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UPDATE ON BONE DISEASE: THE IMPACT OF SKELETAL DISEASE ON ATHLETIC PERFORMANCE

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Introduction

Bone is a critical component of the equine musculoskeletal system. It not only provides strength to the legs, but also acts as the foundation for cartilage in the moveable joints. Much of the clinical disease in the horse associated with bone involves the subchondral bone immediately under the articular cartilage leading to problems in the joint. These conditions can be divided into developmental problems of bone and traumatic problems of bone. They will be considered separately.

Developmental Problems of Bone

The term developmental orthopedic disease (DOD) was coined in 1986 to encompass all orthopedic problems seen in the growing foal and is a term that encompasses all general growth disturbances of horses and is therefore nonspecific (McIlwraith, 1986). The term should not be used synonymously with osteochondrosis, and it is inappropriate for subchondral cystic lesions, physitis, angular limb deformities, and cervical vertebral malformations, all to be presumed as manifestations of osteochondrosis. When the term developmental orthopedic disease was first coined, it was categorized to include the following:

1. Osteochondrosis. Osteochondrosis is a defect in endochondral ossification that can result in a number of different manifestations depending on the site of the endochondral ossification defect. These manifestations include osteochondritis dissecans (OCD) and some subchondral cystic lesions. Not all subchondral cystic lesions or osseous cyst-like lesions are necessarily manifestations of osteochondrosis. Another manifestation is some physitis (but we now recognize that most clinical swelling associated with the physis has no pathologic change involving the physis itself).
2. Acquired angular limb deformities.
3. Physitis.

4. Subchondral cystic lesions.
5. Flexural deformities.
6. Cuboidal bone malformation.

Osteochondrosis

Osteochondrosis (dyschondroplasia) was initially defined as a disturbance of cellular differentiation in the growing cartilage (Olsson, 1978). Osteochondrosis is considered to be the result of a failure of endochondral ossification and therefore may affect either the articular epiphyseal cartilage complex or the metaphyseal growth cartilage. It is usually in the articular epiphyseal cartilage. It can have three consequences: (1)These areas of retained cartilage, due to a lack of endochondral ossification, can heal; (2) They can break out and form flaps of cartilage and bone or fragments of cartilage and bone (called osteochondritis dissecans or OCD); or (3) The retained cartilage can undergo necrosis and form a subchondral cystic lesion (subchondral bone cyst).

The majority of cases of OCD and subchondral bone cysts are considered to be the result of necrosis occurring in the basal layers of the thickened retained cartilage with subsequent pressure and strain within the joint giving rise to fissures in the damaged cartilage.

OSTEOCHONDRITIS DISSECANS

There is a general agreement that this condition involves a dissecting lesion with the formation of a chondral or osteochondral flap. Flaps may become detached and form joint mice. In some instances, lesions have been found at arthroscopy that consist of cartilage separated from bone, and the cartilage does not appear to be thickened (McIlwraith, 1993). Based on these observations, the author questions whether persistence of hypertrophied cartilage is a necessary event prior to the development of an OCD lesion. This question is based on instances seen at arthroscopic examination or in follow-up histologic examination where dissection or separation occurs close to the cartilage-bone interface, rather than in the underlying cancellous bone between normal cartilage and a normal bone-cartilage junction, as it commonly does in humans. The clinical manifestations and treatment of the common entities of OCD are discussed in a separate lecture.

SUBCHONDRAL CYSTIC LESIONS

Subchondral bone cysts were first reported as a clinical entity in 1968 (Pettersson, 1968). Subchondral cystic lesions have also been proposed as manifestations of osteochondrosis by a number of authors (Rooney, 1975; Stromberg, 1979), and there is some pathologic support for this (Rejno and Stromberg, 1978). However, more recent work has demonstrated that subchondral cystic lesions can be produced from a small

defect in the bone, and other work has shown that the lining of clinical subchondral cystic lesions contained increased quantities of neutral metalloproteinases, PGE-2, and interleukin-1, and also are capable of osteoclastic resorption of bone.

The Cause of OCD

Despite the instances where there is no evidence of thickened cartilage, it is generally accepted that most OCD lesions are manifestations of osteochondrosis. In the one- to two-year-old horse, most cases of subchondral cystic lesions are also related to osteochondrosis. For that reason, the following discussion is on various etiologic factors associated with osteochondrosis.

GENETIC PREDISPOSITION

There have been a number of genetic studies on the heredity of OCD in the hock in Standardbreds and Scandinavian cold-blooded horses. A radiographic survey by Hoppe and Phillipson in Standardbred trotters and Swedish Warmbloods showed that one stallion of each breed had a significantly higher frequency of OCD among its progeny, compared with the progeny groups of the other stallions ($p < 0.001$) (1985). In another study, Schougaard et al. (1990) showed radiographic evidence of a significantly higher proportion of OCD in the progeny of one of eight stallions, even though the stallion itself did not show radiographic signs of OCD. Since that time, there have been two additional studies on the heritability of osteochondrosis in the tibiotarsal joint (Grondahl and Dolvik, 1993; Phillipsson et al., 1993). Both of these studies were in Standardbred trotters but did show significant heritability with OCD. Studies in other breeds are markedly lacking. In the Dutch Warmblood, there has been a protocol preventing breeding of stallions with any OCD for ten years, but whether this has lowered the incidence of the disease is questionable.

GROWTH AND BODY SIZE

An association has been made between body weight and OCD by Pagan and Jackson (1996). Foals in Kentucky that had to have arthroscopic surgery for OCD were significantly heavier than foals that did not have OCD.

MECHANICAL STRESS AND TRAUMA

It has long been recognized clinically that mechanical stresses precipitate the onset of clinical signs, presumably by avulsing an OCD flap or fragment (Pagan and Jackson,

1996). The role of trauma as a primary initiator of a lesion is more controversial. Pool pointed out that there are no unique histologic features that will consistently distinguish the lesion of osteochondrosis from that of trauma at a developing osteochondral junction, and that the radial vessels supplying the chondrocytes in the epiphyseal physis may be sheared and cause a primary osteochondrosis lesion (1986). He felt that biomechanical forces are an important factor and are superimposed upon an idiopathic lesion to produce defective cartilage. Reflection back to the classic paper by Konig in 1887 is appropriate in considering the potential role of trauma in the pathogenesis. He claimed that loose bodies in the knee joints of young people had three causes: (1) very severe trauma, (2) lesser trauma causing contusion and necrosis, and (3) minimal trauma acting on an underlying lesion, for which he suggested the name "osteochondritis dissecans" (and for which he is considered the originator) (Barrie, 1987). I feel that these three different syndromes can be seen in the horse.

DEFECTS IN VASCULARIZATION

OCD was initially described as being caused by a vascular or ischemic necrosis of the subchondral bone (Adams, 1974). Although recent work in the pig suggested that the viability of epiphyseal cartilage in the articular-epiphyseal cartilage complex is highly dependent on an adequate blood supply from cartilage canal vessels, and strongly implicates a defect in blood supply in the pathogenesis of osteochondrosis, there is no evidence yet documented in the horse (Carlson et al., 1991).

NUTRITION

Osteochondrosis-like lesions have been induced in horses by feeding 130% of National Research Council (NRC) carbohydrate and protein (Glade and Belling, 1986). More recently, further work has defined that 130% of NRC digestible energy will certainly significantly increase the incidence of osteochondrosis lesions, but increasing the protein content does not (Savage et al., 1993).

MINERAL IMBALANCES

Various mineral imbalances have been implicated in the pathogenesis, including high calcium, high phosphorus, low copper, and high zinc. There is no good equine-specific support for high calcium causing problems, but three times the NRC levels of phosphorus significantly increased the number of OCD lesions (Savage et al., 1993).

Low copper has been implicated as a cause. In experimental studies, it has been reported that a marked copper deficiency (1.7 ppm) produced both flexural deformities

and osteochondrosis-like lesions (Bridges and Harris, 1988). Bridges and Harris also noticed a softening of articular cartilage and suggested that the low copper status may lead to reduced cross-linking of collagen by lysyl oxidase, predisposing to physeal and articular fractures. Hurtig et al. conducted a controlled experiment with high (30 ppm) and low (7 ppm) copper diets (1990). A much higher incidence of lesions of osteochondrosis was seen in the foals fed the low copper diet. Many of the changes were present in the cervical spine. Hurtig and coworkers considered the cause as one of reduced structural strength rather than arrested or abnormal endochondral ossification (1990). Further work has been done in copper by Pearce and coworkers in New Zealand (Pearce et al., 1998a; Pearce et al., 1998b; Pearce et al., 1998c). The absolute levels of copper at which OCD can be produced have been questioned, or at least it appears clear that there are differences between different countries. Pearce and coworkers (1998a) failed to produce significant clinical OCD with low copper diets. They also showed that, while oral supplementation of mares could enhance the foals' copper status, parental administration could not.

Excessive zinc intake has been related to equine osteochondrosis (Messer, 1981). The effects of environmental exposure to zinc and cadmium were studied in pregnant pony mares following observations of lameness, swollen joints, and unthriftiness, particularly in foals (Gunson et al., 1982).

ENDOCRINE FACTORS

It has been postulated by Glade (1986) that the production of osteochondrosis lesions in association with overfeeding is mediated by the endocrine system. Glade has proposed that feeding initiates increased concentrations of insulin and T4 and high concentrations of insulin could inhibit growth hormone, although the exact mechanism is not known (Glade et al., 1983). A long-term administration of dexamethasone has been associated with the production of osteochondrosis-like lesions (Glade et al., 1983). More recent work showing an association between high-glycemic feed, insulin secretion, and osteochondrosis has been made by Ralston and Pagan.

SITE VULNERABILITY

Because the lesions of equine osteochondrosis occur at specific anatomic sites, this does suggest vulnerability that could be related back to trauma or excessive stress and interference with blood supply as originally suggested by Pool (1986). Lesions are frequently bilateral in the femoropatellar and tarsocrural joints and quadrilateral in the fetlock joint, although they infrequently involve different joints in the same animal. This observation could perhaps suggest a "window of vulnerability" in the endochondral ossification of that specific joint at that specific location.

Natural History of Osteochondrosis Lesions

Recent work done by the workers at Utrecht has shown that many lesions in the stifle and the hock will heal. In this study, foals were radiographed every month, and lesions developed (defects developed, signifying a lack of endochondral ossification) and then the lesions healed (Dik et al., 1999). Relatively few of them became clinical, but the times at which they were going to persist were established. This study emphasized that we need to be careful of radiographic surveys in deciding that we have a problem with OCD. This author feels that only when we have clinical signs associated with it should we be intervening. This study also clarified the age at which surgical treatment was appropriate. If surgical intervention is carried out at a very young age, it is likely that it is unnecessary in many instances.

Further work by McIntosh and McIlwraith (1993) showed that it was certainly possible to have lesions heal beyond this time if foals were confined. Definition of what lesions can heal with conservative management has greatly progressed treatment, and this is discussed elsewhere.

Management of OCD Lesions and Subchondral Bone Cysts that Do Not Respond to Conservative Management

OSTEOCHONDRITIS DISSECANS OF THE (STIFLE) JOINT

The stifle joint is one of the principal joints affected with OCD. Although stifle OCD can be diagnosed in almost any breed, it seems to be more common in Thoroughbreds than in other breeds. Approximately 60% of affected horses will be one year of age or less at the time the condition becomes symptomatic, and younger animals that develop clinical signs often have more severe damage within the joint. However, incidental lesions are sometimes identified in older horses where no clinical signs have ever been observed.

Clinical and radiographic signs

Animals usually present with a sudden onset of joint swelling and lameness. A recent increase in the level of exercise is sometimes part of the history. Lameness sometimes may be very mild, with a stiff action and shortened stride being observed, rather than the horse having a prominent lameness. Some more severely affected horses will have a "bunny hop" action behind that can initially be confused with a neurologic problem.

Joint distention, however, is the most consistent sign seen with OCD of the stifle. Careful palpation of the joint may identify free bodies, or the surface irregularity associated with the damage within the joint. Bilateral involvement is common in the

stifle, so careful examination of both stifles should be completed. In one study, 57% of affected animals had bilateral involvement.

Breed Distribution of 161 Horses Presented for Femoropatellar OCD

Breed	*Number*	*Percentage*
Thoroughbred	82	50.9
Quarter Horse	39	24.2
Arabian	16	9.9
Warmblood	9	5.6
Crossbred	5	3.1
Paint Horse	3	1.9
Appaloosa	3	1.9
Other	4	2.5

Age Distribution of 161 Horses Presented for Femoropatellar OCD

Age (yr)	*Number*	*Percentage*
<1	22	13.7
1	68	42.2
2	36	22.4
3	21	13
>4	14	8.7

Flexion of the limb will usually exacerbate the lameness, and anesthetic placed into the joint will improve or eliminate the lameness. However, intra-articular anesthesia is usually not necessary to confirm a diagnosis.

Lateral to medial radiographs provide the most useful information regarding specific lesion location and size. The most common defect identified is a variably sized irregularity or flattening of the lateral trochlear ridge of the femur. The area of the ridge that comes in contact with the bottom portion of the patella is most commonly involved. Partial calcification of the tissue within the defect is sometimes seen, and free bodies are also occasionally identified. It is rare to see OCD primarily affecting the patella, but secondary radiographic change in the patella resulting from the trochlear ridge damage can be seen. The medial ridge of the femur is much less commonly involved.

Generally, the extent of damage to the joint identified at surgery is more extensive than would be predicted from radiographs. Although other joints can be involved

concurrently, this is uncommon. In one study of 161 horses with stifle OCD, five horses also had OCD affecting the rear fetlocks, four had hock OCD, and one had OCD of a shoulder joint.

Treatment

It is generally accepted that surgical debridement of the lesions using arthroscopic technique is the treatment of choice. However, smaller lesions identified in younger horses may respond to rest and resolve radiographically. These are generally lesions that are not causing severe clinical signs. If lameness and swelling are prominent, arthroscopic surgery is indicated.

As for all joint surgery, the joint is thoroughly explored, and suspicious lesions are probed. Loose or detached tissue is elevated and removed. Loose bodies are also removed. The defect site is then debrided down to healthy tissue. Care must be taken to not be overly aggressive with bone debridement in young animals having soft subchondral bone.

Animals are usually stall-rested for 2 weeks after surgery, at which time hand-walking is started. Restricted exercise is usually continued for 2-3 months after surgery, at which time training is started or the horse is turned out.

Prognosis

In one study of 252 stifle joints in 161 horses, follow-up information was available for 134 horses (McIlwraith et al., 1991). Of these 134 horses, 64% returned to their previous use, 7% were in training, 16% were unsuccessful, and 13% were unsuccessful due to reasons unrelated to the stifle. The success rate was higher in horses having smaller lesions, and it was also higher for older horses. However, this age factor was considered to be due to the fact that the most severe lesions were generally identified in the younger horses.

OSTEOCHONDRITIS DISSECANS OF THE TARSOCRURAL (HOCK) JOINT

Hock OCD usually affects the intermediate ridge of the tibia in the proximal and cranial portion of the joint. However, lesions can also develop along the trochlear ridges (lateral ridge much more common than medial ridge) and the medial malleolus of the tibia. Hock OCD is very common in Standardbreds but can be diagnosed in most breeds.

Clinical and radiographic signs

The most common clinical sign of hock OCD is effusion of the tarsocrural joint. This is manifested clinically as a “bog” spavin, which simply refers to the prominent

swelling seen most readily along the medial or inside aspect of the joint. Lameness can also be seen but it is not common and is rarely prominent. Racehorses usually present as two-year-olds, but non-racehorses usually present as yearlings prior to going into training.

On radiographs, most attention is paid to the intermediate ridge of the tibia, followed by the lateral trochlear ridge, and then the medial malleolus of the tibia. Lesions are identified as fragments still in place (intermediate ridge) or surface irregularities of the trochlear ridge(s) or malleolus. The radiographic appearance often underestimates the extent of damage identified at surgery, particularly for lateral trochlear ridge lesions. The hock is also a joint where radiographically silent lesions (lesions identified at surgery where no abnormality was seen on radiographs) occur more commonly than in other joints.

Location of OCD Lesions in 318 Tarsocrural Joints

Number of Joints	*Location*
244	Intermediate ridge (dorsal aspect) of distal tibia
37	Lateral trochlear ridge of talus
12	Medial malleolus (dorsal aspect) of tibia
11	Intermediate ridge of tibia plus lateral trochlear ridge of talus
4	Intermediate ridge plus medial malleolus of tibia
3	Intermediate ridge plus medial trochlear ridge of talus
3	Medial trochlear ridge of talus
3	Lateral trochlear ridge of talus plus medial malleolus of tibia
1	Lateral and medial trochlear ridge of talus
318	**Total**

Treatment

Although lameness is usually minimal with hock OCD, surgery is the recommended treatment. Lameness may only be a problem at racing speeds or at upper levels of performance that cannot be observed during a clinical examination. As well, resolution of the effusion cannot be expected without removal of the abnormal tissue. That is not to say, however, that all horses having hock OCD need to have surgery. Horses with small lesions, minimal effusion, no lameness, and a potential career as a pleasure horse or light-use horse may not require surgery. Surgery should be considered early enough in the course of the disease so that the joint capsule is not unduly stretched, making resolution of the joint effusion less likely.

Arthroscopic identification and removal of fragments is recommended, although an arthrotomy (surgical incision into the joint) can be used successfully and is preferred by some for certain OCD lesions in this joint. Postoperative management is similar to that for OCD of the stifle, and for small lesions the time period for restricted exercise

may be decreased. Maintenance of good bandages is more difficult for the hocks and must be taken in the early postoperative period to avoid subsequent infection in the joint through the small surgical incisions.

Prognosis

In a study involving 183 horses, 76% raced successfully and performed at their intended use after surgery (McIlwraith et al., 1991). If degenerative changes were identified at surgery in the cartilage surrounding the OCD lesion, the prognosis was less favorable. Resolution of effusion was inferior for lesions involving the lateral trochlear ridge compared to the intermediate ridge of the tibia; however, this seemed to have no effect on subsequent performance.

OSTEOCHONDRITIS DISSECANS OF THE FETLOCK JOINT

The most common manifestation of OCD in the fetlock joint is fragmentation and irregularity that occurs on the dorsal aspect of the sagittal ridge and the condyles of the metacarpus or metatarsus (cannon bone). A second condition involving the fetlock that may be OCD is fragmentation of the proximal palmar-plantar aspect of the first phalanx or long pastern bones. Debate continues as to whether these fragments are truly OCD-related or whether they represent small avulsion fractures. A final entity is OCD of the palmar aspect of the metacarpal condyles, which does seem to be a trauma-related condition of racehorses. Although this condition has been referred to as OCD, it does not fit with the developmental etiology. The remainder of this discussion will include the first two entities.

OSTEOCHONDRITIS DISSECANS OF THE DORSAL ASPECT OF THE DISTAL METACARPUS/METATARSUS (FETLOCK JOINT)

Clinical and radiographic signs

Joint swelling (effusion) is the most common clinical sign, with lameness being variable in both appearance and severity. Fetlock flexion tests are usually positive. It is not unusual for all four fetlocks to be involved, and bilateral forelimb or hind limb involvement is quite common.

The diagnosis is confirmed on radiographs, and clinically silent lesions (no effusion or baseline lameness) are often identified along with the lesions causing clinical signs. Lameness can sometimes be induced by flexion in these clinically silent joints. A variety of radiographic presentations are seen with fetlock OCD. Some joints will show only flattening of the sagittal ridge (Type I OCD), others will have a fragment in place within the area of flattening (Type II OCD), and others have flattening with

or without a fragment in place but also have free or loose bodies within the joint (Type III OCD).

Treatment

A conservative approach is initially recommended where only flattening without fragmentation is identified. Many of these cases will have resolution of clinical signs, as well as improvement or disappearance of radiographic signs; however, surgery will eventually be necessary in some of these cases. Surgical debridement is recommended for lesions where fragmentation or loose bodies are present. The prognosis is quite favorable for Type I lesions using conservative treatment but more guarded for Type II and Type III lesions (McIlwraith and Vorhees, 1990). Horses having other signs of articular cartilage erosion or wear lines within the joint had a less favorable prognosis. If the lesion extended out onto the condyle of the metacarpus/metatarsus from the sagittal ridge, the prognosis was also less favorable. It was determined that clinical signs would persist in approximately 25% of cases.

PROXIMAL PALMAR/PLANTAR FRAGMENTS OF THE FIRST PHALANX

Two types of fragments have been identified in this location. Type I fragments usually involve the hind fetlock joints and are located between the midline of the bone and its caudomedial (most common) or caudolateral (less common) borders. Type II fragments are also called ununited proximoplantar tuberosities of the proximal phalanx, as these lesions occur almost exclusively in the hind limb. These fragments are located at the most lateral (most common) or medial (much less common) borders of the bone.

Both of these entities have been identified frequently in radiographic surveys completed on yearling Standardbreds, supporting a developmental concept.

Clinical and radiographic signs

With Type I fragments, effusion is uncommon, and typically lameness is identified only as a somewhat vague problem at racing speeds or at the upper levels of performance. Flexion tests are often positive and anesthetic placed within the joint will usually eliminate any clinical signs that may be present. Regular oblique radiographs will usually demonstrate the lesions, although a special view is often used to highlight their location. Most fragments are present medially. Lameness and effusion are rare with Type II fragments.

Treatment

Arthroscopic surgery is recommended for Type I fragments where clinical signs are present. If these lesions are identified incidentally on fetlock radiographs, treatment is

based on what the intended use is for the horse. If vigorous athletic activity is planned, prophylactic surgery is justified. If less rigorous pursuits are planned, most horses will not require surgery and the fragment will not lead to further arthritic changes within the joint.

Surgery is rarely indicated for Type II fragments, and most of these fragments will unite with the parent bone over a period of many months. However, Type I and II fragments can occasionally occur together in the same joint, and the Type I fragment may require surgery. The prognosis for Type I fragments with surgery is favorable. Most Type II fragments are self-limiting.

OSTEOCHONDRITIS DISSECANS OF THE SHOULDER JOINT

OCD involving the shoulder joint is probably the most debilitating type of OCD affecting horses. Generally, large areas of the joint surfaces are involved, and secondary joint disease is common. However, it is unusual to have free or loose bodies develop. OCD of the shoulder is less common than for the other joints described, and seems to affect Quarter Horses and Thoroughbreds with a similar incidence.

Clinical and radiographic signs

Most horses with shoulder OCD present at one year of age or younger with a history of forelimb lameness of variable severity. Many of these horses will have prominent lameness. If lameness has been present for many weeks, muscle atrophy will also be seen. Because of the altered gait and use of the limb, many cases develop an upright or club-footed appearance to the foot, and the foot may appear smaller on the affected limb. Deep pressure over the shoulder joint will often cause discomfort, and forced flexion/extension of the limb will sometimes accentuate the lameness that is seen. Intra-articular anesthesia will improve or eliminate the lameness.

On radiographs, the most common sign is flattening or indentation of the humeral head. Often, cystic type lesions are also identified in the glenoid cavity of the scapula. Productive remodeling changes are also commonly identified along the caudal border of the glenoid cavity.

Treatment

Conservative treatment is rarely associated with a successful outcome, and sufficient numbers having surgery have not yet been accumulated to accurately identify the prognosis with surgery. However, there is little doubt that surgery dramatically improves the clinical signs in most cases. If extensive degenerative arthritic change is present on radiographs at the time of initial examination, the prognosis for an athletic career is unfavorable, and surgery should only be considered for relative improvement

in the degree of lameness. However, for more localized lesions, the prognosis is favorable for a successful outcome.

The shoulder is probably the most difficult joint on which to perform arthroscopic surgery, due to the depth of the joint below the muscles in the area. Surgery is easier on younger animals due to their relative muscle mass. Problems encountered in the shoulder are inaccessibility of lesions due to their location within the joint, and extravasation or leakage of fluid outside the joint, which impairs visibility within the joint.

Prognosis

A large series of cases having surgery has not yet been reported although preliminary results from such a series being compiled at CSU suggest that the overall prognosis is approximately 50%. The prognosis seems to be less favorable if lesions are present on both the humeral head and the glenoid cavity. In unsuccessful cases, further deterioration of the joint surfaces on radiographs is common.

Traumatic Lesions of the Subchondral Bone

In recent years, good evidence has been provided that intra-articular fractures are preceded by subchondral bone disease. This subchondral bone disease consists of a spectrum of microcracks, diffuse microdamage, cell loss (apoptosis or necrosis), and accompanying subchondral bone sclerosis.

CAUSE OF SUBCHONDRAL BONE DISEASE

The development of microdamage is presumed to be associated as a consequence of cyclic trauma. The repeated wear and tear has been noted with radiographic study and, more recently, CT to contribute to subchondral bone sclerosis. However, the direct association between sclerosis leading to the necrosis of bone has not been totally demonstrated. The development of lytic lesions in the subchondral bone, however, is presumed to be associated with microdamage. Factors involved in the predisposition of horses to damage based on the cyclic trauma of an athletic career include racetrack or arena surface, conformation, genetic predisposition, and a destabilizing traumatic injury.

CONSEQUENCES OF TRAUMATIC SUBCHONDRAL BONE DISEASE

Subchondral bone disease creates an environment for pathologic fractures. The most common manifestations are osteochondral chip fractures, which can be career-ending

if not treated successfully. However, the overall success with arthroscopic surgery is high. Slab fractures represent a more severe injury requiring internal fixation. Some of these cases can return to athletic activities. However, in other instances such as collapsing slab fractures in the carpus, the failure to treat adequately can lead to loss of life. The third level of fracture injury in terms of severity is made up of catastrophic injuries that can be life-threatening. Surgical treatments of such conditions are salvage procedures.

Diagnosis

Early diagnosis is critical. The recognition that early disease in the subchondral bone can lead to fractures has resulted in research efforts to diagnose bone disease early.

Treatment

By 1975 the arthroscope began to achieve real clinical use in human orthopedics, and diagnostic arthroscopy of equine carpal joints in three horses was reported in 1975 (Hall and Keeran, 1975). Experience with the limitations of what could be achieved with arthroscopy surgery has fed back into attempts to develop novel treatment techniques, as well as recognizing the need for early diagnosis and prevention of injury (McIlwraith, 1990a; McIlwraith and Bramlage, 1996).

The application of arthroscopic techniques to the horse has revolutionized the treatment of traumatic joint injuries. The first detailed paper on diagnostic arthroscopy in the horse was published in 1978 (McIlwraith and Fessler, 1978), and it is important to recognize that arthroscopic surgery is the diagnostic method of choice to evaluate articular cartilage and remains the gold standard for assessing pathologic joints. As in human orthopedics, use of the arthroscope in horses extended into surgical practice as technology and techniques of triangulation developed. These techniques were first detailed in textbook form in 1984 (McIlwraith, 1984). Diagnostic arthroscopy is especially valuable when response to medical treatment of a joint is suboptimal. In many instances, articular cartilage lesions are more extensive than what is insinuated on radiographs, but these lesions can sometimes be better related to physical examination and the extent of clinical signs.

By 1990, arthroscopy in the horse had gone from being a diagnostic technique used by a few veterinarians to the accepted way of performing joint surgery (McIlwraith, 1990a). Prospective and retrospective data substantiated the value of the technique in the treatment of carpal chip fractures (McIlwraith et al., 1987), fragmentation of the dorsal margin of the proximal phalanx (Yovich and McIlwraith, 1986), carpal slab fractures (Richardson, 1986), osteochondritis dissecans (OCD) of the femoropatellar joint (Martin and McIlwraith, 1985; McIlwraith and Martin, 1985), OCD of the shoulder (Bertone et al., 1987), and subchondral cystic lesions of the femur (Lewis, 1987). The results with tarsocrural OCD were published in 1991 (McIlwraith et al.,

1991). During this period, the use of diagnostic arthroscopy led to the recognition of previously undescribed articular lesions, many of which are treated using arthroscopic techniques.

Since 1990 there has been further sophistication of techniques. New ones have been developed and treatment principles have been changed based on new pathobiologic knowledge and further prospective and retrospective studies defining the success of various procedures. Many of these advances have been recorded in a recent publication (McIlwraith, 2002). For example, there has been further documentation of success rates following arthroscopic removal of fragments from the dorsoproximal margin of the proximal phalanx (Kawcak and McIlwraith, 1994; Colon et al., 2000). Advances and understanding of the pathogenesis of osteochondral disease and fragmentation in the carpus and fetlock have been reported (Norrdin et al., 1998; Kawcak et al., 2001), which naturally led to progression and diagnosis and treatment. Parameters for the surgical treatment of joint injury have been carefully defined (McIlwraith and Bramlage, 1996). Arthroscopic treatment of fractures in the previously considered inaccessible palmar aspect of the carpus have been described (Wilke et al., 2001), together with arthroscopy of the palmar aspect of the distal interphalangeal joint (Brommer et al., 2001; Vacek et al., 1992). Arthroscopy has also led to understanding of the contribution of soft tissue lesions to joint disease. In the carpus, tearing of the medial palmar intercarpal ligament (MPICL) was first reported in 1992 (McIlwraith, 1992) and its implications discussed by Phillips and Wright (1994) and others (Whitton and Rose, 1997; Whitton et al., 1997a,b).

In the fetlock joints, success rates following arthroscopic removal of osteochondral fragments of the palmar/plantar aspect of the proximal phalanx have now been documented (Foerner et al., 1987; Fortier et al., 1995). Results for arthroscopic treatment of osteochondritis dissecans for the distal/dorsal aspect of the third metacarpal/metatarsal bones (McIlwraith et al., 1990) and results of arthroscopic surgery to treat apical, abaxial, and basilar fragments of the sesamoid bones have also been reported (Southwood et al., 1998; Southwood and McIlwraith, 2000).

The results of arthroscopic surgery for the treatment of OCD in the tarsocrural joint have been documented (McIlwraith et al., 1991), and the arthroscopic approach and intra-articular anatomy of the plantar pouch of this joint have also been described (Zamos et al., 1994).

Considerable advances have been made in arthroscopic surgery of the stifle joint. Results of arthroscopic surgery for the treatment of OCD of the femoropatellar joint were reported in 1992 (Foland et al., 1992), and the syndrome of fragmentation of the distal apex of the patellar was recognized and its treatment reported in the same year (McIlwraith, 1990b). The use of arthroscopic surgery for treating certain patellar fractures was discussed in 1990 and reported in the refereed literature in 2000 (Marble and Sullins, 2000).

In the femorotibial joints, the use of arthroscopic surgery to treat subchondral cystic lesions of the medial condyle of the femur and proximal tibia has been reported

(Howard et al., 1995; Textor et al., 2001). Research has led to alternative methods of treating subchondral cystic lesions. After an initial demonstration that subchondral cystic lesions could develop on the medial condyle after 3 mm deep, 5 mm wide penetration of the subchondral bone plate (Ray et al., 1996), examination of the fibrous tissue of subchondral cystic lesions in horses demonstrated that it produced local mediators and neutral metalloproteinases and caused bone resorption in vitro (von Rechenberg et al., 2000a). Production of nitric oxide (NO), PGE2, and MMPs in media of explant cultures of equine synovial membrane and articular cartilage has also been demonstrated in normal and osteoarthritic joints (von Rechenberg et al., 2000b). Injection of corticosteroids into the lining membrane of subchondral cysts has therefore been carried into the clinical arena.

Cartilage lesions of the medial femoral condyle have been described (Schneider et al., 1997). Arthroscopy has allowed great advances in the recognition and treatment of meniscal tears and cruciate injuries (Walmsley, 1995; Walmsley, 2002; Walmsley et al., 2003). Successful treatment of grade I and grade II meniscal tears has been achieved and documented as well as lack of success recognized with lesions that are not completely accessible. Arthroscopy has also been used to remove fragments from the intercondylar eminence of the tibia (Mueller et al., 1994), and internal fixation of one case has been reported (Walmsley, 1997). Techniques have also been developed for diagnostic and surgical arthroscopy of the caudal pouches of the femorotibial joints (Stick et al., 1992; Hance et al., 1993; Trumble et al., 1994; Walmsley, 2002).

Diagnostic and surgical arthroscopy of the coxofemoral joint has been described, lesions identified, and some surgical treatments performed (Honnas et al., 1993; Nixon, 1994). The use of the arthroscope is no longer confined to the limbs and the arthroscopic anatomy of the temporomandibular joint has been described recently (Weller et al., 2002).

References

Adams, O.R. 1974. Lameness in Horses (3rd Ed.) Lea and Febiger, Philadelphia.

Barrie, H.J. 1987. Osteochondritis dissecans: 1887-1987-A Centennial look at Konig's memorable phrase. J. Bone Joint Surg. Br. 69B:693-695.

Bertone, A.L., C.W. McIlwraith, B.E. Powers, G.W. Trotter, and T.S. Stashak. 1987. Arthroscopic surgery for the treatment of osteochondrosis in the equine shoulder joint. Vet. Surg. 16:303-311.

Bridges, C.H., and E.D. Harris. 1988. Experimentally induced cartilaginous fractures (osteochondritis dissecans) in foals fed low-copper diets. J. Amer. Vet. Med. Assoc. 193:215-221.

Brommer, H., A.M. Rijkenhuizen, H.A.M. van den Belt, et al. 2001. Arthroscopic removal of an osteochondral fragment at the palmaroproximal aspect of the distal interphalangeal joint. Equine Vet. Educ. 13:294-297.

Carlson, C.S., D.J. Meuten, and D.C. Richardson. 1991. Ischemic necrosis of cartilage in spontaneous and experimental lesions of osteochondrosis. J. Orthop. Res. 9:317-329.

Colon, J.L., L.R. Bramlage, S.R. Hance, and R.M. Embertson. 2000. Qualitative and quantitative documentation of the racing performance of Thoroughbred racehorses after arthroscopic removal of dorsoproximal first phalanx osteochondral fractures (1986-1995). Equine Vet. J. 32:475-481.

Dik, K.J., E. Enzerink, and R. van Weeran. 1999. Radiologic development of osteochondral abnormalities in the hock and stifle of Dutch Warmblood foals from age 1 to 11 months. Equine Vet. J. Suppl. 31:9-15.

Foerner, J.J., W.P. Barclay, T.N. Phillips, et al. 1987. Osteochondral fragments of the palmar/plantar aspect of the fetlock joint. In: Proc. Amer. Assoc. Equine Practnr. 33:739-744.

Foland, J.W., C.W. McIlwraith, and G.W. Trotter. 1992. Arthroscopic surgery for osteochondritis dissecans of the femoropatellar joint. Equine Vet. J. 24:419-423.

Fortier, L.A., J.J. Foerner, and A.J. Nixon. 1995. Arthroscopic removal of axial osteochondral fragments of the plantar/palmar proximal aspect of the proximal phalanx in horses: 119 cases (1988-1992). J. Amer. Vet. Med. Assoc. 206:71-74.

Glade, M.J. 1986. Control of cartilage growth in osteochondrosis: A review. J. Equine Vet. Sci. 6:175-187.

Glade, M.J., and T.H. Belling. 1986. A dietary etiology for osteochondrotic cartilage. J. Equine Vet. Sci. 6:151-155.

Glade, M.J., L. Krook, H.F. Schryver, et al. 1983. Morphologic and biochemical changes in cartilage of foals treated with dexamethasone. Cornell Vet. 73:170-192.

Grondahl, A.M., and N.I. Dolvik. 1993. Hereditability estimations of osteochondrosis in the tibiotarsal joint and of bony fragments in the palmar/plantar portion of the metacarpophalangeal and metatarsophalangeal joints of horses. J. Amer. Vet. Med. Assoc. 203:101-104.

Gunson, D.E., D.F. Kowalczyk, C.R. Shoop, et al. 1982. Environmental zinc and cadmium pollution associated with generalized osteochondrosis, osteoporosis and nephrocalcinosis in horses. J. Amer. Vet. Med. Assoc. 180:295-299.

Hall, M.E., and R.J. Keeran. 1975. Use of the arthroscope in the horse. Vet. Med. Small Animal Clin. 70:705-706.

Hance, R., R.K. Schneider, R.M. Embertson, et al. 1993. Lesions of the caudal aspect of the femoral condyles in foals: 20 cases (1980-1990). J. Amer. Vet. Med. Assoc. 202:637-646.

Honnas, C.M., D.T. Zamos, and T.S. Ford. 1993. Arthroscopy of the coxofemoral joint of foals. Vet. Surg. 22;115-121.

Hoppe, F., and J. Phillipsson. 1985. A genetic study of osteochondrosis in Swedish

horses. Equine Pract. 7:7-15.
Howard, R.D., C.W. McIlwraith, and G.W. Trotter. 1995. Arthroscopic surgery for subchondral cystic lesions of the medial femoral condyle in horses: 41 cases (1988-1991). J. Amer. Vet. Med. Assoc. 206:846-850.
Hurtig, M.B., S.L. Green, H. Dobson, et al. 1990. Defective bone and cartilage in foals fed a low copper diet. In: Proc. Amer. Assoc. Equine Practnr. 35:637-643.
Kawcak C.E., and C.W. McIlwraith. 1994. Proximodorsal first phalanx osteochondral chip fragments in 320 horses. Equine Vet. J. 26:392-396.
Kawcak, C.E., C.W. McIlwraith, R.W. Norrdin, R.D. Park, and S.D. James. 2001. The role of subchondral bone in joint disease: A review. Equine Vet. J. 33:120-126.
Lewis, R.B. 1987. Treatment of subchondral bone cysts of the medial condyle of the femur using arthroscopic surgery. In: Proc. Amer. Assoc. Equine Pract. 33:887-893.
Marble, G.P., and K.E. Sullins. 2000. Arthroscopic removal of patellar fracture fragments in horses: 5 cases (1989-1998). J. Amer. Vet. Med. Assoc. 216:1799-1801.
Martin, G.S., and C.W. McIlwraith. 1985. Arthroscopic anatomy of the equine femoropatellar joint and approaches for treatment of osteochondritis dissecans. Vet. Surg. 14:99-104.
McIlwraith, C.W. 1984. Diagnostic and surgical arthroscopy in the horse. Veterinary Medicine Publishing Co.
McIlwraith, C.W. 1986. AQHA Developmental Orthopedic Disease Symposium. American Quarter Horse Association. Amarillo, TX. 1-77.
McIlwraith, C.W. 1990a. Diagnostic and Surgical Arthroscopy in the Horse (2nd Ed.) Lea and Febiger, Philadelphia.
McIlwraith CW. 1990b. Osteochondral fragmentation or the distal aspect of the patella in horses. Equine Vet. J. 22:157-163.
McIlwraith, C.W. 1992. Tearing of the medial palmar intercarpal ligament in the equine mid-carpal joint. Equine Vet. J. 24:367-371.
McIlwraith, C.W. 1993. What is developmental orthopedic disease, osteochondrosis, osteochondritis, metabolic bone disease? In: Proc. Amer. Assoc. Equine Practnr. 39:35-44.
McIlwraith, C.W. 2002. Arthroscopy: An Update. In: Clinical Techniques in Equine Practice. p. 199-281. WB Saunders, Philadelphia.
McIlwraith, C.W., and L.R. Bramlage. 1996. Surgical treatment of joint injuries. In: C.W. McIlwraith and G.W. Trotter (Eds.) Joint Disease in the Horse. WB Saunders, Philadelphia.
McIlwraith, C.W., and J.F. Fessler. 1978. Arthroscopy in the diagnosis of equine joint disease. J. Amer. Vet. Med. Assoc. 172:263-268.
McIlwraith, C.W., and G.S. Martin. 1985. Arthroscopic surgery for the treatment of osteochondritis dissecans in the equine femoropatellar joint. Vet. Surg. 14:105-116.

McIlwraith, C.W., and M. Vorhees. 1990. Management of osteochondritis dissecans of the dorsal aspect of the distal metacarpus and metatarsus. In: Proc. Amer. Assoc. Equine Practnr. 35:547-550.

McIlwraith, C.W., J.J. Foerner, and D.M. Davis. 1991. Osteochondritis dissecans of the tarsocrural joint: Results of treatment with arthroscopic surgery. Equine Vet. J. 23:155-162.

McIlwraith, C.W., J.V. Yovich, and G.S. Martin. 1987. Arthroscopic surgery for the treatment of osteochondral chip fractures in the equine carpus. J. Amer. Vet. Med. Assoc. 191:531-540.

McIntosh, S.C., and C.W. McIlwraith. 1993. Natural history of femoropatellar osteochondrosis in three crops of Thoroughbreds. Equine Vet. J. 16:54-61.

Messer, N.T. 1981. Tibiotarsal effusion associated with chronic zinc intoxication in three horses. J. Amer. Vet. Med. Assoc. 178:294.

Mueller, P.O.E., D. Allen, E. Watson, et al. 1994. Arthroscopic removal of a fragment from an intercondylar eminence fracture of the tibia in 2-year-old horse. J. Amer. Vet. Med. Assoc. 204:1793-1795.

Nixon, A.J. 1994. Diagnostic and operative arthroscopy of the coxofemoral joint in horses. Vet. Surg. 23:377-385.

Norrdin, R.W., C.E. Kawcak, B.A. Capwell, and C.W. McIlwraith. 1998. Subchondral bone failure in an equine model of overload arthrosis. Bone 22:133-139.

Olsson, S.E. 1978. Introduction. Acta. Radiol. Supp. 358:9-14.

Pagan, J.D., and S.G. Jackson. 1996. The incidence of developmental orthopedic disease on a Kentucky Thoroughbred farm. World Equine Vet. Rev. 20-26.

Pearce, SG., E.C. Firth, N.D. Grace, and P.F. Fennessy. 1998a. Effect of copper supplementation on the evidence of developmental orthopaedic disease in pasture-fed New Zealand Thoroughbreds. Equine Vet. J. 30:211-218.

Pearce, S.G., N.D. Grace, E.C. Firth EC, et al. 1998b. Effect of copper supplementation on the copper status of pasture-fed young Thoroughbreds. Equine Vet. J. 30:204-210.

Pearce, S.G., N.D. Grace, J.J. Wichtel, et al. 1998c. Effect of copper supplementation on copper status of pregnant mares and foals. Equine Vet. J. 30:200-203.

Pettersson, H., and F. Sevelius. 1968. Subchondral bone cysts in the horse: A clinical study. Equine Vet. J. 1:75.

Phillips, T.J., and I.M. Wright. 1994. Observations on the anatomy and pathology of the palmar intercarpal ligaments in the middle carpal joints of Thoroughbred racehorses. Equine Vet. J. 26:486-491.

Phillipsson, J., E. Andreasson, B. Sandgren, et al. 1993. Osteochondrosis in the tarsocrural joint and osteochondral fragments in the fetlock joints in Standardbred trotters. II Hereditability. Equine Vet. J. 16:38-41.

Pool, R.R. 1986. Pathologic manifestations of osteochondrosis. In: C.W. McIlwraith (Ed.) p. 3-7. AQHA Developmental Orthopedic Disease Symposium. American Quarter Horse Association. Amarillo, TX.

Ray, C.S., G.M. Baxter, C.W. McIlwraith, et al. 1996. Development of subchondral cystic lesions after articular cartilage and subchondral bone damage in young horses. Equine Vet. J. 28:225-232.

Rejno, S., and B. Stromberg. 1978. Osteochondrosis in the horse. II. Pathology. Acta Radiol. Suppl. 358:153-178.

Richardson, D.W. 1986. Technique for arthroscopic repair of third carpal bone slab fractures in horses. J. Amer. Vet. Med. Assoc. 188:288-291.

Rooney, J.R. 1975. Osteochondrosis in the horse. Mod. Vet. Pract. 56:41-43, 113-116.

Savage, C.J., R.N. McCarthy, and L.B. Jeffcott. 1993. Effects of dietary phosphorus and calcium on induction of dyschondroplasia in foals. Equine Vet. J. 516:80-83.

Schneider, R.K., P. Jenson, and R.M. Moore. 1997. Evaluation of cartilage lesions on the medial femoral condyle as a cause of lameness in horses: 11 cases (1988-1994). J. Amer.Vet. Med. Assoc. 210:1649-1652.

Schougaard, H., J. Falk-Ronne, and J. Phillipsson. 1990. A radiographic survey of tibiotarsal osteochondrosis in a selected population of trotting horses in Denmark and its possible genetic significance. Equine Vet. J. 22:288-289.

Southwood, L.L., and C.W. McIlwraith. 2000. Arthroscopic removal of fracture fragments involving a portion of the base of the sesamoid bone in horses. J. Amer. Vet. Med. Assoc. 217:236-240.

Southwood, L..L., G.W. Trotter, and C.W. McIlwraith. 1998. Arthroscopic removal of abaxial fracture fragments of the proximal sesamoid bone in horses: 47 cases (1989-1997). J. Amer. Vet. Med. Assoc. 213:1016-1021.

Stick, J.A., L.A. Borg, F.A. Nickels, et al. 1992. Arthroscopic removal of osteochondral fragment from the caudal pouch of the lateral femorotibial joint in a colt. J. Amer. Vet. Assoc. 200:1695-1697.

Stromberg, J. 1979. A review of the salient features of osteochondrosis in the horse. Equine Vet. J. 11:211-214.

Textor, J.A., A.J. Nixon, J. Lumsden, et al. 2001. Subchondral cystic lesions of the proximal extremity or the tibia in horses: 12 cases (1983-2000). J. Amer. Vet. Med. Assoc. 218:408-413.

Trumble, T.N., A.J. Stick, S.P. Arnoczky, et al. 1994. Consideration of anatomic and radiographic features of the caudal pouches of the femorotibial joints of horses for the purpose of arthroscopy. Amer. J. Vet. Res. 55:1682-1689.

Vacek, J.R., R.D. Welch, and C.M. Honnas. 1992. Arthroscopic approach and intra-articular anatomy of the palmaroproximal and plantaroproximal aspect of distal interphalangeal joints. Vet. Surg. 4:257-260.

von Rechenberg, B., H. Guenther, C.W. McIlwraith, et al. 2000a. Fibrous tissue of subchondral cystic lesions in horses produce local mediators in neutral metalloproteinases and cause bone resorption in vitro. Vet. Surg. 29:420.429.

von Rechenberg, B., C.W. McIlwraith, and M. Akens. 2000b. Spontaneous production of nitric oxide (NO) prostaglandins (PGE2) in neutral metalloproteinases

(MMPs) in media of explant cultures of equine synovial membrane and articular cartilage from normal and osteoarthritic joints. Equine Vet. J. 32:140-150.

Walmsley, J.P. 1995. Vertical tears in the cranial horn of the meniscus and its cranial ligament in the equine femorotibial joint: 7 cases and their treatment by arthroscopic surgery. Equine Vet. J. 27:20-25.

Walmsley, J.P. 1997. Fracture of the intercondylar eminence of the tibia treated by arthroscopic internal fixation. Equine Vet. J. 29:148-150.

Walmsley, J.P. 2002. Arthroscopic surgery of the femorotibial joint. Clin. Tech. Equine Pract. 1:226-233.

Walmsley, J.P., T.J. Phillips, and H.G. Townsend. 2003. Meniscal tears in horses: An evaluation of clinical signs and arthroscopic treatment of 80 cases. Equine Vet. J. 35:402-406.

Weller, R.R., L.J. Maieler, I.M. Bowen, S.A. May, and H.G. Liebich. 2002. The arthroscopic approach and intra-articular anatomy of the equine temporomandibular joint. Equine Vet. J. 34:421-424.

Whitton, R.C., and R.J. Rose. 1997. The intercarpal ligaments of the equine mid-carpal joint. Part II: the role of the palmar intercarpal ligaments in the restraint of dorsal displacement of the proximal row of carpal bones. Vet. Surg. 26:367-373.

Whitton, R.C., N.J. Kannegieter, and R.J. Rose. 1997a. The intercarpal ligaments of the equine mid-carpal joint. Part III: clinical observations in 32 racing horses with mid-carpal joint disease. Vet. Surg. 26:374-381.

Whitton, R.C., P.H. McCarthy, and R.J. Rose. 1997b. The intercarpal ligaments of equine mid-carpal joint. Part I: the anatomy of the palmar and dorsomedial intercarpal ligaments of the mid-carpal joint. Vet. Surg. 26:359-366.

Wilke, M., A.J. Nixon, and J. Malark. 2001. Fractures of the palmar aspect of carpal 7 bones in horses: 10 cases (1984-2000). J. Amer. Vet. Med. Assoc. 219:801-804.

Yovich, J.V., and C.W. McIlwraith. 1986. Arthroscopic surgery for osteochondral fractures of the proximal phalanx of the metacarpophalangeal and metatarsophalangeal (fetlock) joints in horses. J. Amer. Vet. Med. Assoc. 188:243-279.

Zamos, D.T., C.M. Honnas, and A.G. Hoffman. 1994. Arthroscopic approach and intra-articular anatomy of the plantar pouch of the equine tarsocrural joint. Vet. Surg. 23:161-166.