthroscopically care must be taken not to over interpret cartilage lesions due to magnification (McIlwraith 1984, McIlwraith 1990). Hurtig *et al* (1985) examined the accuracy of arthroscopic identification of lesions in articular cartilage. They found that accurate quantitative assessment of articular defects was possible. Some loss of accuracy occurred when the arthroscope was positioned directly over the lesion. Cartilage fibrillation, and partial and full thickness erosion can be detected readily with arthroscopy (McIlwraith 1990). In many cases it is important to use a probe to further define lesions (McIlwraith 1990). In particular palpation of the intercarpal ligaments with a probe allows assessment of the number of intact fibres remaining (Kannegieter and Colgan 1993), and lesion size can be determined by comparison with graduations on the probe.

The use of two arthroscopic portals in the carpus improves visualisation of the whole joint. This is not as critical in the midcarpal joint as in the antebrachiocarpal joint (McIlwraith 1990). Positioning the horse in dorsal recumbency allows easy swapping of portals (McIlwraith *et al* 1987). Visualisation of the midcarpal joint is generally better than the antebrachiocarpal joint due to the close attachment of the joint capsule to the proximal margin of the intermediate and radial carpal bones and the kinematics of the joint (McIlwraith 1990).

Two grading systems for articular cartilage damage have been described. McIlwraith *et al* (1987) used the following four grades:

Grade 1 - minimal fibrillation or fragmentation at the edge of the defect left by the bone fragment, extending no more than 5 mm from the fracture line.

Grade 2 - articular cartilage degeneration extending more than 5 mm back from the defect and including up to 30% of the articular surface of that bone.

Grade 3 - loss of 50% or more of the articular cartilage from the affected carpal bone.

Grade 4 - significant loss of subchondral bone.

An alternative grading system was described by Kannegieter and Ryan (1991) and involved the following three grades:

Grade 1 - cartilage damage extending 2 to 3 mm from the fracture and no or minimal cartilage damage on the opposing articular surface.

Grade 2 - cartilage damage extending up to 3 to 8 mm from the fracture site

and obvious damage on the opposing articular surface.

Grade 3 - cartilage damage extended greater than 8 mm from the fracture line and severe lesions on the opposing articular surface.

Both these grading systems apply only to joints with osteochondral fragments and give an indication of the overall joint compromise. No grading system has been reported which describes articular cartilage damage and subchondral bone damage separately and which can be applied to joints with no articular fractures.

Clinical significance of intra-articular lesions

As diagnostic aids become more sensitive, previously unrecognised musculoskeletal pathologies are detected, as are changes in musculoskeletal tissues in response to training. There is often no clear cut division between what is a normal response to training and what is a significant clinical problem. A good example of this is sclerosis of C3 (O'Brien *et al* 1985). In racing horses, the effect of a clinical condition on performance is the only true guide to its clinical significance. Ideally the race records of individual horses would be examined without the condition, and then compared to performance with the condition. In practice this is almost impossible as the exact time of onset of the condition is unknown or the horse is not raced once a problem is identified. Also there are multiple factors which affect racing performance (Speirs 1983) and trying to control for a single factor is impossible.

Three techniques have been used to assess the effect of a clinical condition on performance. A group of horses with an identified problem can be compared with a group of horses which are known not to have the problem (Grøndahl and Engeland 1995), or a group taken from the general population (Speirs *et al* 1986, Raidal and Wright 1996). This requires a very large number of control horses to remove the effects of variation in class between the two groups of horses (McIlwraith and Turner 1986). Also many orthopaedic injuries involve a number of structures, so it is not possible to determine the effect of injury to individual structures on performance. Alternatively, performance has been compared preoperatively and postoperatively if the problem can be surgically corrected (McIlwraith *et al* 1987), however in most orthopaedic conditions complete surgical correction is not possible (McIlwraith and Turner 1986). There is also the effect of increasing age and other acquired injuries which will affect performance over time and this is especially important where part of the postoperative treatment involves long periods of rest as is often the case with carpal surgery (Spiers 1983). Martinelli *et al* (1996) demonstrated that there is a natural decrease in performance with age in Standardbred racehorses.

A number of indices have been used to evaluate performance: ability to race and win (Wyburn and Goulden 1974, Kannegieter and Ryan 1991), prize money (Grøndahl and Engeland 1995, Linford *et al* 1993), or position in race (Martin *et al* 1988). In Standardbreds race times and claiming value (Stephens *et al* 1988, Martin *et al* 1988) have also been used. Ability to race and win takes no account of any change in class that the horse may experience. It is also dependant on the will of the trainer to get the horse to the racetrack, which varies depending on the breeding potential of the horse (Martin *et al* 1988). Prize money does give

an indication of both the class of race and position in race, but the large variations in prize money make comparisons difficult (McIlwraith and Turner 1986). Also, data is usually skewed by extremely large amounts of money won by a small number of horses (McIlwraith and Turner 1986). A common method is to classify horses as returning to a level of performance equal to or better than before surgery, or worse than before surgery (Lindsay and Horney 1981, Dabareiner *et al* 1996, McIlwraith *et al* 1987). This is probably the best available method, but although generally determined from race records, often no objective criteria are given for determining performance.

Conclusions

The carpus is a complex joint with multiple articulations and one of its functions is to dissipate axial force. Transverse forces can also be demonstrated at the carpus and how the carpus dissipates these forces requires further investigation. Carpal disease in racing horses is most often due to soft tissue inflammation or fatigue failure of the subchondral bone, particularly of distal CR, radial facet of C3 and the distal radius. Articular cartilage damage in most cases is probably a secondary change. Pathology of the intra-articular ligaments is common in horses with midcarpal joint disease but its significance is not clear. In particular the relationship between ligament pathology and osteochondral damage, and the effect of ligament tearing on performance needs to be investigated. Also the frequency of ligament tearing in the general population of horses is unknown. Investigation of the cause of enlargement of the DMICL of the midcarpal joint and its role in midcarpal joint disease is

also needed.

The diagnosis of carpal disease is primarily based on clinical signs, radiographs and arthroscopic surgery. In some situations scintigraphy and CT scanning may also be useful. Routine synovial fluid analysis adds little information in most degenerative conditions but does indicate the degree of synovitis. Although the measurement of synovial fluid proteoglycans has shown some promise there are questions regarding their use in the clinical situation. Further work is required looking at the effect of training, changes in synovial fluid volume and stage of disease on proteoglycan concentration in synovial fluid. Like any new diagnostic technique their ability to predict articular cartilage damage needs to be compared with the presently available diagnostic techniques.

CHAPTER 2

GENERAL MATERIALS

AND

METHODS

Clinical evaluation of horses admitted for carpal surgery and undergoing training program

History

Owners or trainers were asked how long their horse had shown clinical signs of carpal joint disease. The number of weeks the horse had been in training was recorded as well as the number of days since it had last raced or galloped. If there had been any intra-articular medications, these were recorded as well as the time since the last treatment.

Clinical examination

Conformation abnormalities were assessed visually with the horse standing squarely. Abnormalities, graded according to their severity, were grouped into three categories: carpal hyperextension, valgus and rotational deformity. Where no deformity was evident a score of 0 was given. Slight deformity, where the leg had to be examined closely to detect an abnormality, was given a score of 1. Moderate deformity, one that was obvious to the observer, was given a score of 2, and severe deformities were given a score of 3. Carpal hyperextension was defined as an angle formed by the midline of the radius and metacarpus of greater than 180°. Carpal valgus was defined as lateral angulation of the distal aspect of the metacarpus relative to the midline of the radius when viewed from the cranial aspect, whereas rotational deformity was defined as outward (lateral) rotation of the dorsal aspect of the metacarpus relative to the radius.

All horses were examined at the trot on a firm flat surface. Lameness was graded from 0 to 4

(Stashak 1987). Both forelegs were examined for swelling of the midcarpal and antebrachiocarpal joints with the horse weight bearing, and for pain on flexion of the carpus. Swelling was defined as bulging of the soft tissues over the dorsal aspect of the individual joint and was graded from 0-3 with 0 defined as no detectable swelling, 1 as swelling only evident on close examination, 2 as easily detectable swelling and 3 as marked swelling. Pain on carpal flexion was graded 0 if no pain was detectable, 1 if pain was detectable on extreme flexion, 2 if pain was detected in approximately the last 15° of flexion, and grade 3 if pain was detected before the last 15° of flexion. Lameness evaluation performed after carpal flexion for one minute (flexion test) was graded from 0-2; if there was no increase in lameness, a grade of 0 was given, whereas grade 1 was defined as an increase in lameness of 1 grade after flexion, and grade 2 as an increase of 2 grades. For horses involved in the treadmill training program (Chapter 3) a total lameness score was calculated by adding the four scores determined above together, to determine the overall clinical compromise for each joint.

Radiographic examination

Radiographs of carpi of clinical cases included a flexed lateral, dorsomedial palmarolateral oblique, dorsolateral palmaromedial oblique and flexed dorsoproximal dorsodistal skyline views. All radiographs were assessed for the number, type, position and size of fractures, the degree of subchondral bone lucency and the size, number and extent of enthesiophytes and osteophytes. The radiographic grading system is shown in table 1.

Table 1.

The grading systems used to assess radiographs of diseased carpi.

Score	Osteophytes Enthesiophytes	Subchondral lysis	Fractures
0	none	none	none
1	small and present in only one view	less than 5 mm^2 on one view	single small chip fracture (<5mm wide)
2	large **or** present in more than one view	more than 5 mm^2 or detectable on more than one view	two small chip fractures (<5mm wide) or one large chip fracture (>5mm wide)
3	large **and** present in more than one view	-	three or more chip fractures or a slab fracture

Arthroscopic surgery

Arthroscopic surgery was performed using the method described by McIlwraith (1990). All articular surfaces of the bones of the midcarpal joint were closely examined. Cartilage damage was graded from 0 to 3 according to the area of full thickness cartilage erosion. If no cartilage erosion, either full thickness or partial thickness, was observed the joint was given a grade 0. If any cartilage erosion and less than 1.0 cm^2 full thickness erosion was observed it was given a grade 1. Grade 2 was between 1.0 and 2.5 cm^2 and grade 3 was greater than 2.5 cm^2 full thickness

erosion. After each surgery the areas of damage were drawn on scale diagrams of the articular surfaces and these were compared with sample diagrams on which the above described areas were drawn.

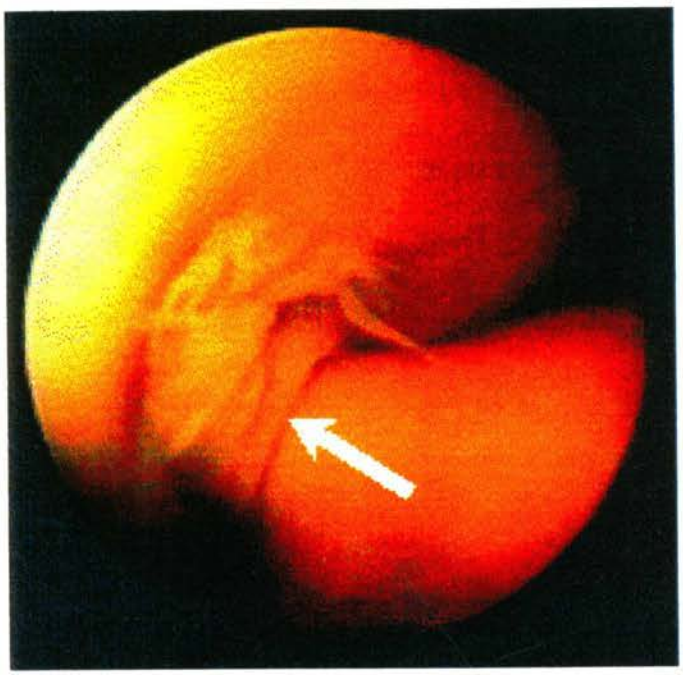

Figure 1.

A left midcarpal joint showing a Grade 3 medial palmar intercarpal ligament tear viewed from the dorsolateral aspect. Between one third and two thirds of the ligament is damaged. Note the intact dorsomedial bundle (white arrow)

Subchondral bone damage was defined as intra-articular fractures or soft subchondral bone that was easily removed with a curette. Any areas of cartilage erosion were gently curetted to determine the firmness of the underlying bone. If soft bone was detected it was curetted back to firm bone. The degree of subchondral bone damage was graded according to the area of joint surface affected as for articular cartilage damage with slight modification. Grade 0 was were no subchondral bone damage was detected. Grade 1 was a single chip or less than 1.0 cm^2 of soft

subchondral bone. Grade 2 was a slab fracture or 1.0 to 2.5 cm^2 of chip fractures and/or soft subchondral bone and grade 3 if more than 2.5 cm^2 soft subchondral bone and or chip fractures.

To assess the palmar intercarpal ligaments the leg was positioned with the carpus in full flexion. Careful probing of the ligaments was then performed to determine the proportion of cross-sectional area intact. The grading system used has been previously described (Kannegieter and Colgan 1993). Mild damage with rupture, fraying or stretching of a small number of fibres was graded as 1; grade 2, included those ligaments with up to one third damaged; grade 3, one to two thirds of the ligament damaged (Figure 1), whereas grade 4 was used for complete rupture of the ligament (Figure 2).

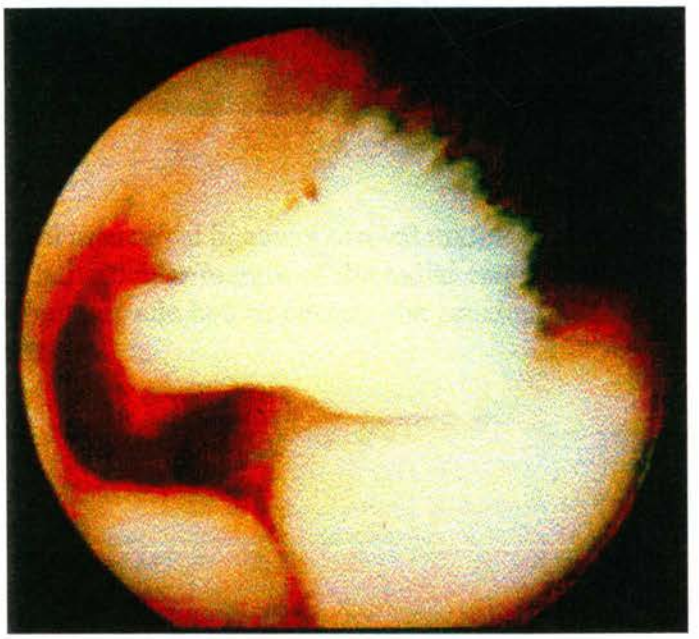

Figure 2.

A left midcarpal joint viewed from the dorsolateral aspect showing a grade 4 medial palmar intercarpal ligament tear. Complete rupture of the dorsal portion of the ligament has occurred.

The intercarpal ligament on the dorsomedial aspect of the midcarpal joint (Selway 1992) was assessed and graded from 0 to 3 based on size. Measurements were estimated by comparison with a graduated probe. If no ligament was detected a grade of 0 was used.

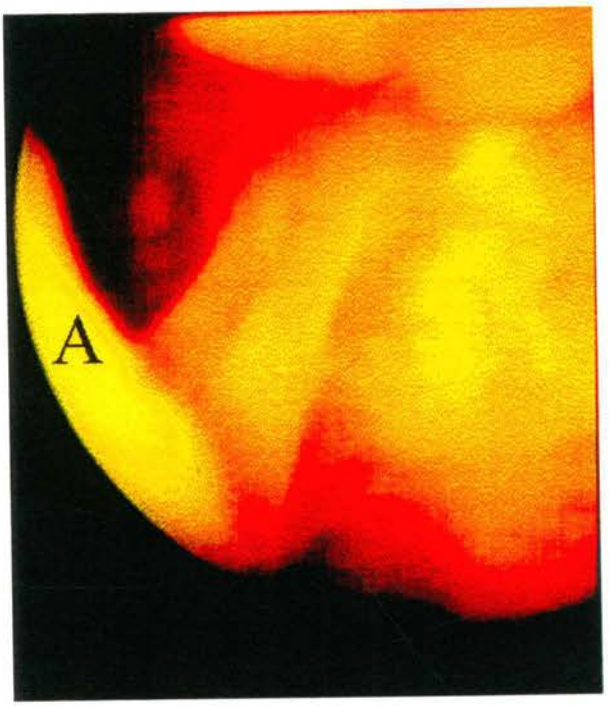

Figure 3.

Grade 1 dorsomedial intercarpal ligament of a left midcarpal joint viewed by looking over the dorsomedial articular margin of the radial carpal bone (A). The ligament blends with the joint capsule before passing the proximal articular margin of the second carpal bone.

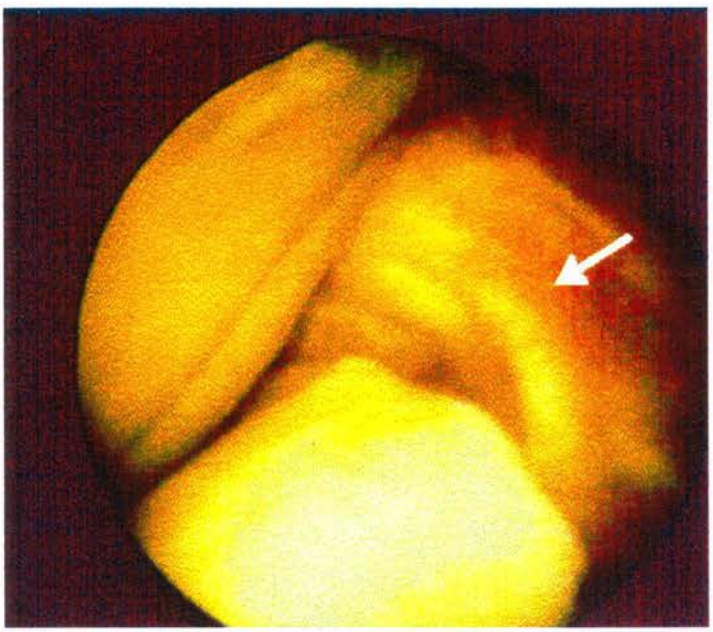

Figure 4.

The dorsomedial aspect of a midcarpal joint showing a grade 2 dorsomedial intercarpal ligament (white arrow). This ligament is easily differentiated from the joint capsule over its whole length.

Ligaments less than 2 mm thick (lateral-medial) that blended into the joint capsule before passing the proximal medial articular margin of the second carpal bone (C2) were graded as 1 (Figure 3), whereas those more than 2 mm thick or easily differentiated from the joint capsule over their whole length were graded as 2 (Figure 4), and ligaments more than 5 mm thick were graded as 3 (Figure 5).

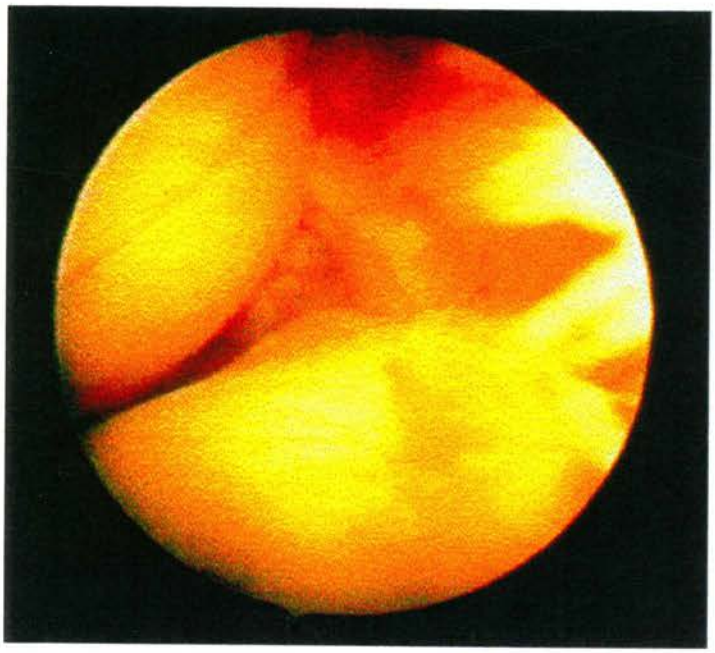

Figure 5.

The dorsomedial aspect of a left midcarpal joint showing an enlarged (grade 3) dorsomedial intercarpal ligament partially obscured by synovial villi.

Synovial fluid collection and storage

The skin over the medial aspect of the midcarpal joint was shaved and prepared in a sterile manner. An 18 g needle was inserted into the midcarpal joint and the maximum amount of synovial fluid was withdrawn without using undue negative pressure. The volume of fluid was recorded and it was then transferred into EDTA Vacutainer tubes.

Synovial fluid analysis.

One ml of fluid was removed for total nucleated cell counts[1] and the remainder centrifuged at 10 000 g for 20 minutes to remove cells and debris. The supernatant was stored in aliquots at -80°C and the sediment smeared for differential cell counts.

Synovial fluid colour was assessed visually and graded according to the degree of xanthochromia. This was done after centrifugation to minimize the effect of blood contamination during collection. Fluid that was clear was graded 0. Grade 1 was defined as the minimal detectable xanthochromia. Grades 2, 3 and 4 were defined as pale straw, medium straw and dark straw respectively, while blood stained fluid was graded 5.

Sulfated glycosaminoglycan (GAG) concentration was measured using the dimethylmethylene blue (DMB) assay. Fifty µl of synovial fluid was added to 450 µl of papain, giving a 1:10 dilution, and incubated at 65°C for two hours. Ten µl of this solution was added to 200 µl of Farndale's reagent and absorbence at 525 nm read immediately. The assay was calibrated with chondroitin sulphate. Total sulfated GAG content of the joint fluid was calculated by multiplying the volume of synovial fluid by the sulfated GAG concentration.

Synovial fluid protein concentration was determined by the biuret method (Unimate 7, Roche). Absorbence was measured at 546 nm using a Cobas Mira spectrophotometer. Lactate dehydrogenase (LDH) activity was measured using pyruvate as the (Trace Scientific Pty Ltd).

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[1]Unopipette method

Absorbence was measured at 340 nm. Alkaline phosphatase activity was determined using the IFCC method with absorbence measured at 405 nm (Trace Scientific Pty Ltd).

Carpal dissection technique

Joints were stored at -10°C until required. After thawing overnight, detailed dissections of the midcarpal joint were performed, paying particular attention to the MPICL and LPICL, and the structure variously described as a normal synovial plica (McIlwraith 1990) and an intercarpal ligament, (Selway 1991, Martin and McIlwraith 1985) located on the dorsomedial aspect of the joint.

After skin removal, the tendons of the extensor carpi radialis, common digital extensor, extensor carpi obliquus and lateral digital extensor muscles were dissected free and discarded. The dorsal joint capsule was removed as far as the collateral ligaments laterally and medially, and carefully separated from the DMICL. Any tearing of the intra-articular portion of the palmar intercarpal ligaments was noted, as was cartilage damage within the joint. The palmar carpal ligament was carefully dissected free of its attachments to the palmar aspects of the carpal bones. Then the joint was passively flexed and extended and the behaviour of the intercarpal ligaments recorded. The radius was then removed by transection of the joint capsule and the ligaments of the antebrachiocarpal joint. Dissection was continued through the intercarpal ligaments attaching the intermediate carpal bone (CI) to the radial carpal bone (CR) and ulnar carpal bone (CU), taking care to avoid damage to the palmar intercarpal ligaments. Any attachment of these ligaments to

CI was recorded before its removal. The palmar intercarpal ligaments could now be observed and their attachments and dimensions recorded.

Further observations were made of the palmar intercarpal ligaments with all other restraining attachments to CU and CR removed to determine their effect on the restraint of these bones in the extended position. The normal position of these bones with the joint extended was determined before removal of the radius. Dimensions of the ligaments were measured with a digital caliper (Mitutoyo, Series 500, Tokyo, Japan) while the ligaments were under moderate tension. Cross-sectional measurements were made at the midpoint of the ligaments.

Treadmill training program

Thirteen Standardbred horses between four and five years of age that were free from lameness and signs of midcarpal joint disease and had been out of training for at least four months underwent a graded treadmill training program (Table 2). This training program was part of a much larger study into the effects of overtraining on horses. Also in further studies the articular cartilage from the mid carpal joints was analysed biochemically at the end of the training protocol. The experimental protocol was approved by the Animal Care and Ethics Committee of the University of Sydney. The program commenced with an endurance phase lasting seven weeks (phase 1), followed by a high intensity phase of eight weeks (phase 2). The horses were then randomly divided into two groups, one of six and one of seven (phase 3). One group (group 1) continued on a training program in which the duration and intensity of exercise was increased

every four weeks. The second group (group 2) were subjected to a much more intense training program with increases in duration and intensity every two weeks. The duration of phase 3 was 16 weeks. All training sessions were conducted with the treadmill (Mustang, Kagra, Sweden) on a 10% slope. At the end of the training program the horses were rested for 12 weeks, with only walking exercise twice a week (phase 4).

Table 2

An overview of the 31 week treadmill training program. * This speed corresponds to the speed at which each horse reached maximum oxygen uptake.

Training Phase		Duration	Type of training session	Number of days per week	Distance per session	Speed at 10% slope
1 (13 horses)		7 weeks	Low intensity	5	2000 - 4000 m	6 m/s
2 (13 horses)		8 weeks	Low intensity	3	4000 m	8 m/s
			High intensity	2	2500 - 4000	9.8-11.6 m/s*
3	Group 1 (6 horses)	16 weeks	Low intensity	3	Increased by 500 m every 4 weeks	8 m/s
			High intensity	2	Increased by 600 m every 4 weeks	9.8-11.6 m/s*
	Group 2 (7 horses)	16 weeks	Low intensity	3	Increased by 500 m every 2 weeks	8 m/s
			High intensity	2	Increased by 600 m every 2 weeks	9.8-11.6 m/s*

During phase 1 the horses were trained three days a week, commencing with a session of 1000 m at 4 m/s followed by 2000 m at 6 m/s, increasing up to 1000 m at 4 m/s and 4000 m at 6 m/s.

During phase 2 horses were trained five days a week with three low intensity sessions and two high intensity sessions. The low intensity sessions increased from 1000 m at 4 m/s and 3000 m at 8 m/s, to 1000 m at 4 m/s and 4000 m at 8 m/s. During high intensity sessions the horses were exercised at the speed at which they reached maximum oxygen uptake (VO_{2max}) for two periods of 2 min, following a warm up of 1000 m at 4 m/s. This was increased to three periods of 2 min at 100% of VO_{2max} by the end of the high intensity phase. The speed at VO_{2max} ranged from 9.8 m/s to 11.6 m/s for all horses.

During the first 11 weeks of phase 3 both groups were exercised five days a week. Both groups underwent two low intensity and three high intensity training sessions a week. For group 1, low intensity training sessions were increased by 500 m every four weeks, while the high intensity training sessions were increased by 1 minute every four weeks. For group 2, low intensity training sessions were increased by 500 m every two weeks and the high intensity training sessions by 1 min every two weeks. The number of high intensity training sessions performed by group 2 was increased to four per week in the last six weeks of phase 3. During the high intensity training sessions group 2 horses were run at 110% VO_{2max} until they could no longer keep pace with the treadmill. These sprints were repeated until their duration was less than 30 seconds. Total sprinting times ranged from five to 12 minutes.

During phase 4 horses were kept in small yards or stables and were not exercised. However, testing runs, in which the horses were run to fatigue, were performed every two weeks, with the last test performed seven days before sampling.

CHAPTER 3

THE EFFECT OF PROLONGED TRAINING ON SYNOVIAL FLUID VARIABLES OF THE EQUINE MIDCARPAL JOINT

Introduction

Synovial fluid analysis has been well described as an aid in the diagnosis of joint disease in the horse (Nilsson and Persson 1973, Van Pelt 1974, Tew 1982 and Moyer 1982). Van Pelt (1974) described the synovial fluid variables that can be measured and their normal values, while Moyer (1982) described the changes that occur with various disease states. It has also been shown that findings from horses in training differ from resting horses (Persson 1971, Yovich *et al* 1993), therefore an understanding of the response of synovial fluid variables to training is important when interpreting findings.

It is generally accepted that predicting the degree of joint compromise, particularly articular cartilage damage, from routine synovial fluid evaluation is unreliable (Moyer 1982, Dyson 1987). In an attempt to find a more accurate indicator of the state of articular cartilage, markers of articular cartilage metabolism have been investigated. Articular cartilage is comprised of proteoglycans embedded in a network of type II collagen fibres. Loss of proteoglycans from the tissue is one of the hallmarks of early degenerative change. Quantification of synovial fluid proteoglycan levels therefore has been proposed as a method of evaluating the biochemical integrity of articular cartilage. In previous studies in the horse, comparisons of synovial fluid proteoglycan concentration between normal joints and those with overt signs of degenerative joint disease have not shown consistent differences (Little *et al* 1990, Alwan *et al* 1990, and Yovich *et al* 1991). Alwan *et al* (1990) demonstrated an increase in sulfated GAGs and keratan sulphate in horses with clinical signs of joint disease, while Yovich *et al* (1991) could find no significant difference. Little *et al* (1990) found there

was no correlation between sulfated GAG levels and the degree of cartilage erosion. One of the reasons for this may be that the horses were exercising at different levels. It has been demonstrated that synovial fluid keratan sulphate levels increased after a single period of exercise in the horse and that horses in training have higher concentrations than untrained horses (Yovich *et al* 1993). Therefore to allow better interpretation of proteoglycan levels in individual cases normal values need to be established for different intensities and durations of exercise. The effect the intensity and duration of training has on the release of proteoglycans into synovial fluid has not previously been investigated.

The purpose of this study was to define the changes that occur in routinely measured synovial fluid variables, as well as sulfated GAG levels, with various levels of training. This may indicate why others have reported inconsistent results with proteoglycan markers (Little *et al* 1990, Alwan *et al* 1990, and Yovich *et al* 1991). Also, as it was anticipated that some signs of midcarpal joint disease would develop due to the intensity of the exercise, the association between the early signs of joint disease and synovial fluid findings were also examined.

Materials and methods

Synovial fluid samples were collected from left and right midcarpal joints of all horses before the exercise program commenced, at the end of phase one, at the end of phase two, and at six weeks, 11 weeks and 15 weeks into phase three, as well as eight weeks after training was discontinued. All samples during the training program were taken within four hours of a low

intensity training session. Methods of synovial fluid collection, storage, and analysis are described in Chapter 2.

All horses were examined for lameness at the beginning of the training program, following phase two, and at sampling times during phase three. The method of examination and the grading system used are detailed in Chapter 2. To determine the overall clinical compromise for each joint a total lameness score was calculated by adding together the four scores determined. If lameness could be clinically attributed to another cause it was not included in the total score. Horses were rested if lameness was graded greater that one out of four in any limb.

Eleven weeks after training was discontinued 11 of the horses were euthanased and their midcarpal joints dissected and examined for cartilage and intercarpal ligament damage. The grading systems for intercarpal ligament tearing and dorsomedial intercarpal ligament hypertrophy are described in Chapter 2. Full thickness articular cartilage was collected from the radial and intermediate facets of the third carpal bone and its palmar condyle. These were fixed in 10% neutral buffered formalin for a maximum of 24 hours at room temperature. Osteochondral slabs approximately 5 mm in width and 5 mm deep, extending 10 mm from the joint margin were removed from the radial facet of the third carpal bone of horses in both groups for histological examination. These were decalcified for 72 hours in 5% formic acid and 5% formalin with continuous agitation. Full thickness articular cartilage was collected form the radial and intermediate facet of the third carpal bone and its palmar condyle. These were fixed in 10% neutral buffered formalin for a maximum of 24 hours at room temperature.

Deparaffinized chondral and osteochondral sections were stained with toluidine blue O and counterstained with fast green as described by Getzy *et al* (1982).

Statistical analysis

Data from horses that were resting due to lameness was excluded. Data were analysed by repeated measures analysis of variance with time the repeated measures factor. Where the data was not of normal distribution log transformation was performed. When the F value was significant a post hoc test of least significant difference was performed. A Mann-Whitney U test was used to compare sulfated GAG concentrations and total sulfated GAGs between groups one and two at sampling times during phase three. Spearman rank order correlations were used to compare lameness scores and synovial fluid values and the level of significance was set at $p<0.05$.

Results

Twelve of the 13 horses were able to complete the training program, while three horses underwent low intensity training for varying periods during the training program due to lameness that did not originate from the carpus. Horse 11 failed to complete training due to a hindlimb lameness that developed during the last five weeks of phase three. During phase three, the training program of horses four, seven and eight were limited to low intensity sessions for eight, five and two weeks respectively, due to hindleg lamenesses. Results from

two synovial fluid samples were disregarded for horse four, while one sample was disregarded in each of horses seven and eight. All of these horses were in group two and were treated with short courses of phenylbutazone and subsequently returned to high intensity training.

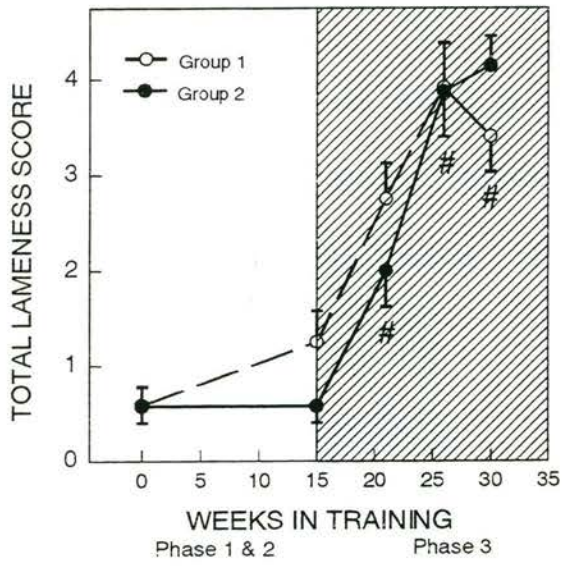

Figure 1.

Total lameness score (mean±sem) during training. Shaded area represents phase 3, where the intensity and duration of training for group 2 was increased at a greater rate than in group 1. Significant differences ($p<0.000001$) from pretraining denoted by #.

Lameness examinations

Signs of midcarpal joint disease developed in all joints during training, evidenced by a significant increase in total lameness scores from the beginning to the end of the training program ($p<0.001$) (Figure 1), although overt lameness was never graded more than one out

of four. There was no significant difference between groups one and two. Synovial fluid variables that significantly correlated with lameness score were TP ($R=0.67$, $p<0.00001$), LDH ($R=0.58$, $p<0.00001$) and degree of xanthochromia ($R=0.53$, $p<0.00001$).

Effect of training on synovial fluid variables

Synovial fluid volume changed significantly with training ($p<0.01$) (Figure 2), but there was no significant difference between groups one and two. The mean difference between left and right joints of individual horses at each sampling time was 0.76 ml.

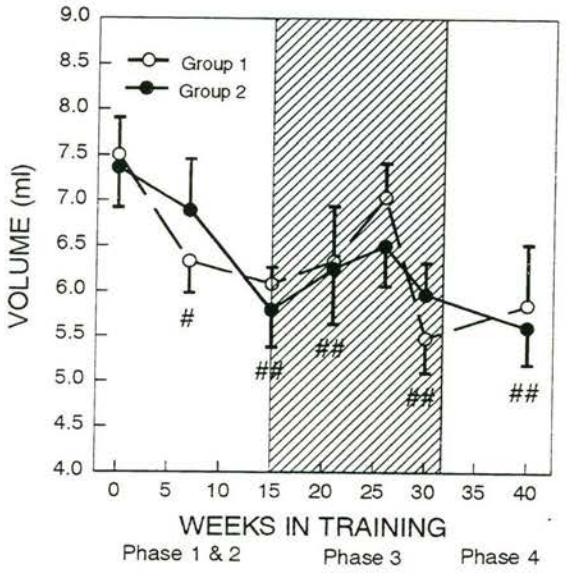

Figure 2.

Synovial fluid volume (mean±sem) of the midcarpal joint during training. Shaded area represents phase 3, where the intensity and duration of training for group 2 was increased at a greater rate than in group 1. Significant differences from pretraining denoted by # ($p<0.01$), and ## ($p<0.0001$).

There was a significant effect of both training ($p<0.001$) and group ($p<0.05$) on synovial fluid colour with the scores of group two higher than group one during phase three (Figure 3). Synovial fluid leukocyte counts remained low throughout the study and there was no significant change with training and no significant difference between groups one and two (Figure 4). Synovial fluid TP changed significantly with training ($p<0.001$) and there was a significant difference between groups ($p<0.01$) (Figure 5).

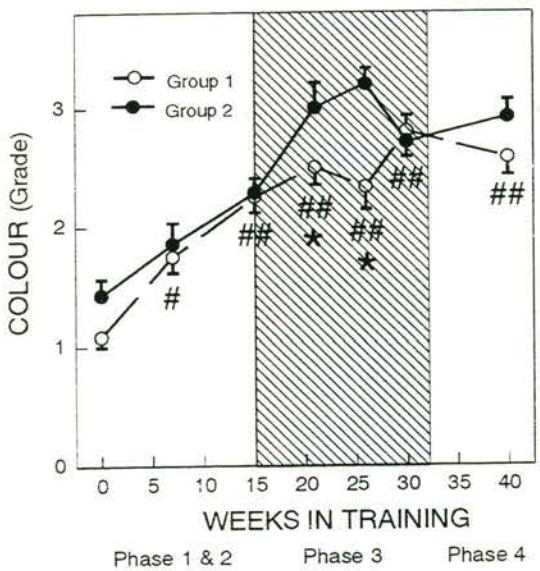

Figure 3.

Synovial fluid colour (mean±sem) as assessed visually during training. Shaded area represents phase 3, where the intensity and duration of training for group 2 was increased at a greater rate than in group 1. Significant differences from pretraining denoted by # ($p<0.0001$), and ## ($p<0.000001$). Significant differences between group 1 and group 2 denoted by *.

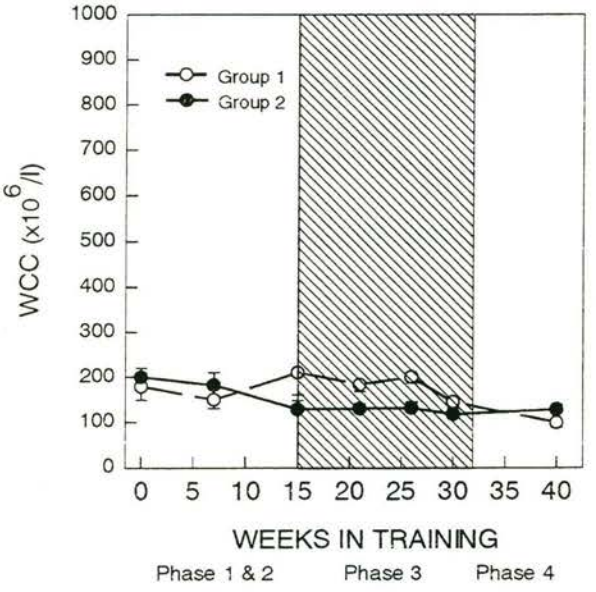

Figure 4.

Synovial fluid total leukocyte count (mean±sem) during training. Shaded area represents phase 3, where the intensity and duration of training for group 2 was increase at a greater rate than in group 1.

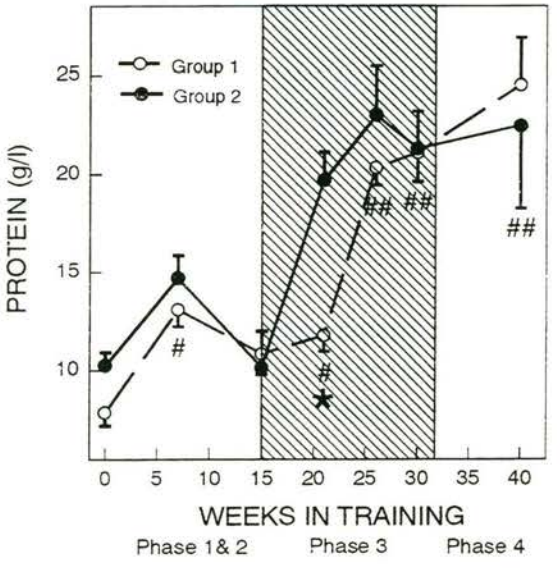

Figure 5.

Synovial fluid protein concentration (mean±sem) during training. Shaded area represents phase 3, where the intensity and duration of training for group 2 was increased at a greater rate than in group 1. Significant differences from pretraining denoted by # ($p<0.05$), and ## ($p<0.0001$). Significant differences between groups 1 and group 2 denoted by *.

There was a significant effect of training on synovial fluid LDH activity ($p<0.001$), but there was no significant effect of group (Figure 5). There was no significant change in synovial fluid AP concentrations with training, but there was a significant increase with resting during phase four ($p<0.001$). There was no significant difference between groups one and two (Figure 6).

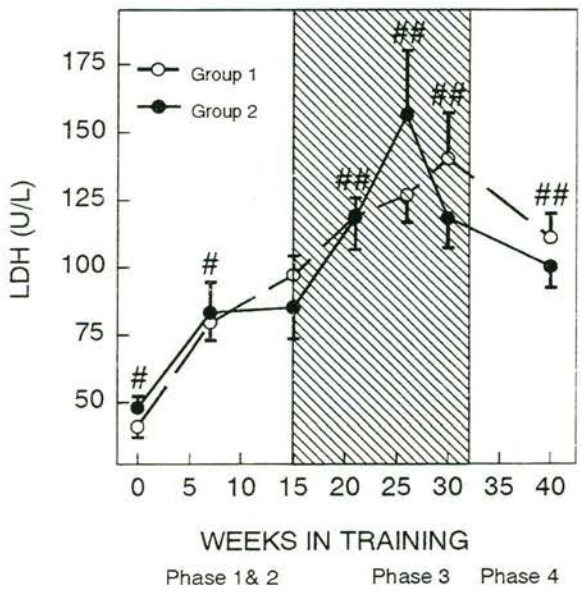

Figure 6.

Synovial fluid LDH activity (mean±sem) during training. Shaded area represents phase 3, where the intensity and duration of training for group 2 was increased at a greater rate than in group 1. Significant differences from pretraining denoted by # ($p<0.01$), and ## ($p<0.000001$).

Sulfated GAG concentrations changed significantly with training ($p<0.001$) but overall there was no significant difference between groups one and two. However after six weeks of phase three there was a significant difference between groups one and two ($p<0.01$) (Figure 7). There was also a significant effect of training on total sulfated GAGs ($p<0.0001$) (Figure 8).

Although there was no overall difference between groups one and two, total sulfated GAG levels were significantly higher in group one at the end of phase one and phase three ($p<0.05$), while they were significantly higher in group two after six weeks of phase three ($p<0.05$)

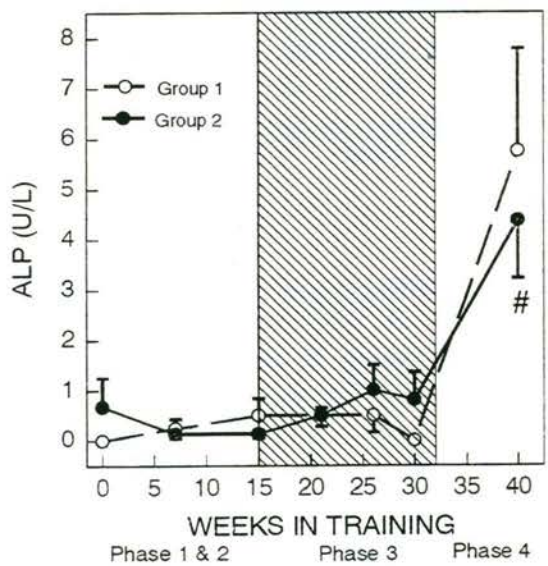

Figure 7.

Synovial fluid AP activity (mean±sem) during training. Shaded area represents phase 3, where the intensity and duration of training for group 2 was increased at a greater rate than in group 1. Significant differences from pretraining denoted by # ($p<0.0001$).

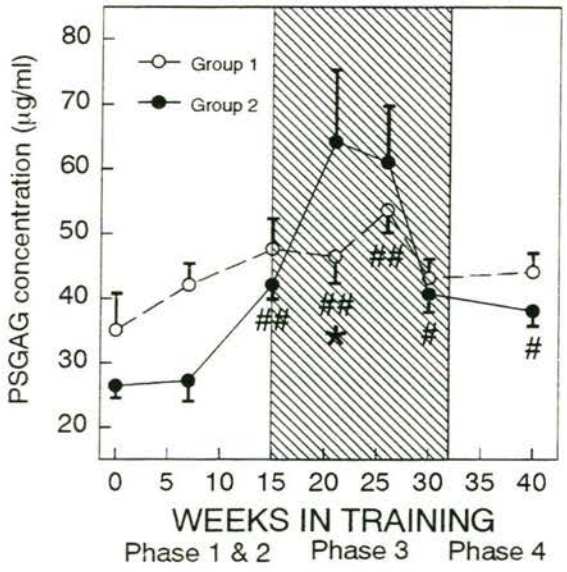

Figure 8.

Synovial fluid sulfated GAG concentration (mean±sem) during training. Shaded area represents phase 3, where the intensity and duration of training for group 2 was increased at a greater rate than in group 1. Significant differences from pretraining denoted by # ($p<0.05$), and ## ($p<0.00001$). Significant differences between groups 1 and group 2 denoted by *.

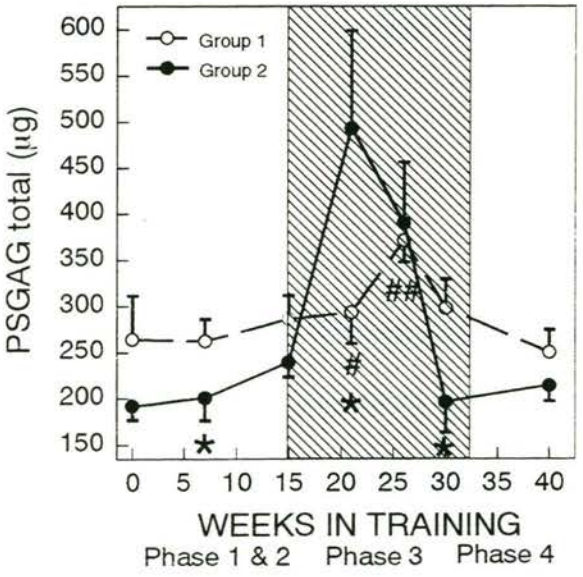

Figure 9.

Synovial fluid total sulfated GAGs (mean±sem) during training. Shaded area represents phase 3, where the intensity and duration of training for group 2 was increased at a greater rate than in group 1. Significant differences from pretraining denoted by # ($p<0.05$), and ## ($p<0.001$). Significant differences between groups 1 and group 2 denoted by *.

Gross pathology and histopathology at postmortem

Postmortem examination of the midcarpal joints of 11 of the horses in the study revealed only minor articular cartilage damage. In horses of both groups there were focal areas (1 mm diameter) of articular cartilage erosions on the radial facet of C3. There was some MPICL tearing in 13 of the 22 joints examined. Grade three tears were found in three joints, grade two tears in seven joints and grade one tears in five joints. Slight fraying of the LPICL was observed in five joints. There was no significant difference in the degree of intercarpal ligament damage between groups one and two. Histological examination of osteochondral segments demonstrated that the cartilage lesions described above were merely depressions overlying collapsed subchondral bone. All articular cartilage sections showed normal architecture and toluidine blue staining. In all samples of synovium the intimal layer was one to two cells thick and very few inflammatory cells were observed.

Correlations between variables

There was a significant but weak correlation between sulfated GAG concentration in synovial fluid and the severity of response to flexion tests ($R=0.19$, $p<0.05$). There was also a trend for legs with higher total lameness scores (all clinical variables added together) to have higher sulfated GAG concentrations ($R=0.18$, $p=0.06$) and legs with higher lameness scores to have higher synovial fluid total sulfated GAGs ($R=0.17$, $p=0.06$). Synovial fluid sulfated GAG concentration was also significantly correlated with the degree of synovial fluid xanthochromia ($R=0.25$, $p<0.001$), TP ($R=0.17$, $p<0.05$), and LDH activity ($R=0.15$, $p<0.05$).

Synovial fluid variables which were significantly correlated with total lameness score were TP ($R=0.67$, $p<0.00001$), LDH ($R=0.58$, $p<0.00001$), and colour ($R=0.53$, $p<0.00001$). Synovial fluid TP was also significantly correlated with the degree of midcarpal joint swelling ($R=0.52$, $p<0.0001$). There was a strong correlation between the sulfated GAG concentrations of left and right midcarpal joints throughout the study ($R=0.72$). This was also the case during phase three ($R=0.60$), despite there being no significant correlation between total lameness scores of left and right legs for this same period ($R=0.15$).

Discussion

Training program

This appears to be the first longitudinal study of synovial fluid variables in horses in training. The horses were trained continuously for 31 weeks which is more prolonged than the training program of many normal racing horses. The intensity of exercise was gradually increased throughout the program and, for the horses in group two, the intensity of the final phase was far in excess of that to which a normal racehorse is subjected. A standard training program of an average Thoroughbred racehorse would be roughly equivalent to the first 16 weeks of this training program, while a Standardbred racehorse is likely to undergo more prolonged but less intense training (Evans 1994, Lovell 1994). Therefore the changes in synovial fluid variables observed in phase three may not reflect normal responses to exercise, but those that occur in the joint under excessive stress.

Synovial fluid volume

Even though it is impossible to withdraw all the synovial fluid form the joint cavity, aspiration of the maximum volume was used to give a guide to trends in synovial fluid volume. In this study a large diameter needle was used and it was passed deep into the joint to minimise the chance of blockage with synovial villi. The accuracy of volume estimation by this method has been shown to be dependant on the volumes retrieved. A mean error of 10% for volumes over 10 ml, but up to 48% for volumes less than 10 ml has been demonstrated in humans (Geborek *et al* 1988). Mean volume in this study ranged form 5.5-7.5 ml which is similar to previous studies (Van Pelt 1962). Despite large variations in volume throughout the study, volumes in individual horses varied very little between left and right joints suggesting that this technique may provide a reliable measure of synovial fluid volume. More accurate techniques involve measuring the dilution of various substances (Wallis et al 1985). This was not practical in the present study as the effects of these compounds on the synovial membrane is unknown.

The changes in synovial fluid volume with training were complex. Previous work has demonstrated that a single period of exercise has either no effect on synovial fluid volume (Yovich *et al* 1993), or increases synovial fluid volume (Persson 1971), but the techniques used were based on the assumption that lymphatic drainage does not increase with exercise. Research in other species suggests that it will (Levick 1987). The movement of fluid into and out of the joint cavity is considered a passive process (McDonald and Levick 1993). Volume is determined by both oncotic and hydrostatic pressure, as well as the distensibility of the

joint capsule and the rate of lymphatic drainage (Levick 1987, Jayson and Dixon 1970). Exercise has been shown to increase hydrostatic pressure (Jayson and Dixon 1970) in joints as well as improving lymphatic drainage, and decreasing venous pressure, which reduces filtration across the synovial membrane (Levick 1987). Synovial fluid oncotic pressure is primarily determined by the albumin concentration (McDonald and Levick 1993) which, based on TP concentration, changed throughout the training program..

Synovial fluid colour

Xanthochromia is caused by the accumulation of haemoglobin in the fluid and indicates previous haemorrhage into the joint space (McIlwraith 1987). It was decided to determine the degree of xanthochromia visually, as is done by most practitioners, although it would be more accurate to determine haemoglobin concentration in the fluid by spectrophotometry as described by Harboe (1959). There was a consistent increase in xanthochromia of the synovial fluid samples with exercise and a significant difference between group 1 and group 2. Some of the increase may have been due to changes in volume as there was a significant, although weak, negative correlation between volume and colour. All joints were repeatedly sampled, so it is possible that haemorrhage at the time of previous sampling caused an increase in xanthochromia. However, there was a significant difference between groups 1 and 2 during phase 3, and these groups were sampled with the same frequency. Also there was no increase in xanthochromia during the last nine weeks of phase 3 when sampling frequency was at its highest. The possible sources of haemorrhage are the synovial membrane, intra-articular ligaments or subchondral bone. Subchondral bone was not a source in this study as

articular cartilage was intact in the joints examined at post mortem.

Synovial fluid nucleated cell counts

The results of this study confirms that training has no effect on nucleated cell numbers (Persson 1971). This was despite the development of swelling and pain in most joints during very high intensity exercise in the present study. Most samples had nucleated cell counts less than 200×10^6/ml which is comparable to the findings of Tew (1982) in normal racing horses. The lack of correlation with synovial fluid protein and the degree of swelling indicates that nucleated cell numbers are an insensitive indicator of low grade synovitis. This is consistent with the findings of others who have stated that increases in nucleated cell counts only occur with more severe synovitis (Persson 1971).

Synovial fluid total protein

Slightly higher synovial fluid TP concentrations have been observed in racing horses in training compared to those that were resting (Persson 1971, Tew 1982). TP concentrations in horses in training ranged from five to 12 g/l. Rose and Paris (1979) reported total protein concentrations of 13.84±4.2 g/l for normal Thoroughbreds in training. This is slightly higher than the levels measured during phase one and two in both groups, and in group one during the first six weeks of phase three. At these times the horses were exercising at a similar level to normal horses in training. Again, repeated sampling may have contributed to the increase in synovial fluid protein concentration during the training program, but there was a

significant difference between groups one and two which were sampled at the same frequency. Mean TP concentrations of 17±8 g/l have been reported in joints with synovitis (Nilsson and Persson 1973) while Tew (1982) suggested that more that 15 g/l meant active synovitis. The question remains whether exercise induces a physiologic release of protein into the synovial fluid or causes synovitis and subsequent increased vascular permeability.

Synovial fluid enzymes

Enzymes are sensitive indicators of cell damage. Changes in cell membrane permeability can cause elevations, particularly those which are highly soluble such as LDH. Both LDH and AP are found in almost all tissues of the body (Rejnö 1976). Therefore sources of these enzymes in the synovial fluid include chondrocytes, synoviocytes and plasma. Increased LDH concentrations may occur by leakage of plasma or release of the enzyme from synovial tissue. Increased xanthochromia and protein suggest that leakage of plasma occurred. There was however no corresponding increase in synovial fluid AP concentration. Therefore, either AP is much more rapidly mobilised from the synovial cavity or the synovial membrane is not the primary source. Except for the pretraining sample, mean LDH activity was higher than recorded by Rose and Paris (1979) in the midcarpal joint of Thoroughbreds in training. Comparisons of LDH levels between joints with intra-articular lesions and normal joints have demonstrated mixed results (Rejnö 1976, Yancik *et al* 1987). For this reason it has been difficult to determine reference values for LDH (Yancik *et al* 1987). AP concentrations in this study were much lower than those observed by Rose and Paris (1979).

Clinical signs of joint disease

During phase 3, clinical signs of carpal joint compromise, although mild, developed in the majority of horses. The synovial fluid variables which were significantly correlated with these signs (i.e. total lameness score) were synovial fluid colour, protein and LDH. Protein leakage and haemorrhage from synovium is the obvious source of these changes. However, as the most significant pathology observed at post mortem in these horses was intercarpal ligament tearing, it is possible that tearing of these ligaments and their overlying synovium, with subsequent intra-articular haemorrhage was another source of synovial fluid changes.

Role of synovitis in joint disease

Synovitis has been incriminated in the pathogenesis of equine degenerative joint disease (Tew 1982, Spiers *et al* 1994). Inflamed synovium is a rich source of proteolytic enzymes which are active against cartilage proteoglycans (Spiers *et al* 1994) and cytokines such as interleukin-1 and tumour necrosis factor (Brown *et al* 1987) which have been shown to stimulate the production of the metalloproteinases. The metalloproteinases have in turn been incriminated in the breakdown of cartilage proteoglycan (Pelletier *et al* 1988). Despite this, both Little *et al* (1990) and Alwan *et al* (1990) could find no relationship between indicators of synovial membrane inflammation and proteoglycan fragments in synovial fluid in horses with degenerative joint disease. Both these studies involved cases with advanced osteoarthritis. In contrast in this study of normal joints undergoing treadmill training a correlation between synovial fluid protein concentration and sulfated GAG concentration was

observed, although it is acknowledged that it was weak. As stated previously, whether the increase in synovial fluid TP reflects synovial inflammation is unknown. The association between TP concentration and the severity of joint swelling suggests that synovitis occurred in these horses. There was no histopathological evidence of synovial inflammation at the conclusion of the study but the horses had not been trained for 11 weeks. Synovial fluid TP concentrations were still elevated at this time so either synovial histopathology is also an insensitive indicator of low grade synovial inflammation or elevations in TP were not due to synovitis. There was however clinical evidence of synovial inflammation.

There is also the possibility that the inflamed synovium releases its own sulfated GAGs into the joint fluid. Carroll (1989) and Ratcliffe *et al* (1988), in the human knee, and Alwan *et al* (1990) in equine joints showed that sulfated GAG concentration was strongly correlated with proteoglycans of cartilage origin in the synovial fluid, indicating that proteoglycans from the synovial membrane were of only minor importance.

Synovial fluid sulfated GAGs

In this study sulfated GAG concentration in synovial fluid initially increased with moderate and high intensity training levels, then decreased with prolonged training at very high intensity, and the response of sulfated GAG concentration and an estimate of total sulfated GAG content were very similar. The changes in response to exercise was most marked in group two but was also observed in group 1.

Total sulfated GAGs were calculated in an attempt to improve the correlation between sulfated GAG levels and articular cartilage damage with a simple method that could be used in the field. Despite apparently large changes in synovial fluid volume there appeared to be little advantage in calculating total sulfated GAG levels as described.

A number of factors other than exercise may have affected sulfated GAG concentrations. The effect of repeated synovial fluid sampling on sulfated GAG concentration is likely to be small as it has been shown that the removal of synovial fluid from human knees had no effect on subsequent proteoglycan concentrations (Dahlberg *et al* 1991). It is also possible that intraarticular haemorrhage at the time of sampling may affect sulfated GAG release from cartilage. It has been demonstrated that a single episode of haemarthrosis caused proteoglycan degradation in rat knees, but this was limited to seven days post injection (Niibayashi *et al* 1995). The interval between sampling in the present study was never less than four weeks.

Yovich *et al* (1993) also showed that keratan sulphate concentration increases immediately after exercise. It is thought that this is due to the augmentation of proteoglycan molecule transport out of the cartilage matrix by mechanical stimulation (Dahlberg *et al* 1992). However exercise in humans has no effect on synovial fluid proteoglycan concentration (Dahlberg *et al* 1992). To standardise any immediate effect of exercise, all samples in the present study, except the pretraining and detraining samples, were taken at a similar time following a low intensity exercise session.

Synovial fluid sulfated GAG concentration is determined by the total mass of sulfated GAG

present in the joint, the metabolic activity of chondrocytes (Dahlberg *et al* 1992), the rate of clearance of sulfated GAGs from the joint cavity (Page-Thomas *et al* 1987) and the activity of degradative enzymes in the synovial fluid (Lohmander *et al* 1992). The effect of exercise on the clearance rate of sulfated GAGs from the synovial cavity is unknown. However it has been shown to be unchanged by experimentally induced synovitis in rabbits (Page-Thomas *et al* 1987). Exercise has been shown to stimulate articular cartilage proteoglycan synthesis in horses (Palmer *et al* 1995). If, as has been shown by Yovich *et al* (1993), exercise stimulates the release of sulfated GAGs from the cartilage, then it is likely that synovial fluid sulfated GAG concentration will increase with moderate exercise as observed in the present study. However, strenuous exercise (20 and 40 km/day) reduces cartilage thickness and proteoglycan content of cartilage in dog stifle joints (Kiviranta *et al* 1992, Arokoski *et al* 1993). Therefore the decrease in synovial fluid sulfated GAG concentration observed after prolonged training at high intensity in the present study, could be explained by a depletion in the proteoglycan content of the articular cartilage and therefore the proteoglycan available to be released into the joint. The normal toluidine blue staining of the cartilage at the end of the study indicates that if this occurred cartilage proteoglycan levels returned to normal during the rest period.

The good correlation of sulfated GAG concentrations between left and right joints is probably because articular cartilage from joints in the same horse is subject to very similar biomechanical and biochemical environments. This relationship continued in phase 3 despite many horses developing unilateral lameness. Morris and Seeherman (1987) demonstrated increased ground reaction forces in the sound limb of horses with carpal lameness. It is

therefore possible that proteoglycan metabolism will be more greatly affected in the sound limb. However, most of the changes they observed were in horizontal rather than vertical ground reaction force. Vertical force would have the greatest effect on articular cartilage (Radin *et al* 1984). There is also the possibility of some central control of chondrocyte metabolism. Hormones such as the corticosteroids have been shown to influence cartilage proteoglycan metabolism (Pelletier *et al* 1994). However to my knowledge levels of endogenous hormones have not been measured in the synovial fluid of horses.

Changes in sulfated GAGs with early signs of joint disease

One of the aims of this study was to determine if sulfated GAG concentration changed with the early signs of joint disease. For this purpose total lameness scores were used to give a measure of overall joint compromise. Obviously lameness, and to some extent pain on carpal flexion and carpal flexion tests, are not specific for the midcarpal joint, but radiocarpal swelling was not observed in these horses and other causes of lameness were ruled out. There was no relationship between sulfated GAG concentration and total lameness score, but there was a correlation with lameness alone, as well as the results of flexion tests. Clearly there are forms of joint compromise which do not involve articular cartilage such as inflammation of the joint capsule and intercarpal ligaments. This was highlighted by the large number of horses with intercarpal ligament tearing at the conclusion of the study. It was not possible to determine if ligament tearing occurred during the study as the joints were not examined arthroscopically at the commencement of training. Also the large changes in sulfated GAG levels due to training were superimposed on those due to pathology. However these results

suggest that in some cases with early signs of joint disease there is an increase in synovial fluid sulfated GAG concentration. However, the fact that sulfated GAG concentrations rise with both training and disease, indicates that the measurement of sulfated GAG concentration is unlikely to be a useful clinical indicator.

Conclusion

This study has demonstrated a number of changes in synovial fluid variables in response to training. The large changes in sulfated GAG concentrations with training and the large variation between individuals indicates that the measurement of sulfated GAG concentration is unlikely to be a useful clinical indicator of early articular cartilage degeneration. It also highlights the need for the effect of exercise to be examined when investigating other potential markers of the status of articular cartilage.

CHAPTER 4

CLINICAL ASPECTS OF CARPAL DISEASE IN RACING HORSES, A PROSPECTIVE STUDY

Introduction

Intra-articular lesions of the antebrachiocarpal and midcarpal joints are common causes of lameness in racing horses. Unfortunately prediction of the extent or severity of these lesions without direct examination can be difficult. One report indicated that there was only a weak correlation between radiographic and arthroscopic assessment of osteoarthritis (Kannegieter and Burbidge 1990). Also, arthroscopic examination of the carpal joints has resulted in lesions being recognised that were not apparent prior to surgery (Moore and Schneider 1994). Despite this there is evidence that the prediction of intra-articular pathology may be improved by more careful interpretation of subtle radiographic changes (Dabareiner *et al* 1996, Moore and Schneider 1994)

Better assessment of the extent of joint pathology has been a hope of those who have investigated the use of synovial fluid analysis (Van Pelt 1974, Moyer 1982). The main use of this technique is as an indicator of the degree of synovitis (Stashak 1987), but synovial fluid analysis is unreliable as a guide to articular cartilage damage (Nilsson and Persson 1973). In an attempt to overcome this limitation Tew and Hackett (1981) reported on the identification of cartilage particles in synovial fluid. Although their initial results were encouraging, this technique has not gained widespread use in clinical cases.

In other species, markers of chondrocyte metabolism in the synovial fluid, have been investigated as indicators of the state of articular cartilage. These include cartilage oligomeric matrix protein (Saxne and Heingård 1992), link protein (Ratcliffe *et al* 1994) and markers of

type II collagen synthesis (Lohmander and Shinmei 1994). In the horse most published work has examined proteoglycans as markers (Alwan *et al* 1990, Yovich *et al* 1991, Little *et al* 1992). As stated in Chapter 3 the results from these studies have been inconsistent. Alwan *et al* (1990) demonstrated an increase in sulfated GAGs and keratan sulphate in horses with clinical signs of joint disease, while Yovich *et al* (1991) in a similar study could find no significant difference. Little *et al* (1990) found there was no correlation between sulfated GAG levels in synovial fluid and the severity of cartilage damage.

There are a number of possible reasons for these inconsistencies. In humans it has been demonstrated that levels of proteoglycans are dependant on the time after joint injury that the synovial fluid sample is collected (Lohmander *et al* 1989). Also exercise has been shown to effect synovial fluid proteoglycan levels in horses (Yovich *et al* 1993, Chapter 3). Therefore it is not surprising that correlations between articular cartilage damage and proteoglycan levels were poor in a group of horses at varying levels of training and with joint disease at a variety of stages (Little *et al* 1990).

In previous studies in the horse, synovial fluid proteoglycan levels were not compared with established diagnostic techniques. This has made it difficult to determine whether this technique offers any advantage over others. The purpose of this study was to determine the clinical, radiographic and synovial fluid variables that best indicated the degree of damage to the intraarticular structures of the midcarpal and antebrachiocarpal joints of racing horses with carpal disease and to directly compare these with sulfated GAG levels in synovial fluid. The effect of training and chronicity of disease on the usefulness of sulfated GAG levels in

synovial fluid as a diagnostic aid were also examined.

Materials and Methods

All horses undergoing midcarpal or antebrachiocarpal joint arthroscopy between April 1993 and September 1994 were included in the study. Clinical examinations, radiographic technique, synovial fluid collection methods, and grading systems are described in Chapter 2. Normal values for synovial fluid variables were established using synovial fluid samples from the midcarpal joints of 13 Standardbred horses between 4 and 5 years of age, with no clinical signs of carpal disease, exercised on a treadmill as described in Chapter 2. Synovial fluid was collected before the commencement of training, and at eight and 16 weeks of training. All horses were examined for signs of carpal joint compromise before training and again after 16 weeks of training.

Prediction of the grade of subchondral bone damage at each site was made based on the extent of subchondral lysis and bone fragmentation on radiographs, and graded 0-3 as for subchondral bone damage. This was then compared with the extent of subchondral bone damage as assessed arthroscopically.

Joint disease was classified as either acute or chronic. Acute joint disease was defined as joints with no detectable osteophytes or enthesiophytes on radiographs, whereas joints were designated as chronically affected if osteophytes or enthesiophytes were observed.

Statistical analysis

Where the diagnostic variable was categorical, contingency tables were generated and Pearson's Chi-square test used to determine the strength of the relationship. Where numbers in one category were low, data was pooled. For the antebrachiocarpal joint these were all 2×2 tables. For continuous diagnostic variables, Spearman rank order correlations were used to test for a significant relationship. Due to the small numbers of antebrachiocarpal joints examined, continuous variables were examined using a Mann Whitney U test. Results were reported as mean ± SEM and significance was set at $p<0.05$. Variables for clinical cases were compared with controls using a Man Whitney U test.

Results

Arthroscopic examination was performed on a total of 81 joints of 43 horses. Thirty nine of the horses were racing Thoroughbreds (74 joints) and four were racing Standardbreds (seven joints). Ages ranged from two to five years with a mean of 3.0±0.1 years. The severity of subchondral bone damage in the midcarpal joint decreased with the age of the horse ($\chi^2_1=5.8$, $p<0.05$), but this was not the case in the antebrachiocarpal joint. No clinical signs of lameness or carpal joint disease developed in control horses after 16 weeks of training.

The mean grade of lameness was 0.9±0.1. Mean synovial fluid leukocyte counts were 378±31×10^6/L, TP 27±1 g/L, LDH activity 210±31 U/L and AP activity 6.0±1.6 U/L.

Comparisons between joints

In the Thoroughbred horses, 23 left and 24 right midcarpal joints were examined. Although the mean grade of articular cartilage and subchondral bone damage was greater in left midcarpal joints, this was not statistically significant. Eleven left and 16 right antebrachiocarpal joints were examined. A wide range of cartilage and subchondral bone damage was observed including three joints in which the only pathology was mild articular cartilage damage (Grade 1). There was no significant difference in the degree of articular cartilage or subchondral bone damage between left and right antebrachiocarpal joints. When comparing the midcarpal and antebrachiocarpal joints examined, antebrachiocarpal joints had significantly more osteophytosis ($p<0.001$) and lower synovial fluid volumes ($p<0.05$). For all other variables examined there was no significant difference.

Conformation abnormalities

Valgus was the most commonly observed conformational deformity with 36 joints of 21 horses affected. Rotational deformity was observed in 30 joints of 18 horses, whereas hyperextension was observed in only seven joints of four horses, and offset metacarpus in four joints of three horses. There was no correlation between intra-articular pathology and the degree of valgus or rotational deformity. Too few cases of carpal hyperextension and offset metacarpus were observed to allow meaningful statistical analysis.

Correlation of diagnostic variables with intra-articular lesions

Articular cartilage damage- In midcarpal joints the degree of enthesiophytosis was the diagnostic variable which best predicted articular cartilage damage (χ^2_6=32, p=0.00002). In all joints given a grade 2 or 3 for enthesiophytes, there was grade 2 or 3 cartilage damage. There were however two joints with grade 3 cartilage damage where the enthesiophyte grading was 0 or 1. There was also a significant relationship between articular cartilage damage and the extent of subchondral bone lysis (χ^2_6=18, p=0.007) and synovial fluid AP activity (R=0.31, p=0.03). No other diagnostic variable was significantly related to articular cartilage damage in the midcarpal joint.

In contrast, in the antebrachiocarpal joint the size and number of fractures observed on radiographs was the best indicator of articular cartilage damage (χ^2_1=7.7, p=0.006). Other diagnostic variables significantly associated with articular cartilage damage were the extent of osteophytes (χ^2_1=5.2, p=0.01), degree of joint swelling (χ^2_1=5.2, p=0.02), and total sulfated GAGs (z=-2.1, p=0.04).

Subchondral bone damage- In the midcarpal joints the best predictor of subchondral bone damage were the size and number of fractures detected on radiographs (χ^2_6=28, p=0.00008) and the size and extent of enthesiophytes (χ^2_6=26, p=0.0003). Other variables associated with subchondral bone damage were the extent of radiographic subchondral lysis (χ^2_6=22, p=0.008), and the degree of joint swelling (χ^2_1=5.4, p=0.02).

Fracture score was the best indicator of subchondral bone damage in the antebrachiocarpal joint (χ^2_1=9.6, p=0.002). Other variables significantly associated with subchondral bone damage in the antebrachiocarpal joint were pain on flexion (χ^2_1=5.6, p=0.02), size and extent of osteophytes (χ^2_1=5.0, p=0.03), degree of joint swelling (χ^2_1=3.8, p=0.04) and total sulfated GAGs (z=-2.0, p=0.04).

Site of lesion- In the midcarpal joint 29 distal CR osteochondral lesions were identified during arthroscopy. Of these 21 were graded the same radiographically, while three were graded one grade greater and three one grade less. In two joints mild subchondral bone damage (Grade 1) was predicted based on radiographic findings, whereas severe subchondral bone damage (Grade 3) was found arthroscopically. Of 12 proximal C3 lesions, five were graded the same radiographically and arthroscopically, four were graded one grade more and three were graded one grade less arthroscopically. Only three distal CI lesions were observed and these were all grade 1 both arthroscopically and radiographically. In the antebrachiocarpal joint twenty distal radius osteochondral lesions were observed. Seventeen of these were graded identically with radiographs and arthroscopy and two were given one grade more and one was given one grade less arthroscopically. Of 12 proximal CI lesions, seven were given the same, four one grade more and one one grade less arthroscopically. Only one of five proximal CR lesions were graded the same radiographically and arthroscopically, whereas two were given one grade more and two were given one grade less.

Proteoglycan levels in synovial fluid

There was no significant difference in both sulfated GAG concentration and total sulfated GAGs between midcarpal joints and antebrachiocarpal joints. Mean synovial fluid sulfated GAG concentration was 59.1±4.3 μg/ml in all joints examined. Mean total sulfated GAG per joint was 463.2±42.2 μg. Mean sulfated GAG concentration in joints with acute disease was 62.1 ±11.1 μg/ml and mean total sulfated GAG content 524.1±114.3 μg, while mean sulfated GAG concentration in joints with chronic disease was 58.2±4.6 μg/ml and mean total sulfated GAG was 445.2±43.4 μg. The difference between total sulfated GAGs in acute and chronically diseased joints was not significant. Mean sulfated GAG concentration in joints from horses that galloped or raced within the previous two weeks was 60.9±5.1 μg/ml, and mean total sulfated GAGs was 537.5±64.5 μg. In joints of horses not galloped or raced within the previous two weeks mean sulfated GAG concentration was 52.2±6.9 μg/ml and total sulfated GAGs were 357.2±48.1 μg. The difference between total sulfated GAGs in these groups was significant ($p<0.05$).

There was no significant correlation between either sulfated GAG concentration and total sulfated GAGs and the extent of subchondral bone defect or articular cartilage damage overall joints (antebrachiocarpal and midcarpal). Antebrachiocarpal joints with grade 2 and 3 articular cartilage damage had significantly higher total sulfated GAGs than joints with grade 0 and 1 articular cartilage damage ($p<0.05$). This was also the case for subchondral bone damage ($p<0.05$).

Comparisons with controls- When sulfated GAG concentrations for joints from clinical cases were compared with those of horses trained on a treadmill at various stages of training, the mean concentration for clinical cases was significantly greater than control horses before training ($p<0.001$), and after eight weeks of low intensity training ($p<0.01$). However there was no significant difference between clinical cases and control horses which had a further eight weeks of high intensity training (Figure 1).

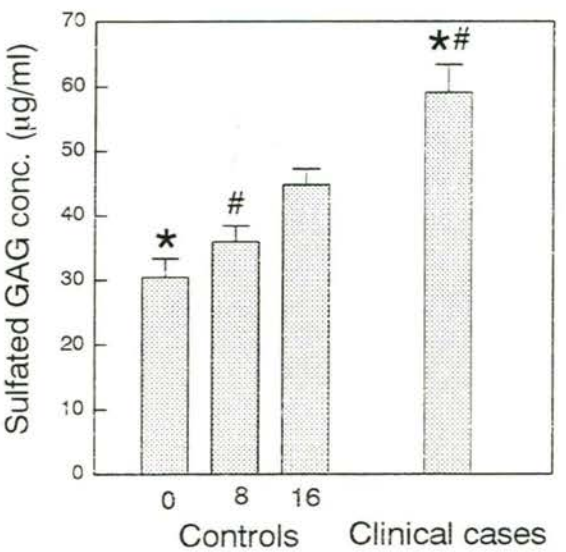

Figure 1.

Sulfated glycosaminoglycan concentration (±sem) in synovial fluid from clinical cases of carpal joint disease and normal sound horses untrained, and at eight and 16 weeks of a controlled treadmill training program. (* significant difference $p<0.001$, # significant difference $p<0.01$).

When only those horses that had galloped in the last two weeks were included there was no change in the results. Total sulfated GAG concentrations were significantly higher in joints of clinically affected horses than control horses at eight ($p<0.01$) and 16 weeks of training

($p<0.05$) (Figure 2). When only those horses that had galloped in the last two weeks were included the differences between clinical cases and controls were greater ($p<0.001$ and $p<0.01$ respectively).

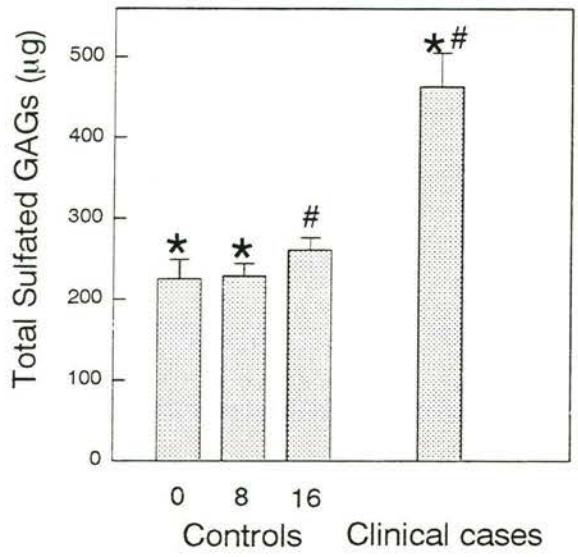

Figure 2.

Total sulfated glycosaminoglycans (±sem) in synovial fluid from clinical cases of carpal joint disease and normal sound horses untrained and at eight and 16 weeks of a controlled treadmill training program (* significant difference $p<0.01$, # significant difference $p<0.05$).

Correlations with other variables

Overall joints there were significant but weak correlations between sulfated GAG concentration and pain on flexion ($R=0.34$, $p<0.01$) as well as degree of lameness ($R=0.24$, $p<0.05$) and synovial colour ($R=0.32$, $p<0.01$). Sulfated GAG concentration was also negatively correlated with the number of weeks in training ($R=-0.23$, $p<0.05$). When only

those horses that had galloped or raced within the previous two weeks were selected, the correlation ($R=0.56$, $p<0.001$) with pain on flexion was stronger than for all horses.

Discussion

Prediction of extent of intra-articular pathology

A number of studies have examined the accuracy of a single diagnostic variable such as synovial fluid proteoglycans (Little *et al* 1990), cartilage wear fragments (Tew and Hackett 1981), and radiography (McIlwraith *et al* 1987, Kannegieter and Burbidge 1990, Dabareiner *et al* 1996, Moore and Schneider 1994) for predicting intra-articular pathology in the carpus. To my knowledge this is the first time a number of diagnostic variables have been compared in a prospective study. This has provided baseline information on how useful existing diagnostic techniques are and allowed direct comparison with a relatively new technique, the measurement of synovial fluid sulfated GAGs. Radiography was the most useful diagnostic aid and synovial fluid sulfated GAG concentration and total GAGs were of limited value in the preoperative assessment of intra-articular pathology. It is acknowledged that there are a number of other synovial fluid markers that have been investigated, but the purpose of this study was to more fully investigate a marker where the literature was unclear on its usefulness.

Articular cartilage damage- Grading systems for articular cartilage damage in the equine

carpus have been described by McIlwraith *et al* (1987), Kannegieter and Ryan (1991) and Gibson *et al* (1996). All of these systems can only be applied to joints with osteochondral fragments and give an indication of overall joint compromise. With the recognition of other types of joint pathology it was necessary to develop separate systems for both bone and articular cartilage. Although subchondral bone and articular cartilage damage tend to occur concurrently, there are also joints with cartilage damage and no subchondral bone damage, as well as joints with very soft subchondral bone but minimally affected overlying cartilage. It was therefore decided to grade cartilage and bone damage separately, based purely on the area of each involved.

In both midcarpal and antebrachiocarpal joints, careful examination of radiographs was the most useful diagnostic tool for predicting the degree of cartilage damage. It may be that by combining individual radiographic variables, prediction of articular cartilage damage can be improved. Greater numbers of cases would be required to test this. As most of the cartilage changes are likely to be secondary to changes in the subchondral bone (Radin *et al* 1984), it was expected that articular fractures and subchondral lysis would be the best indicators of articular cartilage damage. However, in the midcarpal joint it was found that the size and extent of enthesiophytes was the best indicator of articular cartilage damage. In fact there was no significant relationship between the size and number of fractures and articular cartilage damage. This suggests that articular cartilage damage in the midcarpal joint is more dependant on the degeneration of the subchondral bone rather than acute damage from fractures. Based on radiographic observations, Barr (1994b) has suggested that subchondral bone degeneration precedes fracture in this joint, which is consistent with the findings in the

present study. In contrast, fractures played a more important role in the antebrachiocarpal joint indicating that bone degeneration was less likely to precede fractures in this joint.

Enthesiophytes are thought to be the result of stress applied to soft tissue attachments on bone (Rufai *et al* 1995). Those observed in the present study were on the dorsal aspect of CR. Soft tissue attachments at this site include the dorsal carpal joint capsule and the dorsal intercarpal ligament between CR and CI. It is possible that axial forces that produce chronic stress in the dorsal intercarpal ligament and the joint capsule, also cause articular cartilage and subchondral bone damage in the midcarpal joint. Also, in many of the more severe cases, subchondral bone damage extended into the joint capsule attachment and this may have stimulated enthesiophyte production. However, no relationship was found between enthesiophytes and cartilage damage in the antebrachiocarpal joint. This is despite the fact that many distal radius fractures are large and involve the joint capsular attachment. Again this may be because fractures in this joint tend to be simple, while fractures in the midcarpal joint are associated with extensive subchondral bone degeneration which may affect the soft tissue attachments to a greater degree.

In the midcarpal joint, clinical signs were a relatively poor indicator of the degree of articular cartilage damage. The reason for this is that joint compromise is due to many factors, of which cartilage degeneration is only one. Other possible causes of pain, swelling and lameness include intercarpal ligament inflammation and tearing, synovitis and capsulitis, and increased intraosseous pressure. Although flexion tests are widely used as an aid to diagnosis of intra-articular disease, the evidence from the present study indicates that they are of little

use in predicting the degree of intra-articular compromise in the carpus.

Subchondral bone damage- Subchondral bone damage was not divided into articular fractures and soft bone because the distinction between these is not always clear. Carpal fractures in racing horses rarely are due to a single traumatic event, but rather fatigue fractures from cyclical loading (Bramlage *et al* 1988). Bone remodelling in response to this loading initially results in increased porosity, and structurally weakened bone (Martin 1995). The end result may be a chip fracture, slab fracture, crush fracture (Moore and Schneider 1994), soft bone (Dabareiner *et al* 1996) or, as in most cases, a combination of these. It would be both difficult and artificial to divide these findings, so it was decided to assess subchondral bone damage as a whole. The inverse relationship between subchondral bone damage and age in the midcarpal joint was consistent with this damage being caused by bone fatigue. Fatigue fractures are more common in the young, when bone turnover is at its highest (Morris 1980) and bones are subjected to greater strains (Nunamaker *et al* 1990). This relationship was not observed in the antebrachiocarpal joint, suggesting that a reduction in the rate of bone turnover may be delayed in the distal radius, or that fatigue is less important in the bones of this joint.

As expected, radiographic findings, particularly fracture score, were the best indicators of subchondral bone damage in the midcarpal joint, although the association with the degree of enthesiophytosis was unexpected. If enthesiophytes are a response of the bone to microdamage rather than a response to trauma of soft tissue attachments, this suggests that degeneration of bone precedes fractures in the midcarpal joint. In contrast, fracture score was the only useful radiographic indicator of subchondral bone damage in the antebrachiocarpal joint.

Although overall, radiographs were useful indicators of the site of subchondral bone damage in the present study, some deficiencies were highlighted. McIlwraith *et al* (1987) and Kannegieter and Burbidge (1990) have compared radiographic and arthroscopic findings for chip fractures in the carpus. McIlwraith *et al* (1987) found a very weak correlation for fractures of distal CR, and proximal CI and CR, however Kannegieter and Burbidge (1990) found that proximal C3 and distal CI were the only sites where radiographic and arthroscopic findings were not correlated. Dabareiner *et al* (1996) demonstrated that in cases where subchondral lucency of distal CR was the only radiographic sign, osteochondral fragments were often present at this site. This stresses the importance of carefully examining radiographs for subtle signs of subchondral bone damage. Interestingly the two sites identified in the present study where there were large discrepancies between radiographic and arthroscopic findings were the distal CR and proximal C3, demonstrating the need for caution when interpreting radiographs of these sites.

Synovial fluid as a diagnostic aid in the carpus

It was found that synovial fluid analysis was of little use in predicting the degree of both cartilage and bone damage within the midcarpal and antebrachiocarpal joints. Apart from sulfated GAGs, AP activity was the only synovial fluid variable associated with articular cartilage damage and this was far from being a useful clinical relationship. Stashak (1987) suggested that changes in synovial fluid variables mostly reflected the degree of synovitis. Mean synovial fluid protein concentrations and total leukocyte counts were only mildly elevated in the horses in this study, indicating that synovitis was mild in most of the joints

examined. Tew (1982) reported normal protein values of 5-12 mg/ml and leukocyte counts $<200\times10^6$/L for horses in training, but higher levels (protein up to 22 g/l, leukocyte counts $<450\times10^6$/L) were reported by Rose and Paris (1979). It may be that low grade inflammation of the synovial membrane is normal in racehorses. Mean LDH concentrations were elevated compared to those of normal racehorses in training (Rose and Paris 1979). Conflicting results have been obtained previously when LDH activity from normal joints and joints with pathology have been compared. Rejnö (1976) found that LDH activity increased with intra-articular pathology while Yancik *et al* (1987) found that activities in normal joints were highly variable and there was no significant change with pathology. The findings of the present study are consistent with this. This is probably because LDH is found in a wide range of tissues (Rejnö 1976) so there are a number of possible sources of the enzyme including extra-articular ones. Also it has been shown that large changes in LDH concentration occur with training alone (Chapter 3).

Synovial fluid proteoglycans

This study demonstrated no significant difference between the sulfated GAG concentrations in the synovial fluid of horses with arthroscopically confirmed articular cartilage damage and joints from normal horses in full training on a treadmill. There was also no association between synovial fluid sulfated GAG concentrations and the degree of articular cartilage damage. This is consistent with the findings of Yovich *et al* (1991) and Little *et al* (1992). One important reason for this was the wide range of sulfated GAG concentrations in joints with clinical disease which is also consistent with the findings of others (Alwan *et al* 1990).

The response of articular cartilage to damage and degeneration is complex. In humans it has been demonstrated that proteoglycan concentrations increase with acute joint disease, but more chronic joint disease is characterised by levels similar to controls (Ratcliffe *et al* 1988), and high concentrations occur soon after traumatic joint injury, but then slowly decline over time (Lohmander *et al* 1989). Therefore another reason for the poor correlation with extent of joint disease in horses could be that the degree of chronicity has not been addressed. Better correlations between the extent of joint damage and proteoglycan levels have been achieved when joints are grouped according to disease type and stage (Messner *et al* 1993). In this study the presence of osteophytes or enthesiophytes was used to define chronicity. This may not be an ideal method, but due to the difficulty of getting accurate histories was the only one available. There was no advantage in grouping joints in this way. Dalhberg *et al* (1992) found that there was an inverse relationship between the degree of articular cartilage damage and synovial fluid proteoglycan levels in humans with osteoarthritis. They concluded that this was due to a depletion of the proteoglycan pool in the articular cartilage. In contrast, in studies of meniscectomised rabbits Messner *et al* (1993) found that in the early stage of disease synovial fluid proteoglycan concentration was directly proportional to cartilage loss. Therefore the relationship between proteoglycan concentration and articular cartilage damage is complex and time dependant.

There was an effect of rest time on sulfated GAG concentrations. Exercise has been shown to increase synovial fluid proteoglycan concentrations (Yovich *et al* 1993) and this is thought to be due to augmentation of proteoglycan molecule transport out of the cartilage matrix by mechanical stimulation (Dahlberg *et al* 1992). It would therefore be expected that in joints

where cartilage breakdown is occurring, that proteoglycan levels would be highest following exercise and ideally should be sampled very soon after as the half life for proteoglycans in synovial fluid is about 12 hours. Unfortunately most of the cases in the present study were referrals and had often been rested for some time before being presented. But even horses that had two weeks rest had lower proteoglycan levels than those presented within two weeks of fast exercise. Perhaps better correlations between proteoglycan concentrations and cartilage damage may have been obtained if samples were taken immediately following fast exercise.

Measurement of total sulfated GAGs- The use of total proteoglycan levels has not been previously reported in horses, but has been described by Lohmander *et al* (1989) and Silverman *et al* (1990) in human joints. Calculation of total sulfated GAGs relies on the accurate estimation of synovial fluid volume. As discussed in Chapter 3 the accuracy of volume measurement by aspiration is dependant on the volume retrieved. There were a number of joints with low volumes in the present study so the results obtained can only be regarded as estimates. Theoretically, total sulfated GAGs should more accurately reflect cartilage metabolism than sulfated GAG concentration as the total value is not dependant on synovial fluid volume. This is supported by the results of this study, as there was a significant difference in total sulfated GAGs between diseased joints and normal joints after 16 weeks of training, but not sulfated GAG concentration. Also total sulfated GAGs were shown to be correlated with cartilage and subchondral bone damage in the antebrachiocarpal joint, while sulfated GAG concentrations were not. In contrast Lohmander *et al* (1989) found in humans with knee injury that the measurement of total proteoglycans gave no more information than proteoglycan concentration. The use of the albumin dilution technique would provide a more

accurate means of determining synovial fluid volume (Wallis *et al* 1985).

Response of synovial fluid sulfated GAGs to training- Normal values for sulfated GAG concentrations and total sulfated GAGs were established using horses trained on a treadmill. This enabled the exercise level of these horses to be closely controlled while serial synovial fluid samples were taken and signs of joint disease monitored. Clearly treadmill training differs from conventional training and is therefore not ideal as a control. Treadmill surfaces have been shown to be relatively unforgiving, and these horses were training on an uphill slope, while clinical horses were generally trained on the flat. Horses running on a treadmill have also been shown to have different gait patterns from horses running on a track (Barrey *et al* 1993). Both these factors are likely to result in different joint forces which could affect intra-articular metabolism. The control group were all Standardbred horses compared to the predominantly Thoroughbred horses in the clinical group. The effect of breed on synovial fluid GAG concentrations is unknown.

Horses presenting with carpal disease tended to have lower sulfated GAG concentrations if they had been in training for longer periods which contrasts with the findings in Chapter 3 where sulfated GAG concentrations increased with training. Therefore it appears that diseased joints respond differently to training than normal joints. It is possible that joints with osteoarthritis lose larger amounts of proteoglycan early in a training program, depleting the proteoglycan pool and therefore the proteoglycan available to be released into the synovial fluid.

Differences between antebrachiocarpal and midcarpal joints- There are three possible reasons for the differences in associations between sulfated GAGs and articular cartilage degeneration observed between midcarpal and antebrachiocarpal joints. Firstly, the midcarpal joint communicates with the carpometacarpal joint which was not available for examination arthroscopically. Cartilage in the carpometacarpal joint can potentially contribute to the sulfated GAGs in the synovial fluid of the midcarpal joint, but was not included in the evaluation of cartilage damage. Secondly, severe degenerative joint disease was not as much a feature of the antebrachiocarpal joint as in the midcarpal joint. Cartilage damage tended to be confined to the immediate area of subchondral bone damage. Therefore cartilage damage was more acute and cartilage proteoglycan levels were less likely to be depleted. Finally, lower numbers of antebrachiocarpal joints may have influenced the correlations in this joint.

Sulfated GAG concentrations were correlated with synovial fluid colour, LDH and protein concentration which is consistent with findings in horses trained on a treadmill (Chapter 3). This is further evidence that synovitis in involved in proteoglycan breakdown as discussed in Chapter 3.

Conformation

Conformation abnormalities have generally been regarded as predisposing to carpal joint disease (Auer 1980, Palmer 1986, Schneider 1979) but the objective evidence is contradictory (Dolvik and Klemetsdal 1994, Barr 1994a). Dolvik and Klemetsdal (1994) reported an increased incidence of carpitis in Norwegian trotters with carpal hyperextension, but Barr

(1994a) found that the degree of carpal hyperextension, determined radiographically, was not significantly different in racehorses with carpal chip fractures and horses with normal carpi. The low number of horses presenting with carpal hyperextension making conclusions difficult, however carpal valgus and rotational deformities were not associated with increased severity of carpal disease in this study.

Previous reports have shown a unequal distribution of intra-articular fractures between left and right carpal joints (Larsen and Dixon 1970, Dehaan *et al* 1987, Park *et al* 1970, Raidal and Wright 1996). Peloso *et al* (1994) found no difference between left and right legs in the number of carpal injuries identified at the racetrack, and McIlwraith *et al* (1987) observed significantly more fractures in the right midcarpal joint but no difference between left and right antebrachiocarpal joints of Thoroughbred racehorses. The findings of the present study were similar, although there was a trend for increased severity of both subchondral bone and articular cartilage damage in the left midcarpal joint and a higher incidence of pathology in the right antebrachiocarpal joint of Thoroughbreds. Thoroughbred horses involved in this study raced clockwise. Two reasons have been proposed for an unequal distribution of carpal injuries. Greater vertical forces have been observed in the lead limb of galloping horses both on the turn and in the straight (Ratzlaff *et al* 1987). Although there appear to be no studies to my knowledge which have examined whether the direction of racing affects with which leg a horse will predominantly lead, it is generally accepted that horses prefer leading with their inside foreleg on the turn (Schneider 1979). Therefore a higher incidence and severity of joint disease would be expected in the right foreleg in this study, which was not the case. Alternatively Lindholm (1987) and Schneider (1979) have suggested that there are greater

axial forces on the lateral aspect of the inside, and medial aspect of the outside leg, but there are no *in vivo* studies to confirm this. The trends in incidence and severity of intra-articular damage observed in the present study, and the fact that midcarpal pathology occurs predominantly on the medial aspect while antebrachiocarpal pathology occurs predominantly on the lateral aspect, lends weight to this theory. It may be that the relatively symmetrical distribution of carpal damage observed is due to a combination of these two factors.

Conclusion

The findings of this study confirm the difficulty in predicting the degree of joint compromise, particularly articular cartilage damage, based upon clinical findings, radiographic findings and synovial fluid analysis. Radiography remains the most useful diagnostic tool and the use of sulfated GAG concentration in synovial fluid does not appear to improve the accuracy of detecting articular cartilage damage. Despite this there does appear to be some potential for the use of sulfated GAGs as an indicator of the degree of intra-articular damage and this is probably greatest in joints with acute articular cartilage damage. The estimation of the total sulfated GAG content of the joint appears to be more useful than sulfated GAG concentration but requires an accurate method of measuring synovial fluid volume. Other markers of the state of articular cartilage need to be investigated. For the midcarpal joint, evaluation of the extent of enthesiophytes is the most important preoperative factor for predicting the extent of articular cartilage damage, whereas for the antebrachiocarpal joint, the number and size of fractures played a more important role.

CHAPTER 5

THE INTERCARPAL LIGAMENTS OF THE MIDCARPAL JOINT: ANATOMICAL AND BIOMECHANICAL FEATURES

Part 1.

THE ANATOMY OF THE PALMAR INTERCARPAL LIGAMENTS AND RELATED STRUCTURES OF THE MIDCARPAL JOINT.

Introduction

The important role of the intercarpal ligaments in carpal biomechanics and lameness in the horse was first proposed by Bramlage *et al* (1988). With the routine use of arthroscopy the awareness of pathological conditions of these ligaments has increased. Tearing of the palmar intercarpal ligaments of the midcarpal joint is now commonly recognised (McIlwraith 1992, Kannegieter and Colgan 1993, Phillips and Wright 1994), while enlargement of a dorsomedial intercarpal ligament (DMICL) has been described by Selway (1991).

The anatomy of the medial palmar intercarpal ligament (MPICL) and lateral palmar intercarpal ligament (LPICL) of the midcarpal joint have been recently described (Phillips and Wright 1994). McIlwraith (1992) and Kannegieter and Colgan (1993) have described the arthroscopic anatomy of the MPICL. However the behaviour of these ligaments during joint movement has not been described and there appears to be no detailed anatomical descriptions of the DMICL described by Selway (1991).

In this study anatomical dissections were performed to describe in greater detail the structure

and function of the MPICL, the LPICL and the DMICL of the midcarpal joint. Observations were made on the behaviour of these ligaments during flexion and extension of the carpus and their role in the restraint of movement of the proximal row of carpal bones.

Materials and methods

Ten carpal joints were obtained from eight Thoroughbred horses, aged between two and five years that were euthanased for reasons unrelated to carpal disease. Dissections were performed as described in Chapter 2.

Results

Dorsomedial intercarpal ligament

The DMICL was present in all joints. This ligament arose from a triangular area of which the lateral border was a longitudinal ridge on the distal part of the dorsomedial surface of the radial carpal bone (CR). The ligament did not attach to, or impinge on, the articular surface of CR in any of the joints examined. From the CR attachment it passed medially and distally, in close association with the joint capsule, and inserted on the proximodorsal aspect of the large eminence on the dorsomedial aspect of the second carpal bone (C2), immediately dorsal to the deep part of the medial collateral ligament (MCL). In one joint some fibres of the

ligament inserted on the third carpal bone (C3). The deep part of the MCL attached to the proximal aspect of the same eminence of C2, and its fibres diverged, passing both proximally and dorsally, to attach on the medial aspect of CR. Thus the DMICL formed the dorsodistal part of this fibre complex (Figure 1).

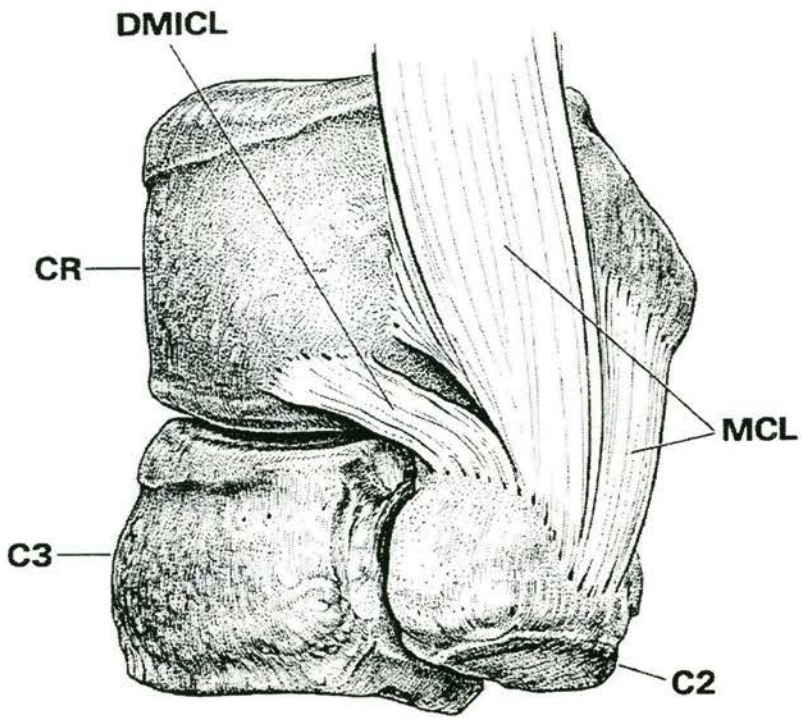

Figure 1.

A medial view of the dorsomedial intercarpal ligament of the right midcarpal joint showing its distal attachment to the dorsomedial aspect of the second carpal bone (C2), immediately dorsal to that of the deep part of the medial collateral ligament (MCL). CR=radial carpal bone, C3=third carpal bone.

Synovial membrane covered the entire palmar aspect of the DMICL and the dorsal surface extending approximately 15 mm from the origin. The mean length (±sem) of the DMICL was

26.7±1.0 mm, mean width 7.2±0.6 mm at its midpoint and mean thickness 1.5±0.02 mm. There was slight spreading of the fibres at both attachments. With the joint extended the DMICL ran in a palmar and slightly distal direction from CR curving more distally immediately before inserting on C2. It was flaccid at all angles of flexion and extension, but this was most pronounced in extension where it was often kinked.

Lateral palmar intercarpal ligament

In all joints the proximal attachment of the LPICL was predominantly on the distal part of the palmaromedial surface of the ulnar carpal bone (CU). A few fibres also attached to the palmarolateral surface of the intermediate carpal bone (CI). From here the ligament ran in a distomedial and slightly palmar direction to the proximal palmarolateral surface of C3 with a few fibres attaching to the palmaromedial surface of fourth carpal bone (C4) in all joints (Figure 2).

The LPICL was either triangular or oval in cross section. In eight joints the LPICL was undivided while in one joint the ligament was divided into a large palmarolateral part and a thin dorsomedial part, and in another, a thin palmarolateral part which inserted on C4 was divided from the main body of the ligament. From its distal attachment on C3 the fibres of the ligament diverged towards CU giving the proximal attachment a greater cross-sectional area. The mean width of the ligament (lateral-medial) at its midpoint was 5.9±0.5 mm, mean depth (dorsal-palmar) was 10.2±0.5 mm and mean length 14±1.4 mm. With the joint extended, the

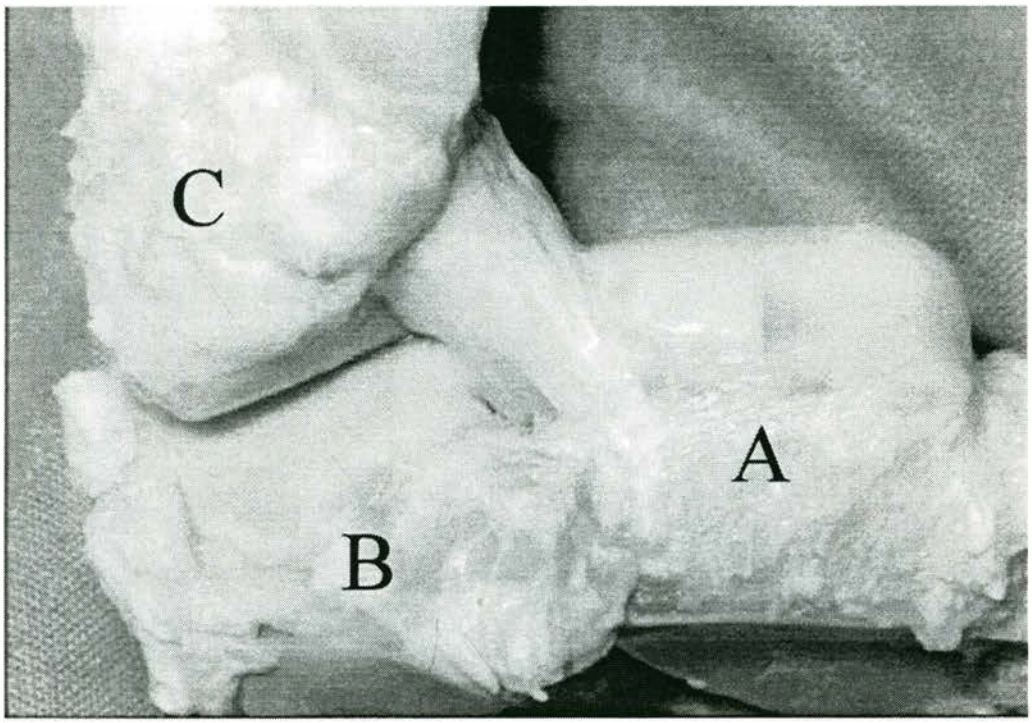

Figure 2.

A palmar view of the lateral palmar intercarpal ligament of the left midcarpal joint showing its distal attachment predominantly on the third carpal bone (A), and its proximolateral to distomedial alignment. B=fourth carpal bone, C=ulnar carpal bone

palmar half to two-thirds of the LPICL was under tension while the dorsal half or one third appeared flaccid (Figure 3). With both extension and dorsal displacement of the CU, greater tension was evident in the palmar aspect of the ligament. In this position the dorsal surface of the ligament closely opposed the articular margin of C3 (Figure 3). Slight fraying of the dorsal surface of the LPICL was observed in one joint and a two mm tear of the most dorsomedial aspect of the same ligament was present in another joint.

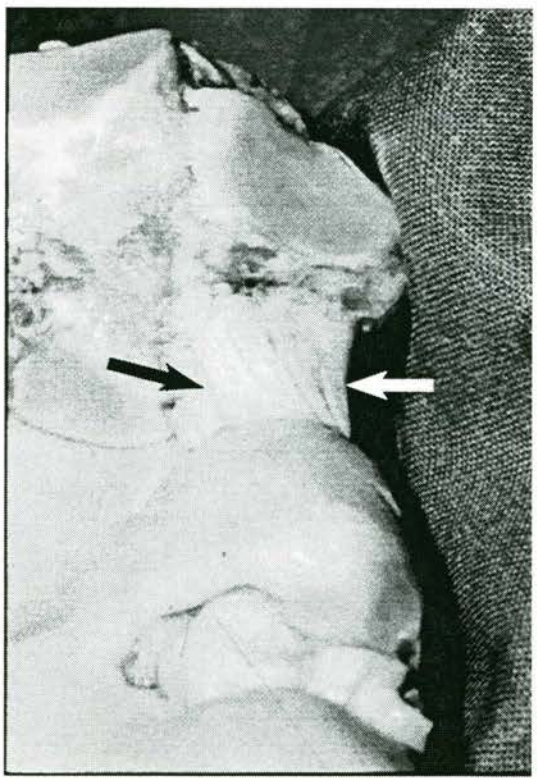

Figure 3.

Medial view of the lateral palmar intercarpal ligament of the right midcarpal joint with the ulnar carpal bone held in extension. The synovial membrane lining has been removed. There is tension on the palmar fibres (white arrow) but not the dorsal fibres (black arrow).

Medial palmar intercarpal ligament

The structure of the MPICL was complex with considerable variation between joints. It attached proximally to the distolateral surface of CR and ran distally to insert on the proximal palmaromedial surface of C3 and the proximal palmarolateral aspect of C2. The ligament was sheet-like; in cross-section the distal attachment formed a roughly semicircular arc, and the proximal attachment was more linear so that the proximal attachment had a greater

dorsopalmar depth than the distal attachment. At its midpoint the mean depth was 18±1.5 mm.

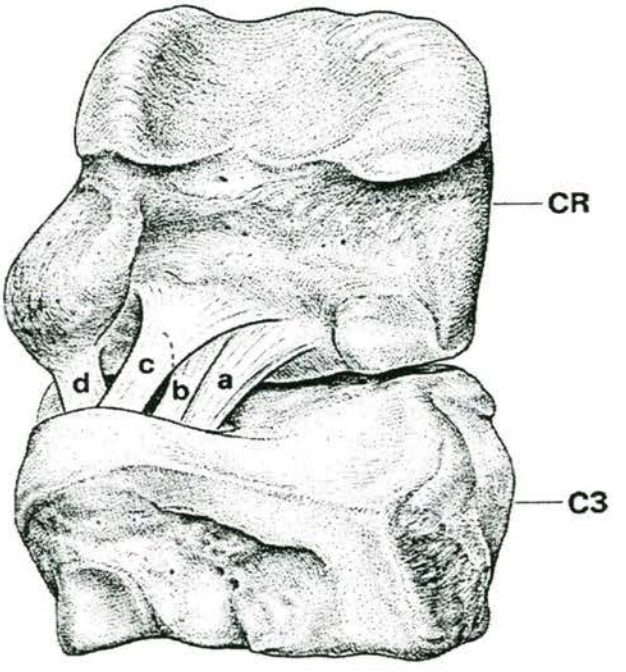

Figure 4.

Lateral view of the medial palmar intercarpal ligament of the right midcarpal joint. The dorsolateral (a), dorsomedial (b) and palmarolateral bundles (c) are inclined proximodorsal to distopalmar, whereas the palmaromedial bundle (d) is inclined proximopalmar to distodorsal. The dotted line represents the palmar border of the dorsomedial bundle. CR=radial carpal bone, C3=third carpal bone.

The MPICL could be divided into four fibre bundles in all horses (dorsolateral bundle, dorsomedial bundle, palmarolateral bundle, palmaromedial bundle, Figure 4), although the division between these was variably defined. In six joints, bilaterally in one horse, the

attachment to CR was easily separated into dorsal and palmar parts. The dorsolateral bundle was longer than the dorsomedial, attaching slightly more proximally on CR. From here it ran distally and palmarolaterally to the fossa at the palmaromedial aspect of C3. The dorsomedial bundle was shorter and attached more distally on CR. Its fibres passed palmarodistally to the lateral aspect of C2. The palmarolateral bundle attached proximally, immediately palmar to the dorsal bundles on the lateral surface of CR. Its fibres ran palmarolaterally and distally to the palmaromedial surface of C3. The proximal attachment of the palmaromedial bundle was more palmar, distal to the large eminence on the palmaromedial aspect of CR. From here it ran dorsodistally to the lateral surface of C2. The fibres of both bundles blended with the palmar carpal ligament at their palmar aspects. The intra-articular surfaces of the MPICL were covered with a thin layer of synovial membrane.

Sizes of the four fibre bundles of the MPICL varied between joints. In all joints the dorsolateral bundle was thinnest dorsally. The dorsolateral bundle was thinner (lateral-medial) than the dorsomedial bundle in four joints, thicker in three joints and the same thickness in three joints (Table 1). Of the two palmar bundles the palmaromedial was larger in cross section than the palmarolateral.

With the joint flexed, only the dorsolateral and dorsomedial bundle could be observed intra-articularly from the dorsal aspect. Both were slightly flaccid in this position. With the joint extended all parts of the ligament were in tension except in five joints where one to two thirds of the dorsolateral bundle were flaccid. All of these MPICLs had some tearing of the dorsolateral bundle. Most tension appeared to be in the palmaromedial bundle. With

extension and dorsal displacement of CR all parts of the ligament were in equal tension.

Table 1.

Mean dimensions (±sem) of the parts of the MPICL.

Fibre bundle	Lateromedial width (mm)	Dorsopalmar depth (mm)	Length (mm)
Dorsolateral	2.8 ±0.6	8.1 ±1.6	15.5 ±1.0
Dorsomedial	2.3 ±0.1	5.8 ±0.7	10.5 ±0.2
Palmarolateral	2.4 ±0.2	5.25 ±0.7	11.5 ±0.2
Palmaromedial	2.7 ±0.3	7.5 ±0.2	8.0 ±0.5

With all attachments to CR removed except for the MPICL, it was not possible to move CR dorsally or palmarly relative to C3 and C2, but a small degree of hyperextension was possible. The fibres of both lateral bundles ran in a proximodorsal to palmarodistal direction, while those of the dorsomedial bundle ran in a more proximodistal direction. The fibres of the palmaromedial bundle ran proximopalmar to distodorsal (Figure 4). Six of 10 joints had some degree of MPICL tearing. Two of these joints were from the same horse. All tears extended palmarly 1-3 mm from the most dorsal margin of the ligament and involved only the dorsolateral bundle. The most dorsal aspect of the ligament often touched or closely apposed the articular margin of C3 and all but one of the tears was at this location. In all joints a substantial intercarpal ligament was present between CR and CI. This ligament attached to the palmaromedial aspect of CI and ran dorsomedially to attach to the dorsolateral aspect of CR.

Discussion

The findings of the present study differ from those of Phillips and Wright (1994) who described the distal attachment of the LPICL as predominantly on C4, with a smaller attachment on C3. This is in direct contrast to the present study where the distal attachment was observed almost wholly on C3, with only a few fibres attached to C4 (Figure 2). Phillips and Wright (1994) also described only two separate fibre orientations of the MPICL, which correspond to the dorsomedial and dorsolateral bundles identified in the present study. Although the attachments of the palmaromedial and palmarolateral bundles were described (Phillips and Wright 1994), the separate orientation of the palmaromedial bundle was not. In the specimens in the present study the four bundles of the MPICL were variably defined, but they were consistently observed in all 10 joints.

Despite the increasing recognition of pathology in the MPICL and LPICL (Kannegieter and Colgan 1993, Phillips and Wright 1994), the function of these ligaments, and the cause of ligament damage remains unclear. Bramlage *et al* (1988) proposed that the intercarpal ligaments dissipate axial forces by allowing abaxial translation of the carpal bones. This hypothesis has been confirmed in a biomechanical study of the loaded carpus (Young *et al* 1994). Bramlage *et al* (1988) also suggested that carpitis develops if the intercarpal ligaments do not adapt to increased loads associated with training. It has also been suggested that tearing of the intercarpal ligaments was caused by overextension with concomitant damage to the palmar carpal ligament (McIlwraith 1992). A more specific role has been suggested by Phillips and Wright (1994). Based on the predominant direction of fibres, they proposed that

the MPICL resists lateral and dorsal displacement of CR, at the limit of midcarpal joint extension.

The equine carpus is locked in extension during the stance phase (Hjertén and Drevemo 1994). There appears to be no studies measuring the forces at the joint during this phase, however Hjertén an Drevemo (1987) reported a method for calculating both transverse and longitudinal external force at the joint from ground reaction forces. Using their data, obtained from a galloping horse, it would appear that a transverse force, acting palmarly on the proximal metacarpus, develops rapidly just before midstance and reaches a maximum, of approximately 2.1 kN just after midstance. Unfortunately the calculation of internal forces from ground reaction forces is not possible (Hjertén and Drevemo 1987). In a further study Hjertén and Drevemo (1994) demonstrated shear forces at the carpus at heel strike. Therefore there are transverse forces generated at the midcarpal joint which must be resisted by the soft tissue structures spanning the joint.

Medial palmar intercarpal ligament

From its attachment on C2 and C3, the MPICL runs predominantly in a proximodorsal direction, while the lateral bundles run medially as well. From this observation it is reasonable to assume that the ligament resists dorsal, and to some degree medial displacement of CR. This was confirmed by sectioning all other attachments of CR. On the medial side CR is strongly anchored by the medial collateral ligament, the deep fibres of which diverge proximodorsally from a palmar attachment on C2 to the medial aspect of CR, mirroring the

dorsal and palmarolateral bundles of the MPICL. Therefore dorsal displacement of CR is prevented by the MPICL on its lateral aspect and by the MCL on its medial aspect (Figures 1 and 4). The orientation of the fibres of the palmaromedial bundle suggest that it resists palmar displacement of CR. This was demonstrated when CR was held in extension.

The intercarpal ligament joining CR and CI runs from the palmar aspect of CI to the dorsal aspect of CR. This allows CR to flex and extend about C3 and C2 independently of CI, while being prevented from displacing dorsally relative to CI when in full extension. It also indicates that there must be strong forces acting to displace CR dorsally as well as medially in relation to the CI. Compared to the medial collateral ligament and the intercarpal ligament between CR and CI, the MPICL is relatively insubstantial. It would therefore appear that the MPICL is the weak link in the restraining apparatus preventing dorsal displacement of CR. This could explain the predisposition for injury of the MPICL. Also, isolated injury to the MPICL is likely to cause medial rotation of the dorsal face of CR which would still be firmly anchored on the medial side by the medial collateral ligament. Overriding of the dorsomedial articular margin of CR relative to C3 could produce cartilage erosion and subchondral bone changes on the dorsomedial aspect of CR and radial facet of C3. Remodelling of the dorsal articular margin of CR in joints with MPICL tearing has been observed by Phillips and Wright (1994).

The MPICL probably has only a minor role in preventing overextension of the midcarpal joint. Except for the palmaromedial bundle, most of the ligament is positioned too far dorsally to act with any mechanical advantage (Phillips and Wright 1994). The primary

structures limiting overextension of the midcarpal joint are the collateral ligaments and the palmar carpal ligament (Bramlage *et al* 1988) all of which are considerably larger than the MPICL and are positioned more palmarly.

Lateral palmar intercarpal ligament

The LPICL is a simpler structure than the MPICL and although there are no obvious anatomical divisions, the fact that there may be differences in fibre tension in extension suggests that there are some differences in fibre function between the dorsal and palmar parts. The proximal attachment is broader than the distal attachment and it tilts laterally and dorsally as it courses towards CU, suggesting that it restricts both dorsal and lateral displacement of CU and CI, which function as a unit (Leach and Dyson 1988).

Dorsomedial intercarpal ligament

The DMICL was identified in all specimens. It has been described as an intercarpal ligament (Martin and McIlwraith 1985, Selway 1991), and as a synovial plica (McIlwraith 1990). In all specimens there was a distinct proximal and distal attachment of fibres indicating that it was a discrete ligament. The observed proximal attachment was similar to that described by Selway (1991). However, distally the ligament was said to blend with the fibrous joint capsule (Selway 1991). In the present study it was found to run in close association with the joint capsule for the distal two thirds of its length, but it remained a discrete ligament. It was also closely associated with the medial collateral ligament and appeared to form the most dorsal

part of that structure.

The function of the DMICL is more difficult to determine. Its position and the direction of its fibres suggest it may also prevent dorsal displacement of CR, but the ligament was flaccid when the joint was in extension. Selway (1991) observed enlargement of the ligament in a number of horses during arthroscopy and this was suggested as a primary cause of joint disease. This was not observed in any of the specimens in the present study. However, I have observed this in clinical cases, commonly in association with MPICL tearing (Chapter 6). It is therefore possible that excessive dorsal displacement of CR caused by rupture of the MPICL could bring this ligament into tension, with subsequent hypertrophy, in an attempt to resist this abnormal motion but such conclusions cannot be drawn from the present study. Enlargement of the ligament may be secondary to joint instability and not a primary cause of joint disease. Enlargement may also be an indication of generalised hypertrophy of the MCL.

The DMICL was not observed to impinge on the articular surface of CR as described by Selway (1991). In all specimens the DMICL was observed to attach proximal to the articular surface of CR. The site of attachment is a common site for osteochondral damage. Adherence of synovial membrane to osteochondral defects often occurs and it is possible that this is why the ligament is observed to attach directly to the articular surface of CR during arthroscopy (Selway 1991, Wright 1996).

Ligament injury

Determining the frequency of palmar intercarpal ligament damage was not a primary objective of this study, but a high incidence of MPICL tearing was noted. Kannegieter and Colgan (1993) found some damage in 41%, while Phillips and Wright (1994) observed some damage in 70% of joints undergoing midcarpal joint arthroscopy. A similar incidence was found in this study although these injuries tended to be minor with the most severe only involving 17% of the ligament. Detailed histories of the horses used were not available, but none were euthanased for carpal lameness and none had obvious signs of carpal disease. It is therefore possible that slight tearing of the dorsal aspect of the MPICL is clinically insignificant.

Tearing of the MPICL was always observed at the most dorsal aspect of the dorsolateral bundle, as has been noted previously by McIlwraith (1992). Interestingly the ligament is at its thinnest at this point and the injured part of the ligament passed very close to the palmar proximal articular margin of C3. It is possible that stretching of the ligament, caused by minor damage could allow CR to displace dorsally and the MPICL to abrade on the articular margin of C3. If tearing of the ligament is caused by dorsal displacement of CR, the dorsal fibres of the ligament will tear before the more palmar fibres, as they are inclined more dorsopalmar than the rest of the ligament. If overextension was the cause of ligament tearing as suggested by McIlwraith (1992), damage to the most palmar aspect of the ligament would be more common. Tearing of the palmar aspect of the ligament was not observed in any of the specimens studied.

There is evidence that more subtle forms of damage to the dorsolateral bundle of the MPICL occur. Laxity of the dorsal aspect of the dorsolateral bundle was observed in a number of joints with damaged ligaments. These fibres had probably reached their yield point but had not completely failed, therefore surgeons need to be aware that damaged ligaments may not appear grossly torn. Also the greater variation in dimensions of the dorsolateral bundle, when compared to the rest of the ligament, may reflect thickening in some specimens due to chronic damage and fibrosis. McIlwraith (1992) attempted to assess ligament laxity with the joint in flexion during arthroscopic surgery. The findings of the present study indicate that it is normal for the ligament to be lax in this position. It is probably more relevant to assess laxity with the joint in extension, but this is impossible with arthroscopy.

Damage to the LPICL was observed less frequently, which is consistent with the findings of Kannegieter and Colgan (1993) and Phillips and Wright (1994). McIlwraith (1992) found no LPICL tears in 45 midcarpal joints with MPICL tearing, but did not state whether all lateral ligaments were examined. There are several possible reasons for the lower incidence of observed LPICL damage. The LPICL is both a wider and simpler structure than the MPICL and it should therefore be intrinsically more resistant to injury. A much smaller proportion of the ligament can be observed arthroscopically, and because the palmar aspect is under the most tension when the joint is in extension, excessive dorsal displacement of CI and CU unit, or overextension of the midcarpal joint will result in tearing of the palmar aspect of the ligament first. This area cannot be visualised arthroscopically, so may go undiagnosed.

Conclusion

This study has defined in greater detail the structure and function of the palmar intercarpal ligaments and the DMICL. The structure of the MPICL is complex and it is proposed that its primary role is as a lateral restraint to dorsal and medial displacement of CR. The LPICL is simpler in structure but performs a similar role with CI and CU unit. As conclusions based on passive range of motion studies in separated cadaver specimens have limitations, further studies of the biomechanics of these ligaments are necessary to confirm and better define their function.

Part 2.

THE ROLE OF THE PALMAR INTERCARPAL LIGAMENTS IN THE RESTRAINT OF DORSAL DISPLACEMENT OF THE PROXIMAL ROW OF CARPAL BONES.

Introduction

Tearing of the medial and lateral palmar intercarpal ligaments (MPICL and LPICL) in the midcarpal joint of racing horses has only recently been recognised (Kannegieter and Burbidge 1990). The function of these ligaments and their clinical significance has been the subject of speculation, based on clinical observations (McIlwraith 1992, Kannegieter and Colgan 1993, Phillips and Wright 1994). Their role and importance in carpal biomechanics is unknown, yet pathology is commonly observed. One of the reasons for a lack of information about the role of the intercarpal ligaments in biomechanics of the carpus is because there are very few biomechanical studies of midcarpal ligament function in the horse (Young *et al* 1994).

In part 1 of Chapter 5 it was proposed that the main function of the MPICL and LPICL in the midcarpal joint is to prevent dorsal displacement of the proximal row of carpal bones and that it is this motion that causes tearing of the MPICL. Descriptions of carpal function have focused on axial forces (Bramlage *et al* 1988), yet transverse forces at the carpus have been reported (Hjertén and Drevemo 1994) and must be attenuated by the soft tissue structures

spanning the joint. This paper describes a biomechanical study of cadaver equine carpi in which the relative contributions of the ligaments of the midcarpal joint to the restraint of dorsal displacement of the proximal row of carpal bones were measured.

Materials and methods

Specimen preparation

Eight equine forelegs were obtained from Thoroughbred horses between two and five years of age euthanased for reasons unrelated to carpal disease. These were stored at -10°C until required. After thawing overnight, the skin and all soft tissue structures above and below the carpus were removed, including the palmar retinaculum of the carpal canal, and the contents of the canal. Using a band saw the radius was cut 25 cm above the antebrachiocarpal joint and third metacarpal bone was cut 25 cm below the carpometacarpal joint. The accessory carpal bone was removed by severing its ligamentous attachments as close to its surface as possible. The joints were then moistened in 0.9% saline, wrapped in plastic and stored at 4°C until tested the following day. Immediately before testing the dorsal capsule of the midcarpal joint was removed and a flattened area created on the dorsal aspect of CR, and immediately adjacent on C3, with a scalpel and file. The medial and lateral palmar intercarpal ligaments were examined and any signs of tearing recorded. During testing joints were kept moist with swabs soaked in 0.9% saline.

Biomechanical testing

Testing was performed with a universal testing machine (model 4302, Instron Ltd, Buckinghamshire, England). A 10 kN static load cell measured restraining force. Output of the cell was set at 50%. Joints were mounted in the machine in full extension using two clamps which were designed to firmly hold both the radius and metacarpus (Figure 1). A

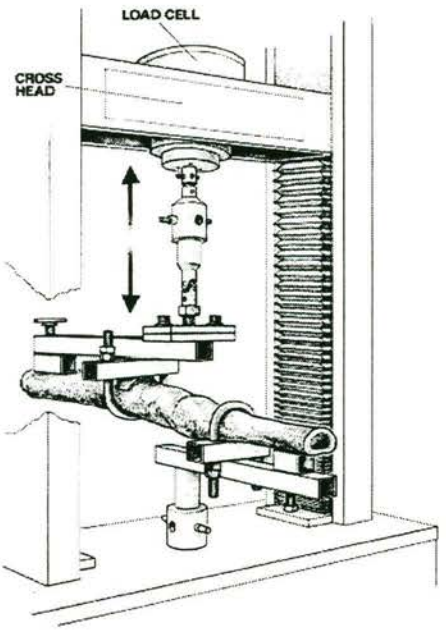

Figure 1.

An equine carpus mounted in the testing machine.

universal joint was incorporated into the upper clamp so that forces acting on the load cell were unidirectional. The lower clamp holding the metacarpus was fixed while the upper clamp attached to the load cell on the crosshead. Care was taken to align the joints so that no

rotation occurred when load was applied.

With the joint firmly held in the machine, the crosshead was displaced upwards until full extension was reached as determined visually. From this position a palmar-dorsal displacement of the radius was performed to a maximum dorsal displacement of 10 mm. This was repeated 30 times to minimise time-dependant force relaxation (Butler *et al* 1980). During the last five cycles the change in maximum restraining force was less than 0.02% in all joints. The joint was again positioned at the point of maximum extension and two dial gauges (Mercer, type 51, Sheffield, England), accurate to 0.01 mm, were placed on the flattened areas on the dorsal surface of CR and C3.

The radius was dorsally displaced to 10 mm and the resulting displacement at the joint surface measured. The joint was then cycled four times at a displacement rate of 200 mm/min, with force and displacement recorded 10 times a second. Following this the measurement of maximum joint displacement was repeated. The fourth cycle was used for all measurements.

The joint was removed from the testing machine, with the clamps still attached, and a ligament or pair of ligaments were sectioned. It was then replaced and the measuring procedure repeated. This was done until no ligaments remained. The ligaments tested were the medial collateral ligament and lateral collateral ligament, the palmar carpal ligament and the MPICL and LPICL. The dorsomedial intercarpal ligament was included as part of the medial collateral ligament. In four joints the medial collateral ligament and lateral collateral

ligament were sectioned first, followed by the MPICL and LPICL, while in four joints the medial collateral ligament and lateral collateral ligament were sectioned followed by the palmar carpal ligament. Displacement of the crosshead was reduced to eight mm following sectioning of the collateral ligaments and six mm following sectioning of the palmar carpal ligament or MPICL and LPICL because actual joint displacement increased.

Data analysis

The data generated from the fourth cycle of each testing procedure was used to produce a load-displacement curve. Displacement data from the crosshead movement was converted to joint displacement by dividing the maximum joint displacement by the maximum crosshead displacement. Maximum joint displacement was taken as the average of the two displacements measured before and after the measurement cycle. This produced a constant with which all the other crosshead displacement readings were multiplied. A specimen prepared identically to those tested was used to determine the relationship between crosshead and joint displacement. Joint displacement was measured with crosshead displacement increased by 1 mm increments up to 10 mm. The results showed linearity with r^2 values ranging from 0.997 with the PCL and collateral ligaments sectioned to 0.999 for the intact joint. Linear regression was performed on the load-displacement curves for displacements greater than 1 mm to produce an equation for the relationship between load and displacement at which point all appeared linear.

For each test the restraining force at a joint displacement of 1.5 mm was determined using the

regression equation. A displacement of 1.5 mm was chosen retrospectively as this was the maximum displacement reached by all joints. The contribution of each ligament to restraining dorsal displacement of the proximal row of carpal bones at a displacement of 1.5 mm could then be calculated as the percentage change in restraining force after that ligament was sectioned.

The relative contributions of each ligament or ligament pair tested were compared with a one way analysis of variance and post hoc comparisons were performed by determining the least significant difference. Analysis of variance was also used to determine the effect of the order of sectioning

Cross-sectional area

To determine the relative cross sectional area of the ligaments tested, five frozen Thoroughbred carpi from horses aged 2-5 years were sectioned at the level of the midcarpal joint with a band saw. Dimensions of the ligaments were measured with a digital calliper (Mitutoyo Series 500, Tokyo, Japan). Both width (lateral-medial) and depth (dorsal-palmar) were measured at 1 mm intervals and mean width multiplied by mean depth to derive cross-sectional area.

Results

Eight joints were tested. Before testing a tear extending 4 mm from the dorsal aspect of the MPICL was observed in joint 4, while 1 mm dorsal tears were detected in joints 1 and 3. In joint 3 slight tearing of the intercarpal ligaments was audible at a dorsal joint displacement of 1.95 mm, with all other ligaments sectioned.

In all joints tested, displacement was observed in the three carpal articulations. The CI appeared to displace dorsally slightly more than CR. The CR was observed to rotate medially as it moved dorsally independent of CI. Slight bending of the radius and third metacarpal bone was also apparent.

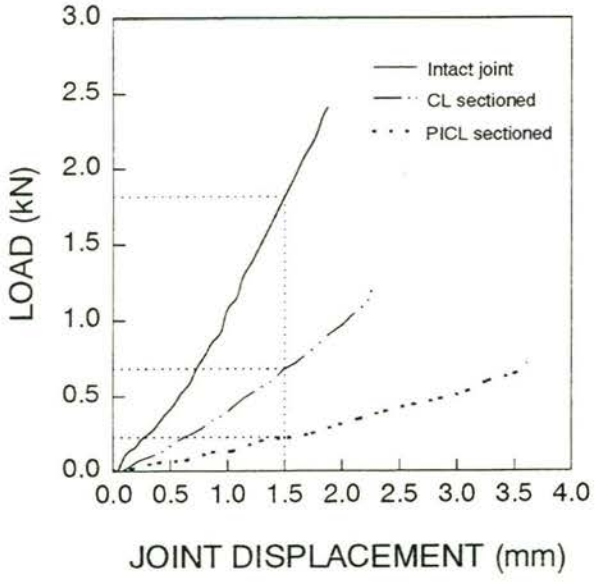

Figure 2.

A typical load displacement curve for dorsal displacement of the proximal row of carpal bones of the midcarpal joint.

In all joints load increased exponentially with initial displacement, but for displacements greater than 1 mm, r^2 values for linear regression analysis ranged from 0.978 to 0.999, indicating a strong linear relationship (Figure 2). The mean restraining force (±sem) for the intact joints was 1.92±0.12 kN. The mean percentage (±sem) contributions of the ligaments studied to the restraint of dorsal displacement were 62.8±3.4 for the collateral ligaments, 14.5±1.4 for the PCL and 22.7±2.2 for the palmar intercarpal ligaments (Figure 3).

In all joints the collateral ligaments were the major contributors to restraining dorsal displacement of the proximal row of carpal bones, with the percentage contribution significantly greater than the other ligaments ($p<0.001$). In all joints the palmar intercarpal ligaments contributed a greater proportion than the PCL, and this was significant ($p<0.05$). There was no significant effect of the order of sectioning on the percentage contributions of each ligament and no significant difference between joints with MPICL tearing and those without. Mean percentage contribution of the palmar intercarpal ligaments in joints with tearing of the MPICL was 20.2%, while in those without it was 26.9%.

When the mean cross-sectional area of each ligament or ligament pair was expressed as a percentage (±sem) of the total ligamentous cross-sectional area at the level of the midcarpal joint, the palmar intercarpal ligaments made up 9.0±0.3%, the PCL 27.1±3.0% and the collateral ligaments 63.8±2.8%. Figure 3 shows both mean percentage contribution to restraint of dorsal displacement of the proximal row of carpal bones and mean percentage of the total ligamentous cross-sectional area of the carpus for each ligament.

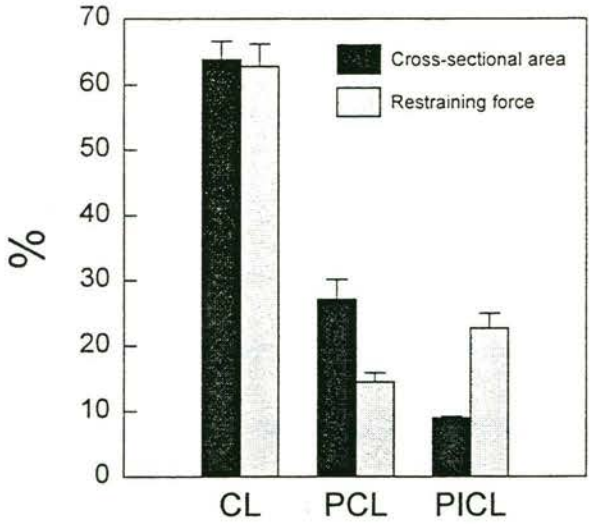

Figure 3.

Mean percentage cross-sectional area and percentage contribution to restraint of dorsal displacement of the proximal row of carpal bones of the ligaments of the midcarpal joint.

Discussion

Methodology

The method used to determine the contributions of the ligaments of the midcarpal joint to the restraint of dorsal displacement of the proximal row of carpal bones, is based on that used by Butler *et al* (1980) to determine the ligamentous restraints to anterior and posterior drawer in the human knee. By using a set joint displacement, and measuring the change in restraining force before and after cutting a ligament, the relative contribution of that ligament can be

determined. The same starting point and the same end point is used for each ligament so the results are the same regardless of the order in which they are sectioned (Butler *et al* 1980). The results here support this, although only two orders of sectioning were possible due to difficult access to the palmar intercarpal ligaments of the midcarpal joint prior to sectioning the collateral ligaments.

The collateral and intercarpal ligaments were sectioned as pairs to maintain joint symmetry. If only one of these ligament pairs was sectioned, the joint would tend to rotate when the radius was displaced dorsally, making the measurement of joint displacement difficult. For the same reason the ligamentous attachments of the accessory carpal bone were not included in the study. The PCL is unpaired but is positioned axially. Therefore soft tissue structures which span the midcarpal joint that were not assessed were the dorsal joint capsule, accessorioquartal ligament and accessoriometacarpal ligament. The dorsal capsule is flaccid with the joint in extension so it is likely to provide very little restraining force. The two ligaments of the accessory carpal bone are also unlikely to have any major effect as they do not attach to the proximal row of carpal bones (Sisson 1975).

Joints with intercarpal ligament tearing were included in the study for two reasons. Firstly there was no significant difference between joints with ligament tearing and joints without ligament tearing. In fact, the mean percentage contribution of the palmar intercarpal ligaments was slightly greater in joints with ligament tearing. The tears involved only a very small proportion of the total palmar intercarpal ligament cross-sectional area. Secondly, it is impossible to say which ligaments are normal just by gross examination. The large variation

in size of the dorsolateral bundle of the MPICL, observed in Part 1 may be caused by pathology rather than individual variation. In some joints the dorsolateral bundle is very small. Degeneration and complete loss of intra-articular ligaments has been observed in other species (Hefti *et al* 1991).

Joint Displacements

Measurements of maximal joint displacement were made at the joint surface. To determine joint displacement at all points, it was assumed that the relationship between crosshead displacement and joint displacement was linear. In the equine carpus the antebrachiocarpal and carpometacarpal joints are interposed between the testing machine and the midcarpal joint, meaning that the compliance of the ligaments of these joints and the metacarpus and radius need to be taken into account. Despite this the relationship between crosshead displacement and joint displacement was shown to be linear with and without ligaments sectioned.

Joint displacement was measured at the dorsolateral surface of CR and the immediately adjacent area on C3. This site was chosen in preference to the dorsomedial surface of CI because most pathology in the midcarpal joint occurs in this area and because the MPICL restrains CR, and is the most commonly damaged ligamentous structure. Therefore it was considered that displacement of CR was more clinically relevant. During testing CI was observed to displace further than CR indicating it is less well restrained than CR. Even though slight medial rotation of CR was observed, the dial gauges were positioned to measure

only dorsal displacement.

A maximum joint displacement of 1.5 mm was chosen because this was the maximum displacement that all joints were able to reach at all testing cycles. It also corresponded to a mean maximal restraining force of 1.92 kN which was thought to be similar to the forces generated in the carpus of galloping horses. Hjertén and Drevemo (1987) reported a method for calculating the external transverse force at the joint from ground reaction forces. Using their data, obtained from a 640 kg horse, the maximum transverse force at the carpus was approximately 2.1 kN in a caudal direction. Being obtained from ground reaction forces there will be some dissipation of this force as it passes up the leg, but it does give an indication of the magnitude and direction of forces. Unfortunately the calculation of internal joint forces from ground reaction forces is not possible (Hjertén and Drevemo 1987). A normal 450 kg Thoroughbred should generate transverse forces of lower magnitude. The slight tearing of the palmar intercarpal ligaments in joint 3 occurred at 1.95 mm indicating that a displacement of 1.5 mm was near the yield point of the ligaments.

Cross sectional area

The measurement of cross-sectional area of ligaments is difficult. A number of techniques have been described including the use of an area micrometer (Butler *et al* 1986), laser micrometer (Lee and Woo 1988), or polymethylmethacrylate casts (Race and Amis 1994). None of these methods was considered practical because of inaccessibility of the palmar aspect of the midcarpal joint. Access to all surfaces of each ligament was not possible without

extensive dissection. By using frozen sections of joints, all ligamentous structures could be accessed without disturbing their anatomical relationships and with a large proportion of each ligament under tension. However this meant that some parts of some ligaments were not sectioned perpendicular to their long axis. The majority of the fibres of the collateral ligaments and the PCL are aligned proximodistally, but both the MPICL and LPICL have large proportions which are aligned obliquely to the joint surface (Chapter 5, Part 1). The cross-sectional area of the palmar intercarpal ligaments was therefore likely to be overestimated.

Clinical relevance

It has been proposed, based on clinical observations, that one of the main functions of the intercarpal ligaments is dissipation of axial force during high joint loading (Bramlage *et al* 1988). This has been confirmed in a biomechanical study (Young *et al* 1994). Transverse or shearing forces, primarily acting to displace the radius dorsally in relation to the proximal metacarpus, have also been demonstrated at the carpus at and soon after heel strike (Hjertén and Drevemo 1987). It was proposed that the palmar intercarpal ligaments play a role in restraint of these forces based on the predominant direction of their fibres. However, it is important to understand that the restraint of dorsal displacement of the proximal row of carpal bones in the live horse is not solely because of ligamentous structures. Joint congruency is also an important factor in joint stability and is particularly important under high axial loading as demonstrated in the human knee (Hsieh and Walker 1976). Consistent with this, transverse accelerations at the carpus have the greatest magnitude very early in the stride

phase before axial loading has peaked (Hjertén and Drevemo 1987). This early phase of the stride may therefore be a time when the joint is relatively unstable and reliant on the ligamentous structures of the joint for restraint of transverse forces. The testing procedure described did not allow determination of the effect of axial loading on dorsopalmar stability of the midcarpal joint.

The force-displacement curves obtained were typical for bone-ligament-bone units (Noyes *et al* 1974a). The initial non-linear toe region correspond to straightening of collagen fibres and loss of crimp. This was followed by a linear region which ended with failure of some fibres (Noyes *et al* 1974a). The mean length of the LPICL and the various bundles of the MPICL range from 10.5 to 15.5 mm (Chapter 5, Part 1). Therefore the tearing detected in joint 3 at 1.95 mm corresponds to a strain of 12% to 19% in these ligaments. Primate anterior cruciate ligaments have been shown to begin to fail at about 40% strain (Noyes *et al* 1974b). The relatively low compliance of the palmar intercarpal ligaments reflected the high stability of the midcarpal joint in the extended position and may explain their susceptibility to injury. The collagen fibril diameter of the dorsolateral bundle of the MPICL is less than other carpal ligaments, and this has been proposed as a reason for its low compliance (Firth *et al* 1991).

These results demonstrate that despite the relative small size of the palmar intercarpal ligaments, they play an important role in the midcarpal joint, along with the collateral ligaments, in the restraint of dorsal displacement of the proximal row of carpal bones. Together their cross sectional area makes up 9% of the total ligamentous structure of the midcarpal joint, yet their contribution to the total restraint of dorsal displacement is 22.7%. In

all joints, the palmar intercarpal ligaments contributed a greater proportion of the restraining force than the PCL which is three times as large, indicating that prevention of dorsal displacement is not the primary role of the PCL. The CLs were the major contributors to the restraint of dorsal displacement. Together they were seven times larger than the palmar intercarpal ligaments combined, yet contributed less than three times the restraining force.

Dorsal displacement of CR is restricted on the medial side by the MCL and on the lateral side by the MPICL (Chapter 5, Part 1). If, as shown in the present study, the MCL is the stronger of the two ligaments, then CR should rotate medially with dorsal displacement, as we observed. These observations also emphasise that the MPICL is likely the weak link in the restraining apparatus of CR and the most prone to injury with excessive forces on CR.

Intra-articular ligament damage has been reported in other species to cause degenerative joint disease (Brandt *et al* 1991). However the rate of degeneration in cranial cruciate deficient dogs is very slow, despite it being the major contributor to restraint of cranial drawer, as demonstrated in the human knee where it provides 85% of the restraining force (Butler *et al* 1980). As shown in the present study the MPICL of the horse is relatively less important in overall joint function than the cranial cruciate ligament in dogs, so it is possible that degeneration resulting from MPICL tears would proceed extremely slowly. This is supported by unpublished work of CB Little (personal communication) in which no cartilage damage could be produced in horses exercised following arthroscopic sectioning of the MPICL.

Conclusion

The present study has demonstrated that the major restraint of dorsal displacement of the proximal row of carpal bones is the collateral ligaments, while the palmar intercarpal ligaments play an important role despite their relatively small cross sectional area. These observations are consistent with the theorized role of the intercarpal ligaments based on the predominant orientation of their fibres (Phillips and Wright 1994, Chapter 5, Part 1).

CHAPTER 6

THE INTERCARPAL LIGAMENTS OF THE MIDCARPAL JOINT: CLINICAL OBSERVATIONS IN 32 RACING HORSES WITH MIDCARPAL JOINT DISEASE

Introduction

Tearing of the medial palmar intercarpal ligament (MPICL) and lateral palmar intercarpal ligament (LPICL) of the midcarpal joint, has only been recognised with the advent of routine arthroscopic surgery. However, MPICL tearing is common in horses with midcarpal joint disease. The frequency of MPICL tearing has been reported as high as 41% (Kannegieter and Colgan 1993) and 70% (Phillips and Wright 1994), but tearing of the LPICL is much less commonly observed. Despite the increasing recognition of MPICL injury, its cause is poorly understood. Both McIlwraith (1992) and Kannegieter and Colgan (1993) found that complete rupture of the ligament caused lameness which could be localised to the midcarpal joint, but the mechanism for this is unknown and the relationship between ligament tearing and other forms of joint pathology is unclear.

An intercarpal ligament on the dorsomedial aspect of the midcarpal joint has been described by Martin and McIlwraith (1985) and Selway (1991). Hypertrophy of the ligament was observed in a high proportion of horses during arthroscopic examination, and it was proposed as a cause of midcarpal joint disease in racing horses (Selway 1991). Despite this, evidence for its role in the pathogenesis of midcarpal joint disease is lacking.

The aim of this prospective study was to characterise the clinical features of intercarpal ligament pathology and to determine the relationship between palmar intercarpal ligament tearing, DMICL hypertrophy and other intra-articular lesions.

Materials and methods

Intercarpal ligament injury, associated bone or cartilage pathology and clinical signs of carpal joint disease were recorded in 32 horses admitted to the University of Sydney Veterinary Teaching Hospital for arthroscopy of the midcarpal joint between April 1993 and September 1994. The observations and grading systems are detailed in the general materials and methods section (Chapter 2).

Data analysis

Spearman rank order correlations were performed between the various observations with significance set at $p<0.05$. As individual joints from bilaterally affected horses were not independent observations the analysis was performed both with all joints included, and with only the left or right joint from bilaterally affected horses included on separate occasions. A Mann-Whitney U test was used to compare the findings in joints with grade 1, or no visible MPICL tearing, with joints with grade 2-4 MPICL tearing, as well as the severity of injury between left and right joints.

Results

Fifty three midcarpal joints of 32 horses were examined. Four horses were racing Standardbred pacers (seven joints) and 28 horses were racing Thoroughbreds (47 joints). Horse ages ranged

from two to five years. Palmar intercarpal ligament tearing was identified in 30 joints of 22 horses. Tearing of the MPICL alone occurred in 21 joints of 16 horses, tearing of the LPICL alone occurred in three joints of three horses, and tearing of both ligaments in the same joint occurred in six joints of five horses.

Some tearing of the MPICL was present in 27 joints in 20 horses, and tearing of the LPICL was evident in nine joints in seven horses. Of the 27 MPICL injuries, 14 were grade 1 tears, two were grade 2 tears, six were grade 3 tears and five were grade 4 tears. The LPICL injuries consisted of seven grade 1 tears and two grade 2 tears. Both grade 2 tears occurred in horses with grade 4 MPICL injuries. Moderate to severe subchondral bone damage and no MPICL tearing was observed in one joint, with grade 2-4 ligament tearing and no or mild subchondral bone damage in the opposite joint in two horses.

Tearing of the MPICL was observed in 13 left and 14 right midcarpal joints. For Thoroughbred racehorses, there was no significant difference between the severity of tearing in left and right midcarpal joints. There was also no significant difference in the degree of articular cartilage and subchondral bone damage between left and right joints. The frequency of MPICL tearing was similar in Thoroughbred and Standardbred carpi. Of the 47 Thoroughbred midcarpal joints examined, 23 had evidence of MPICL damage, whereas four of the seven Standardbred joints had evidence of damage. One horse with a grade 3 MPICL tear was re-examined by arthroscopy six months after the initial diagnosis. The ligament was not debrided at the initial examination. There was no evidence of healing at the second examination. The ligament was not debrided at the initial examination.

There was no significant correlation between the degree of MPICL damage and the severity of carpal valgus or carpal rotation. Too few horses were observed with carpal hyperextension conformation to allow statistical analysis. Of the three horses with grade 2 carpal hyperextension, one had a grade 4 and a grade 2 MPICL tear, while another had a grade 4 and a grade 1 MPICL tear.

There was also no correlation between the severity of clinical signs recorded and the degree of MPICL or LPICL tearing. However, there was a significant correlation between the degree of lameness and articular cartilage damage ($R=0.30$, $p<0.05$), and the degree of swelling ($R=0.32$, $p<0.05$) and pain on flexion ($R=0.27$, $p<0.05$) with subchondral bone damage. In 22 horses where bilateral midcarpal joint arthroscopy was performed, damage to the left MPICL was different from damage to the right MPICL in 12 horses. In five of these horses the lameness was worse in the limb with the more severely affected ligament, whereas in seven horses the lameness was more evident in the limb with the less severely affected ligament.

No significant correlation was found between the degree of damage to the MPICL and the degree of articular cartilage or bone damage, but joints with grade 2-4 MPICL tearing had significantly less ($p<0.05$) cartilage and bone damage than joints with grade 1 or no detectable ligament damage. There was a significant inverse relationship between fracture score, as assessed radiographically, and ligament damage ($R=-0.31$, $p<0.05$). When normal joints (no MPICL tearing or subchondral bone damage) were excluded, there was a significant negative correlation between ligament damage and fracture score ($R=-0.53$, $p<0.001$), bone defect ($R=-0.55$, $p<0.0001$) and cartilage damage ($R=-0.36$, $p<0.05$), (Table 1). When only the left joints from

bilaterally affected horses were included, the significant negative correlation between ligament damage and fracture score ($R=-0.66$, $p<0.0001$), and bone defect ($R=-0.49$, $p<0.01$) remained. When only right joints were included fracture score ($R=-0.53$, $p<0.01$), bone defect ($R=-0.65$, $p<0.001$) and cartilage defect ($R=-0.44$, $p<0.05$) were significantly negatively correlated with ligament damage.

Table 1.

Relationship between medial palmar intercarpal ligament damage and subchondral bone damage in the midcarpal joint of racing horses.

Medial intercarpal ligament damage	Subchondral bone damage			
	0	1	2	3
0	7	4	9	6
1	2	4	7	1
2	1	1	-	-
3	0	4	1	1
4	2	3	-	-

The DMICL was identified in all joints. In 18 joints, the ligament was enlarged (grade 2 or 3), with marked enlargement noted in five of these joints. There was a significant correlation

between MPICL damage and hypertrophy of the DMICL ($R=0.35$, $p<0.01$). The degree of enlargement of the DMICL was significantly greater ($p<0.001$) in joints with grade 2-4 tearing, than joints with no, or grade 1 tearing. There was no correlation between DMICL hypertrophy and articular cartilage damage or subchondral bone damage. A significant correlation between the number of weeks in training and DMICL hypertrophy was observed ($R=0.41$, $p<0.01$). The DMICL was not observed to impinge on the articular surface in any of the joints studied, although in a number of joints it adhered closely to damaged subchondral bone at the dorsal articular margin of the distal aspect of the radial carpal bone (CR).

Discussion

This paper describes what appears to be the first prospective study of intercarpal ligament tearing in the midcarpal joint of racehorses undergoing arthroscopy. The frequency of MPICL tearing was 51%, while the incidence of LPICL tearing was 18.5%. In previous studies the highest frequency of MPICL tearing (70%) has been observed in the United Kingdom (Phillips and Wright 1994), while the frequency in Australia (41%) (Kannegieter and Colgan 1993) is greater than in the United States (8.7%) (McIlwraith 1992). These differences may be attributed to a number of factors including racing surfaces, race distances, breeds involved and differing interpretation of tearing by surgeons.

Very few horses in this study had carpal hyperextension, but two of the three most affected horses had complete MPICL tears. Although carpal hyperextension did not appear to be

associated with osteochondral damage (Chapter 4) it may be associated with intra-articular ligament damage. Greater numbers of horses need to be examined to determine this. Differences in frequency and severity of ligament damage between left and right midcarpal joints may indicate an effect of racing direction on the pathogenesis of ligament injury. Standardbreds were not included in this comparison because they race in an anticlockwise direction in our practice area while Thoroughbreds race in a clockwise direction. There was no predisposition to injury for left or right joints in Thoroughbred racehorses in this study.

Arthroscopy remains the only means of diagnosing intercarpal ligament damage. The present study confirms that there are no characteristic clinical signs which will differentiate intercarpal ligament tearing from other carpal injuries. McIlwraith (1992) observed that horses with ligament tearing and osteochondral fragments had a greater degree of clinical compromise than was expected for the degree of osteochondral fragmentation. It was also noted that joints with ligament tearing and no osteochondral damage, had a greater degree of effusion than the opposite joint with osteochondral fragments (McIlwraith 1992). This was not observed in the present study. Even in horses with bilateral midcarpal joint disease there was no possibility of predicting which joint would have ligament damage. Despite these difficulties, intercarpal ligament tearing must be considered in horses with lameness localised to the midcarpal joint and no radiographic changes (McIlwraith 1991, Moore and Schneider 1994). Ultrasound has proven invaluable in the diagnosis of orthopaedic soft tissue disease, but the position of the MPICL, deep within the carpus and surrounded by bone, along with its complex structure make ultrasonic examination extremely difficult. The use of more advanced imaging techniques such as magnetic resonance imaging, which is used in the diagnosis of cruciate ligament tearing in humans (Liu *et al* 1994),

needs to be investigated.

Only two grade 2 LPICL tears were observed but these occurred in joints with grade 4 MPICL tears suggesting that moderate to severe intercarpal ligament injuries tend to go together. It was proposed in part 1 of Chapter 6 that the low incidence of LPICL tears observed by arthroscopy was because of the difficulty in viewing the ligament and that tearing of the palmar aspect of the ligament would tend to occur before the dorsal aspect. Therefore it is possible that some LPICL tears are undiagnosed. The observation of combined intercarpal ligament injury also demonstrates that injury to the MPICL is not always isolated, and injury to other extra-articular soft tissue structures may occur concurrently. Some of these structures are accessible with ultrasound, particularly the medial collateral ligament. However, Denoix and Audigié (1993) stated that desmitis of the medial collateral ligament was not easily observed with ultrasound due to its normal irregular echogenicity caused by the different orientation of fibrous planes.

An inverse relationship was demonstrated between MPICL tearing and osteochondral damage. This relationship was more pronounced when only those joints with some form of injury to either the subchondral bone, articular cartilage or MPICL damage, were considered. This was an unexpected finding as ligamentous damage and the resulting instability is usually associated with articular cartilage degeneration (Brandt *et al* 1991). However, as the relationship was stronger for subchondral bone damage, particularly fractures, than articular cartilage damage, it is probably more a reflection of bony change rather than cartilage degeneration. This relationship has not been previously demonstrated, but would explain why others have found no correlation between articular cartilage and ligament damage (McIlwraith 1992, Phillips and Wright 1994).

The only other study to specifically examine subchondral bone damage only recorded the presence or absence of fractures. The number and size of fractures was not recorded, or the extent of soft subchondral bone, so a true evaluation of subchondral bone was not made. Kannegieter and Colgan (1994) found that the frequency of ligament damage was higher in joints with severe osteochondral damage, but could find no correlation between the severity of osteochondral and ligament injuries. In the present study both the frequency and severity of ligament tearing was lower in joints with severe osteochondral damage.

In the horses in this study there were two common sites of failure in the midcarpal joint. The subchondral bone of the dorsal aspect of CR, and the MPICL. Rather than ligament rupture resulting in osteochondral damage due to joint instability and subsequent abnormal loading, it appears that the forces generated at racing speeds within the carpus result in either one site or the other predominantly failing (Table 2). This is consistent with observations that no articular cartilage damage occurred in horses trained on a treadmill following sectioning of the LPICL and MPICL (CB Little, personal communication). Our results also indicate that this apparent inverse relationship is due to both a direct interaction between ligament and subchondral bone damage within the same joint, and an interaction between both left and right joints. McIlwraith reported six cases similar to the two in our study which had moderate to severe subchondral bone damage to the distal margin of CR with mild or no detectable ligament tearing, but in the opposite joint there was a grade 3 or 4 ligament tear with no intra-articular fractures (McIlwraith 1992). There is evidence that carpal lameness in one limb changes the biomechanical variables in the opposite forelimb by increasing the horizontal ground reaction force, with no change in vertical force (Morris and Seeherman 1987). This could increase the risk of intercarpal ligament tearing in the

contralateral limb to one with chip fractures, without increasing the risk of subchondral bone damage.

Although there was no evidence that MPICL rupture results in significant osteochondral damage in the midcarpal joint it is highly unlikely that dorsomedial displacement of CR resulting from MPICL tearing is innocuous. Firstly, most of the joints with severe MPICL tearing had some degree of cartilage and subchondral bone damage. Also, although not observed in the present study, remodelling of the dorsodistal articular margin of CR has been shown to be associated with MPICL tearing (Phillips and Wright 1994). Degeneration of cartilage secondary to MPICL tearing is likely to develop slowly as the rate of degeneration following intra-articular ligament damage in other species is very slow (Brandt 1991). Secondly, MPICL rupture will place increased stress on other intercarpal ligaments, as demonstrated by the enlargement observed of the DMICL. Bramlage *et al* (1988) have proposed that desmitis of the intercarpal ligaments is a major cause of lameness.

There are few reports of the findings of followup arthroscopic examinations of torn MPICLs. Only one joint in the present study was re-examined, four months following the diagnosis, with no evidence of healing. Healing after six months rest has been observed by Firth *et al* (1991) in three of four joints re-examined and after 12 months in one joint by Kannegieter and Colgan (1993). The potential for, and the factors affecting healing need further investigation.

Enlargement of the DMICL was first described by Selway (1991) as a congenital condition, and proposed as a primary cause of carpal disease. The findings of the present study suggest that this

lesion is probably acquired in response to MPICL tearing and training. There was no evidence that DMICL enlargement was a primary cause of osteochondral fractures or articular cartilage damage. In part 1 of chapter 5 it was demonstrated that the DMICL formed the dorsal part of the deep part of the medial collateral ligament and the MPICL and the medial collateral ligament are the major ligaments preventing dorsal displacement of CR. In the normal joint the DMICL is flaccid in extension. With rupture of the MPICL the lateral restraint of CR is lost. The resulting increase in dorsomedial displacement of CR could induce changes in the medial collateral ligament and therefore in the DMICL. With acute MPICL tears, DMICL enlargement will not have sufficient time to occur, explaining the lower incidence of this latter lesion compared with MPICL tearing. Also a certain amount of exercise is required as suggested by the relationship between the duration of training and DMICL enlargement. Selway (1991) observed enlargement of the DMICL in 74% of cases undergoing midcarpal joint arthroscopy but did not examine the MPICL. The much lower incidence (33%) in the present study may be due to a difference in definition of ligament enlargement. In the present study there was no evidence of the ligament impinging on the articular surface as described by Selway (1991). Admittedly joint distension with fluid during arthroscopy will force the joint capsule and DMICL away from the articular surfaces, but some secondary changes in articular cartilage should have been observed where the DMICL is said to impinge palmar to CR attachment.

Conclusion

This study has confirmed that MPICL tearing is common in racing horses with midcarpal joint

disease. Tearing of the LPICL is less commonly observed, but this may not be a true indication of its prevalence. The prevalence of these lesions in the racing horses is unknown and should be further investigated. There was an apparent inverse relationship between the severity of MPICL tearing and osteochondral damage. Enlargement of the DMICL appears to be a secondary change rather than a primary cause of carpal disease.

CHAPTER 7

THE INTERCARPAL LIGAMENTS OF THE MIDCARPAL JOINT: A SURVEY OF GROSS PATHOLOGY IN RACING AND NON-RACING HORSES

Introduction

A high frequency of abnormalities of the intercarpal ligaments of the midcarpal joint has been observed arthroscopically in joints of racing horses presenting with clinical signs of midcarpal joint disease (Selway 1991, Kannegieter and Colgan 1993, Phillips and Wright 1994, Chapter 6). This observation has led to the suggestion that both tearing of the medial palmar intercarpal ligament (MPICL) (Kannegieter and Colgan 1994) and enlargement of the dorsomedial intercarpal ligament (DMICL) (Selway 1991) contribute to osteochondral damage in the midcarpal joint and are therefore significant clinical findings. There is however no direct evidence that DMICL enlargement is a significant problem in the carpus and only limited evidence that MPICL tearing is. McIlwraith (1992) found that horses that had joints with MPICL tearing had more severe clinical signs of joint compromise than would be expected based on the severity of osteochondral damage, although this was not supported by a prospective clinical study (Chapter 6). McIlwraith (1992) also reported that when greater than 50% of that part of the MPICL visible arthroscopically was torn, the prognosis for future racing was poor.

Although one of the major reasons that intercarpal ligament abnormalities are thought to be a significant finding is their high frequency in clinical cases, to my knowledge there are no reports of the frequency of intercarpal ligament tearing in the wider population of horses, and particularly in horses without evidence of carpal disease. If the incidence of intercarpal ligament tearing in this latter group of horses is high, it is less likely that these abnormalities are clinically important.

In Chapter 5 it was shown that there was a large variation in size of the dorsolateral bundle of the MPICL. It is not known whether this is individual variation or due to damage to the ligament and subsequent degeneration in some horses. The high incidence of tearing of the MPICL may be due to degeneration and weakening of the ligament as is observed in other intra-articular ligaments of other species (Vasseur *et al* 1985).

The purpose of this study was to determine the frequency of damage to the MPICL and the range of size of the DMICL and the bundles of the MPICL in Thoroughbred and Standardbred race horses, and horses of other breeds not subjected to high levels of training, excluding those with know carpal disease.

Materials and Methods

Carpi were collected from horses of a variety of ages which were euthanased for reasons unrelated to carpal joint disease. These were dissected as described in Chapter 2 to allow close examination of the intercarpal ligaments of the midcarpal joint. The dimensions of the ligaments and the individual bundles of the MPICL were measured as described in Chapter 2. Comparison of ligament dimensions between horses of various ages is complicated by the overall increase in size of the carpus with growth. To correct for the size of each carpus the dorsopalmar depth of C3 was measured in each joint and ligament dimensions were divided by this value. An estimate of cross-sectional area of each bundle of the MPICL was made by multiplying the lateromedial and dorsopalmar dimensions together. For each bundle the coefficient of variation (CV) of cross-

sectional area was calculated for horses two years and older.

Information obtained for each horse included the age, breed and sex, whether the horse had commenced training and whether it had raced. If the owner or trainer reported any history of carpal problems, the joints were not included in the study.

Statistical analysis

The effect of age and breed on ligament findings was determined by one way analysis of variance. Log transformation was performed where the data was not of normal distribution. Where transformation was not appropriate the Kruskal-Wallis ANOVA by ranks and median test was used. Relationships between variables were determined by Spearman rank order correlations.

Results

One hundred and forty two joints were examined from 72 horses. Age ranged from an unborn term foal to 18 years. There were 40 Thoroughbreds, 16 Standardbreds, and 15 horses of non-racing breeds which included four Arabs, two Appaloosas, four Ponies, one Australian Stockhorse, two Quarter horses and two Thoroughbred crossbred horses.

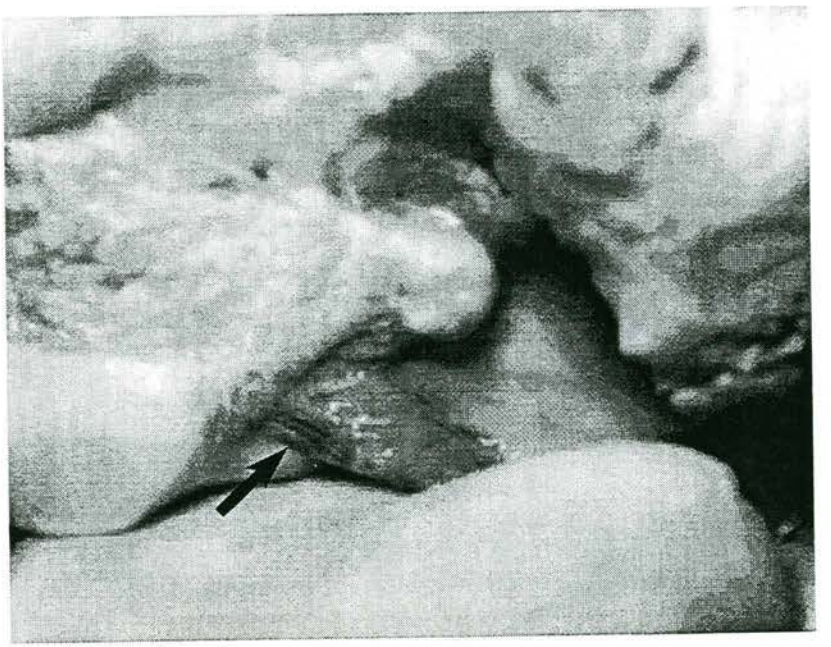

Figure 1.

Lateral view of a left adult carpus (intermediate carpal bone removed) showing subtle tearing of the dorsolateral bundle of the medial palmar intercarpal ligament at its attachment to the radial carpal bone (arrow). This is the most common site of damage.

Medial palmar intercarpal ligament

Tearing of the MPICL was observed in 99 of the 142 joints examined. In horses more than two years of age tearing was observed in 88 of 96 joints. All tears were of the most dorsal part of the ligament and in all but 12 joints, involved the dorsolateral part of the attachment to CR (Figure 1). In the remaining joints tearing was observed in the dorsal margin of the ligament midway between the proximal and distal attachments. The most severe tear extended 12.5 mm palmarly through the dorsolateral bundle into the dorsomedial bundle. Complete rupture of both dorsolateral and dorsomedial bundles (grade 4) was not observed. Severity of tearing in adult Standardbreds was significantly greater than in adult Thoroughbreds ($p<0.01$) and there was a

trend for a greater severity of tearing in Standardbreds than non-racing breeds ($p=0.06$) (Table 1). Tearing was not the only abnormality observed in MPICLs. Localised thickenings were also observed in a number of grossly intact dorsolateral bundles as well as longitudinal separation of fibres.

Table 1.

The frequency and extent of medial palmar intercarpal ligament tearing in the midcarpal joints of horses of different breeds and ages.

	Joints examined	Joints with MPICL tearing	Joints with grade 2-4 tearing	Mean depth of tearing (mm)
Total	142	101(71%)	56 (39%)	1.5 (±0.17)
Horses > 2 years	96	88 (92%)	55 (57%)	2.1 (±0.22)
Horses < 2 years	46	13 (28%)	1 (4%)	0.2 (±0.05)
Adult TBs	45	38 (84%)	23 (51%)	1.5 (±0.21)
Adult SBs	30	30 (100%)	20 (67%)	3.2 (±0.55)
Adult non racing breeds	21	20 (95%)	12 (62%)	1.7 (±0.27)

Tearing of the MPICL was not observed in foals less than four months of age but increased in severity significantly with age up to three years ($p<0.000001$) (Figure 2). The largest tear observed in a joint of a one year old horse extended 1.3 mm from the dorsal margin. The four previously described fibre bundles (Chapter 5) of the MPICL were identified in 120 joints. Greatest variation in cross sectional area in horses two years and over was observed in the

dorsolateral bundle (CV 71%) and palmarolateral bundle (CV 95%) with somewhat less variation observed in the palmaromedial (CV 63%) and the dorsomedial bundles (CV 42%). The dorsomedial and palmarolateral bundles were present in all joints, but in eight joints of five horses no dorsolateral bundle was present and in 14 joints of nine horses no palmarolateral bundle was present. All of these horses were two years or older except one 18 month old Standardbred in which one joint had no dorsolateral bundle. This horse had begun training. In foals the proximal attachment of the dorsolateral bundle extended dorsally to the palmar edge of the articular cartilage on the lateral aspect of CR (Figure 3) while in most horses over one year of age the dorsal extent of the proximal attachment was more palmar (Figure 1). Dorsopalmar depth of the dorsolateral bundle decreased significantly with age even when corrected for the size of the carpus (Figure 4). There was a significant correlation between dorsopalmar depth of the dorsolateral bundle and size of tearing of the bundle ($R=0.33$, $p<0.01$).

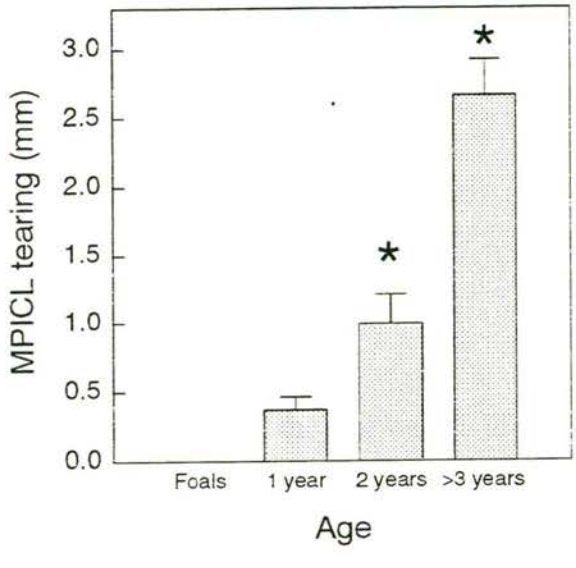

Figure 2.

Size (±sem) of medial palmar intercarpal ligament tears grouped by age of horse. (* denotes significant difference at $p<0.001$)

For horses of racing age (2-6 years) there was a significant but weak correlation between the extent of MPICL tearing and the area of partial thickness cartilage erosion ($R=0.23$, $p<0.05$) but not the area of full thickness erosion, subchondral bone damage or dorsomedial remodelling.

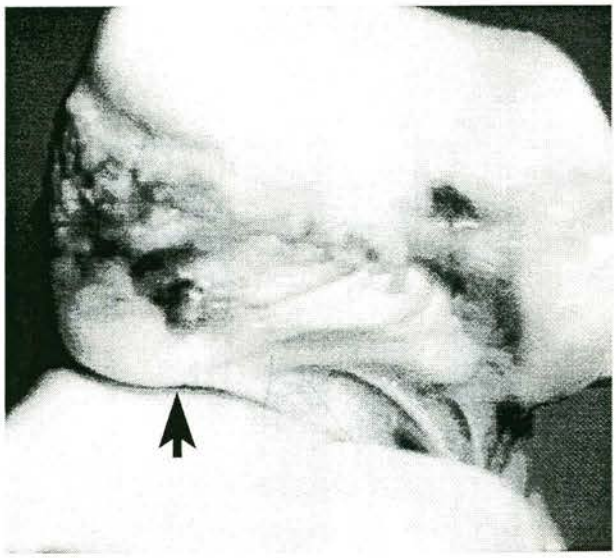

Figure 3.

Lateral view of the medial palmar intercarpal ligament of an unborn term foal. The dorsolateral bundle extends to the palmar aspect of the articular cartilage on the lateral aspect of the radial carpal bone (arrow).

Dorsomedial intercarpal ligament

The DMICL was identified in all joints but the size was highly variable, ranging from 0.4 mm to 2.6 mm thick (lateromedial) in horses two years and over. Gross enlargement as seen in clinical cases (Grade 3, Chapter 6) was only observed in three racing Standardbreds, two racing Thoroughbreds and two horses of non-racing breeds. In some joints the DMICL appeared as just a fold of synovial tissue with no distinct collagen bundles and no obvious distal border. Enlarged

ligaments consisted of parallel fibre bundles which were easily differentiated from the joint capsule over its full length. Lateromedial thickness of the DMICL increased significantly with age even when corrected for the size of the carpus (Figure 5). DMICL thickness in Standardbreds was greater than Thoroughbreds ($p<0.01$), and non-racing breeds, although this was not significant ($p=0.05$).

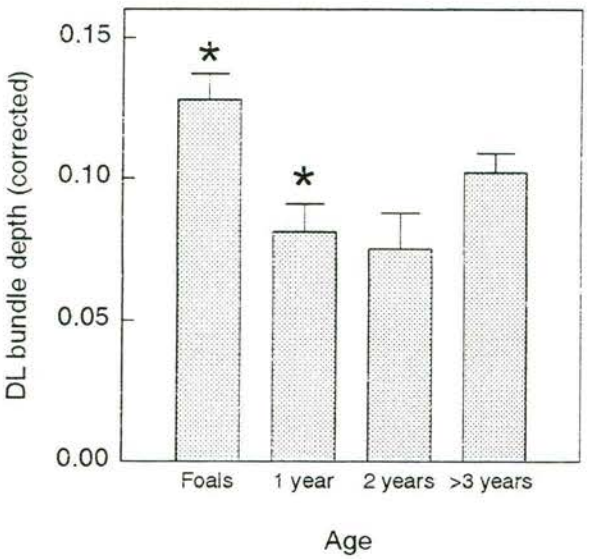

Figure 4.

Dorsopalmar depth (±sem) of the dorsolateral bundle of the medial palmar intercarpal ligament corrected for the size of the carpus, grouped by age. (* denotes significant difference at $p<0.05$)

Lateral palmar intercarpal ligament

Tearing was observed in eight joints of seven horses. Most tears were at the dorsal aspect. Five were Thoroughbreds, and there was one Standardbred and one other breed. Most tears involved only very superficial damage. In four joints from three horses small osseous fragments

approximately 3 mm in diameter were present in the CU attachment at the lateral aspect. In these joints there was an irregularity in the surface of the ligament but it remained firmly attached to CU.

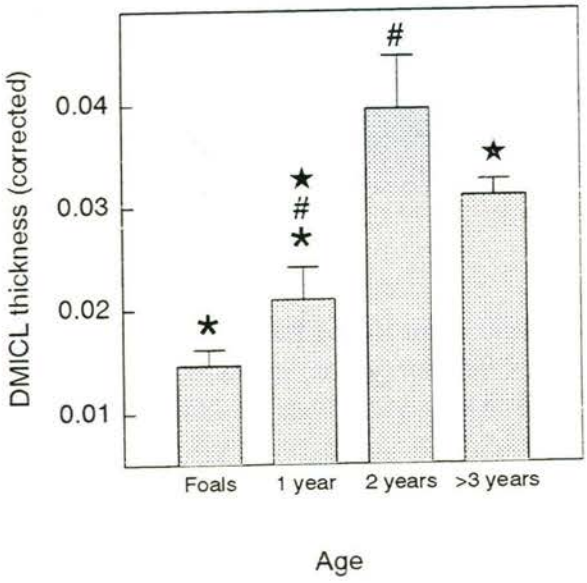

Figure 5.

Lateromedial thickness (±sem) of the dorsomedial intercarpal ligament corrected for the size of the carpus and grouped by age. (★ denotes significant difference at $p<0.05$, * denotes significant difference at $p<0.01$, # denotes significant difference at $p<0.0001$)

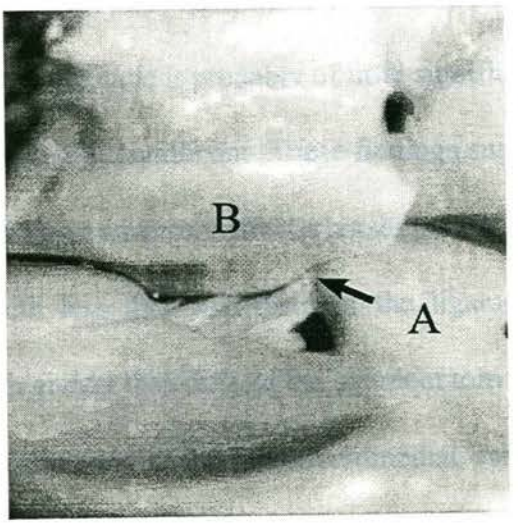

Figure 6.

Dorsal view of a partially flexed left adult carpus showing the proximal aspect of the medial palmar intercarpal ligament (arrow) trapped between the articular surfaces of the third carpal bone (A) and the radial carpal bone (B).

Discussion

Medial palmar intercarpal ligament

This survey has demonstrated a high frequency of tearing of the MPICL in horses of all breeds over two years of age. Although not reported, a similar frequency was found in racing Thoroughbreds in England (T Phillips pers comm). These findings question the clinical significance of tearing of the MPICL in racing horses. If tearing is as common in the general population of horses as this study suggests, tearing observed during arthroscopy of horses presented with signs of midcarpal joint disease may be an incidental finding rather than a cause of lameness. However it is important to note that no complete tears of the dorsolateral and

dorsomedial bundle (grade 4) were observed in the present survey. Although partial or even complete tearing of the dorsolateral bundle is probably of little significance, this study provides no evidence that grade 4 tears are not significant. These findings support the observation that minor ligament damage is unlikely to adversely affect a horses performance. McIlwraith (1992) reported that most horses with less than one third of the ligament torn performed well postoperatively, but horses with greater than 50% of the ligament torn had a poor prognosis. As the dorsolateral bundle is usually smaller than the dorsomedial bundle and very few of the observed tears extended into the dorsomedial bundle, most tears in the present study involved less than 50% of the MPICL that can be assessed arthroscopically.

Complete assessment of the significance of MPICL tearing with arthroscopy is limited by the inability to fully evaluate the whole ligament and the large variation between horses in the size of the various bundles. The most likely role of the dorsal bundles of the MPICL is stabilisation of CR, preventing dorsal displacement in relation to C3 (Phillips and Wright 1994, Chapter 6). Stability is related to the amount of intact ligament remaining rather than the size of the tear; a small tear in a small dorsolateral bundle may be just as significant as an extensive tear in a large dorsolateral bundle. The extent of remaining ligament needs to be determined before conclusions about the role of the tear in instability can be made. However the palmarolateral bundle cannot be observed arthroscopically because of its palmar position. The palmarolateral bundle has a very similar orientation to the dorsolateral bundle, and of all the bundles has the greatest variation in size. If a large palmarolateral bundle is present, instability from tearing of the dorsolateral bundle may not result, but if no palmarolateral bundle is present instability may result from minor damage.

The frequency of tearing in racing horses in this survey was greater than that observed in horses from the same population with midcarpal joint disease which were examined arthroscopically (Chapter 6). Many of the subtle tears observed by dissection in this study were at the proximal attachment of the MPICL which is not always directly visible with the standard arthroscopic portals. During dissection CI was removed allowing careful examination of the whole ligament. However grade 2 and 3 tears were also more common in this survey than observed in clinical cases and this degree of tearing would have been easily identified with arthroscopy. In Chapter 6 it was demonstrated that once damage occurred to one structure, ligament tearing and subchondral bone damage were inversely correlated. If this relationship holds for all horses it would explain the higher frequency of ligament tearing in the present study where subchondral bone damage was rarely observed, than in a group of horses undergoing arthroscopy that included many horses with significant subchondral bone damage.

Phillips and Wright (1994) suggested that tearing of the MPICL occurred due to cyclical tensing of the ligament during exercise. The high frequency of tearing in racing horses in their study and others (Kannegieter and Colgan 1993, Chapter 6) also suggested that this was a racing injury. The finding in the present study that tearing first appeared at about one year of age and seemed to progress in horses of all breeds as they matured would seem to refute this. But, the significant increase in tearing observed between one and two years of age in all horses also indicates that the commencement of training exacerbates damage. Therefore it appears that although damage and degeneration of the ligament commences in young horses prior to the onset of training, stresses applied to the ligament with training at any level results in further damage to an already compromised ligament. Also, the severity and frequency of tearing was greater in Standardbred

racehorses than Thoroughbred racehorses suggesting an influence of gait on ligament tearing.

A significant reduction in size of the dorsolateral bundle appears to occur at about the same time that ligament tearing was first observed (one year of age). A reduction in ligament size could be a response to increasing exercise, or due to degeneration. Degeneration preceding ligament tearing is observed in other intra-articular ligaments of other species (Vasseur *et al* 1985). Complete loss of intra-articular ligaments has been reported in humans in association with ligament rupture (Hefti *et al* 1991). In some joints in the present survey it appeared that complete loss of the dorsolateral bundle had occurred and it is unclear whether this was abnormal or due to individual variation. Although there was some variation in size of the dorsolateral bundle in young foals it was consistently present and extended further dorsally than was observed in adults. Therefore complete loss of the dorsolateral bundle in older horses is probably acquired. Longitudinal fibre separation is likely to be a gross manifestation of degeneration.

In Chapter 5 it was proposed that abrasion of the MPICL on the palmar articular margin of C3 may be a cause of ligament degeneration and tearing. During dissection of the specimens in the present study it was observed that with flexion of the carpus the most dorsal part of the proximal attachment of the MPICL is trapped between the articular surfaces of CR and C3. This area of the ligament was the most common site of tearing of the MPICL (Figure 1). During flexion CR moves laterally relative to C3 (Phillips and Wright 1994). This movement is stopped when the lateral aspect of CR articulates with the palmaromedial aspect of C3. As this occurs the most dorsoproximal part of the MPICL is drawn over the articular surface of C3 before CR lifts off C3 as full flexion is approached (Figure 6). Repeated compression and abrasion of the ligament

at this site would explain its predisposition for injury. Removal of the synovial lining of intra-articular ligaments has been shown to result in their degeneration (Robinson *et al* 1991). In some of the more mildly affected ligaments it appeared that only the synovial membrane was lost.

Tearing of the other bundles of the MPICL was not observed. However there was also a large degree of variation in size of the palmarolateral bundle. This was probably due in some degree to individual variation as it was also a feature of foals. Interestingly, like the dorsolateral bundle, the palmarolateral bundle is also drawn over the articular surface of C3 during flexion suggesting that ligament abrasion may also play a role.

Others have observed a relationship between tearing of the MPICL and remodelling of the dorsomedial aspect of CR (Phillips and Wright 1994). Although dorsomedial remodelling was observed in a number of joints in the present study, there was no relationship with tearing of the MPICL. This may be due to the lack of grade 4 tears. Also if remodelling is a response to CR instability then, as previously argued, MPICL tearing alone may not be a good indicator of this.

Dorsomedial intercarpal ligament

Enlargement of the DMICL has been described as a congenital condition that is a cause of carpal joint disease in racing horses by impingement on the articular surface of the midcarpal joint (Selway 1991). Although variation in size was observed in foals, none had grade 3 enlargement. Larger numbers of foal joints would need to be examined before a congenital cause of enlargement can be ruled out, but the findings of the present study suggest that enlargement

occurs with the onset of training. In Chapter 6 it was demonstrated that there was a relationship between the size of the DMICL and the time the horse has been in training. In the present survey an increase in thickness of the ligament appeared to occur when horses commenced training, but this response was not consistent in all horses, suggesting that there are other factors involved in DMICL enlargement. Also, enlargement was observed in non-racing breeds so it is possible that other forms of exercise may contribute to enlargement. In Chapter 6 a relationship between MPICL tearing and DMICL enlargement was observed. This was not observed in the present study but no grade 4 MPICL tears were observed. The largest DMICLs in that study were observed in joints with grade 4 MPICL tears.

Lateral palmar intercarpal ligament

Damage to the LPICL was rarely observed and was only mild. It was however interesting to observe bone fragments within the proximal attachment in several joints. These appeared to originate from CU and were possibly avulsions. Avulsion fractures at this site have been observed in clinical cases (A Nixon, pers comm). The significance of these lesions was difficult to determine. In all horses the ligament appeared to have reattached to the defect in CU. The observation of one of these in a one year old Thoroughbred indicates that they may not be a racing injury. It is possible that not all such lesions were detected as they were buried within the substance of the ligament and were only found by dissection.

Conclusion

Damage to the dorsolateral bundle of the MPICL of the midcarpal joint is a common finding in both racing and non-racing horses older than one year of age, but complete rupture of the dorsolateral and dorsomedial bundles is rare. It is likely that damage confined to the dorsolateral bundle of the MPICL is not clinically significant in most horses. Variation in size of the DMICL is observed in horses of all ages but is most marked in two year old racehorses, and gross enlargement is probably acquired. Damage to the LPICL is rare, and when it occurs appears mild and is of unknown significance.

CHAPTER 8

HISTOPATHOLOGICAL FINDINGS IN THE MEDIAL PALMAR AND DORSOMEDIAL INTERCARPAL LIGAMENTS OF THE MIDCARPAL JOINT

Introduction

Tearing of the MPICL is commonly observed, but tearing of other intra-articular ligaments of the midcarpal joint is rare. Phillips and Wright (1994) found that 70% of midcarpal joints of racing horses presenting with carpal disease had some degree of tearing of the MPICL, whereas in Chapter 7 an even greater incidence was demonstrated in racing horses with no history of carpal problems. Tearing of other intercarpal ligaments of the midcarpal joint has been reported (Kannegieter and Colgan 1993) but is relatively uncommon. The MPICL must therefore be under greater stresses than these other ligaments, or it is intrinsically less resistant to applied stress.

Tearing is most commonly observed in the dorsolateral bundle of the MPICL. Of the four bundles of the MPICL, this bundle was shown to have the greatest variation in size and was sometimes completely absent (Chapter 7). These observations could be explained by degeneration of the dorsolateral bundle resulting in both weak and smaller ligaments. Degeneration of intra-articular ligaments in other species is thought to precede gross changes (Vasseur *et al* 1985, Noyes *et al* 1989, Robinson *et al* 1992). Vasseur *et al* (1985) demonstrated degeneration of the cranial cruciate ligament in those dogs that had a high incidence of ligament rupture.

Dramatic variation in the size of DMICL was first described by Selway (1992). Large ligaments were thought to be a congenital abnormality and it was proposed that they caused instability in the midcarpal joint by impinging on the articular surface. However a relationship between the size of the DMICL and the stage of training of the horse was demonstrated in Chapter 6. This

suggests an hypertrophy of the ligament in response to training. Both ligaments and tendons in the horse and other species have been shown to adapt to exercise (Gillis *et al* 1993, Bramlage *et al* 1987), but hypertrophy to the degree seen in the DMICL has not been described to my knowledge. Tearing of the DMICL has been described in occasional cases (Selway 1992) but in my experience is uncommon.

A histopathological study of the dorsolateral bundle of the MPICL, and the DMICL was undertaken. The aim of the study was to determine whether there was evidence of degenerative changes not only in torn MPICLs, but also in those that appeared grossly normal. The DMICL was also examined to determine whether there was evidence that any variation in size was due to a true hypertrophy of some ligaments, or due to degeneration and atrophy of other ligaments. The effect of the intensity of exercise on ligament pathology was also examined.

Materials and Methods

Twenty two midcarpal joints were obtained from 11 horses at the completion of a controlled training program (Chapter 2). The horses were divided into two groups; group 2 was exercised at a higher level than group 1. The joint capsule was opened dorsally, the joint inspected and the MPICL and DMICLs examined. The degree of tearing of the medial palmar intercarpal ligament was graded according to the method described by Kannegieter and Colgan (1993). Using a digital caliper (Mitutoyo, Series 500, Tokyo, Japan) the dorsopalmar depth of the tear was measured, as well as the dorsopalmar depth of the dorsolateral bundle of the MPICL, as defined in Chapter

5. The size of the DMICL was graded as described in Chapter 2 and its mediolateral width measured. From each joint the DMICL and the dorsolateral bundle of the MPICL were severed at their proximal and distal attachments and fixed in 10% formalin.

Longitudinal sections of the full length of the ligament were cut and stained with haematoxylin and eosin, Gomori's Trichrome and Picro-Sirius Red. Sections were examined under a light microscope (Olympus Model BX 40). Using a video adapter and a solid-state video camera (Hitachi, Model KP-143) the image was transferred to a desktop computer (Scan Beam, Denmark) and cell counts and vascularity were determined using image analysis software (Tema version 5.3, Scan Beam, Denmark). Cells were counted in 12 fields from each ligament section. In all ligaments three of the fields used were adjacent to one attachment, and three were adjacent to the opposite attachment. The other six fields were within the body of the ligament. A mean cell density was then calculated from these 12 fields. The coefficient of variation of these 12 figures was also calculated to give an estimate of the variation in cell density throughout the ligament. As indicators of vascularisation, the perimeter of each vessel was traced manually and the percentage area of vessels, the mean diameter of vessels, and vessel density were calculated.

Other variables were determined subjectively using simple grading systems. Synovial membrane was assessed for the thickness of both intimal and subintimal layers, proportion of intimal surface intact, size and number of villi, and inflammatory cell infiltrate. Cellularity was assessed by estimating the degree of pleomorphism, the size and number of acellular areas, clumping or chain formation, and extent of chondroid metaplasia. Three different cell types were identified based on the shape of their nuclei: elongated, spindle and round. Differential counts were performed

to determine their proportions. The proportion of collagen fibres parallel with the longitudinal axis of the ligament was estimated, as well as the proportion of loose and dense connective tissue, and the area of loss of organisation into primary collagen bundles. The amount of adipose tissue in the ligament was also assessed. Degeneration of the ligaments was assessed and graded using the system described by Vasseur *et al* (1985).

Due to a lack of grossly normal MPICLs in adult horses for comparison (Chapter 8), ligaments were also examined histologically from seven young horses ranging in age from unborn term foal to 11 months. Also as there was only one horse in the training program with grade 3 enlargement of the DMICL, ligaments from a further 10 joints of five Thoroughbred and Standardbred horses with grade 3 enlargement of the DMICL were examined histologically.

Statistical analysis

Differences in findings between groups and between DMICLs and MPICLs were determined by a Mann Whitney U test. Relationships between gross and histological findings were determined by Spearman rank order correlations. Variables are expressed as means±sem and differences and associations were considered significant at $p<0.05$.

Results

Trained horses

Gross changes - Tearing of the dorsolateral bundle of the MPICL was observed in 15 of the 22 joints and eight of the 11 horses. There were five grade 1 tears, seven grade 2 tears, three grade 3 tears and no grade 4 tears. The largest tear extended 5 mm palmarly from the dorsal margin into the substance of the ligament. The maximum dorsopalmar width of the dorsolateral bundle of the MPICL was 5.3 mm, and in one joint it was completely absent. There was no significant difference in the grade and size of tears between groups 1 and 2, or in the size of the dorsolateral branch of the MPICL. The lateromedial width of the DMICL varied from 1.0 to 3.8 mm. There was no significant difference in width between groups 1 and 2.

Histological characteristics - The dorsolateral bundle of the MPICL was characterised by a mixture of loose connective tissue, and more dense collagen arranged in bundles. Collagen bundles were generally disorganised and alignment was poor (Figure 1). The ligaments were highly cellular and there were moderate numbers of small vessels generally located in the loose connective tissue. The predominant cell was the fibroblast and there was a wide variety of cell shapes. A narrow zone of chondroid hyperplasia was often present at the attachments of the ligament. Very few inflammatory cells were observed in any ligament. On all sections at least one surface was covered with a thin layer of synovial cells. The subintimal connective tissue layer was very thin or absent. Synovial villi were only occasionally observed.

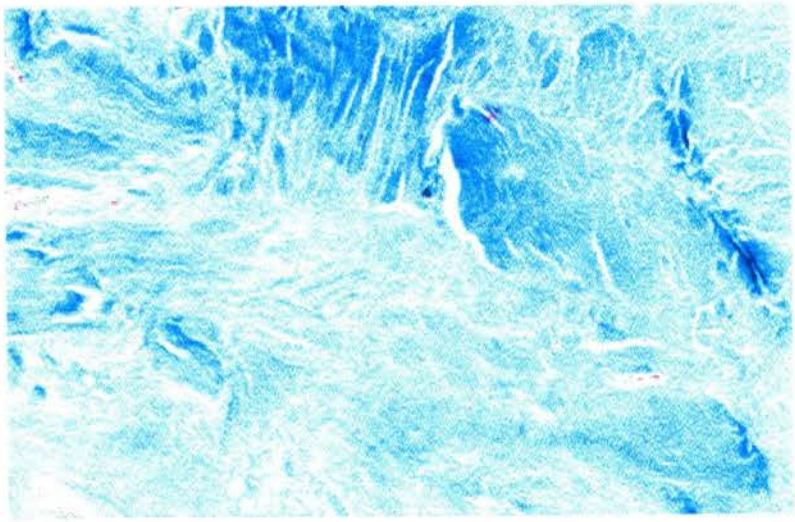

Figure 1.

Longitudinal section of the dorsolateral bundle of a medial palmar intercarpal ligament of an adult horse. Note the poor collagen alignment and organisation. No gross tearing was evident in this ligament (Gomori's Trichrome, ×80)

DMICLs consisted primarily of parallel dense collagen fibre bundles interspersed with thin layers of loose connective tissue containing vessels (Figure 2). Blood vessels were confined to the loose connective tissue layers. An area of chondroid metaplasia was consistently observed at the attachment of the ligament to the radial carpal bone. The synovial intimal layer was present in all sections and overlaid a subintimal layer of connective tissue. As with the MPICLs very few inflammatory cells were observed and the fibroblasts were a variety of shapes. Some sectioning artefact was observed in the grade three DMICLs, but was otherwise minimal.

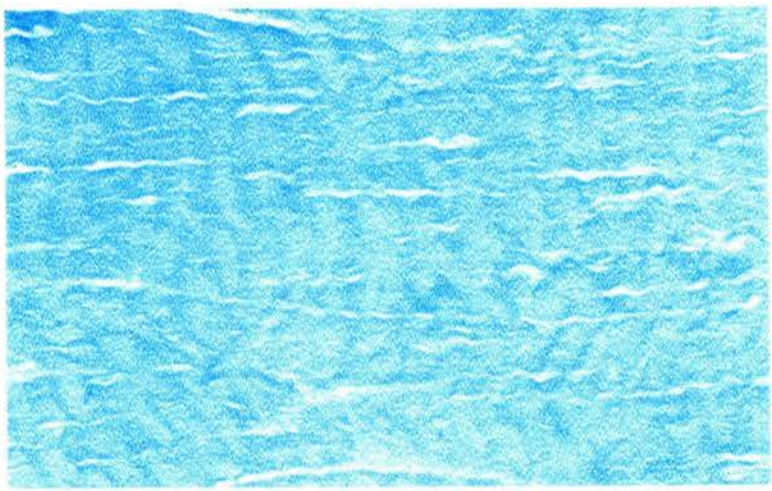

Figure 2.

Longitudinal section of a dorsomedial intercarpal ligament of an adult horse showing parallel dense collagen bundles. (Gomori's Trichrome, ×160)

As with gross findings there was very little difference in the histological findings between groups 1 and 2. There was a trend for MPICLs from horses in group 2 to have less longitudinally oriented collagen (32±5%) than horses in group 1 (42±4%), but this difference was not significant ($p=0.07$). There were also few differences in the histological characteristics of the DMICL between groups 1 and 2. DMICLs of horses from group 2 had lower cell numbers (314±27 cells/mm^2) than group 1 (361±26 cell/mm^2) ($p=0.07$), but again this was not significant.

Histologically there were many differences between the dorsolateral bundle of the MPICL and the DMICL (Table 1). The MPICL had significantly more cells ($p<0.00001$). There was a significantly larger number of round cells ($p<0.01$), while the number of elongated cells was less ($p<0.05$) and acellular areas were fewer and smaller ($p<0.05$) than in the DMICL. Synovial

intimal layer thickness was greater in the MPICL ($p<0.0001$), but synovial subintimal layer thickness was significantly greater in DMICLs than MPICLs ($p<0.05$). Vessels in the MPICL were significantly larger ($p<0.05$) and took up a greater proportion of the area of the ligament ($p<0.05$). There was significantly less longitudinally aligned collagen ($p<0.00001$), more loose connective tissue ($p<0.00001$), more loss of collagen bundle organisation ($p<0.00001$) and the grading of degenerative changes as described by Vasseur *et al* (1985) was higher in the MPICL ($p<0.00001$).

Relationship between gross pathology and histopathology - There was a significant correlation between the size and area of vessels in the ligament and the size of MPICL tears ($R=0.44$, $p<0.05$) but not ligament size. The proportion of elongated fibroblasts within the ligament decreased with MPICL tearing ($R=-0.52$, $p<0.05$). There were no other significant correlations between gross pathology of the MPICL and histopathological changes.

There were significant correlations between the size of the DMICL and the extent of cellular clumping ($R=0.44$, $p<0.05$) and chondroid metaplasia ($R=0.57$, $p<0.01$). There was a negative correlation between DMICL size and the grading of degeneration ($R=0.48$, $p<0.05$) as well as loss of organisation of collagen bundles ($R=0.43$, $p<0.05$). Smaller ligaments tended to have fewer elongated fibroblasts ($R=0.44$, $p<0.05$) and more spindle shaped fibroblasts ($R=0.63$, $p<0.01$) as a proportion of the total cells.

Table 1.

Differences in histological variables between the medial palmar intercarpal ligament (MPICL) and the dorsomedial intercarpal ligament (DMICL) in adult trained Standardbred horses.

Variable	MPICL	DMICL	p value
Cell density (cell/mm^2)	611±41	335±19	<0.00001
Round cells (%)	45±4	32±2	<0.01
Elongated cells (%)	14±2	24±3	<0.05
Vessel area (% of field)	1.8±0.2	1.2±0.3	<0.05
Vessel diameter ($\times 10^{-3}$ mm)	24±0.9	20±0.9	<0.05
Proportion longitudinal collagen (%)	36±4	77±4	<0.00001
Proportion loose connective tissue (%)	48±4	19±3	<0.00001
Degeneration (grade)	2.5±0.1	1.1±0.2	<0.00001

Foal ligaments

The only gross changes observed in the foal MPICLs were bilateral 1 mm tears in the 11 month old horse. The histologic characteristics of MPICLs obtained from foals appeared to be age dependant. MPICLs from the two unborn term foals consisted of well organised mostly parallel collagen bundles separated by thin layers of loose connective tissue (Figure 3). Towards the distal attachment to C3, orientation of collagen bundles was more random but they remained well organised. MPICLs from unborn foals were also highly cellular. MPICLs in very young foals (less than one week) had similar characteristics, while in those of older foals there was some loss of organisation (Figure 4) and a decrease in cellularity.

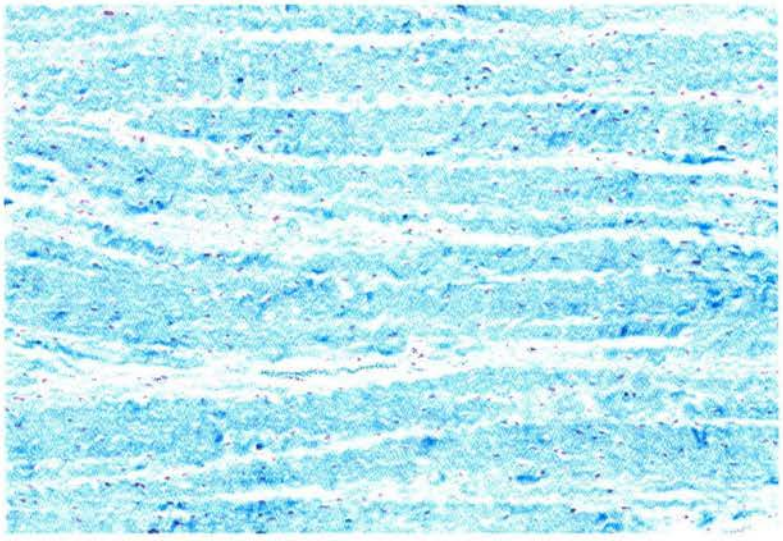

Figure 3.

Longitudinal section of a dorsolateral bundle of a medial palmar intercarpal ligament of an unborn term foal. Collagen bundles are parallel and well organised. (Gomori's Trichrome, ×160)

Mean cell density in MPICLs of foals was 983±55 cells/mm^2 , which was significantly greater than adult trained horses (611±41 cells/mm^2, $p<0.0001$), and the distribution of the cells was more uniform in foal MPICLs ($p<0.01$). The grading of degeneration in foal MPICLs was significantly less than in adult MPICLs (foals 1.7±0.2, adults 2.5±0.1, $p<0.01$), and there was a greater proportion of longitudinal collagen bundles (foals 69±5%, adults 36±4%, $p<0.001$) with less loss of organisation ($p<0.01$).

Histologically the DMICL of foals had a structure very similar to that of fibrous joint capsule. There was less dense longitudinal collagen than in adults and in most foals there were multiple areas of adipose tissue within the structure. Mean cell density in foal DMICL was 1057±95 cells/mm^2, which was significantly greater than DMICLs from adults in training (335±19 cells/mm^2, $p<0.00001$).

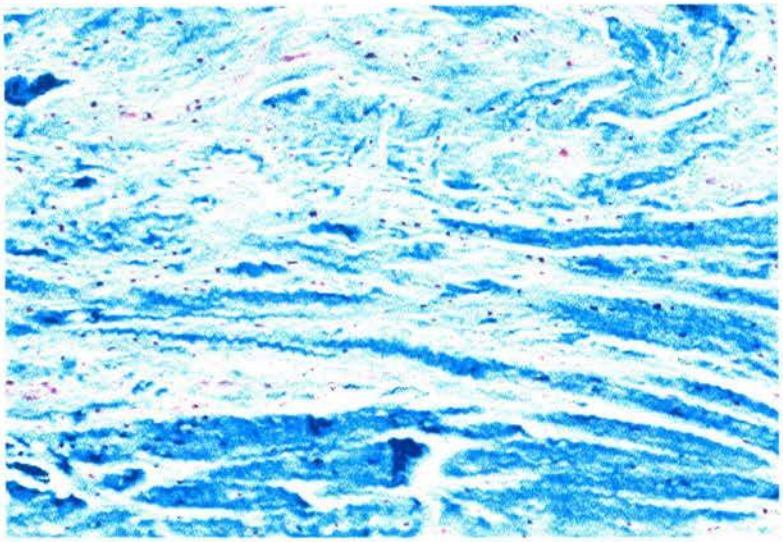

Figure 4.

Longitudinal section of the dorsolateral bundle of a medial palmar intercarpal ligament of a one month old foal. There is some loss of collagen fibre alignment and organisation. (Gomori's Trichrome, ×80)

Grade 3 dorsomedial intercarpal ligaments

Enlarged DMICLs were characterised by very dense parallel collagen bundles with little loose connective tissue. Large acellular areas were observed adjacent to the attachment to CR and the whole ligament appeared relatively avascular. In all enlarged ligaments at least one discrete area of fibrovascular infiltration was observed with resulting loss of collagen fibre continuity (Figure 5).

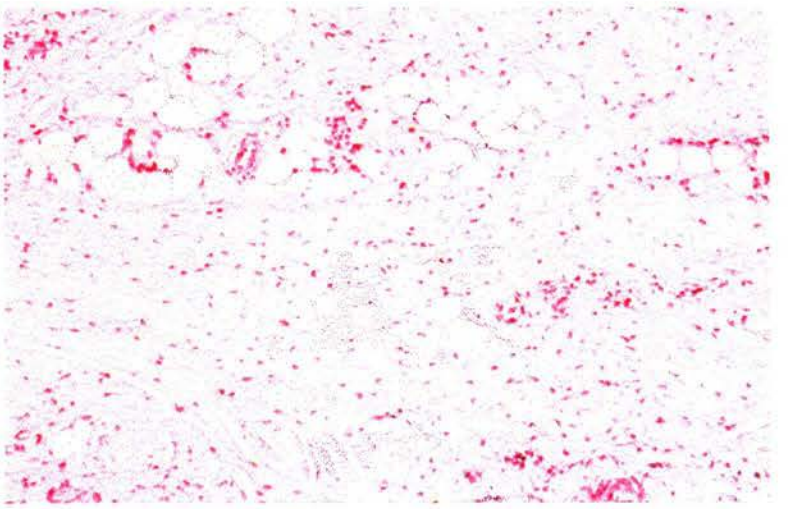

Figure 5.

Longitudinal section of a dorsomedial intercarpal ligament of a one month old foal. Note random collagen arrangement and areas of adipose tissue (haematoxylin and eosin, ×80).

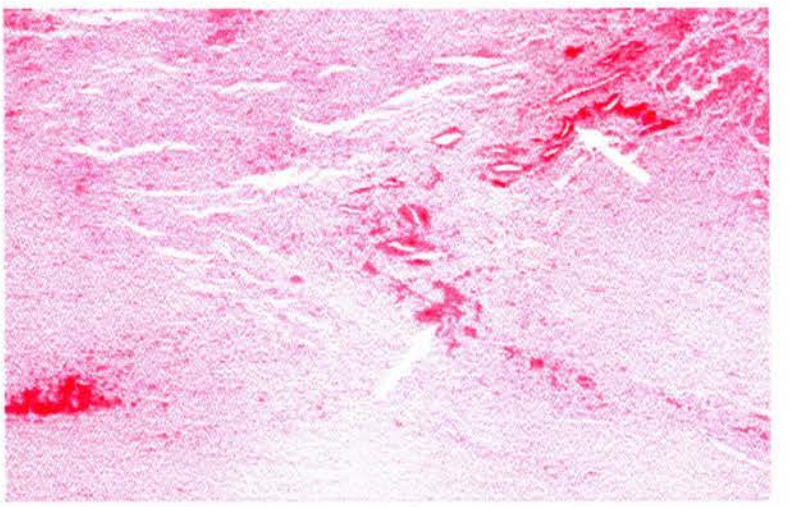

Figure 6.

Longitudinal section of a grade 3 dorsomedial intercarpal ligament showint an area of fibrovascular infiltration (white arrows)(haematoxylin and eosin, ×30).

Discussion

Ligament degeneration

Vasseur *et al* (1985) described a number of histological changes that they considered evidence of intra-articular ligament degeneration in the dog. These included loss of fibroblasts, chondroid metaplasia and calcification, and failure to maintain primary collagen fibrils within collagen bundles. These changes were also associated with inferior material properties, confirming that they were detrimental to function. Also, those dogs which exhibited the most marked degenerative changes were those in which rupture of the ligament was most commonly observed. However, care must be taken in the interpretation of these findings, particularly chondroid metaplasia. Chondroid metaplasia of connective tissue structures is thought to be a normal finding in areas of compression (Matyas *et al* 1994). Compressive forces have been demonstrated close to the insertion of collateral ligaments (Matyas *et al* 1994) and it has been suggested that they occur in cruciate ligaments as well (Clark and Siddles 1990). Chondroid metaplasia was confined to the attachments of both the ligaments examined in the present study and so was considered a normal finding.

The most striking finding of the present study was the marked loss of normal ligamentous collagen architecture in all adult MPICL specimens. Typical intra-articular ligament architecture was described by Clark and Siddles (1990). Cranial cruciate ligaments of dogs, humans and rabbits were made up of fibre bundles separated by cells and formed into fascicles, surrounded by thin sheets of loose connective tissue. This arrangement was observed in the DMICL of adult

horses, but in MPICL specimens was only observed in young foals. Ideally a group of adult ligaments showing no gross pathology would be used to determine the normal structure of this ligament, but the post mortem examination of 96 adult joints could find no MPICLs that were considered completely free from tearing, localised thickenings or vertical fibre separation (Chapter 8). The only ligaments that appeared to be free of gross pathology were from foals less than three months of age. Histological examination demonstrated loss of architecture in MPICLs from foals over one week of age. It could be argued that this change in architecture between foals and adults is a normal response to ageing and/or weight bearing, but similar changes are associated with weakening of the cranial cruciate ligament in dogs (Vasseur *et al* 1985). The replacement of parallel aligned dense connective tissue with loose, randomly arranged connective tissue must result in a weaker structure. In contrast with ligament degeneration in the dog, although there was some loss of cellularity with ageing, this was not a feature of degeneration of the MPICL of the horse. Focal acellular areas were observed in MPICLs, but the ligaments in general showed high cellularity with relatively large numbers of more immature type fibroblasts.

Similar histologic changes in other intra-articular ligaments of other species have been induced experimentally by both partial sectioning or synovial stripping. Hefti *et al* (1991) partially sectioned the cranial cruciate ligament of rabbits and demonstrated prolonged and incomplete healing with increased cellularity and ligament lengthening. Robinson *et al* (1992) demonstrated that stripping of the synovium from the cranial cruciate ligament of rabbits resulted in areas of collagen necrosis and disorganisation, as well as increased cell density. The end result was a smaller and lax ligament. This was proposed as the mechanism by which partial cruciate

ligament rupture progresses to complete rupture in humans (Noyes *et al* 1989). The changes observed in the MPICL could therefore be explained by constant low grade damage to both the ligament and its synovial membrane.

Intra-articular ligaments appear to have very limited healing potential. Although Arnoczky *et al* (1979) observed increased vascularity following sectioning of the cranial cruciate ligament in the dog, this was limited if the synovial lining of the ligament was removed. Lyon *et al* (1991) demonstrated that the healing ability of intra-articular cruciate ligaments was less than that of extra-articular collateral ligaments. In humans, complete resorption of the torn ends of cruciate ligaments has been observed (Hefti *et al* 1991). In the present study, the increased vascularity with increased numbers of fibroblasts, mostly with large rounded nuclei, in ligaments with gross tearing suggests some attempt at healing.

Therefore the increased cellularity, increased vascularity and loss of collagen architecture observed int he MPICL is consistent with the mechanism for damage proposed in Chapter 8 by which the dorsolateral bundle is trapped between CR and C3 during carpal flexion and abraded by their articular surfaces. As this would occur in all horses of all ages, it explains why both gross and histologic evidence of degeneration is observed in horses well before they begin an athletic career.

An electron microscopic study of the intercarpal ligaments of the carpus has demonstrated that the dorsolateral bundle of the MPICL has a different fibril population compared with other intercarpal and collateral ligaments, including the dorsomedial bundle of the MPICL, with a large

number of smaller fibrils observed (Firth *et al* 1988). This was proposed as a reason for the high incidence of tearing in this part of the ligament. Due to the consistency of degeneration in the specimens from the present study, it is possible that this unique fibre pattern is a result of damage and degeneration of the ligament rather than a cause of damage.

Dorsomedial intercarpal ligament

The DMICL varies greatly in size between joints. The ligament has been described as a synovial fold by some authors due to its very thin structure in many joints (McIlwraith 1990). Diameters ranging from 2-16 mm have been reported by Selway (1991) in clinical cases of midcarpal joint disease. This is difficult to interpret as the ligament is rectangular rather than circular in cross-section and assessment of dimensions with arthroscopic visualisation can only give an estimate. In Chapter 7, the lateral-medial thickness of the DMICL ranged from 0.4-2.6 mm in joints with no history of carpal disease. The cause of this variation is unknown. Selway (1991) suggested that enlargement of the ligament was a congenital condition, but in Chapter 6 there was a tendency for increased ligament size in horses which had been in training for a longer period prior to examination. An increase in ligament size with age was also observed in Chapter 7 and this was associated with the onset of training. These findings suggest that the ligament increases in size in response to exercise. Changes in ligament size with training have not been reported in horses. Tendons in the horse have been shown to increase their cross-sectional area in response to exercise, but the changes are small (Gillis *et al* 1993). The other possible reason for an increase in size is due to injury. Healing of connective tissue structures generally results in thickening due to scar tissue (Williams *et al* 1980). In other species, collateral ligaments have

been shown to have greater healing ability than intra-articular ligaments (Lyon *et al* 1991). Although the DMICL is partially intra-articular, it was proposed in Chapter 6 that it is an intra-articular part of the medial collateral ligament based on its position and attachments. Gross tearing of the ligament has been reported (Selway 1991), but was not observed in 54 joints of 32 horses examined arthroscopically for midcarpal joint disease (Chapter 6).

Histologic examination of the DMICL in the present study demonstrated typical ligamentous architecture in adults (Clarke and Siddles 1990, Amiel *et al* 1984) but this was not the case in foals where the ligament had an appearance more like that of joint capsule (Ross and Reith 1985). It may be that some amount of loading is required to stimulate fibre alignment and organisation in this structure. Typical ligamentous architecture was maintained in grossly enlarged ligaments although the relative amount of dense connective tissue was increased.

The areas of fibrovascular infiltration observed in all enlarged ligaments are consistent with focal ligament disruption and subsequent healing. Despite this, evidence of more chronic ligament damage such as poor collagen fibre alignment was not observed. This suggests that the ligament has good potential for healing and remodelling of its collagen structure, as has been observed in collateral ligaments (Lyon *et al* 1991). The observation of tearing with apparent healing in enlarged DMICLs has two possible explanations. Firstly, enlarged ligaments may be more susceptible to tearing. If ligament enlargement is an hypertrophy due to increased loading, then it is also likely that this loading will induce more damage. Alternatively, repetitive low grade tearing of the ligament could result in an increased cross-sectional area due to the accumulation of scar tissue. In equine tendon, trauma results in much greater increases in size (Webbon 1977,

Williams *et al* 1980) than hypertrophy due to exercise. It is therefore more likely that the enlargement observed in the DMICL is a response to repetitive trauma rather than a physiological response to exercise.

Synovial membrane

A synovial layer was observed on both the MPICL and the DMICL. It was interesting to observe that the MPICL tended to have a thicker intimal layer. In other species, synovial stripping of ligaments induced a subsequent synovial hypertrophy of the intimal layer (Robinson *et al* 1992). Synovial hypertrophy of the MPICL and LPICL is often observed during midcarpal joint arthroscopy, and may be due to abrasion of the synovial layer of these ligaments on the bones of the midcarpal joint.

Effect of exercise

Little difference was demonstrated between the histological appearance of the MPICL of each of the training groups. It is important to note that both groups of horses were exercised for the same period of time and both at high intensities. Also degeneration of the MPICL appeared to start at an early age, unrelated to training, so any direct effect of training must be superimposed over age related ligament degeneration. By examining ligaments from a group of horses that had all completed a training program at the same time any variation which may have arisen due to acute inflammatory changes was removed. This was confirmed by the very low numbers of inflammatory cells present in both ligaments. At the time of joint collection, the horses had been

rested for eight weeks. If ligaments had been collected from a general population of horses, the type and amount of exercise the horses received could not be controlled and the effect of this on the structure of the ligament is unknown.

Conclusion

This study has demonstrated that degeneration of the collagen structure of the dorsal aspect of the MPICL occurs in most horses over one month of age, which would explain the high incidence of tearing of the ligament and the large variation in size of the dorsolateral bundle. In contrast degeneration was not a feature of the DMICL. In both ligaments there was evidence of low grade damage, so differences between them were probably due to the ability of each structure to repair. In the MPICL, damage results in loss of collagen fibre architecture, a reduction in ligament size, ligament laxity, and presumably a loss of strength and increased susceptibility to tearing. Low grade damage to the DMICL appears to stimulate a good healing response, and if repeated could cause enlargement of the ligament. The observation of low grade tearing in enlarged DMICLs suggests that the ligament is being loaded excessively.

CHAPTER 9

THE INTERCARPAL LIGAMENTS OF THE MIDCARPAL JOINT: CLINICAL SIGNIFICANCE OF LIGAMENT PATHOLOGY

Introduction

Osteochondral damage in the midcarpal joint is a well established cause of clinical carpal disease and poor performance in racing horses (Spiers *et al* 1986, Kannegieter and Ryan 1991, McIlwraith *et al* 1987, Raidal and Wright 1996). However the clinical significance of other intra-articular lesions is less well understood. Both tearing of the medial palmar intercarpal ligament (MPICL) (Kannegieter and Colgan 1993, Phillips and Wright 1994) and enlargement of the dorsomedial intercarpal ligament (DMICL) (Selway 1991) are commonly observed in horses with midcarpal joint disease. It has been proposed that tearing of the MPICL causes instability in the midcarpal joint and contributes to osteochondral damage (Kannegieter and Colgan 1993). Phillips and Wright (1994) demonstrated a relationship between remodelling of the dorsal aspect of the radial carpal bone (CR) and tearing of the MPICL. McIlwraith (1992) suggested that clinical signs of carpal disease were much more severe in joints with tearing than in joints with similar osteochondral damage but no ligament damage. Also enlargement of the DMICL has been proposed as a primary cause of osteochondral damage in the midcarpal joint due to impingement on the joint surface (Selway 1991).

Although changes in these ligaments seem to be associated with midcarpal joint disease, observations in earlier Chapters of this thesis raise questions about their significance. In Chapter 7 a high incidence of MPICL tearing was observed in the general population of horses. This suggests that MPICL tearing is not always clinically significant. Also in Chapter 6 no relationship was found between osteochondral damage and DMICL enlargement, challenging its proposed role in midcarpal joint disease.

One way to determine the significance of intercarpal ligament pathology is to examine the postoperative performance of horses in which the midcarpal joint has been examined. Unfortunately concurrent osteochondral damage also affects performance (McIlwraith *et al* 1987) and so comparison between horses is difficult. To my knowledge there is only one report of the postoperative performance in a number of horses with MPICL tearing (McIlwraith 1992). Performance of horses with more than 50% of the MPICL torn was found to be very poor, but no consideration was made of concurrent pathology in the joint.

In this prospective study I examined the postoperative performance of horses where the mid carpal joint was examined arthroscopically. The primary aim was to determine the effect of tearing of the MPICL and enlargement of the DMICL, while taking into account other pathology identified in the joint at the time of surgery.

Materials and methods

Clinical observations

All racing horses undergoing midcarpal joint arthroscopy at the University of Sydney Veterinary Teaching Hospital between April 1993 and April 1995 were included in the study. The indications for surgery were intra-articular osteochondral fragments observed on radiographs, areas of subchondral lucency and/or lameness localised to the midcarpal joint with intra-articular anaesthesia. Arthroscopic surgery was performed as described in Chapter 2. Intra-articular

structures assessed were the subchondral bone, articular cartilage, MPICL, LPICL and DMICL. The degree of damage to each of these structures was graded as described in Chapter 2.

Treatment at the time of arthroscopic surgery included removal of osteochondral fragments, debridement of soft subchondral bone and loose articular cartilage tags. Torn ends of the MPICL were not debrided and the DMICL was not trimmed except to remove any osteochondral fragment attached to its proximal insertion.

Race records

Race records of Thoroughbred racehorses were obtained from the Australian Associated Press database while those of Standardbred racehorses were obtained from the Harness Racing Authority of New South Wales. Rest time was defined as the time between surgery and the first postoperative race. Information obtained included the number of races before and after surgery, the position in each race and the class of race. Time to follow up ranged from 15 to 27 months.

A scoring system was developed for the first five placings in each race with values increasing for each class of race. For Thoroughbred racehorses, points for each class were determined based on the weight variations supplied by the Australian Jockey Club handicapper. Winners of a maiden race received four points, second place three points, third place two points and fourth place one point. Points increased with race class up to Group 1 races where winners received 25 points, second place 20 points, third place 15 points, fourth place 10 points and fifth place five points. A similar system was used for Standardbred races based on race class conditions

published by the Harness Racing Authority of NSW. Postoperative performance was determined by comparing the results of up to five races prior to surgery and up to five races following surgery. Each horse had to complete at least two races before and after surgery to be included. The mean of the presurgery scores was calculated and subtracted from the mean postoperative score to give a net score. An improvement of performance was defined as a positive net score.

Statistical analysis

A Mann Whitney U test was used to compare the postoperative results of horses with a least one joint with grade 2-4 MPICL tearing and horses in which the most severe tearing was grade 1. The relationship between rest time and net score was determined by Spearman rank order correlation.

The relationship between net score and the severity of various intra-articular lesions was determined using multiple linear regression. Intra-articular variables that were examined were the degree of subchondral bone damage, the extent of tearing of the MPICL, the extent of articular cartilage damage and the degree of enlargement of the DMICL. In horses where two joints were examined, the highest value of each intra-articular variable was used for the analysis. Each variable was plotted against net score and data transformation performed if the relationship was not linear. Multiple regression was performed with each variable added individually so that all possible combinations were examined. To determine if adding further variables to the model significantly improved the prediction of postoperative performance the F value for inclusion was calculated.

Results

Forty two horses were included in the study. Ages ranged from two to six years. Thirty eight were racing Thoroughbreds and four were racing Standardbred pacers. Time between surgery and the first postoperative race ranged from 21 to 84 weeks. Of the 42 horses, 32 (76.2%) raced postoperatively and 23 (54.8%) were able to win at least one race. Twenty six horses completed at least two races prior to surgery and two races after surgery. Of these, 12 (46%) performed at the same or higher level than prior to surgery.

Fourteen horses had grade 2-4 tearing of at least one MPICL. Of these horses, 10 (71%) raced and five (36%) won at least one race. Ten of these horses raced at least twice before and after surgery and only three (30%) of these were able to perform at the same level or better. The mean net score for these horses was significantly less than those with no, or grade 1 tearing ($p<0.05$). Four horses had grade three DMICL enlargement. Of these, two horses raced and one horse won postoperatively. Two horses raced at least twice pre- and postoperatively and both could not perform at their previous level.

The relationship between subchondral bone score and net score did not appear linear (Figure 1). There is evidence that only the more severe subchondral bone damage significantly affects performance (McIlwraith *et al* 1987). For this reason the data for this variable was transformed exponentially and this normalised the distributions and allowed least squares regression to be undertaken with net score. There appeared to be no advantage in transforming the data for MPICL tearing (Figure 2) or articular cartilage damage (Figures 3). Due to the low numbers of

horses with grade 3 DMICL enlargement, this variable could not be included in the multiple regression model.

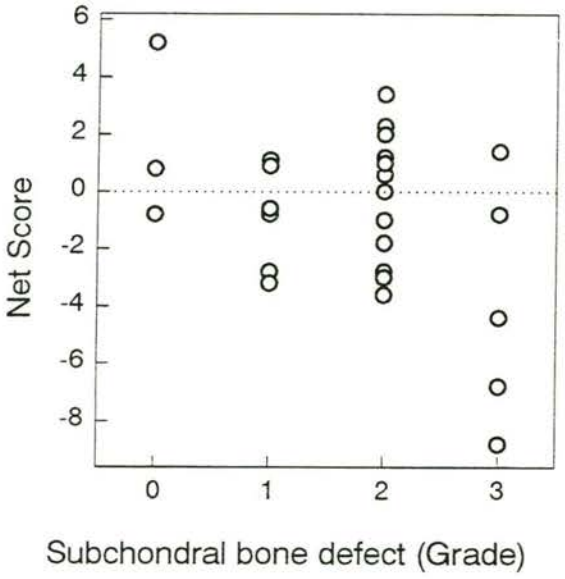

Figure 1.

The relationship between subchondral bone damage and postoperative performance as determined by net score.

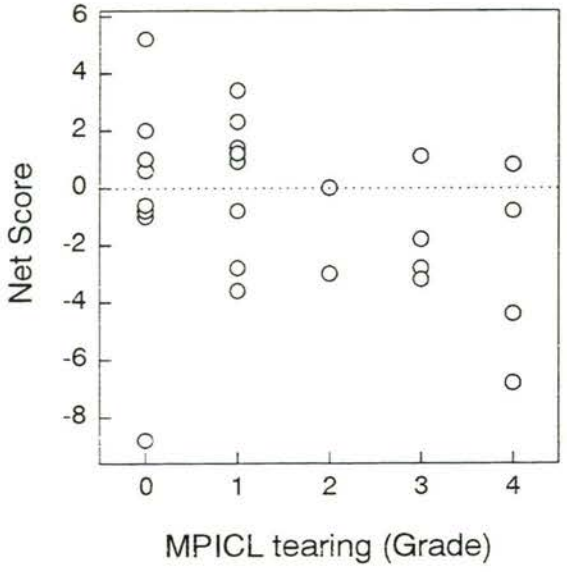

Figure 2.

The relationship between medial palmar intercarpal ligament (MPICL) tearing and postoperative performance as determined by net score.

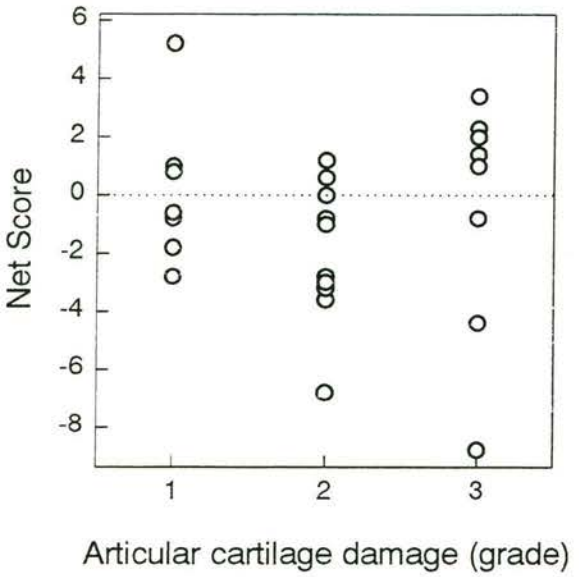

Figure 3.

The relationship between articular cartilage damage in the midcarpal joint and postoperative performance as determined by net score.

Multiple linear regression

The variable which alone best predicted postoperative performance was the extent of subchondral bone damage. This accounted for 23% of the variation in net score ($p<0.05$) and it was the only variable significant on its own (Table 1). The addition of the grade of MPICL tearing to subchondral bone damage significantly improved the prediction of postoperative performance ($F_{2,23}=3.85$, $p<0.05$), but the addition of grade of articular cartilage damage did not.

Table 1.

Results of multiple linear regression model for the relationship between intra-articular variable and net score.

Variables included in model	R^2	p
Subchondral bone damage only	0.23	0.01
MPICL tearing only	0.10	0.10
Cartilage damage only	0.01	0.64
Bone damage and MPICL tearing	0.35	0.007

Although there was no significant effect of rest time on net score the two horses that had the longest rest time (62 and 84 weeks) were the only horses with grade 2-4 MPICL tearing to improve their performance postoperatively.

Discussion

Interpretation of results

Postoperative performance is affected by many factors (Spiers 1983). Following carpal surgery horses are often rested for prolonged periods to allow subchondral bone healing (Kannegieter and Ryan 1991). During this time and subsequent retraining periods, there is the possibility of further injury, and the horse is also older when it returns to work and may no longer have the desire or ability to perform (Spiers *et al* 1986, McIlwraith and Turner 1986). In fact Martinelli *et al* (1996)

demonstrated that there is a natural decrease in performance with increasing age in Standardbred racehorses. Also lameness is often multifocal and failure to perform may not be due to the condition that was treated. When examining at the effect of an individual component of a joint such as the MPICL, there is the added complication of damage to other components of the joint. It is not possible to determine the effect of MPICL tearing on performance without taking into account the degree of subchondral bone damage and articular cartilage damage in the same joint. The use of multiple linear regression allowed the examination of the effect of each individual component of the joint on postoperative performance independent of the other components of the joint.

Ideally, to use pre- and postoperative performance data to determine the significance of an intra-articular lesion it must be assumed that the lesion developed immediately before the diagnosis was made and was still present when the horse resumed racing. The performance data can then be compared with and without the lesion. In practice this is rarely the case. Many articular problems develop gradually and horses often present with a history of low grade problems prior to the development of a more severe one. Wyburn and Goulden (1974) found that 23% of horses with carpal fractures had a gradual onset, and Moore and Schneider (1995) reported that the mean duration of lameness in horses with carpal disease with minimal radiographic changes was six months. In particular it is unlikely that DMICL enlargement develops rapidly and it has even been suggested that it is a congenital condition (Selway 1991). The time of occurrence of the lesions identified in the present study was unknown but no horses presented with a prolonged history of poor performance. Therefore it was considered that even if the lesions were present, they were not severe enough to affect performance in most of the five races before surgery.

There is also the question of whether the lesions were still present when the horse returned to racing. By adequately debriding loose and degenerate subchondral bone and articular cartilage it was hoped that osteochondral fragmentation was no longer present, but an articular surface defect was likely to remain. Even if repair tissue fills in these defects, it is usually of poor quality and unlikely to provide an adequate joint surface (McIlwraith *et al* 1987). Nothing was done to the DMICL or the MPICL during surgery. Although repair of the MPICL is possible, it appears to be limited (Firth *et al* 1991, Chapter 7) and the size of the DMICL is also unlikely to reduce over time.

Factors affecting postoperative performance

This study demonstrated that although the extent of subchondral bone damage was the major intra-articular lesion which affected postoperative performance, changes observed in the intra-articular ligaments also contributed, and to a much greater degree than did the extent of articular cartilage damage. In fact the best prediction of postoperative performance was achieved when the extent of subchondral bone damage and MPICL tearing were combined, and this accounted for 35% of the variation in net score. Considering the multifactorial nature of postoperative performance this seems to be an important finding. Including the extent of articular cartilage damage in the model was of no value in predicting postoperative performance.

Medial palmar intercarpal ligament tearing- The postoperative performance of horses with more extensive MPICL tearing was worse than those with no tearing or only minor damage to the ligament. Some horses with only mild MPICL damage also performed poorly postoperatively

due to large subchondral bone defects. When this was accounted for with multiple regression there was a significant correlation between ligament damage and postoperative performance. The only other study to examine postoperative performance of a series of horses with MPICL tearing suggested that the prognosis was poor with more substantial damage to the ligament (McIlwraith 1992). In that study, of 15 horses with 50% of the ligament torn, only three had a successful outcome. The results of the present study were very similar with only three of 14 horses with significant tearing of the MPICL ligament able to perform at or above their level of performance prior to surgery. In contrast to the present study McIlwraith (1992) trimmed the torn ligament ends. Although it is very difficult to compare the two studies, the similarity in results suggests that the effect of torn ligament remaining in the joint may be small. Other studies have reported postoperative follow up in limited numbers. Moore and Schneider (1995) reported one horse with a complete tear of the MPICL which raced more than 20 times postoperatively and one horse with a partial tear that raced less than 10 times postoperatively. Kannegieter and Ryan (1991) reported follow up on three cases of which only one raced and failed to win postoperatively. Firth *et al* (1991) reported limited follow up (5-6 months) on six horses with MPICL tearing. Three raced and one won one race.

Arthroscopic assessment of tearing of the MPICL may not indicate the degree of compromise to its function. It is not possible to assess the whole of the MPICL with the arthroscope in the dorsal pouch of the midcarpal joint as both palmar bundles and a significant proportion of the of the dorsomedial bundle cannot be viewed (Chapter 5). Most tears only involve the dorsal bundles (Chapter 6) but a more severe tear may pass more palmarly. Laxity of the ligament with the joint in extension, a position in which it is normally taut, has also been observed (Chapter 5)

suggesting that damage may not always manifest as tearing. It may be that in some horses, MPICL tearing is severe enough to cause instability of CR and therefore affect performance, while in others it is not. Simple assessment of tearing of the dorsal aspect of the ligament may not allow adequate assessment of this.

Osteochondral damage- The striking difference between the extent of the subchondral bone defect and articular cartilage damage in their effect on postoperative performance was unexpected. However this finding may explain some discrepancies between previous studies. This is the first study to my knowledge where articular cartilage and subchondral bone damage have been graded separately. McIlwraith *et al* (1987) found that the severity of lesions significantly affected postoperative performance, but used a grading system that combined both subchondral bone and articular cartilage damage. Because of this it is unclear which component was the most important. Antebrachiocarpal joints were also included in the study of McIlwraith *et al* (1987) and it may be that cartilage degeneration is more important in this joint. Kannegieter and Ryan (1991) found that there was no association between the grading of lesions and postoperative performance using a grading system that was only based on the distance articular cartilage erosion extended away from a fracture. As this system was more a measure of articular cartilage than subchondral bone damage, the findings are consistent with the present study. The study of Kannegieter and Ryan (1991) also appeared to have a higher proportion of midcarpal joints than was reported by McIlwraith *et al* (1987).

It therefore appears that loss of articular cartilage in the midcarpal joint in the absence of other pathology may not adversely affect performance. This is in agreement with observations in

human athletes where extensive articular cartilage defects in high weight bearing areas of the knee, with no injury to other intra-articular structures, do not appear to adversely affect outcome (Messner and Maletius 1996).

As expected, the extent of subchondral bone damage was the variable that had the greatest effect on postoperative performance in the midcarpal joint. The relationship with performance was exponential, demonstrating that minor damage (Grade 1 and 2) had little effect, but severe subchondral bone damage was performance limiting. Possible causes of poor performance in these horses are continued bone degeneration and fragmentation, fragmentation of repair tissue into the joint space and subsequent synovitis, increased intraosseous pressure in surrounding subchondral bone, or joint instability caused by loss of articular surface.

Enlargement of the dorsomedial intercarpal ligament- Unfortunately it was not possible to determine the significance of DMICL enlargement from the present data. Further studies are required with larger numbers of horses with DMICL enlargement. However it was interesting to note that no horse with grade 3 DMICL enlargement was able to perform adequately postoperatively. All of these horses had MPICL tearing as well which may have been the primary reason for the poor performance. Selway (1991) suggested that trimming of the DMICL improved the prognosis for horses with carpal joint disease. This assumes that the ligament is a primary cause of poor performance, for which there is no evidence as yet.

Previous studies of postoperative results

In terms of the proportion of horses which raced postoperatively, the results of the present study are similar to those of Moore and Schneider (1995), Lucas *et al* (1995) and Raidal and Wright (1996), but are slightly worse than McIlwraith *et al* (1987) (89%), Kannegieter and Ryan (1991) (87%) and Dabareiner *et al* (1996) (80%). Kannegieter and Ryan (1991) excluded horses that did not return to racing for reasons other than lameness, while Dabareiner *et al* (1996) only reported horses with radiographic lucency of the distal radial carpal bone, and these are likely to be the less severe cases. The number of horses performing at the same or better level was less than that reported by McIlwraith *et al* (1987) (66% of Thoroughbreds with midcarpal joint disease), and Dabareiner *et al* (1996) (68%). In both these studies the technique for determining return to performance was not described, making comparison difficult. Mizuno (1996) and Lucas *et al* (1995), both using a similar technique to that in the present study, found that 59% and 61% respectively of horses with midcarpal joint chip fractures performed at the same or better level.

Rest Time

It has been suggested that horses with MPICL damage should have a longer convalescence period (Wright 1995). Although there appeared to be no effect of rest time up to 12 months, it is possible that longer periods do benefit these horses and that there may be some advantage in prolonged time off post surgery.

Conclusion

It appears that damage to the subchondral bone and changes in the MPICL are two of the major intra-articular lesions that limit postoperative performance of horses with midcarpal joint disease. As a prognostic indicator, the extent of articular cartilage loss is of little importance in the midcarpal joint of racing horses. Considering the difficulties in determining the full extent of MPICL compromise with arthroscopy and the multitude of factors that affect postoperative performance these findings demonstrate that MPICL integrity is clinically important in racehorses.

CHAPTER 10

GENERAL DISCUSSION

Introduction

The studies described in this thesis have attempted to address two major areas: to fully investigate the usefulness of synovial fluid sulfated GAGs as a diagnostic aid in the carpus of racing horses, and an investigation of the structure, function and pathology of the intercarpal ligaments of the midcarpal joint. To do the first, it was necessary to monitor the effect of training on sulfated GAG concentrations in synovial fluid, as significant changes would affect interpretation in diseased joints. This work also provided information on the response of synovial fluid variables to the early stages of joint disease. Results obtained from normal horses in training were then compared with those from clinical cases of joint disease in racing horses in which the carpus was examined arthroscopically. Sulfated GAGs were compared with other routinely used diagnostic techniques. In the second area of study, examination of the anatomy of the intercarpal ligaments of the midcarpal joint was used to make hypotheses on their function and then to devise a biomechanical study to test these. Histopathological examination of both medial palmar intercarpal ligament (MPICL) and dorsolateral intercarpal ligament (DMICL) gave important information on the pathogenesis of ligament changes, and the clinical significance of ligament pathology was addressed by a survey of midcarpal joints and the examination of postoperative race records from horses with varying degrees of damage.

Diagnosis of carpal joint disease

In Chapters 3 and 4, the problems in accurately diagnosing the extent of intra-articular pathology

of the carpal joints were highlighted. In particular, the degree of damage to the articular cartilage and intra-articular ligaments is very difficult to predict without resorting to arthroscopic examination of the joints. Standard synovial fluid analysis appears to be of little use in predicting the extent of articular cartilage damage, and is at best an indicator of the degree of synovitis. The measurement of synovial fluid proteoglycan concentration shows some promise as an indicator of the state of articular cartilage, but due to changes which occur with training, and great differences in response to training and disease between individual horses, it is far from a clinically useful diagnostic aid. Most clinical cases in Chapter 4 were referrals, and there was often a delay in obtaining synovial fluid. As exercise appears to stimulate proteoglycan release from cartilage (Yovich *et al* 1993), better correlations between the extent of articular cartilage damage and proteoglycan concentration may be obtained if synovial fluid samples are collected immediately after exercise. Despite not being able to image cartilage, radiography remains the most useful diagnostic tool for predicting the extent of articular cartilage damage in joints with traumatic osteoarthritis, probably because in young racing horses cartilage degeneration in the carpus is secondary to subchondral bone compromise.

Although one of the aims of these studies was to investigate which diagnostic techniques best predict the severity of articular cartilage damage in the carpus, the findings of Chapter 9 suggest that, at least in young racing horses, articular cartilage may not be the most critical intra-articular structure to evaluate. It appears that in the midcarpal joint it is more useful to be able to detect the extent of subchondral bone damage and the extent of tearing of the MPICL. Radiography is the best technique that is widely available for evaluating subchondral bone, but the direct detection of subchondral bone damage based on the size of fractures and the extent of

subchondral lysis on radiographs was less accurate than expected, particularly in the midcarpal joint. This was because degeneration of bone often extended much further than the margin of articular fractures and changes in bone density are not detectable on radiographs until 40% of bone density is lost (Dyson 1987).

Interestingly, the degree of enthesiophyte production on the dorsomedial aspect of the radial carpal bone (CR) was the best indicator of damage to the articular surface of CR. Enthesiophytes form at the site of soft tissue insertion to bone. The site where these form on CR is quite localised and the only soft tissue structure attaching there is the carpal joint capsule. If the new bone production was due to tearing of joint capsule insertions it would be expected that enthesiophyte production would be generalised across dorsal CR rather than localised as observed. The area on CR where these form is immediately distal to the area where subchondral bone is most commonly damaged. It is therefore possible that enthesiophyte production is due to weakening of the immediately adjacent bone. The periosteum may therefore be responding to detected changes in the biomechanical properties of the adjacent bone, as is thought to occur in the third metacarpal bone (Nunamaker 1996), or the weakened bone is more easily damaged by tension on the capsule and this stimulates new bone production.

So in the midcarpal joint, not only is the estimation of articular cartilage damage dependant on indirect means of estimation, but to some degree so is subchondral bone damage, and imaging techniques that can objectively assess subchondral bone and articular cartilage better, need to be developed. Computed tomography, by allowing imaging in multiple planes, does improve the assessment of subchondral bone compared with standard radiography, but soft tissue structures

cannot be imaged. Magnetic resonance imaging appears to have the most potential as it allows imaging of bone, articular cartilage and ligaments. Research needs to focus on designing equipment that can be used in horses. Scintigraphy is another diagnostic technique that may be useful in this area. Although primarily used at present for identification of stress fractures in long bones, advances in equipment, particularly with computer manipulation of the image, will permit detection of more subtle changes in bone and this technique could have application in the assessment of subchondral bone remodelling.

It is also important to understand that arthroscopy has limitations in the assessment of carpal joint damage. The two palmar bundles of the MPICL cannot be assessed with the usual dorsal approach. Not only may damage to this area of the ligament go unnoticed, but large variations in size of the palmarolateral bundle complicate the interpretation of tearing of the dorsal bundles (Chapter 7). Subchondral bone damage may also be obscured by intact overlying articular cartilage. Identification of these areas requires careful probing and curettage.

These studies have highlighted the need for more research to improve the accuracy of equine joint disease diagnosis. Not only must new diagnostic aids be tested against the actual pathology in the joint, but they need to be compared objectively with diagnostic aids which already exist, to determine if real advances have been made. Diagnostic aids also need to be assessed for each joint, as even within the carpus there is variation between joints in the ability of different techniques to predict intra-articular damage. As diagnostic methods become more sensitive it becomes even more important to study changes in response to training. The difficulty will be in determining what is a normal response to training and what is early pathology.

Although the measurement of sulfated glycosaminoglycans (GAGs) in synovial fluid was not found to be clinically useful in predicting articular cartilage damage, there may be other markers of articular cartilage degradation that are and deserve closer scrutiny. Keratan sulfate, unlike sulfated GAGs, is specific to articular cartilage (Alwan *et al* 1990, Ratcliffe *et al* 1994), but major advantages over sulfated GAGs have not been shown (Carroll 1989). Cartilage oligomeric matrix protein has been shown to be elevated in the synovial fluid of humans with osteoarthritis (Saxne and Heingård 1992) and warrants further investigation in the horse. Link protein has been shown to increase in experimental osteoarthritis in dogs, but unlike proteoglycans, it does not increase with disuse, suggesting it is more specific for osteoarthritis (Ratcliffe *et al* 1994). Both Caterson *et al* (1995) and Lark *et al* (1995) have developed antibodies which recognise proteoglycan fragments generated by 'aggrecanase' in humans. Whether 'aggrecanase' activity is present in equine joints is unknown. Markers of type II collagen synthesis have also been investigated in human patients with osteoarthritis (Lohmander and Shinmei 1994).

Researchers in human osteoarthritis have tried to identify markers of cartilage degradation in serum (Manicourt *et al* 1991, Saxne and Heingård 1992). Many of the markers identified in synovial fluid are taken up by the circulation and can therefore be detected as in synovial fluid. In humans, osteoarthritis often affects multiple joints and blood sample collection is easier than joint aspiration. Detection of markers in serum is of less importance in horses due to the need to identify individual joints with osteoarthritis and the ease of access to most of the commonly affected joints for synovial fluid aspiration.

Synovitis is a common clinical finding in performance horses. It may be observed in association

with osteoarthritis and osteochondral fragmentation or as an entity on its own. Despite assertions that synovitis plays an important role in the development of osteoarthritis, there is in fact no direct evidence that it does. Severe synovitis, either induced chemically or due to septic arthritis, does have demonstrable effects on articular cartilage, but synovial fluid leukocyte counts in these situations are markedly elevated. In Chapter 3, horses developed clinical signs of midcarpal joint synovitis (swelling, pain on flexion) as well as increased synovial fluid total protein concentrations, but there was no change in their synovial fluid leukocyte numbers. This indicates that the synovitis induced by the repetitive loading of high speed training is relatively mild. Whether this level of synovitis results in clinically significant cartilage damage is unknown. The importance of synovitis in the pathogenesis of osteoarthritis in humans has been questioned (Radin *et al* 1984). In equine midcarpal joints, Little *et al* (1990) found that there was no association between the indicators of synovitis and the level of proteoglycan fragments in synovial fluid. This was a cross-sectional study and most cases had advanced osteoarthritis. The articular cartilage was therefore probably heavily depleted of proteoglycans already, so there was unlikely to be any marked response to synovitis. In contrast, in Chapter 4 in a similar group of horses, it was found that sulfated GAGs were correlated with synovial fluid protein, LDH activity and degree of xanthochromia. Similar associations were observed in Chapter 3 in horses with no evidence of osteoarthritis that were followed longitudinally. This is therefore the first direct evidence of a relationship between low grade clinical synovitis and cartilage matrix breakdown in the horse. Whether this degree of synovitis results in permanent cartilage damage is unknown. Further studies are required into the relationship between synovitis and osteoarthritis in horses. In particular more sensitive indicators of synovial inflammation need to be identified.

As discussed previously, sulfated GAGs are only one of many markers of articular cartilage metabolism. It would certainly have been worthwhile to examine the usefulness of other markers in clinical cases of joint disease and to examine how their levels change with training. Sulfated GAGs were chosen, as previous work in horses had demonstrated some potential (Alwan *et al* 1991), and assays for other markers were not available at the time of these studies. The statistical analysis of the data on diagnostic aids for carpal joint disease was limited due to the relatively small numbers of horses. This was due to the need to perform a prospective trial which allowed standardisation of measures of intra-articular pathology. Larger numbers would have allowed the use of log linear analysis to determine whether better prediction of intra-articular pathology could be achieved by combining several diagnostic variables. The pooling of the antebrachiocarpal and midcarpal joint findings would have increased the numbers but it was apparent that there were marked differences between these two joints. Despite these problems the purpose of the study, to identify clinically useful diagnostic techniques, was achieved. If a diagnostic technique is not accurate even in ten horses, it is unlikely to be of any use to clinicians.

Intercarpal ligaments

The ligaments of the equine carpus play an important role by absorbing forces passing up the forelimb while maintaining the carpus as a relatively rigid strut during the stance phase. They must achieve this while still allowing flexion of the limb during the swing phase. Three intra-articular ligaments of the midcarpal joint are of particular interest due to changes observed during arthroscopic surgery. Tearing of the MPICL is common in all types of horses and tearing has also

been observed occasionally in the lateral palmar intercarpal ligament (LPICL) (Chapter 7). Enlargement of the DMICL is also a common finding in racing horses presented for midcarpal joint disease (Selway 1991 and Chapter 6). The MPICL and the LPICL are both true intra-articular ligaments, whereas the DMICL, although partly intra-articular, appears to be part of the medial collateral ligament of the carpus (Chapter 5). The MPICL has the most complex structure being divided into four separate fibre bundles. The primary orientation of all three ligaments suggests that they resist dorsal displacement of the proximal row of carpal bones relative to the distal row. This was confirmed for both the LPICL and the MPICL in Chapter 5. The palmaromedial bundle of the MPICL is orientated in the opposite direction from the other three bundles which would allow it to resist palmar displacement of CR relative to the distal row of carpal bones. Dorsopalmar oscillations of the carpus have been observed soon after heel strike (Hjertén and Drevemo 1994) and the complex structure of the MPICL would allow it to resist the resulting movements of CR.

The relationship between ligament and bone pathology in clinical cases of joint disease was interesting. It was observed that significant MPICL tearing and severe subchondral bone damage rarely occurred together (Chapter 6). It has been suggested that bone and ligament pathology were related, due to the high incidence of ligament tearing in joints with osteochondral fragments (Kannegieter and Colgan 1993). It was thought that ligament rupture would cause joint instability and this would result in damage to the articular surface. Others could find no such relationship (McIlwraith 1992, Phillips and Wright 1994). The findings in Chapter 6 were partly due to an interaction between left and right joints in the same horse and it was suggested that this could be due to changes in gait due to carpal lameness. An interaction between ligament and bone

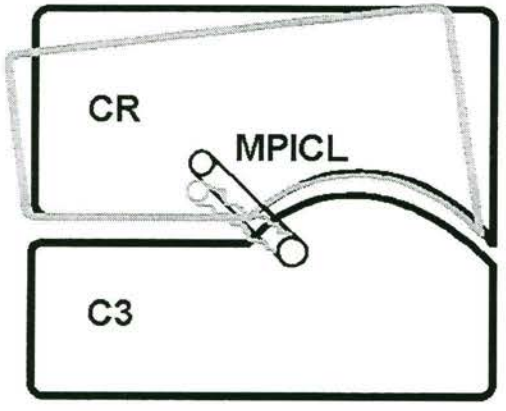

Figure 1.

Proposed mechanism by which extensive loss of subchondral bone from the dorsomedial aspect of the radial carpal bone (CR) results in reduced tension on the medial palmar intercarpal ligament (MPICL) due to dorsodistal rocking of CR.

pathology within individual joints was also observed. It is feasible that extensive loss of subchondral bone on the dorsoproximal aspect of CR could allow dorsodistal rocking of CR during high axial loading. This could reduce tension on the dorsolateral bundle due to its dorsal location within the joint, therefore reducing the risk of ligament rupture (Figure 1). Alternatively, increased dorsal displacement of CR due to MPICL rupture may result in better dissipation of axial forces or move the area of stress concentration palmarly, where the bone may be able to adapt better.

Tearing of the dorsolateral bundle of the MPICL is a common observation in joints of racing horses examined arthroscopically for midcarpal joint disease. However, an even higher incidence of tearing was observed in joints from horses with no history of carpal problems (Chapter 7). This predisposition to damage was explained by a loss of collagen architecture in the dorsolateral bundle in all horses from as early as one month of age, as demonstrated by histological examination (Chapter 8). It was proposed that this was due to abrasion of the synovial membrane by the articular surfaces of the carpal bones during carpal flexion. This results in size reduction, laxity and weakening of the dorsolateral bundle. Because of this, the MPICL is prone to tearing when subjected to tensile forces.

Subchondral bone damage in the midcarpal joint is most commonly observed in Thoroughbred racehorses due to high axial loads generated by galloping at high speeds. The incidence of subchondral bone damage in racing Standardbreds in Chapter 6 was lower, presumably due to lower axial loads. In contrast MPICL tearing was found to be more common and of greater severity in Standardbred racehorses (Chapter 7). Based on the orientation of the dorsolateral bundle of the MPICL, it is likely that transverse forces cause tearing of the ligament (Chapter 5). Transverse oscillations of the carpus have been demonstrated immediately after heel strike in trotting Standardbreds (Hjertén and Drevemo 1994, C Johnston personal communication) and it is these movements which are most likely to damage the dorsal bundles of the MPICL (Chapter 5). At low speed these oscillations occur early in the stance phase, but with increasing speed they continue through to midstance (C Johnson personal communication). Similar kinematic studies have not been performed in the galloping horse, but it is possible that higher axial forces and a shorter stance phase in galloping horses result in stabilisation of the joint earlier than occurs in

trotting horses, thus reducing transverse movements.

Enlargement of the DMICL is also a common observation during routine arthroscopic procedures. It had previously been proposed that this was the cause of osteochondral fragmentation in the midcarpal joint due to impingement on the articular surface (Selway 1991), but in Chapter 6 no association could be found between the severity of osteochondral damage and enlargement of the DMICL. Enlargement was also observed in horses with no history of joint disease (Chapter 7). The cause of enlargement could be either due to hypertrophy of the ligament in response to exercise, or enlargement due to repetitive trauma and scar tissue production. Due to the extent of the enlargement and the observation of focal areas of tearing and repair in enlarged ligaments (Chapter 8), the second cause seems the most likely.

In Chapter 6 a relationship was demonstrated between MPICL tearing and DMICL enlargement. Both ligaments resist dorsal displacement of CR relative to the distal row of carpal bones (Chapter 5). Tearing of the MPICL, resulting in greater dorsal displacement of CR, would subject the DMICL to greater forces and subsequent trauma and enlargement. Larger forces would also be applied to the insertion of the DMICL on CR. It is interesting that this is the site at which remodelling of CR has been observed in association with tearing of the MPICL (Phillips and Wright 1994). Remodelling could also be due to changes in axial loading of the dorsal margin of CR due to instability.

An important aim of the studies described in this thesis was to determine the clinical significance of changes in the intercarpal ligaments. It was demonstrated that the palmar intercarpal ligaments

play an important role in the restraint of transverse forces across the midcarpal joint (Chapter 5). Therefore it is likely that any compromise to their function would cause instability of the carpal bones. However tearing of the dorsolateral bundle of the MPICL was a common observation in horses with no history of midcarpal joint problems (Chapter 7). This would suggest that MPICL tearing was not significant, but was consistent with the findings of McIlwraith (1992) that performance in horses with midcarpal joint disease was only affected when more than 50% of the ligament, visible arthroscopically, was torn. As both the dorsolateral and dorsomedial bundles are visible arthroscopically, tearing of the dorsolateral bundle alone involves less than 50% of the visible ligament. Further, a prospective study of horses with midcarpal joint disease confirmed that MPICL tearing did significantly affect postoperative performance (Chapter 9). Therefore although tearing confined to the dorsolateral bundle was of little clinical significance, more extensive tearing was a significant finding.

The clinical significance of DMICL enlargement remains unclear. Although only small numbers were observed, horses with gross enlargement of the DMICL performed poorly postoperatively. These horses also had extensive MPICL damage so the DMICL may not have been the primary cause of poor performance. Selway (1991) recommended resection of enlarged DMICLs during arthroscopic examination of the midcarpal joint. However, there is no evidence that this would improve postoperative performance.

It appears that assessment of MPICL function (i.e. restraint of dorsal displacement of CR) based on arthroscopic visualisation has limitations due to an inability to observe the whole ligament and large variations in bundle size (Chapters 5 and 7). It is likely that MPICL tearing is only

significant if it causes CR instability. It would therefore be useful if other indicators of CR instability can be identified arthroscopically. As the DMICL and MPICL both appear to restrain dorsal displacement of CR (Chapter 5) and there is a correlation between tearing of the MPICL and DMICL enlargement (Chapter 6), enlargement of the DMICL may be a sign that MPICL tearing has resulted in CR instability. Dorsomedial remodelling of CR has been associated with MPICL tearing so this change may also be an indicator of CR instability as well. I would therefore propose that tearing of the MPICL is only significant if it results in CR instability and indicators of CR instability could be DMICL enlargement and/or dorsomedial remodelling of CR. Both of these changes can be explained by increased loading of the DMICL.

Biomechanical studies using cadaver limbs have many limitations. It is unrealistic to believe that the forces created in the laboratory on an isolated leg are the same as those occurring *in vivo*. However due to the inherent complexity of *in vivo* forces, it can be very difficult to obtain meaningful results in the live animal. Ideally, the study of joint biomechanics should involve the use of a combination of live animal and laboratory testing (Platt and Wilson 1994). In Chapter 5, joint movements and forces which had been identified in the live animal using sophisticated kinematic techniques (Hjertén and Drevemo 1994, C Johnston personnel communication) were applied to cadaver limbs to test a simple hypothesis. Such kinematic studies have only been performed in trotting Standardbreds and need to be extended to galloping Thoroughbreds. Further studies are also required of the movements of the individual carpal bones during the stance phase in the moving horse.

Another problem encountered in the study of the MPICL was the high proportion of adult horses

with damage to the ligament. In fact, careful examination of the MPICL failed to identify any that were considered completely free from gross pathology (Chapter 7). This meant that it was not possible to use joints with normal MPICL for biomechanical testing. However small tears involving only the dorsolateral bundle did not appear to affect the function of the ligament. It was also not possible to have a control group of MPICLs for the study of the ligament's histology. In fact it appears that the histological structure of the ligament deteriorates from a very early age (Chapter 8). Although it was shown that this process does not occur to the same extent in the DMICL, other ligaments of the carpus need to be studied to determine if this is peculiar to the MPICL.

It is a commonly held belief that carpal disease is an inevitable result of racing young horses. However, this was also the case for the "bucked shins" complex and research into the pathogenesis of this problem has led to the development of training regimens which can substantially reduce its occurrence (Nunamaker 1996). Carpal joint disease is one of the common causes of lameness in young racing horses. It is clear that horses with large areas of subchondral bone loss, or loss of restraining ability of the MPICL have a poor prognosis. At present, treatment options are limited due to the poor healing potential of these structures. Although there is a need to develop better treatment modalities, it is also imperative that research is directed towards the development of training programs which reduce the frequency of joint injury.

Conclusions

Carpal joint disease is a common cause of poor performance in racing horses. The studies in this thesis have confirmed that presently available diagnostic techniques do not allow accurate assessment of articular cartilage, intercarpal ligament and, to some extent, subchondral bone pathology. The use of synovial fluid analysis is complicated by marked changes due to training. It was found that radiography remains the most useful technique for evaluating both subchondral bone and articular cartilage damage, but there are important limitations. A relationship was demonstrated between low grade synovitis and sulfated GAG levels in synovial fluid, suggesting that synovitis may play a role in cartilage matrix breakdown.

It was also demonstrated that MPICL pathology is extremely common, probably due to degeneration of the dorsal part of the ligament from an early age in all types of horses. In most horses tearing of the dorsolateral bundle does not appear to affect performance, but complete rupture of both dorsal bundles was clinically significant, possibly due to resulting radiocarpal bone instability. No evidence was found that enlargement of the DMICL is a primary cause of osteochondral damage in the midcarpal joint. It appears that the DMICL enlarges in some joints in response to low grade tearing of its collagen bundles, which is probably a result of increased stress on the ligament.

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THE EFFECT OF TRAINING ON SYNOVIAL FLUID OF THE EQUINE MIDCARPAL JOINT

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Introduction

Musculoskeletal tissues such as bone, tendon and ligament have been shown to undergo changes in response to training in horses. It is also likely that synovial fluid variables change with training, but little is known about this response. The interpretation of synovial fluid variables in performance horses is dependant on understanding these changes. Tew (1982)[1] demonstrated that protein concentration was slightly higher in horses in training, while Yovich *et al* (1993)[2] found that after a single period of exercise, protein was unchanged, but keratan sulfate concentration increased. The aim of the present study was to monitor the response of synovial fluid of the midcarpal joint to prolonged exercise. Also, as it was anticipated that some signs of midcarpal joint disease would develop due to the high intensity of the exercise, the relationship between the levels of one of the markers of articular cartilage metabolism, polysulphated glycosaminoglycan (PSGAG), and the early signs of lameness were examined.

Materials and Methods

Thirteen horses underwent a treadmill training program of 31 weeks duration during which the intensity of exercise was gradually increased. Horses were trained 5 days per week on a treadmill at 10% slope. The training program was divided into three phases. The duration of phase 1 was 7 weeks, during which all horses underwent trot and canter work, up to 4000 m at 6m/s. Phase 2 consisted of 8 weeks of sprint training involving 2 minute sprints at 11m/s. By the end of phase 2 all horses were sprinting for 4000m 3 times a week. At the beginning of phase 3 the horses were randomly split into 2 groups. Group 1 continued sprint training with increases of 500m every 4 weeks, while sprint training in group 2 was increased by 500m every 2 weeks. The duration of phase 3 was 16 weeks.

Synovial fluid was collected from both midcarpal joints at 7, 15, 21, 26 and 30 weeks, and 8 weeks after training was discontinued. An estimate of synovial fluid volume was made by measuring the volume that could be easily

withdrawn from the joint. Synovial fluid colour was evaluated visually and graded based on the degree of xanthochromia. Total nucleated cell counts were performed using the Unopipette method. Total protein was determined using the Biuret method. PSGAG concentration was measured using the dimethylmethylene blue assay. An estimate of the total PSGAGs within the joint was made by multiplying the volume of synovial fluid by the PSGAG concentration.

All horses were examined for lameness at the beginning of the study, and at all sampling times during phase 3. At the end of the training program 11 of the horses were euthanased and their carpal joints dissected and examined for pathology. Data were analysed by repeated measures analysis of variance. Log transformation was used where the data was not of normal distribution. Spearman rank order correlations were used to determine the relationship between the signs of midcarpal joint disease and PSGAG levels.

Results

Post mortem examination of 22 joints revealed minimal gross articular cartilage damage. There was some damage to the medial palmar intercarpal ligaments in 13 joints. When joints of horses from group 1 and 2 were compared there was no significant difference in either articular cartilage or intercarpal ligament damage.

Signs of lameness associated with the midcarpal joint developed in all joints during phase 3 of the training program. There were significant increases in the degree of xanthochromia and protein concentration with training, but leucocyte numbers remained unchanged. Synovial fluid volume decreased during the first 15 weeks of training, but then increased as the intensity of exercise was increased. PSGAG concentration and total PSGAGs changed significantly with training, rising initially, but then declining towards the end of the training program (Figure 1.). Correlations between PSGAG levels and signs of lameness were only weak. There was a significant although weak correlation between total PSGAGs and the degree of articular cartilage damage at post mortem (R=0.20). Synovial fluid variables that were significantly correlated with lameness were xanthochromia (R=0.53), and protein concentration (R=0.67).

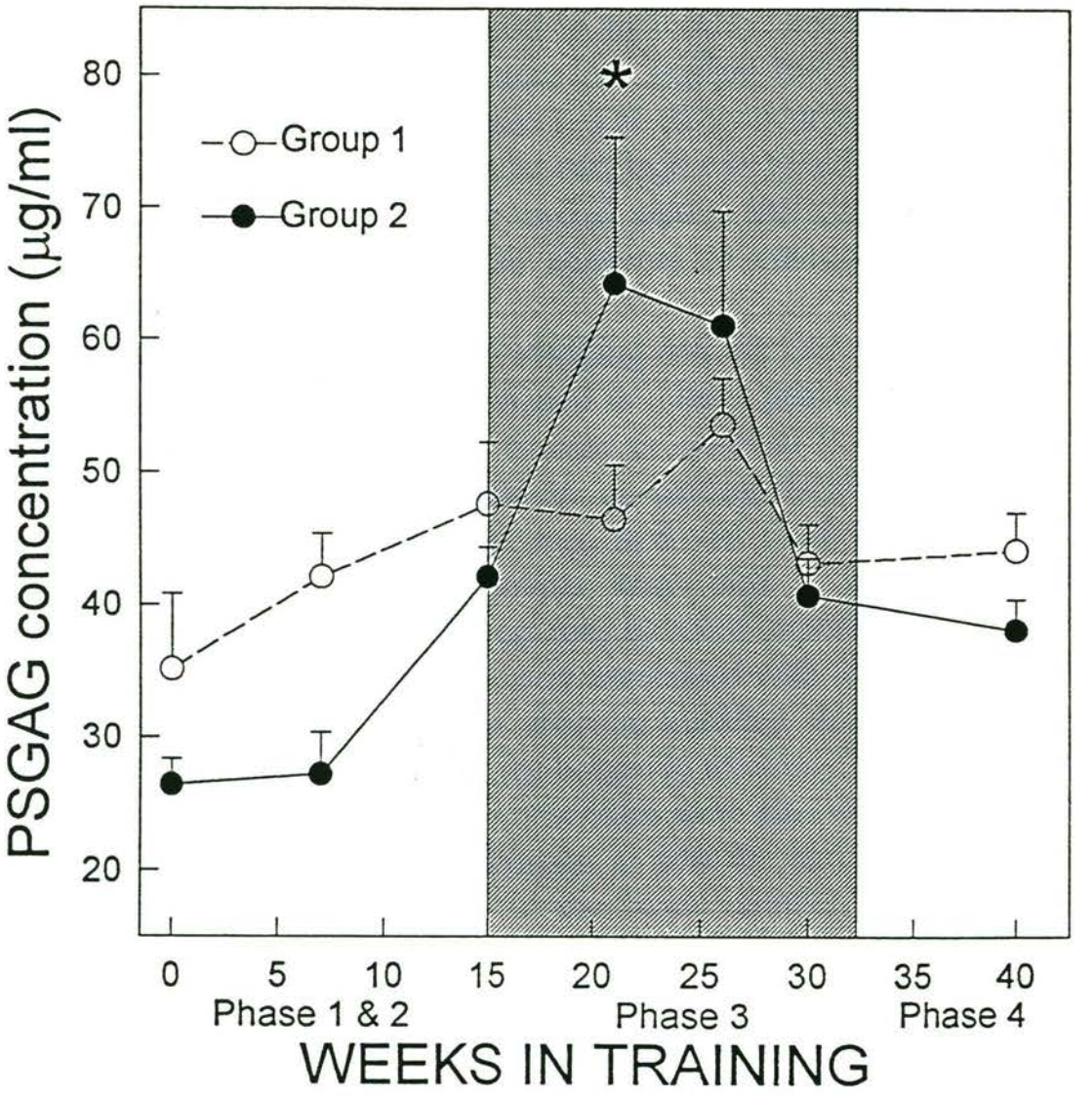

Figure 1. *PSGAG concentrations in groups 1 and 2 during the training program. The shaded area represents phase 3 where groups 1 and 2 are training at differing intensities, (* significant difference between groups 1 and 2).*

Discussion

It is important to understand that this training study was more prolonged, and in phase 3, much more intense than that to which a normal racehorse would be subjected. A normal training program for a Thoroughbred racehorse would be roughly equivalent to the first 15 weeks of this study. The changes in synovial fluid variables observed in phase 3 may therefore not reflect normal responses to exercise but those that occur in the joint under stress.

The changes in synovial fluid volume were complex. Synovial fluid volume is determined by its oncotic pressure, the intraarticular hydrostatic pressure, joint capsule distensibility and the rate of lymphatic drainage. All of these are likely to be affected by exercise and training.

Xanthochromia indicates previous intraarticular haemorrhage. Some of the xanthochromia which developed in this study would have been due to previous arthrocentesis, but that does not explain the significant difference between groups 1 and 2 during phase 3. The likely source of this haemorrhage is the synovial membrane, as articular cartilage was found to be predominantly intact at the end of the study. The medial palmar intercarpal ligament and its overlying synovial membrane also appeared to contribute to intraarticular haemorrhage in these horses.

The signs of midcarpal joint swelling and the increase in synovial fluid protein suggest that synovitis developed in a large proportion of joints in this study. Despite this there was no change in synovial fluid leucocyte counts. This indicates that leucocyte counts are an inadequate indicator of low grade synovitis. The low correlation between synovial fluid protein and PSGAG concentration suggests that synovitis may play a role in chondrocyte metabolism although intrinsic factors may be more important.

PSGAG levels were only weakly associated with the early signs of joint disease. This is understandable as articular cartilage damage is not the only form of joint disease and many of the horses in the study had evidence of other soft tissue damage within the joint. PSGAGs were also weakly associated with the degree of articular cartilage damage at post mortem suggesting they may have a role as a research tool, but the large variation between individuals indicates that it is of little use in clinical cases for predicting cartilage degeneration.

This study demonstrates that prolonged treadmill training at high intensities on an uphill slope compromises the soft tissue structures of the mid-carpal joint with resulting changes in synovial fluid variables. The articular cartilage of the midcarpal joint responds by initially releasing PSGAGs into the synovial fluid, but over time the synovial fluid PSGAG concentration declines, probably due to depletion of cartilage proteoglycan. It appears that articular cartilage of the heavily exercising horse is losing PSGAGs at a very high rate and is probably functioning at or near its physiological limit.

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The Intercarpal Ligaments of the Equine Midcarpal Joint, Part 1: The Anatomy of the Palmar and Dorsomedial Intercarpal Ligaments of the Midcarpal Joint

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Objective—To describe in detail the structure of the medial palmar intercarpal ligament (MPICL), the lateral palmar intercarpal ligament (LPICL), and a dorsomedial intercarpal ligament (DMICL) of the equine midcarpal joint.
Study Design—Dissections of equine midcarpal joints.
Animals and Sample Population—Ten carpal joints from eight thoroughbred horses.
Methods—Detailed dissections of the midcarpal joint were performed, with particular attention paid to the MPICL, the LPICL, and the DMICL. The attachments and dimensions of these structures were recorded, as well as their behavior during joint movement.
Results—The DMICL arose from the dorsomedial surface of the radial carpal bone (CR) and coursed palmarodistally to insert on the dorsomedial aspect of the second carpal bone (C2). The LPICL attached proximally predominantly on the distal part of the palmaromedial surface of the ulnar carpal bone (CU). From here the ligament coursed distomedially and slightly palmarly to the proximal palmarolateral surface of the third carpal bone (C3). The structure of the MPICL was complex. It attached proximally to the distolateral surface of the CR and distally to the proximal palmaromedial surface of C3, and the proximal palmarolateral aspect of the C2. It could be divided into four fiber bundles in all carpi. The predominant direction of fibers was dorsoproximal to palmarodistal, whereas the palmaromedial bundle coursed palmaroproximal to dorsodistal.
Conclusions—The orientation of their fibers indicate that the MPICL and DMICL primarily resist dorsomedial displacement of CR, whereas the LPICL resists dorsolateral displacement of the CU and intermediate carpal bone.
Clinical Relevance—An understanding of the structure of the intercarpal ligaments of the midcarpal joint is important in interpreting their function and the reasons for damage to their structure.

AN IMPORTANT ROLE for the intercarpal ligaments in carpal biomechanics and lameness in horses was proposed by Bramlage et al.[1] With routine use of arthroscopy, awareness of pathological conditions of these ligaments has increased. Tearing of the palmar intercarpal ligaments of the midcarpal joint is commonly recognized,[2-4] and hypertrophy of a dorsomedial intercarpal ligament (DMICL) has been described.[5] The gross[4] and arthroscopic[2,3] anatomy of the medial palmar intercarpal ligament (MPICL) and lateral palmar intercarpal ligament (LPICL) of the midcarpal joint has been described, however the behavior of these ligaments during joint movement has not been examined. There appears to

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be no detailed descriptions of the anatomy of the DMICL of the midcarpal joint.

In the study reported here, anatomic dissections were used to describe in greater detail the structure and function of the MPICL, LPICL and DMICL of the midcarpal joint. Observations were made on the behavior of these ligaments during flexion and extension of the carpus and their role in the restraint of movement of the proximal row of carpal bones.

MATERIALS AND METHODS

Ten carpal joints were obtained from eight thoroughbred horses between 2 and 5 years of age, that were euthanatized for reasons unrelated to carpal disease. Detailed histories were not available, but none had obvious signs of midcarpal joint disease. Joints were stored at −10°C until required. After thawing overnight, detailed dissections of the midcarpal joint were performed, with particular attention paid to the MPICL and LPICL, and the structure variously described as a normal synovial plica[6] and an intercarpal ligament,[5,7] located on the dorsomedial aspect of the joint.

After skin removal, the tendons of the extensor carpi radialis, common digital extensor, extensor carpi obliquus, and lateral digital extensor muscles were dissected free and discarded. The dorsal joint capsule was removed as far as the collateral ligaments laterally and medially, and carefully separated from the DMICL. Any tearing of the intraarticular portion of the palmar intercarpal ligaments was noted, as was cartilage damage within the joint. The palmar carpal ligament was carefully dissected free of its attachments to the palmar aspects of the carpal bones. Then the joint was passively flexed and extended and the behavior of the intercarpal ligaments recorded. The radius was then removed by transection of the joint capsule and the ligaments of the antebrachiocarpal joint. Dissection was continued through the intercarpal ligaments attaching the intermediate carpal bone (CI) to the radial carpal bone (CR) and ulnar carpal bone (CU), taking care to avoid damage to the palmar intercarpal ligaments. Any attachment of these ligaments to CI was recorded before its removal. The palmar intercarpal ligaments could now be observed and their attachments and dimensions recorded.

Further observations were made of the palmar intercarpal ligaments with all other restraining attachments to CU and CR removed to determine their effect on the restraint of these bones in the extended position. The normal position of these bones with the joint extended was determined before removal of the radius. Dimensions of the ligaments were measured with a digital caliper (Mitutoyo, Series 500, Tokyo, Japan) while the ligaments were under moderate tension. Cross-sectional measurements were made at the midpoint of the ligaments.

RESULTS

DMICL

The DMICL, present in all joints, arose from a triangular area, of which the lateral border was a longitudinal ridge on the distal part of the dorsomedial surface of the CR. The ligament did not attach to, or impinge on, the articular surface of CR in any of the joints. From the CR attachment it passed medially and distally, in close association with the joint capsule, and inserted on the proximodorsal aspect of a large eminence on the dorsomedial aspect of the second carpal bone (C2), immediately dorsal to the deep part of the medial collateral ligament (MCL). In one joint some fibers of the ligament inserted on the third carpal bone (C3). The deep part of the MCL attached to the proximal aspect of the same eminence of C2, and its fibers diverged, passing both proximally and dorsally, to attach on the medial aspect of CR. Thus the DMICL formed the dorsodistal part of this fiber complex (Fig 1).

Synovial membrane covered the entire palmar aspect of the DMICL, and on the dorsal surface it extended approximately 15 mm from the origin. The mean length (±SD) of the DMICL was 26.7 ± 2.9 mm, mean width 7.2 ± 1.9 mm at its midpoint, and mean thickness 1.5 ± .05 mm at its midpoint. There was slight spreading of the fibers at both attachments. With the joint extended, the DMICL ran in a palmar and slightly distal direction from the CR curving more distally immediately before inserting on C2. It was flaccid at all angles of flexion and extension, but this was most pronounced in extension where it was often kinked.

LPICL

In all joints, the proximal attachment of the LPICL was predominantly on the distal part of the palmaromedial surface of CU. A few fibers also attached

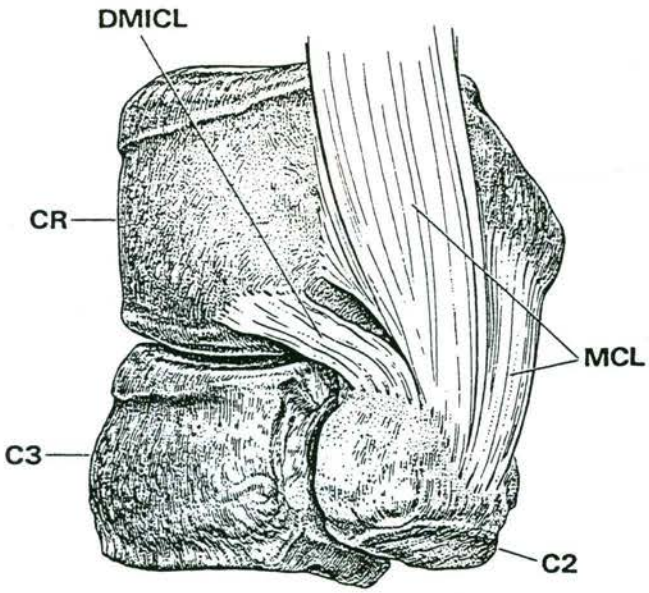

Fig 1. A medial view of the dorsomedial intercarpal ligament (DMICL) of the right midcarpal joint showing its distal attachment to the dorsomedial aspect of the second carpal bone (C2), immediately dorsal to that of the deep part of the medial collateral ligament (MCL). CR, radial carpal bone; C3, third carpal bone.

to the palmarolateral surface of CI. From here the ligament ran in a distomedial and slightly palmar direction to the proximal palmarolateral surface of C3, with a few fibers attaching to the palmaromedial surface of fourth carpal bone (C4) in all joints (Fig 2). The LPICL was either triangular or oval in cross section. In eight joints the LPICL was undivided, whereas in one joint the ligament had a large palmarolateral part and a thin dorsomedial part, and in another the LPICL had a thin palmarolateral part that inserted on C4 and was divided from the main body of the ligament. From its distal attachment on C3, the fibers of the ligament diverged towards CU giving the proximal attachment a greater cross-sectional area. The mean lateral-medial width of the ligament at its midpoint was 5.9 ± 1.5 mm, mean dorsal-palmar depth was 10.2 ± 1.6 mm, and mean length was 14 ± 3.3 mm. With the joint extended, the palmar half to two thirds of the LPICL was under tension while the dorsal half or one third appeared flaccid (Fig 3). With both extension and dorsal displacement of CU, greater tension was evident in the palmar aspect of the ligament. In this position the dorsal surface of the ligament closely opposed the articular margin of C2. Slight fraying of the dorsal surface of the LPICL was observed in one joint and a 2-mm tear of the most dorsomedial aspect of the same ligament was present in another joint.

MPICL

The structure of the MPICL was complex with considerable variation between joints. It attached proximally to the distolateral surface of CR and coursed distally to insert on the proximal palmaromedial surface of C3 and the proximal palmarolateral aspect of C2. The ligament was sheet-like; in cross-section, the distal attachment formed a roughly semicircular arc and the proximal attachment was more linear so that the proximal attachment had a greater dorsopalmar depth than the distal attachment. At its midpoint, the mean dorsopalmar depth was 18 ± 4.6 mm.

The MPICL could be divided into four fiber bundles: dorsolateral, dorsomedial, palmarolateral, and palmaromedial (Fig 4). However, the division was variably defined. In six joints, bilaterally in one horse, the attachment to CR was easily separated into dorsal and palmar parts. The dorsolateral bundle was longer than the dorsomedial, attaching slightly more proximally on CR. From here it coursed distally and palmarolaterally to the fossa at the palmaromedial aspect of C3. The dorsomedial bundle was shorter and attached more distally on CR. Its fibers passed palmarodistally to the lateral aspect of C2. The palmarolateral bundle attached proximally, immediately palmar to the dorsal bundles on the lateral surface of CR. Its fibers coursed palmarolaterally and distally to the palmaromedial surface of C3. The proximal attachment of the palmaromedial bundle was more palmar, distal to the large eminence on the palmaromedial aspect of CR. From here it ran dorsodistally to the lateral surface of C2. Some fibers of both bundles blended with the palmar carpal ligament at their palmar aspects. The intraarticular surfaces of the MPICL were covered with a thin layer of synovial membrane.

Sizes of the four fiber bundles of the MPICL varied between joints. In all joints the dorsolateral bundle was thinnest dorsally. The dorsolateral bundle was thinner (lateral-medial) than the dorsomedial bundle in four joints, thicker in three joints, and the same thickness in three joints (Table 1). Of the two palmar bundles, the palmaromedial was larger in cross section than the palmarolateral.

With the joint flexed, only the dorsolateral and

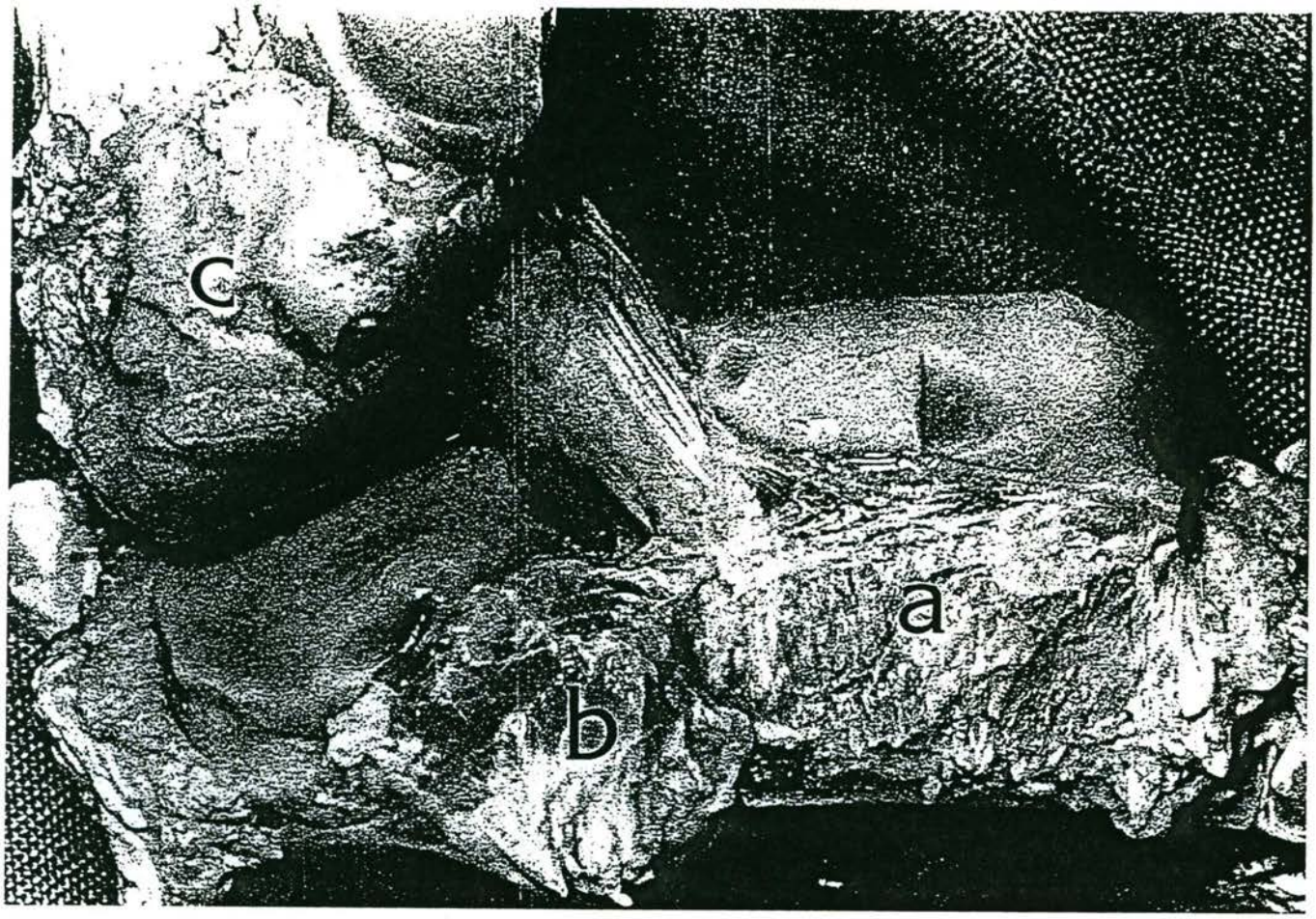

Fig 2. A palmar view of the lateral palmar intercarpal ligament of the left midcarpal joint showing its distal attachment predominantly on the third carpal bone (a), and its proximolateral to distomedial alignment. Also shown are the fourth carpal bone (b) and the ulnar carpal bone (c).

dorsomedial bundle could be observed intraarticularly from the dorsal aspect. Both were slightly flaccid in this position. With the joint extended, all parts of the MPICL were in tension except in five joints in which one to two thirds of the dorsolateral bundle was flaccid. All of these MPICLs had some tearing of the dorsolateral bundle. Most tension seemed to be in the palmaromedial bundle. With extension and dorsal displacement of CR, all parts of the MPICL were in equal tension.

With all attachments to CR removed except for the MPICL, it was not possible to move the CR dorsally or palmarly relative to C3 and C2, but a small degree of hyperextension was possible. The fibers of both lateral bundles coursed in a proximodorsal to palmarodistal direction, whereas those of the dorsomedial bundle coursed in a more proximodistal direction. The fibers of the palmaromedial bundle coursed proximopalmar to distodorsal (Fig 4). Some degree of MPICL tearing was present in 6 of 10 joints. Two of these joints were from the same horse. All tears extended palmarly 1 to 3 mm from the most dorsal margin of the ligament and involved only the dorsolateral bundle. The most dorsal aspect of the ligament often touched or closely apposed the articular margin of C3 and all but one of the tears was at this location. In all joints, a substantial intercarpal ligament was present between CR and CI. This ligament attached to the palmaromedial aspect of CI and coursed dorsomedially to attach to the dorsolateral aspect of CR.

DISCUSSION

Our findings differ from those of Phillips and Wright[4] who described the distal attachment of the

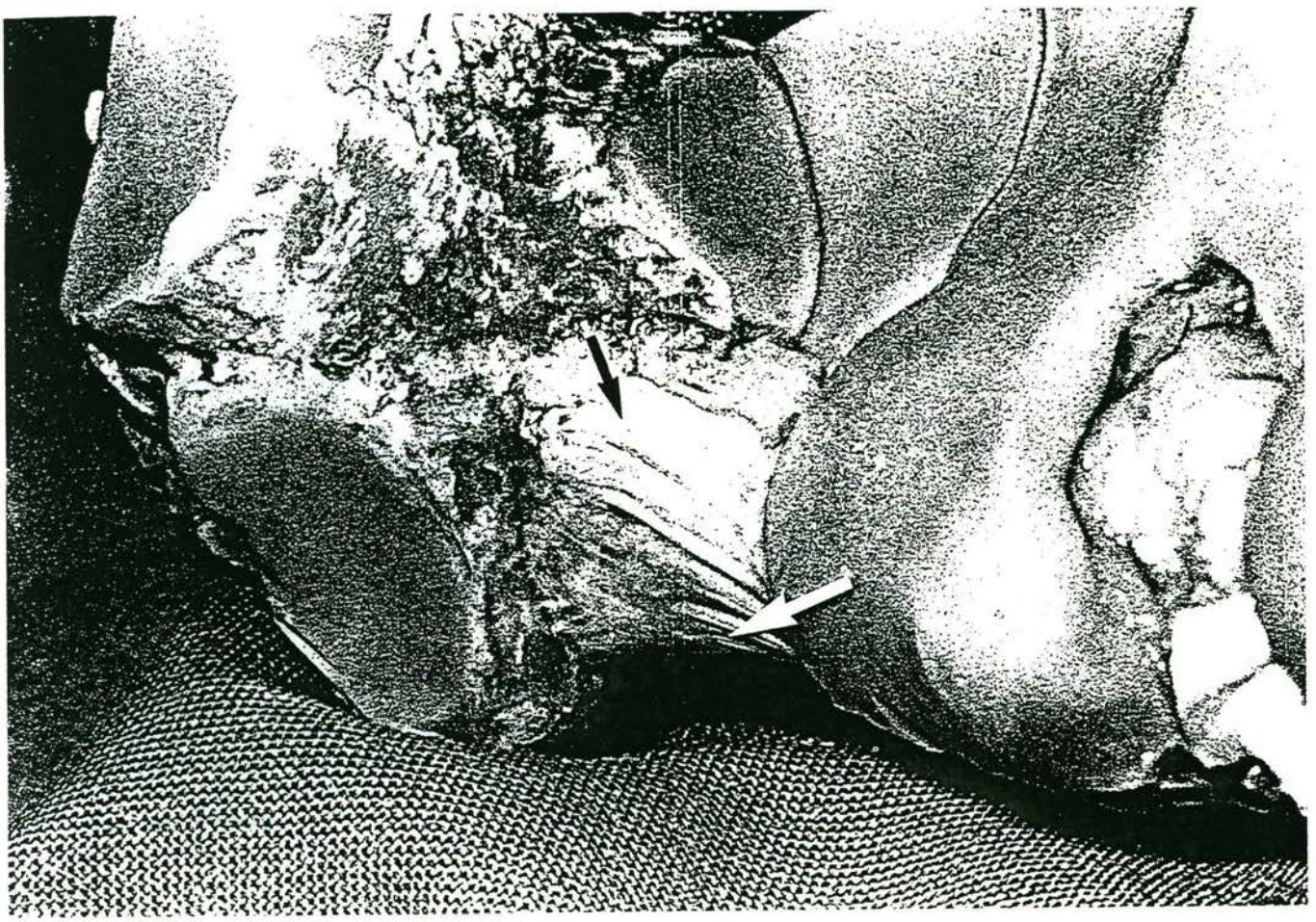

Fig 3. Medial view of the lateral palmar intercarpal ligament of the left midcarpal joint with the ulnar carpal bone held in extension. The synovial membrane lining has been removed. There is tension on the palmar fibers (white arrow) but not the dorsal fibers (black arrow).

LPICL as predominantly on C4, with a smaller attachment on C3. We observed the distal attachment almost wholly on C3, with only a few fibers attached to C4 (Fig 2). Also, two separate fiber orientations of the MPICL were described,[4] which correspond to the dorsomedial and dorsolateral bundles identified in the present study. Although the attachments of the palmaromedial and palmarolateral bundles were described,[4] the separate orientation of the palmaromedial bundle was not. Although in our specimens the four bundles of the MPICL were variably defined, they were consistently observed in all 10 joints.

Despite the increasing recognition of pathology in the MPICL and LPICL,[3,4] the function of these ligaments, and the cause of ligament damage remains unclear. Bramlage et al proposed that the intercarpal ligaments dissipate axial forces by allowing abaxial translation of the carpal bones.[1] This hypothesis was confirmed in a biomechanical study of the loaded carpus.[8] Bramlage et al also suggested that carpitis develops if the intercarpal ligaments do not adapt to increased loads associated with training.[1] It has also been suggested that tearing of the intercarpal ligaments was caused by overextension with concomitant damage to the palmar carpal ligament.[2] Based on the predominant direction of fibers, it has been proposed that the MPICL resists lateral and dorsal displacement of CR, at the limit of midcarpal joint extension.[4]

The equine carpus is locked in extension during the stance phase.[9] There appear to be no studies measuring the forces at the joint during this phase; however, both transverse and axial external force at the joint can be calculated from ground reaction forces.[10] Using data from a galloping horse,[10] it

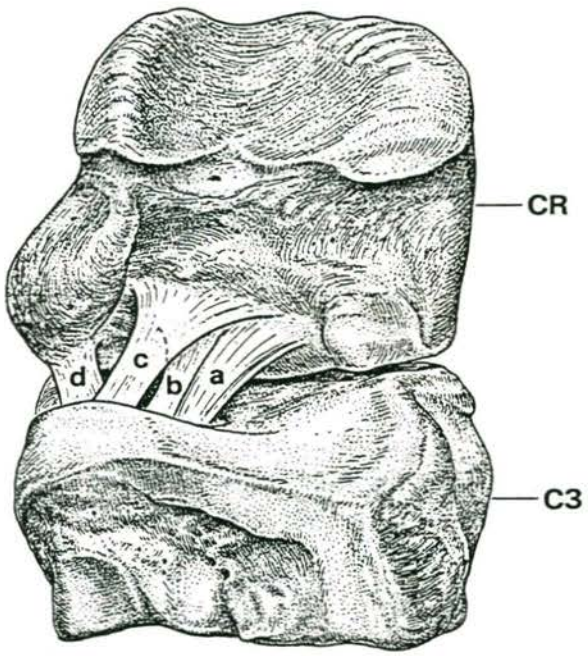

Fig 4. Lateral view of the medial palmar intercarpal ligament of the right midcarpal joint. The dorsolateral (a), dorsomedial (b) and palmarolateral bundles (c) are inclined proximodorsal to distopalmar, whereas the palmaromedial bundle (d) is inclined proximopalmar to distodorsal. The dotted line represents the palmar border of the dorsomedial bundle. CR, radial carpal bone; C3, third carpal bone.

would appear that a transverse force, acting in a palmar direction on the proximal metacarpus, develops rapidly just before midstance and reaches a maximum of approximately 2.1 kN just after midstance. Therefore, axial force is not the only force that must be dissipated by the intercarpal ligaments of the carpus. Unfortunately, the calculation of internal joint forces from ground reaction forces is not possible.[10] Shear forces in a dorsopalmar plane have also been reported at the carpus at heel-strike.[9]

MPICL

From its attachment on C2 and C3, the MPICL courses predominantly in a proximodorsal direction, whereas the lateral bundles pass medially as well. From this observation it is reasonable to assume that the ligament resists dorsal, and to some degree medial displacement of CR. This was confirmed by sectioning all other attachments of CR. On its medial side CR is strongly anchored by the MCL, the deep fibers of which diverge proximodorsally from a palmar attachment on C2 to the medial aspect of CR, mirroring the dorsal and palmarolateral bundles of the MPICL. Therefore, dorsal displacement of CR is prevented by the MPICL on its lateral aspect and by the MCL on its medial aspect (Figs 1 and 4). The orientation of the fibers of the palmaromedial bundle suggest that it resists palmar displacement of CR; this was confirmed when CR was held in extension.

The intercarpal ligament joining CR and CI courses from the palmar aspect of the CI to the dorsal aspect of CR. This allows CR to flex and extend about C3 and C2 independently of CI, while being prevented from displacing dorsally relative to CI when in full extension. It also indicates that there must be strong forces acting to displace CR dorsally as well as medially in relation to CI. Compared with the MCL and the intercarpal ligament between CR and CI, the MPICL is relatively insubstantial. This could explain the predisposition for injury of the MPICL. Also, isolated injury to the MPICL is likely to cause medial rotation of the dorsal face of CR that would still be firmly anchored on the medial side by the MCL. Overriding of the dorsomedial articular margin of CR relative to C3 could produce cartilage erosion and subchondral bone changes on the dorsomedial aspect of CR and the radial facet of C3. Remodeling of the dorsal articular margin of CR in midcarpal joints with MPICL tearing has been observed.[4]

The MPICL probably has only a minor role in preventing overextension of the midcarpal joint. Except for the palmaromedial bundle, most of the ligament is positioned too far dorsally to act with any mechanical advantage.[4] The primary structures limiting overextension of the midcarpal joint are the collateral ligaments[1] and the palmar carpal ligament, all of which are considerably larger than the MPICL and are positioned more palmarly.

LPICL

The LPICL is a simpler structure than the MPICL and, although there are no obvious anatomic divi-

Table 1. Mean Dimensions (±SD) of the Parts of the Medial Palmar Intercarpal Ligament

Fiber Bundle	Lateromedial Width (mm)	Dorsopalmar Depth (mm)	Length (mm)
Dorsolateral	2.8 ± 1.8	8.1 ± 5	15.5 ± 3.5
Dorsomedial	2.3 ± .4	5.8 ± 2.2	10.5 ± .7
Palmarolateral	2.4 ± .5	5.25 ± 2.2	11.5 ± .7
Palmaromedial	2.7 ± .8	7.5 ± .6	8 ± 1.4

sions, the fact that there may be differences in fiber tension in extension suggests that there are differences in fiber function between the dorsal and palmar parts. The proximal attachment of the LPICL is broader than the distal attachment, and tilts laterally and dorsally as it courses towards CU, suggesting that it restricts both dorsal and lateral displacement of CU and CI, which function as a unit.[11]

DMICL

The DMICL[5,7] was identified in all horses. It has been described as an intercarpal ligament[5,7] and as a synovial plica.[6] In all of our specimens there was a distinct proximal and distal attachment of fibers indicating that it was a discrete ligament. The proximal attachment was similar to that described previously.[5] Selway[5] reported that the ligament blended with the fibrous joint capsule distally; however, we observed the DMICL to be in close association with the joint capsule over the distal two thirds of its length, but it remained a discrete ligament. It was also closely associated with the MCL and appeared to form the most dorsal part of that structure.

The function of the DMICL was more difficult to determine. Its position and the direction of its fibers suggest it may also prevent dorsal displacement of CR, but the ligament was flaccid when the joint was in extension. Hypertrophy of the ligament, observed during arthroscopy, has been suggested as a primary cause of joint disease.[5] We did not observe hypertrophy in any of our specimens. However, we have also observed hypertrophy during arthroscopy, commonly in association with MPICL tearing.[12] We speculate that excessive dorsal displacement of CR caused by rupture of the MPICL could bring the DMICL into tension, with subsequent hypertrophy in an attempt to resist this abnormal motion. Enlargement of the ligament may occur because of joint instability and not be a primary cause of joint disease. Enlargement may also be an indication of generalized hypertrophy of the MCL.

The DMICL was not observed to impinge on the articular surface of CR, as has been described.[5] In all of our specimens, the DMICL attached proximal to the articular surface of CR. This site of attachment is a common location for osteochondral damage. Adherence of synovial membrane to osteochondral defects often occurs and it is possible that this is why the ligament has been observed attaching directly to the articular surface of CR during arthroscopy.[5]

Inferences From Observations of Ligament Injury

Determination of the frequency of palmar intercarpal ligament damage was not an objective of our study, but a high frequency of MPICL tearing was noted. The frequency of MPICL tearing observed during arthroscopy has been reported to be 41%[3] and 70%.[4] Injuries of the MPICL occurred in 60% of our specimens but were minor; the most severe injuries only involved 17% of the ligament. Detailed histories of the horses used were not available, but none was euthanatized for carpal lameness and none had obvious signs of carpal disease. Thus, it may be that slight tearing of the dorsal aspect of the MPICL does not cause a clinical problem.

Tearing of the MPICL was always observed at the most dorsal aspect of the dorsolateral bundle, as has been previously noted.[2] The ligament is thinnest at this point and the injured part of the ligament is passed very close to the palmar proximal articular margin of C3. It is possible that stretching of the ligament, caused by minor damage, could allow CR to displace dorsally allowing MPICL abrasion on the articular margin of C3. If tearing of the ligament is caused by dorsal displacement of CR, the dorsal fibers of the ligament should tear before the more palmar fibers as they are inclined more dorsopalmar than the rest of the ligament. If overextension was the cause of ligament tearing,[2] damage to the most palmar aspect of the ligament would be expected to be more common. Tearing of the palmar aspect of the ligament was not observed in any of our specimens.

There is evidence that more subtle forms of damage to the dorsolateral bundle of the MPICL occur. Laxity of the dorsal aspect of the dorsolateral bundle was observed in a number of specimens with damaged ligaments. These fibers had probably reached their yield point but had not completely failed; therefore, surgeons need to be aware that damaged ligaments may not appear grossly torn. Also, the greater variation in dimensions of the dorsolateral bundle, when compared with the rest of the ligament, may reflect thickening because of chronic damage and fibrosis. One investigator attempted to assess MPICL laxity with the joint in flexion during arthroscopic surgery,[2] but our observations suggest that it is normal for the ligament to be lax in the flexed position.

It is probably more relevant to assess laxity with the joint in extension, but this is impossible during arthroscopy.

As reported in other studies[2-4] damage to the LPICL was observed less frequently. We suggest there are three possible reasons for this observation: (1) the LPICL is a wider and simpler structure than the MPICL and it should therefore be intrinsically more resistant to injury; (2) a much smaller proportion of the ligament can be observed arthroscopically so tearing may be underdiagnosed; (3) because the palmar aspect is under the most tension when the joint is in extension, excessive dorsal displacement of the CI and CU unit, or overextension of the midcarpal joint should result in tearing of the palmar aspect of the ligament first. This area cannot be observed by arthroscopy of the dorsal aspect of the joint, so injury may go undiagnosed. The commonly observed superficial fraying of the dorsal aspect of the LPICL[3] may occur because of abrasion on the articular margin of C3.

We have defined in greater detail the structure and function of some of the intercarpal ligaments. The structure of the MPICL is complex and we propose that its primary role is as a lateral restraint to dorsal and medial displacement of CR. The lateral ligament is simpler in structure but performs a similar role with the CI and CU unit. As conclusions based on passive range of motion studies in separated cadaveric specimens have limitations, further studies of the biomechanics of these ligaments are necessary to confirm and better define their function.

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The Intercarpal Ligaments of the Equine Midcarpal Joint, Part 2: The Role of the Palmar Intercarpal Ligaments in the Restraint of Dorsal Displacement of the Proximal Row of Carpal Bones

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Objective—To determine the relative contributions of the palmar intercarpal ligaments in the midcarpal joint to the restraint of dorsal displacement of the proximal row of carpal bones.
Study Design—A biomechanical study of cadaver equine carpi.
Animals or Sample Population—Eight equine forelimbs from six thoroughbred horses.
Methods—With joints in full extension, the radius was dorsally displaced while midcarpal joint displacement was measured. The restraining force at a joint displacement of 1.5 mm was determined from the load-displacement curve. A ligament or pair of ligaments was then cut and the testing procedure repeated. Their contribution to restraining force was calculated as the percentage change in restraining force after the ligament was sectioned. Relative cross-sectional areas of the ligaments tested were measured at the level of the midcarpal joint.
Results—The collateral ligaments were the major contributors to the restraint of dorsal displacement ($P < .001$). In all joints, the palmar intercarpal ligaments contributed a greater proportion than the palmar carpal ligament (PCL) ($P < .05$). The mean percentage (±SEM) contributions to the restraint of dorsal displacement were 62.8 ± 3.4 for the collateral ligaments, 14.5 ± 1.4 for the PCL, and 22.7 ± 2.2 for the palmar intercarpal ligaments. Mean cross-sectional area expressed as a percentage (±SEM) of the total ligamentous area were 9.0 ± 0.3 for the palmar intercarpal ligaments, 27.1 ± 3.0 for the PCL, and 63.8 ± 2.8 for the collateral ligaments.
Conclusions—Despite the small size of the palmar intercarpal ligaments, they play an important role in the restraint of dorsal displacement of the proximal row of carpal bones.
Clinical Relevance—Interpretation, as well as prevention and treatment of intercarpal ligament tearing requires an understanding of their function.

TEARING OF THE medial palmar intercarpal ligaments (MPICL) and lateral palmar intercarpal ligaments (LPICL) in the midcarpal joint of racing horses has been reported.[1] The function of these ligaments and their clinical significance has been the subject of speculation, based on clinical observations.[2-4] Their role and importance in carpal biomechanics is unknown, yet pathology is commonly observed. One of the reasons for a lack of information about the role of the intercarpal ligaments in biomechanics of the carpus is because there are very few biomechanical studies of midcarpal ligament function in the horse.[5]

We have proposed that the main function of the MPICL and LPICL in the midcarpal joint is to prevent dorsal displacement of the proximal row of carpal bones and that it is this motion that causes tearing of the MPICL.[6] Descriptions of carpal function have focused on axial forces,[7] yet transverse forces at the carpus have actually been reported[8] and must be attenuated by the soft tissue structures spanning the joint. The study reported here describes a biome-

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chanical study of cadaver equine carpi in which the relative contributions of the ligaments of the midcarpal joint to the restraint of dorsal displacement of the proximal row of carpal bones were measured.

MATERIALS AND METHODS

Preparation of Specimens

Eight equine forelegs were obtained from six thoroughbred horses between 2 and 5 years of age, euthanatized for reasons unrelated to carpal disease. These were stored at −10°C until required. After thawing overnight, the skin and all soft tissue structures above and below the carpus were removed, including the palmar retinaculum of the carpal canal and the contents of the canal. Using a band saw, the radius was cut 25 cm above the antebrachiocarpal joint and third metacarpal bone was cut 25 cm below the carpometacarpal joint. The accessory carpal bone was removed by severing its ligamentous attachments as close to its surface as possible. The joints were then moistened in 0.9% saline solution, wrapped in plastic, and stored at 4°C until tested the following day. Immediately before testing, the dorsal capsule of the midcarpal joint was removed and a flattened area created on the dorsal aspect of the radial carpal bone (CR), and immediately adjacent on the third carpal bone (C3), with a scalpel and file. The medial and lateral palmar intercarpal ligaments were examined and any signs of tearing recorded. During testing, joints were kept moist with swabs soaked in 0.9% saline solution.

Biomechanical Testing

Testing was performed with a universal testing machine (model 4302, Instron Ltd, Buckinghamshire, England). A 10-kN static load cell measured restraining force. Output of the cell was set at 50%. Joints were mounted in the machine in full extension using two clamps that were designed to firmly hold both the radius and metacarpus (Fig 1). A universal joint was incorporated into the upper clamp so that forces acting on the load cell were unidirectional. The lower clamp holding the metacarpus was fixed, and the upper clamp was attached to the load cell on the crosshead. Care was taken to align the joints so that no rotation occurred when load was applied.

With the joint firmly held in the machine, the crosshead was displaced upwards until full extension

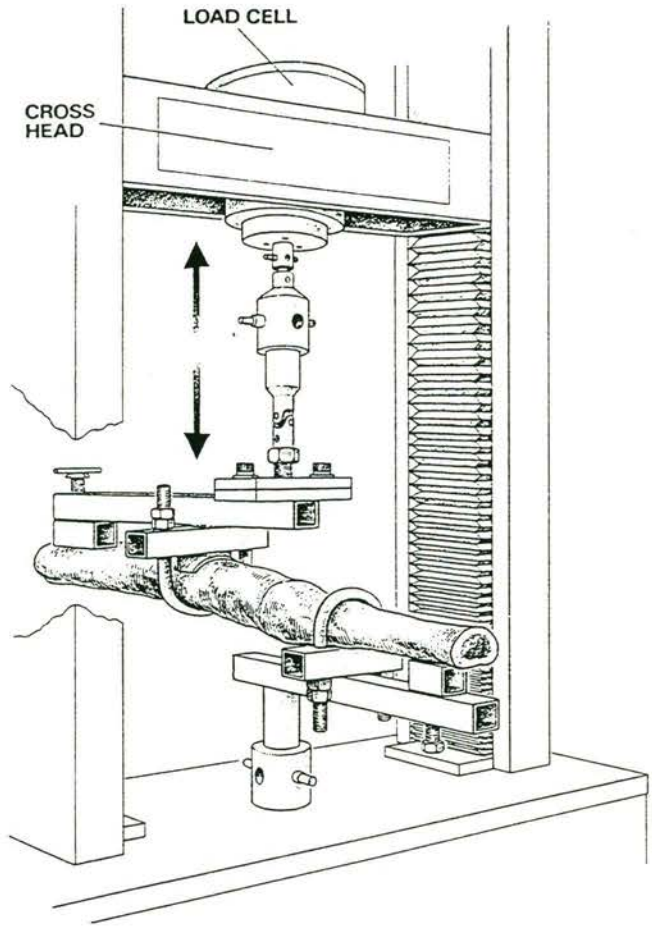

Fig 1. An equine carpus mounted in the testing machine.

of the carpus was reached, as determined by observation. From this position a palmar-dorsal displacement of the radius was performed to a maximum dorsal displacement of 10 mm. This was repeated 30 times to minimize time-dependent force relaxation.[9] During the last five cycles, the change in maximum restraining force was less than .02% in all joints. The joint was again positioned at the point of maximum extension and two dial gauges (Mercer, type 51, Sheffield, England), accurate to .01 mm, were placed on the flattened areas created on the dorsal surface of CR and C3.

The radius was dorsally displaced to 10 mm and the resulting displacement at the joint surface measured. The joint was then cycled four times at a displacement rate of 200 mm/min, with force and crosshead displacement recorded 10 times a second. After this the measurement of maximum joint displacement was repeated. The fourth cycle was used for all measurements.

The joint was removed from the testing machine,

with the clamps still attached, and a ligament or pair of ligaments was sectioned. It was then replaced and the measuring procedure repeated. This process was repeated until no ligaments remained. The ligaments tested were the medial collateral ligament (MCL) and lateral collateral ligament (LCL), the palmar carpal ligament (PCL) and the MPICL, and LPICL. The dorsomedial intercarpal ligament[10] was included as part of the MCL. In four joints the MCL and LCL were sectioned first, followed by the MPICL and LPICL, whereas in four joints the MCL and LCL were sectioned followed by the PCL. Displacement of the crosshead was reduced to 8 mm after sectioning of the collateral ligaments, and 6 mm after sectioning of the PCL, or MPICL and LPICL, because actual joint displacement increased.

Data Analysis

The data generated from the fourth cycle of each testing procedure were used to produce a load-displacement curve. Displacement data from the crosshead movement were converted to joint displacement by dividing the maximum joint displacement by the maximum crosshead displacement. Maximum joint displacement was taken as the average of the two displacements measured before and after the measurement cycle. This produced a constant by which all the other crosshead displacement readings were multiplied. A specimen prepared identically to those tested was used to determine the relationship between crosshead and joint displacement. Joint displacement was measured with crosshead displacement increased by 1 mm increments up to 10 mm. The results showed linearity with r^2 values ranging from .997 with the PCL and collateral ligaments sectioned to .999 for the intact joint. Linear regression was performed on the load-displacement curves for displacements greater than 1 mm to produce an equation for the relationship between load and displacement, at which point all appeared linear. For each test the restraining force at a joint displacement of 1.5 mm was determined using the linear regression equation. A displacement of 1.5 mm was chosen retrospectively because this was the maximum displacement reached by all joints. The contribution of each ligament to restraining dorsal displacement of the proximal row of carpal bones at a displacement of 1.5 mm could then be calculated as the percentage change in restraining force after that ligament was sectioned.

The relative contributions of each ligament or ligament pair tested were compared with a one-way analysis of variance (ANOVA), and post hoc comparisons were performed by determining the least significant difference. ANOVA was also used to determine the effect of the order of sectioning and the effect of ligament tearing. The level of significance was set at $P < .05$.

Cross-Sectional Area

To determine the relative cross-sectional area of the ligaments tested, five frozen thoroughbred carpi from horses aged 2 to 5 years were sectioned at the level of the midcarpal joint with a band saw. Dimensions of the ligaments were measured with a digital caliper (Mitutoyo, Series 500, Tokyo, Japan). Both width (lateral-medial) and depth (dorsal-palmar) were measured at 1-mm intervals and mean width multiplied by mean depth to derive cross-sectional area.

RESULTS

Eight joints were tested. Before testing, a tear extending 4 mm palmarly from the dorsal aspect of the MPICL was observed in joint 4, whereas 1-mm dorsal tears were detected in joints 1 and 3. In joint 3, slight tearing of the intercarpal ligaments was audible at a dorsal displacement of 1.95 mm, with all other ligaments sectioned. In all joints tested, displacement was observed in the three carpal articulations. The intermediate carpal bone (CI) seemed to displace dorsally slightly more than CR. The CR was observed to rotate medially as it moved dorsally, independent of CI. Slight bending of the radius and the third metacarpal bone was also apparent.

In all joints, load increased exponentially with initial displacement, but for joint displacements greater than 1 mm, r^2 values for linear regression analysis ranged from .978 to .999, indicating a strong linear relationship (Fig 2). The mean restraining force (±SEM) at 1.5-mm dorsal displacement of CR for the intact joints was 1.92 ± .12 kN. The mean percentage (±SEM) contributions of the ligaments studied to the restraint of dorsal displacement were 62.8 ± 3.4 for the collateral ligaments, 14.5 ± 1.4 for the PCL, and 22.7 ± 2.2 for the palmar intercarpal ligaments (Fig 3).

In all joints, the collateral ligaments were the ma-

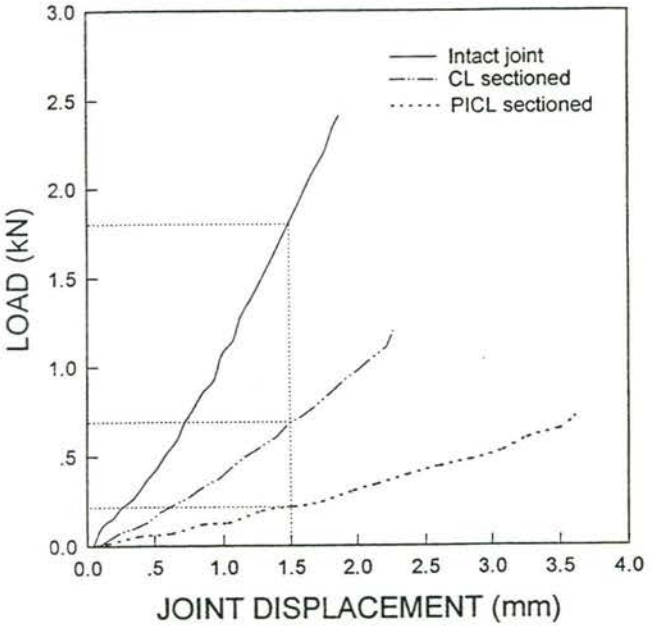

Fig 2. A typical load displacement curve for dorsal displacement of the proximal row of carpal bones of the intact midcarpal joint and with various ligaments sectioned. CL, collateral ligament; PICL, palmar intercarpal ligament.

jor contributors to restraining dorsal displacement of the proximal row of carpal bones, with the percentage contribution significantly greater than the other ligaments ($P < .0001$). In all joints, the palmar intercarpal ligaments contributed a greater proportion than the PCL, and this was significant ($P < .05$). There was no significant effect of the order of sectioning on the percentage contributions of each ligament, and no significant difference between joints with MPICL tearing and those without. Mean percentage contribution of the palmar intercarpal ligaments in joints with tearing of the MPICL was 20.2% whereas in those without tearing it was 26.9%.

When the mean cross-sectional area of each ligament or ligament pair was expressed as a percentage (±SEM) of the total ligamentous cross-sectional area at the level of the midcarpal joint, the palmar intercarpal ligaments made up 9.0 ± 0.3%, the PCL 27.1 ± 3.0%, and the collateral ligaments 63.8 ± 2.8% (Fig 3).

DISCUSSION

Methodology

The method used to determine the contributions of the ligaments of the midcarpal joint to the restraint of dorsal displacement of the proximal row of carpal bones is based on the one described by Butler et al[9] to determine the ligamentous restraints to anterior and posterior drawer in the human knee. By using a set joint displacement and measuring the change in restraining force before and after cutting a ligament, the relative contribution of that ligament can be determined. The same starting point and same end point are used for each ligament so the results are the same regardless of the order in which they are sectioned. This has been shown in the human knee where the results were independent of the order in which the ligaments are sectioned.[9] Our results in the equine carpus are similar, although only two orders of sectioning were possible because of difficult access to the palmar intercarpal ligaments of the midcarpal joint before sectioning the collateral ligaments.

The collateral and intercarpal ligaments were sectioned as pairs to maintain joint symmetry. If only one of these ligaments was sectioned, the joint would tend to rotate when the radius was displaced dorsally, making the measurement of joint displacement difficult. For the same reason, the ligamentous attachments of the accessory carpal bone were not included in the study. The PCL is unpaired but is positioned

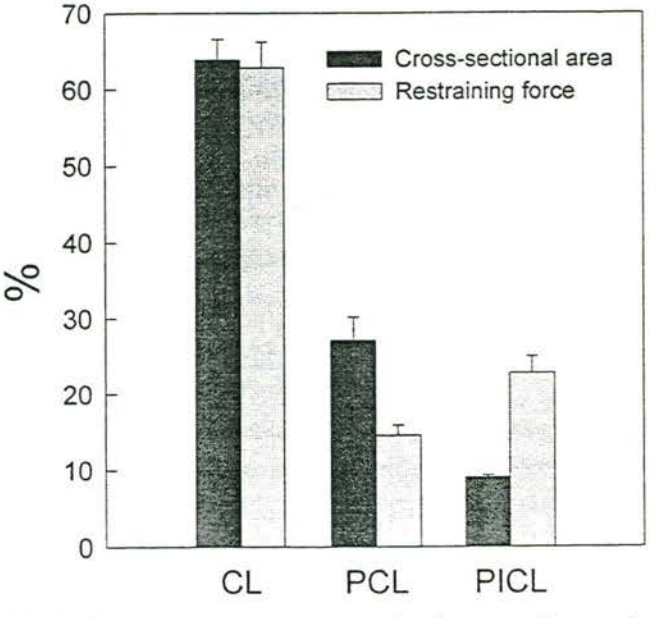

Fig 3. Mean percentage cross-sectional area and percentage contribution to the restraint of dorsal displacement of the proximal row of carpal bones of the ligaments of the midcarpal joint. CL, collateral ligaments; PCL, palmar carpal ligaments; PICL, palmar intercarpal ligaments.

axially. Therefore, soft tissue structures that span the midcarpal joint that were not assessed were the dorsal joint capsule, accessorioquartal ligament, and accessoriometacarpal ligament. The dorsal capsule is flaccid with the joint in extension, so it is likely to provide very little restraining force. The two ligaments of the accessory carpal bone are also unlikely to have any major effect as they do not attach to the proximal row of carpal bones.[11]

Joints with intercarpal ligament tearing were included in the study for two reasons. First, there was no significant difference between joints with ligament tearing and joints without ligament tearing. In fact, the mean percentage contribution of the palmar intercarpal ligaments was greater in joints with ligament tearing. The tears involved only a very small proportion of the total palmar intercarpal ligament cross-sectional area. Second, it is impossible to say which ligaments are normal just by gross examination. The large variation in size of the dorsolateral bundle of the MPICL may be caused by pathology rather than individual variation.[6] In some joints the dorsolateral bundle is very small. Degeneration and complete loss of intra-articular ligaments has been observed in other species.[12]

Joint Displacement

Measurements of maximal joint displacement were made at the joint surface. To determine joint displacement at all points, it was assumed that the relationship between crosshead displacement and joint displacement was linear. In the equine carpus, the antebrachiocarpal and carpometacarpal joints are interposed between the testing machine and the midcarpal joint, meaning that the compliance of the ligaments of these joints and the metacarpus and radius need to be taken into account. Despite this, the relationship between crosshead displacement and joint displacement was shown to be linear with and without ligaments sectioned.

Joint displacement was measured at the dorsolateral surface of CR and the immediately adjacent area on C3. This site was chosen in preference to the dorsomedial surface of CI because most pathology in the midcarpal joint occurs in this area and because the MPICL restrains CR and is the most commonly damaged ligamentous structure. Therefore, we considered that displacement of CR was more clinically relevant. During testing, CI was observed to displace further than CR, indicating that it was not as well restrained as CR. Although slight medial rotation of CR was observed, the dial gauges were positioned to measure only dorsal displacement.

A maximum joint displacement of 1.5 mm was chosen because this was the maximum displacement that all joints were able to reach at all testing cycles. It also corresponded to a mean maximal restraining force of 1.92 kN that was thought to be similar to the forces generated in the carpus of galloping horses. Hjertén and Drevmo[13] reported a method for calculating the external transverse force at the joint from ground reaction forces. Using their data obtained from a 640 kg horse, the maximum transverse force at the carpus was approximately 2.1 kN in a caudal direction. Because these data were obtained from ground reaction forces, there will be some dissipation of this force as it passes up the leg, but it does give an indication of the magnitude and direction of forces. Unfortunately, the calculation of internal joint forces from ground reaction forces is not possible.[13] A typical 450-kg thoroughbred would generate transverse forces of lower magnitude. The slight tearing of the palmar intercarpal ligaments in joint 3 occurred at 1.95 mm, indicating that a displacement of 1.5 mm was near the yield point of the ligaments.

Cross-Sectional Area

The measurement of cross-sectional area of ligaments is difficult. A number of techniques have been described including the use of an area micrometer,[14] laser micrometer,[15] or polymethylmethacrylate casts.[16] None of these methods was considered practical because of inaccessibility of the midcarpal joint and all surfaces of each ligament without extensive dissection. By using frozen sections of joints, all ligamentous structures could be accessed without disturbing their anatomic relationships and with most of each ligament under tension. However, this meant that some parts of some ligaments were not sectioned perpendicular to their long axis. The majority of the fibers of the collateral ligaments and the PCL are aligned proximodistally, but both the MPICL and LPICL have large proportions that are aligned obliquely to the joint surface.[6] The cross-sectional area of the palmar carpal ligaments was, therefore, likely to be overestimated.

Clinical Relevance

It has been proposed, based on clinical observations, that one of the main functions of the intercarpal ligaments is dissipation of axial force during high joint loading.[7] This has been confirmed in a biomechanical study.[5] Transverse or shearing forces, primarily acting to displace the radius dorsally in relation to the proximal metacarpus, have also been shown at the carpus at and soon after heel strike.[8] We have proposed that the palmar intercarpal ligaments play a role in restraint of these forces based on the predominant direction of their fibers.[6] However, it is important to understand that the restraint of dorsal displacement of the proximal row of carpal bones in the live horse is not solely because of ligamentous structures. Joint congruency is also an important factor in joint stability and may be particularly important under high axial loading. Consistent with this, transverse accelerations at the carpus have the greatest magnitude very early in the stride phase before axial loading has peaked.[8] This early phase of the stride may, therefore, be a time when the joint is relatively unstable and reliant on the ligamentous structures of the joint for restraint of transverse forces. The testing procedure described did not allow determination of the effect of axial loading on dorsopalmar stability of the midcarpal joint.

The force-displacement curves obtained were typical for bone-ligament-bone units.[17] The initial nonlinear toe region corresponds to straightening of collagen fibers and loss of crimp. This was followed by a linear region that ended with failure of some fibers.[17] The mean length of the LPICL and the various bundles of the MPICL range from 10.5 to 15.5 mm.[6] Therefore, the tearing detected in joint 3 at 1.95 mm corresponds to a strain of 12% to 19% in these ligaments. Primate anterior cruciate ligaments have been shown to begin to fail at about 40% strain.[18] The relatively low compliance of the palmar intercarpal ligaments reflected the high stability of the midcarpal joint in the extended position and may explain their susceptibility to injury. The collagen fibril diameter of the dorsolateral bundle of the MPICL is less than other carpal ligaments, and this has been proposed as a reason for its low compliance.[19]

These results show that despite the relatively small size of the palmar intercarpal ligaments, they play an important role in the midcarpal joint, along with the collateral ligaments, in the restraint of dorsal displacement of the proximal row of carpal bones. Together their cross-sectional area makes up 9% of the total ligamentous structure of the midcarpal joint, yet their contribution to the total restraint of dorsal displacement is 22.7%. In all joints, the palmar intercarpal ligaments contributed a greater proportion of the restraining force than the PCL, which is three times as large, indicating that prevention of dorsal displacement is not the primary role of the PCL. The CLs were the major contributors to the restraint of dorsal displacement. Together they were seven times larger than the palmar intercarpal ligaments combined, yet contributed less than three times the restraining force.

Dorsal displacement of CR is restricted on the medial side by the MCL and on the lateral side by the MPICL.[6] If, as shown in the present study, the MCL is the stronger of the two ligaments, then CR should rotate medially with dorsal displacement, as we observed. These observations also emphasize that the MPICL is likely the weak link in the restraining apparatus of CR and the most prone to injury with excessive forces on CR.

Intra-articular ligament damage has been reported in other species to cause degenerative joint disease.[20] However, the rate of degeneration in cranial cruciate-deficient dogs is very slow, despite it being the major contributor to restraint of cranial drawer, as shown in the human knee.[7] The MPICL of the horse is relatively less important in overall joint function than the cranial cruciate ligament in dogs, so it follows that degeneration resulting from MPICL tears would proceed extremely slowly. This is supported by other observations (CB Little, University of Sydney, personal communication, 1995) in which no cartilage damage could be produced in horses exercised after arthroscopic sectioning of the MPICL.

The present study has shown that the major restraint of dorsal displacement of the proximal row of carpal bones is the collateral ligaments, whereas the palmar intercarpal ligaments play an important role despite their relatively small cross-sectional area. These observations are consistent with the theorized role of the intercarpal ligaments based on the predominant orientation of their fibers.[4,6]

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The Intercarpal Ligaments of the Equine Midcarpal Joint, Part 3: Clinical Observations in 32 Racing Horses With Midcarpal Joint Disease

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Objective—To characterize the clinical features of intercarpal ligament pathology and to determine the relationship among palmar intercarpal ligament tearing, dorsomedial intercarpal ligament (DMICL) hypertrophy, and other intraarticular lesions.
Study Design—Prospective clinical observations.
Animals or Sample Population—Twenty-eight thoroughbred and four standardbred race horses.
Methods—Clinical, radiographic, and arthroscopic examination of 53 midcarpal joints of 32 horses.
Results—Palmar intercarpal ligament tearing was observed in 30 joints of 22 horses. Some tearing of the medial palmar intercarpal ligament (MPICL) was present in 27 joints of 20 horses, and tearing of the lateral palmar intercarpal ligament in 9 joints of 7 horses. There was no correlation between the severity of clinical signs recorded and the degree of MPICL tearing. Joints with grade 2-4 MPICL tearing had significantly less cartilage and bone damage than joints with grade 1 or no ligament damage ($P < .05$). There was a significant inverse relationship between the number and size of intra-articular fractures, as assessed radiographically, and ligament damage ($R = -.31$). The DMICL was identified in all joints, and in 18 joints the ligament was enlarged. There was a significant correlation between MPICL damage and hypertrophy of the DMICL ($R = .35$). There was no correlation between DMICL hypertrophy and articular cartilage damage or subchondral bone damage.
Conclusions—Severe subchondral bone and MPICL damage rarely occur in the same joint and DMICL hypertrophy may be associated with, rather than a cause of, joint disease.
Clinical Relevance—There are no clinical or radiographic findings that will differentiate intercarpal ligament injury from other carpal injuries. Diagnosis is only possible by arthroscopic examination of the midcarpal joint.

TEARING OF the medial palmar intercarpal ligament (MPICL) and lateral palmar intercarpal ligament (LPICL) of the midcarpal joint of racing horses was first observed by arthroscopy.[1] The frequency of MPICL tearing has been reported to be 41%[2] and 70%[3] in joints examined by arthroscopy, whereas tearing of the LPICL is much less commonly observed. The cause of MPICL injury is poorly understood. Complete rupture of the ligament causes lameness that can be localized to the midcarpal joint,[2,4] but the mechanism for this is unknown and the relationship between ligament tearing and other forms of joint pathology is unclear. An intercarpal ligament on the dorsomedial aspect of the

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midcarpal joint (DMICL) has been described.[5,6] Hypertrophy of this ligament, observed during arthroscopy, has been proposed as a primary cause of midcarpal joint disease in racing horses,[6] but direct evidence for the role of the DMICL in the pathogenesis of midcarpal joint disease is lacking.

The aim of this prospective study was to characterize the clinical features of intercarpal ligament pathology and to determine if there was a relationship among palmar intercarpal ligament tearing, DMICL hypertrophy, and other intra-articular lesions.

MATERIALS AND METHODS

Intercarpal ligament injury, associated bone or cartilage pathology, and clinical signs of carpal joint disease were recorded in 32 horses admitted for arthroscopy of the middle carpal joint between April 1993 and September 1994. Information obtained included the time (in weeks) the horse had been in training since its last rest period.

Clinical Observations

Conformation abnormalities were assessed visually with the horse standing squarely. Abnormalities, graded according to their severity, were grouped into three categories: carpal hyperextension, valgus, and rotational deformity. When no deformity was evident, a score of 0 was given. Slight deformity, when the leg had to be examined closely to detect an abnormality, was given a score of 1. Moderate deformity, one that was obvious to the observer, was given a score of 2. Severe deformities were given a score of 3. Carpal hyperextension was defined as an angle formed by the midline of the radius and metacarpus of greater than 180°. Carpal valgus was defined as lateral angulation of the distal aspect of the metacarpus relative to the midline of the radius when viewed from the cranial aspect, whereas rotational deformity was defined as outward (lateral) rotation of the dorsal aspect of the metacarpus relative to the radius.

All horses were examined at the trot on a firm flat surface. Lameness was graded from 0 to 4.[7] Both forelegs were examined for swelling of the midcarpal joint with the horse weightbearing, and for pain on flexion of the carpus. Swelling was graded from 0 to 3 with 0 defined as no detectable swelling, 1 as swelling only evident on close examination, 2 as easily detectable swelling, and 3 as marked swelling. Pain on carpal flexion was graded 0 if no pain was detectable, 1 if pain was detectable on extreme flexion, 2 if pain was detected in the last 15° of flexion, and 3 if pain was detected before the last 15° of flexion. Lameness evaluation performed after carpal flexion was graded from 0 to 2. If there was no increase in lameness, a grade of 0 was given, whereas grade 1 was defined as an increase in lameness of 1 grade after flexion, and grade 2 as an increase of two grades.

Radiographic Observations

Radiographic examination of all carpi included a flexed lateromedial, a 45° dorsomedial-palmarolateral, 45° dorsolateral-palmaromedial oblique, and flexed dorsoproximal-dorsodistal skyline projections. All radiographs were assessed for the number, type, position, and size of carpal bone fractures. They were also assessed for the degree of subchondral bone lucency and the size, number, and extent of enthesiophytes and osteophytes (Table 1).

Surgical Observations

Articular surfaces of the bones of the midcarpal joint were closely examined by arthroscopy.[8] Cartilage damage was graded from 0 to 3 according to the area of full-thickness cartilage erosion. If no cartilage erosion was observed, the joint was given a grade of 0. Full-thickness erosion with an area less than 1 cm^2 was scored as grade 1, 1 to 2.5 cm^2 as grade 2, and greater than 2.5 cm^2 as grade 3.

Subchondral bone damage was defined as intra-articular fractures or soft subchondral bone that was easily removed with a curette. Areas of cartilage erosion were gently debrided to determine the quality of the underlying bone. If soft bone was detected, it was debrided to firm bone. Subchondral bone damage was graded according to the area affected; grade 0, no subchondral bone damage was detected; grade 1, a single small chip or less than 1 cm^2 of soft subchondral bone; grade 2, a slab fracture or 1 to 2.5 cm^2 of chip fractures or soft subchondral bone; grade 3, more than 2.5 cm^2 soft subchondral bone or chip fractures.

To assess the palmar intercarpal ligaments, the leg was positioned with the carpus in full flexion. Careful probing of the ligaments was then performed

Table 1. Radiographic Grading System Used For Assessment of Carpal Radiographs

Score	Osteophytes or Enthesiophytes	Subchondral Lysis	Fractures
0	None	None	None
1	Small (extending <2 mm) and present in only one view	<5 mm^2 on one view	Single small chip (<10 mm^2)
2	Large (extending >2 mm) or present in more than one view	>5 mm^2 or detectable more than one view	Two small chips or one large chip (>10 mm^2)
3	Large (extending >2 mm) and present in more than one view	Not used	Three or more chips or a slab

to determine the proportion of cross-sectional area intact. The grading system used has been previously described.[2] Mild damage with rupture, fraying, or stretching of a small number of fibers was graded as 1 (Fig 1); grade 2 included those ligaments with up to one third damaged (Fig 2); in grade 3, one to two thirds of the ligament was damaged; grade 4 was used for complete rupture of the ligament (Fig 3).

The intercarpal ligament on the dorsomedial aspect of the midcarpal joint was assessed and graded from 0 to 3 based on size. If no ligament was detected, a grade of 0 was used. Ligaments less than 2 mm thick (lateral-medial) that blended into the joint capsule before passing the proximal medial articular margin of the second carpal bone were graded as 1 (Fig 4), whereas those more than 2 mm thick or easily differentiated from the joint capsule over their whole length were graded as 2 (Fig 5), and ligaments more than 5 mm thick were graded as 3 (Fig 6).

Data Analysis

Spearman rank order correlations were performed between various observations with significance set at $P < .05$. Because individual joints from bilaterally affected horses were not independent observations, the analysis was performed both with all joints included and with only the left or right joints from bilaterally affected horses. A Mann-Whitney U test was used to compare the findings in joints with grade 1 or no visible MPICL tearing, with joints with grade 2 to 4 MPICL tearing, as well as the severity of injury between left and right joints.

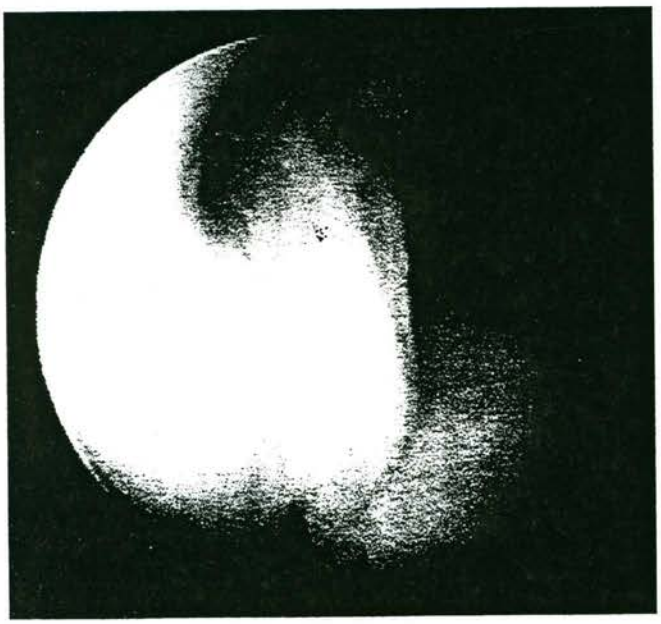

Fig 1. Grade 1 medial palmar intercarpal ligament tear with fraying of a small number of fibers.

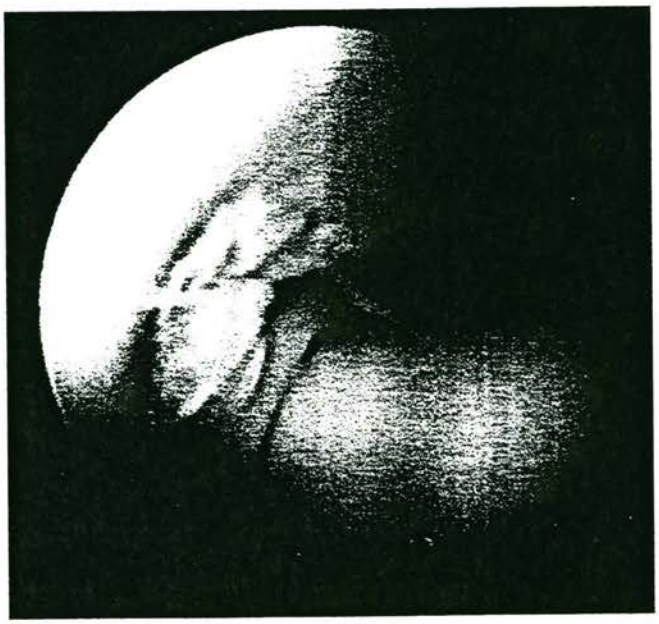

Fig 2. Grade 2 medial palmar intercarpal ligament tear with less than one third of the ligament damaged.

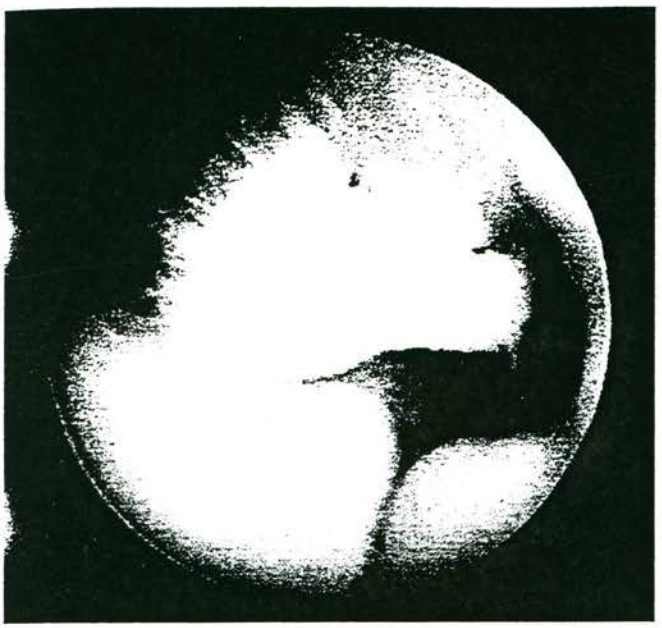

Fig 3. Grade 4 medial palmar intercarpal ligament tear. Complete rupture of the dorsal portion of the ligament has occured.

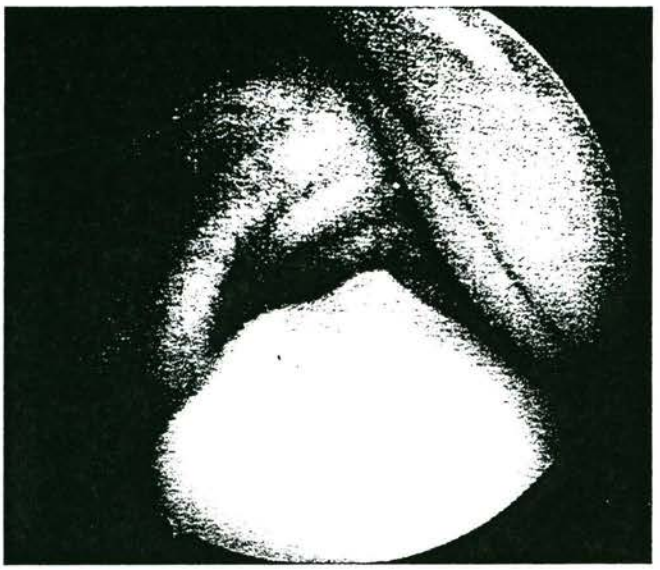

Fig 5. Dorsomedial intercarpal ligament, grade 2. This ligament is easily differentiated from the joint capsule over its whole length.

RESULTS

A total of 53 midcarpal joints of 32 horses were examined. Four horses were racing standardbred pacers (7 joints) and 28 horses were racing thoroughbreds (46 joints). Horse ages ranged from 2 to 5 years. Palmar intercarpal ligament tearing was identified in 30 joints of 22 horses. Tearing of the MPICL alone occurred in 21 joints of 16 horses, tearing of the LPICL alone occurred in 3 joints of 3 horses, and tearing of both ligaments in the same joint occurred in 6 joints of 5 horses.

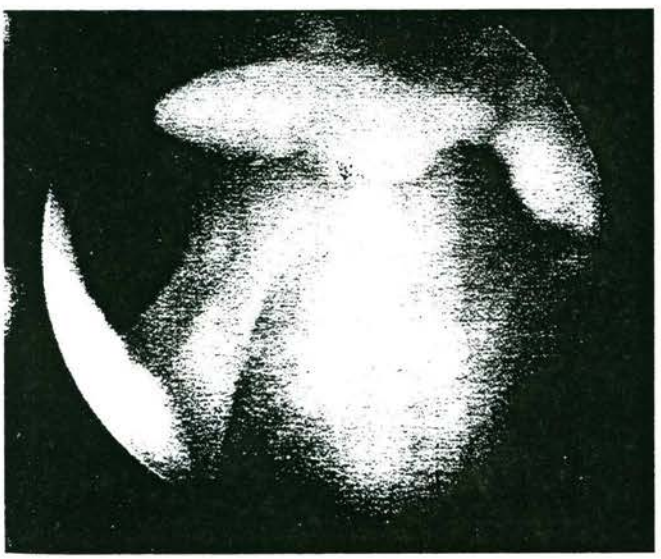

Fig 4. Normal (grade 1) dorsomedial intercarpal ligament. The ligament blends with the joint capsule before passing the proximal articular margin of the second carpal bone.

Fig 6. Enlarged dorsomedial intercarpal ligament, grade 3.

Some tearing of the MPICL was present in 27 joints in 20 horses, and tearing of the LPICL was evident in 9 joints in 7 horses. Of the 27 MPICL injuries, 14 were grade 1 tears, 2 were grade 2 tears, 6 were grade 3 tears, and 5 were grade 4 tears. The LPICL injuries consisted of 7 grade 1 tears and 2 grade 2 tears. Both grade 2 tears occurred in horses with grade 4 MPICL injuries. Moderate to severe subchondral bone damage and no MPICL tearing was observed in one joint, with grade 2 to 4 ligament tearing and no or mild subchondral bone damage in the opposite joint in two horses.

Tearing of the MPICL was observed in 13 left and 14 right midcarpal joints. For thoroughbred racehorses, there was no significant difference between the severity of tearing in left and right midcarpal joints. There was also no significant difference in the degree of articular cartilage and subchondral bone damage between left and right joints. The frequency of MPICL tearing was similar in thoroughbred and standardbred carpi. Of the 47 thoroughbred midcarpal joints examined, 23 had evidence of MPICL damage, whereas 4 of the 7 standardbred joints had evidence of damage. One horse with a grade 3 MPICL tear was re-examined by arthroscopy 6 months after the initial diagnosis. There was no evidence of healing at the second examination.

There was no significant correlation between the degree of MPICL damage and the severity of carpal valgus and carpal rotation. Too few horses were observed with carpal hyperextension conformation to allow statistical analysis. Of the three horses with bilateral grade 2 carpal hyperextension, one had a grade 4 and a grade 2 MPICL tear, while another had a grade 4 and a grade 1 MPICL tear.

There was also no correlation between the severity of clinical signs recorded and the degree of MPICL or LPICL tearing. However, there was a significant correlation between the degree of lameness and articular cartilage damage ($R = .29$, $P < .05$), and the degree of swelling ($R = .32$, $P < .05$) and pain on flexion ($R = .27$, $P < .05$) with subchondral bone damage. When bilateral midcarpal joint arthroscopy was performed on 22 horses, damage to the left MPICL was different from damage to the right MPICL in 12 horses. In five of these horses the lameness was worse in the limb with the more severely affected ligament, whereas in seven horses the lameness was more evident in the limb with the less severely affected ligament.

No significant correlation was found between the degree of damage to the MPICL and the degree of articular cartilage or bone damage, but joints with grade 2 to 4 MPICL tearing had significantly less ($P < .05$) cartilage and bone damage than joints with grade 1 or no detectable ligament damage. There was a significant inverse relationship between fracture score, as assessed radiographically, and ligament damage ($R = -.31$, $P < .05$). A weak correlation ($R = .23$) between MPICL and LPICL damage was shown, but this was not significant ($P = .09$). When normal joints (no MPICL tearing or subchondral bone damage) were excluded, there was a significant negative correlation among ligament damage and fracture score ($R = -.53$, $P < .001$), bone defect ($R = -.55$, $P < .0001$), and cartilage damage ($R = -.36$, $P < .05$) (Table 2). When only the left joints from bilaterally affected horses were included, the significant negative correlation between ligament damage and fracture score ($R = -.66$, $P < .0001$) and bone defect ($R = -.49$, $P < .01$) remained. When only right joints were included, fracture score ($R = -.53$, $P < .01$), bone defect ($R = -.65$, $P < .001$), and cartilage defect ($R = -.44$, $P < .05$) were significantly negatively correlated with ligament damage.

The DMICL was identified in all joints. In 18 joints, the ligament was enlarged, with marked enlargement noted in 5 of these joints. There was a significant correlation between MPICL damage and hypertrophy of the DMICL ($R = .35$, $P < .01$). The degree of hypertrophy of the DMICL was significantly greater ($P < .001$) in joints with grade 2 to 4 tearing, than in joints with no, or grade 1 tearing. There was no correlation between DMICL hypertrophy and articular cartilage damage or subchondral

Table 2. Relationship Between Medial Palmar Intercarpal Ligament Damage and Subchondral Bone Damage in the Midcarpal Joint of Racing Horses

Medial Intercarpal Ligament Damage (grade)*	Subchondral Bone Damage (Grade)*			
	0	1	2	3
0	7	4	9	6
1	2	4	7	1
2	1	1	—	—
3	0	4	1	1
4	2	3	—	—

* For a description of the injury grade, refer to the Materials and Methods section.

bone damage. A significant correlation between the number of weeks in training and DMICL hypertrophy was observed ($R = .41$, $P < .01$). The DMICL was not observed to impinge on the articular surface in any of the joints studied, although in a number of joints it adhered closely to damaged subchondral bone at the dorsal articular margin of the distal aspect of the radial carpal bone (CR).

DISCUSSION

In the present study of racing horses with carpal disease, the frequency of MPICL tearing was 51%, whereas the frequency of LPICL tearing was 18.5%. In previous studies the highest frequency of MPICL tearing has been observed in the United Kingdom (70%),[3] whereas the frequency in Australia (41%)[2] is greater than that in the United States (8.7%).[4] These differences may be attributed to a number of factors including racing surfaces, race distances, breeds involved, and differing interpretation of the degree of tearing by surgeons.

Very few horses in this study had carpal hyperextension conformation, but two of the three most affected horses had complete MPICL tears. Further studies are required to determine if this conformation predisposes to MPICL tearing. Differences in the frequency and severity of ligament damage between left and right midcarpal joints may indicate an effect of racing direction on the pathogenesis of ligament injury. Standardbreds were not included in this comparison because they race in an counterclockwise direction in our practice area, whereas thoroughbreds race in a clockwise direction. There was no predisposition to injury for left or right carpal joints in thoroughbred racehorses in our study.

Arthroscopy remains the only method of diagnosing intercarpal ligament damage. The present study confirmed that there are no characteristic clinical signs that will differentiate intercarpal ligament injury from other carpal injuries. Others have observed that horses with ligament tearing and osteochondral fragments had a greater degree of lameness and effusion than was expected for the degree of osteochondral fragmentation.[4] It has also been noted that joints with ligament tearing and no osteochondral damage had a greater degree of effusion than the opposite joint with only osteochondral fragments.[4] This was not observed in the present study. Even in horses with bilateral midcarpal joint disease there was no possibility of predicting which joint would have ligament damage. Despite these difficulties, intercarpal ligament tearing must be considered in the differential diagnosis for horses with lameness localized to the midcarpal joint and with no radiographic changes.[9,10] Ultrasound has proven invaluable in the diagnosis of orthopedic soft tissue disease, but the position of the MPICL, deep within the carpus and surrounded by bone, together with its complex structure make ultrasonic examination extremely difficult. The use of magnetic resonance imaging, which is used in the diagnosis of cruciate ligament tearing in humans,[11] needs to be investigated.

Only two grade 2 LPICL tears were observed, but these occurred in joints with grade 4 MPICL tears, suggesting that severe intercarpal ligament injuries tend to occur together. We previously proposed that the low frequency of LPICL tears observed during arthroscopy was caused by the difficulty in viewing the ligament and that tearing of the palmar aspect of the ligament would tend to occur before tearing of the dorsal aspect.[12] Therefore, it is possible that many LPICL tears are undiagnosed. The observation of combined intercarpal ligament injury also shows that injury to the MPICL is not always isolated, and injury to other extra-articular soft tissue structures may occur concurrently. Some of these structures are accessible with ultrasound, particularly the medial collateral ligament. However, desmitis of the medial collateral ligament is not easily observed with ultrasound because of its normal irregular echogenicity caused by the different orientation of its fibrous planes.[13]

An inverse relationship was shown between MPICL tearing and osteochondral damage. This relationship was more pronounced when only those joints with some form of injury to either the subchondral bone or MPICL damage were considered. This was an unexpected finding as ligamentous damage and the resulting instability is usually associated with articular cartilage degeneration.[14] However, as the relationship was stronger for subchondral bone damage (particularly fractures) than articular cartilage damage, it is probably more a reflection of bony change rather than cartilage degeneration. This relationship has not been previously shown, but may explain why others have found no correlation between articular cartilage and ligament damage.[3,4] The only other study to specifically examine subchondral bone damage only recorded the presence or absence

of fractures.[3] The number and size of fragments was not recorded, nor was the extent of soft subchondral bone, so a true evaluation of subchondral bone compromise was not made. Kannegieter and Colgan found that the frequency of ligament damage was higher in joints with severe osteochondral damage, but they could find no correlation between the severity of osteochondral and ligament injuries.[2] In the present study, both the frequency and severity of ligament tearing was lower in joints with severe osteochondral damage.

Therefore, there appears to be two common sites of failure in the midcarpal joint. The subchondral bone of the dorsal aspect of CR and the MPICL. Rather than ligament rupture resulting in osteochondral damage because of joint instability and subsequent abnormal loading, it appears that the forces generated at racing speeds within the carpus result in either one site or the other predominantly failing. This is consistent with observations that no articular cartilage damage occurred in horses trained on a treadmill after sectioning of the LPICL and MPICL (CB Little, University of Sydney, personal communication, 1993). Our results also indicate that this apparent inverse relationship is caused by both a direct interaction between ligament and subchondral bone damage within the joint, and an interaction between both left and right joints. McIlwraith reported six cases similar to the two in our study that had moderate to severe subchondral bone damage to the distal margin of CR with mild or no detectable ligament tearing, but in the opposite joint there was a grade 3 or 4 ligament tear with no intra-articular fractures.[4] There is evidence that carpal lameness in one limb changes the biomechanical variables in the opposite forelimb by increasing the horizontal ground reaction force, with no change in vertical force.[15] This could increase the risk of intercarpal ligament tearing in the contralateral limb to one with chip fractures, without increasing the risk of subchondral bone damage.

Although we found no evidence that MPICL rupture results in significant osteochondral damage in the midcarpal joint, it is unlikely that MPICL rupture is innocuous. First, most of the joints with severe MPICL tearing had some degree of cartilage and subchondral bone damage. Also, although not observed in the present study, remodeling of the dorsodistal articular margin of CR has been shown to be associated with MPICL tearing.[3] Degeneration of cartilage and bone secondary to MPICL tearing is likely to develop slowly as the rate of degeneration after intra-articular ligament damage in other species is very slow.[16] Second, MPICL rupture will place increased stress on other intercarpal ligaments, as shown by the hypertrophy observed in the DMICL. Desmitis of the intercarpal ligaments has been proposed as a major cause of lameness.[17]

There are few reports of the findings of follow-up arthroscopic examinations of torn MPICLs. Only one joint in the present study was re-examined 4 months after the diagnosis with no evidence of healing. Healing after 6 months rest has been observed in three of four joints re-examined[18] and after 12 months in 1 joint.[2] The potential for, and the factors affecting healing need further investigation.

Hypertrophy of the DMICL has been described as a congenital condition and proposed as a primary cause of carpal disease.[6] The findings of the present study suggest that this lesion is probably acquired and may be a response to both MPICL tearing and training. There was no evidence that DMICL hypertrophy was a primary cause of osteochondral fractures or articular cartilage damage. We previously showed[12] that the DMICL formed the dorsal part of the medial collateral ligament and that the medial collateral ligament and the MPICL are the major ligaments preventing dorsal displacement of CR.[19] In the normal joint, the DMICL is flaccid in extension. With rupture of the MPICL the lateral restraint of CR is lost. The resulting increase in dorsomedial displacement of CR could induce changes in the medial collateral ligament and therefore in the DMICL. With acute MPICL tears, DMICL hypertrophy will not have sufficient time to occur, which may explain the lower incidence of this latter lesion compared with the frequency of MPICL tearing. Also, a certain amount of exercise is required as suggested by the relationship between the duration of training and DMICL hypertrophy. Selway observed enlargement of the DMICL in 74% of cases undergoing midcarpal joint arthroscopy but did not examine the MPICL. The much lower incidence (33%) in the present study may be related to a difference in definition of ligament hypertrophy. We saw no evidence of the ligament impinging on the articular surface as has been previously described.[6] Admittedly, joint distension with fluid during arthroscopy will force the joint capsule and DMICL away from the articular surfaces, but some secondary changes in

articular cartilage should have been observed where the DMICL is said to impinge, palmar to its CR attachment.

Our study has confirmed that MPICL tearing is common in horses with midcarpal joint disease. Tearing of the LPICL is less commonly observed, but this may not be a true indication of its prevalence. The prevalence of these lesions in racing horses is unknown and should be further investigated. There was an apparent inverse relationship between the severity of MPICL tearing and osteochondral damage, suggesting that rupture of the MPICL does not consistently result in osteochondral damage. Hypertrophy of the DMICL appears to be a response to MPICL tearing and training, and is likely to be a secondary change rather than a primary cause of carpal disease.

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Postmortem lesions in the intercarpal ligaments of the equine midcarpal joint

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Objective To determine the frequency of damage to the medial palmar intercarpal ligament (MPICL), and the range of sizes of the dorsomedial intercarpal ligament (DMICL) of the midcarpal joint in horses with no history of carpal joint disease.

Materials and methods Cadaver limbs were collected from 72 horses with no history of carpal joint disease. One hundred and forty-two midcarpal joints were dissected and the MPICL and DMICL were examined. Measurements were made with a digital micrometer.

Results MPICL tearing was present in 88 of 96 joints from horses 2 years and older. Tears were predominantly of the dorsolateral bundle and complete rupture of the dorsolateral and dorsomedial bundles was not observed. Tearing was not present in foals less than 4 months of age and the severity of tearing increased significantly with age ($P < 0.0001$). Severity of tearing was significantly greater in racing Standardbreds than racing Thoroughbreds ($P < 0.01$), but there was no significant difference between racing and non-racing horses. The lateromedial thickness of the DMICL ranged from 0.4 mm to 2.6 mm in horses 2 years and older. Lateromedial thickness increased significantly with age, and was significantly greater in racing Standardbreds than racing Thoroughbreds ($P < 0.01$). There was no significant difference between racing and nonracing horses.

Conclusions Damage confined to the dorsolateral bundle of the MPICL is a common finding in horses over 1 year of age and is probably of little clinical significance. Complete rupture of both dorsolateral and dorsomedial bundles is uncommon in horses with no history of midcarpal joint disease. Variation in size of the DMICL is observed in horses of all ages, but is most marked in 2-year-old horses.

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Key words: Horse, carpus, ligament, intercarpal ligament, histopathology.

A high frequency of abnormalities of the intercarpal ligaments of the midcarpal joint has been observed arthroscopically in joints of racing horses presenting with clinical signs of midcarpal joint disease.[1-3] This has led to the suggestion that both tearing of the MPICL[2] and enlargement of the DMICL[1] contribute to osteochondral damage in the midcarpal joint and are therefore significant clinical findings. However, there is no direct evidence that DMICL enlargement is a significant problem in the carpus and the evidence for the clinical significance of MPICL tearing is limited. Horses that have joints with MPICL tearing have more severe clinical signs of joint compromise than would be expected based on the severity of osteochondral damage, and when more than half of that part of the MPICL visible arthroscopically is torn, the prognosis for future racing is poor.[4]

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Although one of the major reasons that intercarpal ligament abnormalities are thought to be a significant clinical finding is their high frequency in clinical cases, to our knowledge there are no reports of the frequency of intercarpal ligament tearing in the wider population of horses, and particularly in horses without evidence of carpal disease. If the incidence of intercarpal ligament tearing in this latter group of horses is high, it is less likely that these abnormalities are clinically important.

Variation in size of the dorsolateral bundle of the MPICL has been observed.[3] It is not known whether this is normal individual variation or the result of damage to the ligament and subsequent degeneration in some horses. The high incidence of tearing of the MPICL may be due to degeneration and weakening of the ligament as occurs in other intra-articular ligaments of other species.[5]

The purpose of this study was to determine the frequency of damage to the MPICL and the range of size of the DMICL and the bundles of the MPICL in Thoroughbred and Standardbred race horses, and horses of other breeds not subjected to high levels of training, excluding those with known carpal disease.

Materials and methods

Carpi were collected from horses of a variety of ages that were euthanased for reasons unrelated to carpal joint disease. Joints were stored at -10°C until required. After thawing overnight, detailed dissections of the midcarpal joint were performed, paying particular attention to the MPICL and LPICL, and the DMICL which has been variously described as being a normal synovial plica[6] or an intercarpal ligament on the dorsomedial aspect of the joint passing from the radial to the second carpal bone.[1,7]

After skin removal, the tendons of the extensor carpi radialis, common digital extensor, extensor carpi obliquus and lateral digital extensor muscles were dissected free and discarded. The dorsal joint capsule was removed as far as the collateral ligaments laterally and medially, and carefully separated from the DMICL. The palmar carpal ligament was dissected free of its attachments to the palmar aspects of the carpal bones. Then the joint was passively flexed and extended and the behaviour of the intercarpal ligaments recorded. The radius was then removed by transection of the joint capsule and the ligaments of the antebrachiocarpal joint. Dissection was continued through the intercarpal ligaments that attach CI to CR and CU, taking care to

MPICL	Medial palmar intercarpal ligament
LPICL	Lateral palmar intercarpal ligament
DMICL	Dorsomedial intercarpal ligament
CR	Radial carpal bone
CI	Intermediate carpal bone
CU	Ulnar carpal bone
C3	Third carpal bone
CV	Coefficient of variation

avoid damage to the palmar intercarpal ligaments. The palmar intercarpal ligaments could now be observed and their dimensions measured with a digital micrometer (Mitutoyo, Series 500, Tokyo, Japan) while the ligaments were under moderate tension. Cross-sectional measurements were made at the midpoint of the ligaments. The extent of MPICL tearing was graded on a scale of 0 to 4 as previously described[2] and the dorsopalmar depth of the tear measured.

Comparison of ligament dimensions between horses of various ages was complicated by the overall increase in size of the carpus with growth. To correct for the size of the carpus, the dorsopalmar depth of C3 was measured and ligament dimensions were divided by this value. An estimate of cross-sectional area of the four previously described bundles of the MPICL[8] was made by multiplying the lateromedial and dorsopalmar dimensions together. For each bundle the CV of cross-sectional area was calculated for horses two years and older.

Information obtained for each horse included the age, breed and sex, whether training had commenced and whether it had raced. If the owner or trainer reported any history of carpal problems, both joints were excluded from the study.

Statistical analysis

The effects of age and breed on ligaments were determined by one way analysis of variance. Before statistical analysis, log transformation was performed where the data was not of normal distribution. Where transformation did not result in normalisation of the data, the Kruskal-Wallis ANOVA by ranks and median test was used. Relationships between variables were determined by Spearman rank order correlations.

Results

One hundred and forty-two joints were examined from 72 horses. Ages ranged from an unborn term foal to 18 years. There were 40 Thoroughbreds, 16 Standardbreds, and 15 horses of non-racing breeds, including four Arabs, two Appaloosas, four Ponies, two Thoroughbred crossbred horses, two Quarter horses and one Australian Stockhorse. Two carpal joints were not available for examination but the contralateral joint was included.

Medial palmar intercarpal ligament

Tearing of the MPICL was present in 99 of the 142 joints examined. In horses two years of age or older, tearing was present in 88 of 96 joints. All tears were of the most dorsal part of the ligament and in all but 12 joints involved the dorsolateral part of the attachment to CR (Figure 1). In the remaining joints, tearing was present in the dorsal margin of the ligament midway between the proximal and distal attachments. The most severe tear extended 12.5 mm palmarly through the dorsolateral bundle into the dorsomedial bundle. Complete rupture of both dorsolateral and dorsomedial bundles (grade 4) was not observed. Severity of tearing in adult Standardbreds was significantly greater than in adult Thoroughbreds ($P < 0.01$) and there was a trend for a greater severity of tearing in Standardbreds than in non-racing breeds ($P = 0.06$) (Table 1). Tearing was not the only abnormality recorded in MPICLs. Localised thickenings were also found in a number of grossly intact dorsolateral bundles, as well as longitudinal separation of fibres. During flexion and extension of the midcarpal joint the most dorsal part of the proximal attachment of the MPICL was observed to be trapped between the articular surfaces of CR and C3 (Figure 2) and this corresponded to the most common site of MPICL tearing.

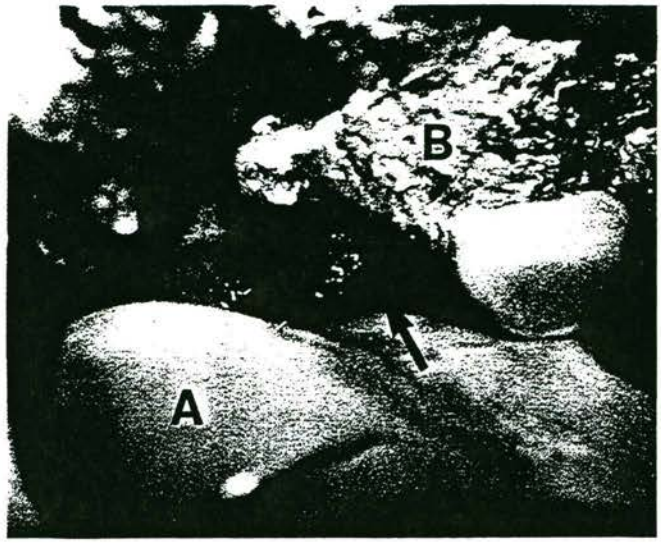

Figure 1. Lateral view of a right adult carpus (intermediate carpal bone removed) showing minor tearing (grade 1) of the dorsolateral bundle of the medial palmar intercarpal ligament (arrow) at its attachment to the radial carpal bone (B). This is the most common site of damage.

Table 1. Frequency and extent of medial palmar intercarpal ligament tearing in the midcarpal joints of horses of different breeds and ages.

Class of horse	No. of joints examined	No. of joints with MPICL tearing	No. of joints with grade 2-4 tearing (mm)	Mean (± SEM) depth of tearing (mm)
Total	142	101(71%)	56 (39%)	1.5 (± 0.17)
Horses ≥ 2 years	96	88 (92%)	55 (57%)	2.1 (± 0.22)
Horses < 2 years	46	13 (28%)	1 (4%)	0.2 (± 0.05)
TB ≥ 2 years	45	38 (84%)	23 (51%)	1.5 (± 0.21)
SB ≥ 2 years	30	30 (100%)	20 (67%)	3.2 (± 0.55)
Non racing breeds ≥ 2 years	21	20 (95%)	12 (62%)	1.7 (± 0.27)

TB = Thoroughbred, SB = Standardbred

Tearing of the MPICL was not present in foals less than 4 months of age but increased significantly in severity with age up to 3 years ($P < 0.0001$) (Figure 2). The largest tear in a 1-year-old horse extended 1.3 mm from the dorsal margin. The four previously described fibre bundles[8] of the MPICL were identified in 120 joints. Greatest variation in cross sectional area in horses 2 years and older occurred in the dorsolateral bundle (CV 71%) and palmarolateral bundle (CV 95%), with somewhat less variation in the palmaromedial (CV 63%) and the dorsomedial bundles (CV 42%). The dorsomedial and palmarolateral bundles were present in all joints, but in 8 joints of five horses no dorsolateral bundle was present and in 14 joints of nine horses no palmarolateral bundle was present. All of these horses were 2 years or older, except one 18-month-old Standardbred in which one joint had no dorsolateral bundle. This horse had begun training. In foals the proximal attachment of the dorsolateral bundle extended dorsally to the palmar edge of the articular cartilage on the lateral aspect of CR (Figure 3), while in most horses over 1 year of age, the dorsal extent of the proximal attach-

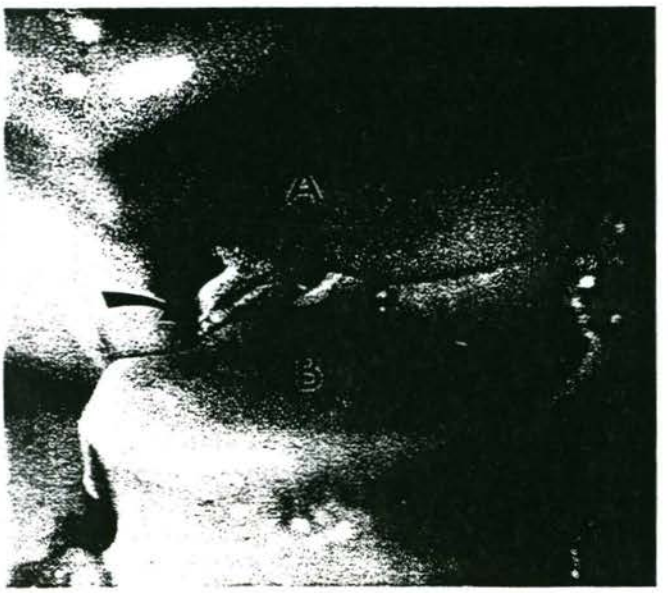

Figure 2. Dorsal view of a partially flexed right adult carpus showing the proximal aspect of the medial palmar intercarpal ligament (arrow) trapped between the articular surfaces of the third carpal bone (A) and the radial carpal bone (B).

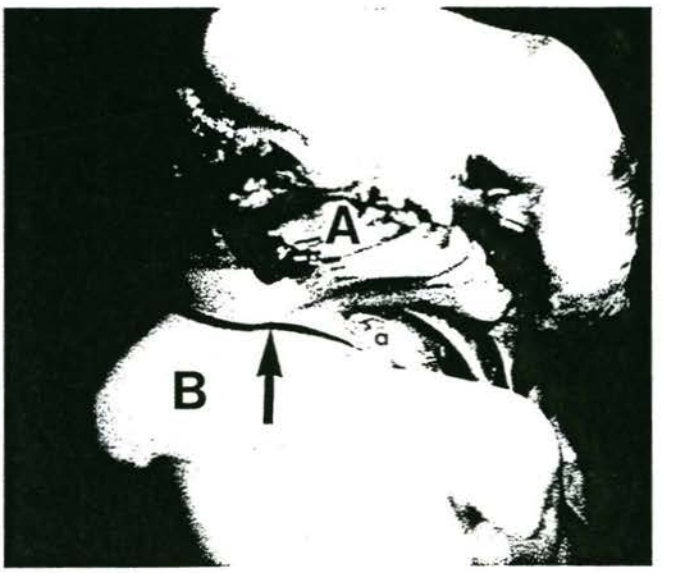

Figure 4. Lateral view of the medial palmar intercarpal ligament of a left carpus of an unborn term foal (intermediate and ulnar carpal bones removed). The dorsolateral bundle (α) extends to the palmar aspect of the articular cartilage (arrow) of the radial carpal bone (A).

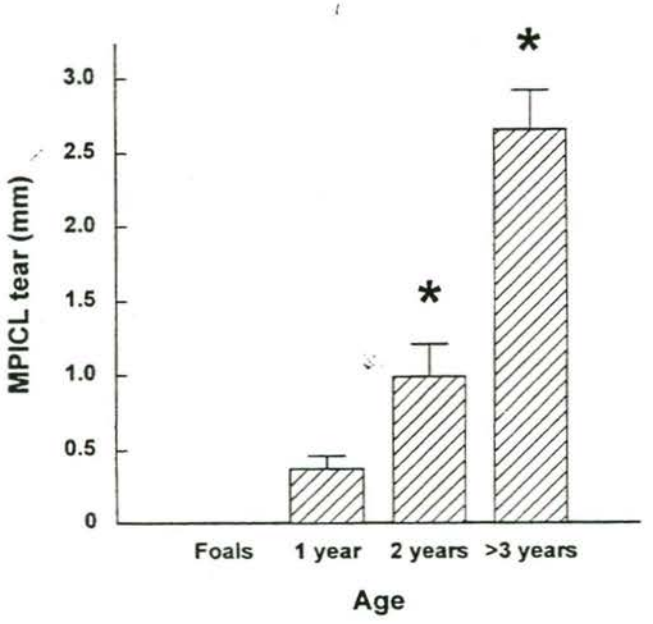

Figure 3. Depth of medial palmar intercarpal ligament (MPICL) tears grouped by age of horse. Bars indicate SEM; * = P < 0.001 for difference.

ment was more palmar (Figure 1). Dorsopalmar thickness of the dorsolateral bundle decreased significantly with age even when corrected for the size of the carpus ($P < 0.0001$) (Figure 4). There was a significant correlation between dorsopalmar thickness of the dorsolateral bundle and both depth and grade of tearing of the bundle ($R = 0.33$, $P < 0.01$).

Dorsomedial intercarpal ligament

The DMICL was identified in all joints but the size was highly variable, ranging from 0.4 mm to 2.6 mm thick (lateromedial) in horses 2 years and over. Marked enlargement, as seen in clinical cases of carpal joint disease,[9] was only observed in three racing Standardbreds, two racing Thoroughbreds and two horses of non-racing breeds. In some joints the DMICL appeared as just a fold of synovial tissue with no distinct collagen bundles and no obvious distal border. Enlarged ligaments consisted of parallel fibre bundles that were easily differentiated from the joint capsule over its full length. Lateromedial thickness of the DMICL increased significantly with age even when corrected for the size of the carpus ($P < 0.0001$) (Figure 6). DMICL thickness in Standardbreds was greater than in Thoroughbreds ($P < 0.01$) and non-racing breeds, although the difference was not significant ($P = 0.05$).

Lateral palmar intercarpal ligament

Tearing was present in eight joints of seven horses. Most tears were at the dorsal aspect. Five of the affected horses were Thoroughbreds, and there was one Standardbred and one other breed. Most tears involved only very superficial damage. In four joints from three horses, small osseous fragments, about 3 mm in diameter, were present in the CU attachment at the lateral aspect. In these joints there was an irregularity in the surface of the ligament, but it remained firmly attached to CU.

Discussion

Medial palmar intercarpal ligament

This study has demonstrated a high frequency of tearing of the MPICL in horses 2 years of age and over, of all breeds. Although not reported, a similar frequency was found in racing Thoroughbreds in England (T Phillips personal communication). These findings question the clinical significance of tearing

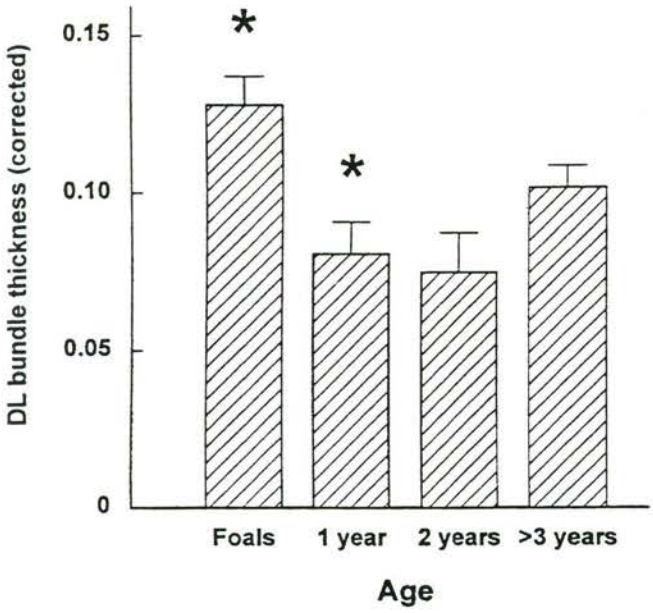

Figure 5. Dorsopalmar thickness of the dorsolateral (DL) bundle of the medial palmar intercarpal ligament corrected for the size of the carpus, grouped by age. Bar indicates SEM; * = P < 0.05 for difference.

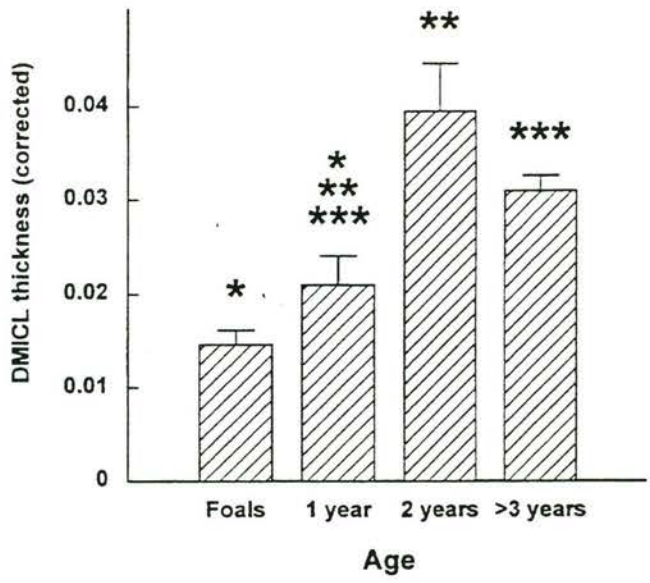

Figure 6. Lateromedial thickness of the dorsomedial intercarpal ligament (DMICL) corrected for the size of the carpus and grouped by age. Bar indicates SEM; (i = P < 0.05, * = P < 0.01, # = P < 0.0001 for difference between corresponding pairs.

of the MPICL in racing horses. If tearing is as common in the general population of horses as this study indicates, tearing observed during arthroscopy of horses that present with signs of midcarpal joint disease may be an incidental finding rather than a cause of lameness. However, it is important to note that complete disruption of the dorsolateral and dorsomedial bundles, as occurs in clinical cases of midcarpal joint disease,[2,4,9] was not recorded in the present survey. Although partial tearing of the dorsolateral bundle is probably of little significance, this study provides no evidence that grade 4 tears[2] (dorsolateral and dorsomedial bundles) are not significant. Also, although horses had no history of carpal disease, they were not clinically examined, so low-grade lameness may have been present in some. In the only study that has examined postoperative performance of horses with MPICL tearing[4] it was reported that most horses with less than one third of the ligament torn performed well postoperatively, but horses with more than 50% of the ligament torn had a poor prognosis. As the dorsolateral bundle is usually smaller than the dorsomedial bundle and very few of the observed tears extended into the dorsomedial, most tears in the present study involved less than 50% of the MPICL that can be seen arthroscopically.

Complete assessment by arthroscopy of the significance of MPICL tearing is limited by the inability to fully evaluate the whole ligament and by the large variation between horses in the size of the various bundles. The most likely role of the dorsal bundles of the MPICL is stabilisation of CR, preventing dorsal and, to some degree, medial displacement in relation to C3.[3] Stability is related to the amount of intact ligament remaining, rather than the size of the tear; a small tear in a small dorsolateral bundle may be just as significant as an extensive tear in a large dorsolateral bundle. The extent of remaining ligament needs to be determined before conclusions about the role of the tear in instability can be made. However, the palmarolateral bundle cannot be observed arthroscopically, because of its palmar position.[8] The palmarolateral bundle has a very similar orientation to the dorsolateral, and of all the bundles has the greatest variation in size. If a large palmarolateral bundle is present, instability from tearing of the dorsolateral bundle may not result, but if no palmarolateral bundle is present instability may result from minor damage.

The frequency of tearing in racing horses in this survey was greater than in racing horses from the same geographical area that had midcarpal joint disease that was examined arthroscopically.[2,9] Many of the minor tears observed by dissection in this study were at the proximal attachment of the MPICL, which is not always directly visible with the standard arthroscopic portals. During dissection CI was removed allowing careful examination of the whole ligament. However, grade 2 and 3 tears were also more common in this survey than is recorded in clinical cases, and this degree of tearing would have been identified easily with arthroscopy.

It has been suggested that tearing of the MPICL occurs as a result of cyclical tensing of the ligament during exercise.[3] The high frequency of tearing in racing horses[2] also suggests that this is a racing injury. The finding in the present study that tearing first appears at about 1 year of age and seems to progress in horses of all breeds as they mature would seem to refute this. Nevertheless, the significant increase in tearing between 1 and 2 years of age in all horses also indicates that the commencement of training does exacerbate damage. Therefore it is probable that although damage and degeneration of the ligament commences in young horses before the onset of training, stresses applied to the ligament with training at any level result in further damage to an already compromised ligament. Also, the greater severity and frequency of tearing in Standardbred racehorses than in Thoroughbred racehorses, suggests an influence of gait on ligament tearing. Based on the alignment of fibres of the dorsolateral

bundle of the MPICL, tearing is most likely due to transverse forces across the midcarpal joint rather than axial force.[9] The higher axial forces in the carpi of Thoroughbred racehorses may produce greater transverse stability than in the pacing horse, where axial forces are lower, but transverse vibrations at the carpus have been demonstrated during full extension.[10]

A significant reduction in the size of the dorsolateral bundle appeared to occur at about the same time as the first ligament tearing was observed (one year of age). A reduction in ligament size could be a response to increasing exercise, or as a result of degeneration. Degeneration preceding ligament tearing has been described in intra-articular ligaments in other species[5] and complete loss has been reported in humans in association with ligament rupture.[11] In some joints in the present survey there was a complete absence of the dorsolateral bundle and it is unclear whether this was abnormal or an indication of normal anatomical variation. Although there was some variation in size of the dorsolateral bundle in young foals, it was consistently present and extended further dorsally than in adults. This strongly supports the view that the absence of a dorsolateral bundle in older horses is acquired. Longitudinal fibre separation is likely to be a gross manifestation of degeneration.

The observation that the most dorsal part of the proximal attachment of the MPICL was trapped between the articular surfaces of CR and C3 during midcarpal joint flexion may explain the MPICL's predisposition to injury. During flexion CR moves laterally relative to C3.[3] This movement is stopped when the lateral aspect of CR articulates with the palmaromedial aspect of C3. As this occurs, the most dorsoproximal part of the MPICL is drawn over the articular surface of C3 before CR lifts off C3 as full flexion is approached (Figure 2). Repeated compression and abrasion of the ligament at this site would explain its predisposition to injury. Removal of the synovial lining of intra-articular ligaments has been shown to result in their degeneration.[12] In some of the more mildly affected ligaments it appeared that only the synovial membrane was lost.

Tearing of the other bundles of the MPICL was not observed. However there was also a large variation in size of the palmarolateral bundle, probably indicative of individual variation, as it was also a feature of foals. Like the dorsolateral bundle, the palmarolateral bundle also is drawn over the articular surface of C3 during flexion suggesting that ligament abrasion may play a role.

Dorsomedial intercarpal ligament

Enlargement of the DMICL has been described as a congenital condition that is a cause of carpal joint disease in racing horses by impingement on the articular surface of the midcarpal joint.[1] Although variation in size was observed in foals, none had marked enlargement of the ligament. Larger numbers of foal joints would need to be examined before a congenital cause of enlargement could be ruled out, but our findings suggest that enlargement occurs with the onset of training, and possibly in response to, training. This was not consistent in all horses, suggesting that there are other factors involved in DMICL enlargement. Also, enlargement was observed in non-racing breeds, so it is possible that other forms of exercise at lower intensities than those involved in Standardbred and Thoroughbred training may contribute to enlargement.

As with MPICL tearing, the finding of enlarged ligaments in horses with no history of midcarpal joint disease and no evidence of intra-articular osteochondral fractures questions the clinical significance of this lesion, although low grade lameness cannot be ruled out in some horses.

Lateral palmar intercarpal ligament

Damage to the LPICL was rarely recorded and was only mild, which is consistent with observations in clinical cases of midcarpal joint disease.[2] The bone fragments that were found within the proximal attachment in several joints appeared to originate from CU and were possibly avulsions. Avulsion fractures at this site have been seen in clinical cases (A Nixon personal communication). The significance of these lesions is difficult to determine. In all horses the ligament appeared to have reattached to the defect in CU. The occurrence of one of these lesions in a 1-year-old Thoroughbred indicates that they may not be a racing injury. It is possible that not all such lesions were detected as they were buried within the substance of the ligament and were only found by dissection.

Conclusion

Damage to the dorsolateral bundle of the MPICL of the midcarpal joint is a common finding in both racing and non-racing horses older than one year of age, but complete rupture of the dorsolateral and dorsomedial bundles is uncommon. The high frequency of this lesion appears to be related to abrasion of the ligament between the articular surfaces of the carpal bones during joint flexion. It is likely that damage confined to the dorsolateral bundle of the MPICL is not clinically significant in most horses. Variation in size of the DMICL occurs in horses of all ages, but is most marked in 2-year-old racehorses, and gross enlargement is probably acquired. Damage to the LPICL is rare, and when it occurs it is mild and of unknown significance.

Acknowledgments

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