Kentucky Equine Research®
World Leaders In Equine Nutrition

# Advances in Equine Nutrition
## Volume III

Edited by
J.D. Pagan

# SURGICAL AND MEDICAL MANAGEMENT OF OSTEOCHONDRITIS DISSECANS (OCD)

C. WAYNE MCILWRAITH
*Colorado State University, Fort Collins, Colorado, USA*

## Introduction

Osteochondritis dissecans (OCD) is an important entity within the developmental orthopedic disease complex. It is a frequent cause of lameness in young athletic horses and is the most frequent condition of the complex requiring surgical intervention. OCD has been classically considered as a manifestation of osteochondrosis (McIlwraith, 1993b). Rejno and Stromberg described the first stage of osteochondrosis as a disturbance of cellular differentiation in the growing cartilage, and the second as involving necrosis of the basal layers of the thickened retained cartilage with subsequent pressure and strain within the joint, giving rise to fissures in the damaged cartilage (1978). The terms osteochondrosis, osteochondritis dissecans, and osteochondrosis dissecans have been regularly used as synonyms, and their meaning is still somewhat controversial. The terms have been distinguished as follows: osteochondrosis is the disease, osteochondritis is the inflammatory response to the disease, and OCD is the condition in which a flap can be demonstrated (Poulos, 1986). This is a simple but fairly appropriate representation. This presentation addresses the clinical aspects of OCD, including the clinical signs and diagnosis, as well as treatment options and prognosis. Although arthroscopic surgery is the most commonly recommended treatment to achieve athletic activity and prevent degenerative joint disease, certain situations in which conservative treatment is successful have been recognized.

Three categories of OCD lesions are recognized: (1) those showing clinical and radiographic signs, (2) those showing clinical without radiographic (but arthroscopic) signs, and (3) those showing radiographic but no clinical signs. Data from the first two categories of disease have been tabulated for the most commonly selected joints from the author's surgical case reports (McIlwraith, 1993a). The relative incidence of clinical signs versus radiographic lesions has also been documented in the femoropatellar joint by McIntosh and McIlwraith (1993). Similar data in other joints are needed.

The clinical aspects of OCD are presented next for the individual joints in which OCD is most common.

## Osteochondritis Dissecans of the Femoropatellar Joint

OCD was first described in the femoropatellar joint in the horse by Nilsson in 1947 (Nilsson, 1947). The lesion described by Nilsson is believed to be similar to lesions currently referred to as OCD. Similar lesions were described as osteochondral fractures in 1973 (O'Brien, 1973). Since that time there have been a number of reports concerning pathologic and surgical aspects of the condition (Moore and McIlwraith, 1977; Wyburn, 1977; Rejno, 1978; Stromberg and Rejno, 1978; Trotter et al., 1983; Pascoe et al., 1984; McIlwraith and Martin, 1985; Foland et al., 1992; McIlwraith, 1987; Wright and Pickles, 1991). The clinical signs have been well defined, but the treatment is more controversial. The femoropatellar joint is one of the principal sites of OCD in the horse.

### *INCIDENCE AND CLINICAL SIGNS*

In a recent study, more than 50% of the horses operated upon for femoropatellar OCD were Thoroughbreds (Table 1) (Foland et al., 1992). There were 53 females in the same group and 108 males (82 intact and 26 gelded). The age distribution of 161 horses presented for femoropatellar OCD is presented in Table 2.

**Table 1.** Breed disposition of 161 horses operated on for femoropatellar osteochondritis dissecans (Foland et al., 1992).

| *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 |

**Table 2.** Age distribution of 161 horses presented for femoropatellar osteochondritis dissecans (Foland et al., 1992).

| *Age (Years)* | *Number* | *Percentage* |
|---|---|---|
| <1 | 22 | 13.7 |
| 1 | 68 | 42.2 |
| 2 | 36 | 22.4 |
| 3 | 21 | 13.0 |
| ≥4 | 14 | 8.7 |

Approximately 55% of the horses were 1 year of age or less at presentation, and the younger animals tended to have more severe lesions (Foland et al., 1992).

Clinical signs may develop at any age. More mature animals frequently present with a sudden onset of clinical signs thought to be associated with the displacement of osteochondral fragments. Less frequently, clinically silent lesions may be identified in mature horses, and a sudden onset of clinical signs may also occasionally be seen in cases in which fragmentation has not yet developed. In one study on one farm, the average age of identification of femoropatellar OCD problems was 12.6, 9, and 6 months in 3 consecutive years (McIntosh and McIlwraith, 1993).

Horses with OCD of the femoropatellar joint usually present with differing degrees of distension of the femoropatellar joint and differing degrees of lameness, depending on the severity of the lesions. Distension of the femoropatellar joint is the more consistent presenting sign. However, the clinicopathologic changes in the synovial fluid are usually minor. Lameness varies from nondiscernible to severe. Other common abnormalities of gait include reduced cranial phase to the stride and lowered foot arc. In young animals with severe lesions, there may be difficulty in rising. Concurrent flexural deformities have also been reported (Moore, 1977; McIlwraith and Martin, 1985). Lateral luxation of the patella in association with OCD of the lateral trochlear ridge of the femur has been seen (McIlwraith, 1987). Unilaterally affected animals are often asymmetrically muscled, whereas bilateral cases frequently exhibit poor hind limb muscle development (Wright and Pickles, 1991). The disease is commonly bilateral. In one recent series, 91 horses (57%) were bilaterally affected and 70 horses (43%) had unilateral disease (Foland et al., 1992).

Lateromedial radiographs provide the most useful information with regard to the location and nature of the lesions. Caudolateral to craniomedial oblique projections may also provide additional information with regard to the depth of the lesion on the lateral trochlear ridge of the femur. The most common defect seen radiographically is an irregularity or flattening in the subchondral bone of the lateral trochlear ridge of the femur that may be localized or generalized. In either situation, the area of the lateral trochlear ridge that articulates with the distal aspect of the patella in the standing position is involved. Lesions manifesting as defects on radiographs usually manifest with an OCD flap or elevated cartilage. Partially mineralized flaps may be observed radiographically in some instances. There are islands of mineralized tissue in other defects. Mineralized free bodies or joint mice that have detached from the primary defect may be present loose within the joint or attached to synovial membrane. The presence of irregular subchondral defects without joint mice tends to be seen in younger horses. Cystic lesions and undermined lytic lesions within the subchondral bone are observed in other instances. Similar lesions may be seen on the medial trochlear ridge but are usually limited to irregularities of contour and are not as extensive. In a number of instances, a separation of cartilage from the medial trochlear ridge without any defect in the subchondral bone (and therefore no radiographic signs) is seen at surgery. Primary OCD of the patella is relatively rare but is seen in some instances. Articular cartilage degenerative changes may be seen secondary to severe OCD of the lateral trochlear ridge of the femur (usually on the lateral facet of patella). In severe cases of OCD of the trochlear ridges, remodeling of the patellar contour may be visible. Localized defects of the patellar contour visualized on radiographs may represent primary osteochondrosis or secondary changes. Care must be taken not to diagnose the radiographic changes of endochondral ossification on the trochlear ridges of young foals as OCD lesions (Adams and Thilsted, 1985).

The most common location for OCD lesions is the lateral trochlear ridge of the femur. The overall incidence in a series of cases (Foland et al., 1992) is presented in Table 3. Thirty-two horses had loose bodies in at least one joint in this series. Grades as determined from surgery reports were equal to the radiographic grades in 111 cases, but at surgery 46 horses had lesions that were worse than those seen radiographically. In a related study, the radiographs of 72 femoropatellar and femorotibial joints from 50 horses that had arthroscopic surgery were evaluated (Steinheimer et al., 1996). Ninety-four arthroscopically evaluated areas were graded according to a predetermined system (based on surgery reports).

**Table 3.** Location of osteochondritis dissecans lesions in 252 femoropatellar joints (Foland et al., 1992).

| *Location* | *No. of joints affected* |
|---|---|
| LTR | 161 |
| LTR and patella | 31 |
| MTR | 17 |
| LTR and MTR | 17 |
| LTR, patella and TG | 4 |
| LTR, MTR and patella | 3 |
| MTR and patella | 3 |
| Patella | 3 |
| LTR, MTR, TG and patella | 3 |
| LTR and TG | 3 |
| LTR, MTR and TG | 3 |
| MTR and TG | 3 |
| TG | 1 |

LTR = lateral trochlear ridge; MTR = medial trochlear ridge ; TG = trochlear groove.

The radiographic grade was then compared with arthroscopic findings in the same location and statistical analysis performed to determine the association between radiographic subchondral bone changes and arthroscopic findings. Radiographically normal areas in the femoropatellar joint were arthroscopically positive for cartilaginous changes in 40% of the femoropatellar joints. Areas of mild subchondral bone flattening (grade I) in the lateral trochlear ridge of the femur were arthroscopically positive for cartilage changes 78% of the time. Ninety-six percent of moderate to severe subchondral bone changes (grades II to V) were arthroscopically positive for cartilage damage. This research demonstrated that (1) a significant number of radiographically normal joints have cartilage changes, (2) areas of mild subchondral bone flattening have cartilage changes in most cases, and (3) areas of moderate to severe subchondral bone change have arthroscopically detectable cartilage changes.

Lesions of OCD in joints other than the femoropatellar joint may occur at the same time. In the previously mentioned series of 161 horses, 10 horses underwent surgery for other OCD lesions at the time of the femoropatellar arthroscopy (Foland et al., 1992). Five had OCD of both metatarsophalangeal joints, four had OCD of the tarsocrural joint, and one had OCD of the scapulohumeral joint. Two other horses had subchondral cystic lesions of the medial femoral condyle.

### *TREATMENT*

Conflicting reports have been published concerning the management of OCD of the equine femoropatellar joint. In 1977, Wyburn reported that satisfactory results were achieved with conservative therapy if osteochondral fragments were not seen radiographically, but that surgery was indicated if free bodies were noted (Wyburn, 1977). However, two later reports showed that at surgery fragments that could not be detected radiographically were often found (Pasco et al., 1984; McIlwraith, 1985). Another group of authors also felt that 54% of horses with stifle problems (OCD of the femoropatellar joint and subchondral cystic lesions of the femorotibial joint were not distinguished) were improved after conservative therapy (Rose et al., 1985), but an examination shows the results to be inferior to those reported with surgery (Foland et al., 1992). Stromberg and Rejno reported radiographic evidence that young horses that had large subchondral defects and were not treated surgically developed degenerative joint disease (1978). Steenhaut et al. also considered the outcome without surgical treatment to be poor (1982). Favorable results after surgical treatment using arthrotomy have been reported (Stromberg and Rejno, 1978; Pasco et al., 1984). The advantages of using arthroscopic surgery to treat femoropatellar OCD are now well recognized, and the results of surgery with this technique have been published (McIlwraith, 1985; Foland et al., 1992; Martin and McIlwraith, 1985).

More recent work indicates that conservative treatment can be appropriate in some instances. A recent study demonstrated considerable success in Thoroughbreds (based on subsequent racing performance) with conservative treatments. In this study a careful assessment of foals led to the detection of lesions at an early age, and it was concluded that with confinement early radiographic lesions of OCD (subchondral bone contour irregularity and subchondral lysis) are potentially reversible. On a farm affected with a high incidence of femoropatellar OCD, three crops of foals were evaluated. Of 11 horses not operated on in 1989 (some other horses were operated on), 6 raced, 3 had no follow-up, 1 became a jumper, and 1 was operated on later. In 1990, 9 foals with clinical and radiographic lesions of OCD were treated conservatively; at the time of the report, 2 had raced, 2 were still unraced, 3 had lameness, and 2 were used for nonracing careers. In 1991, 10 cases were diagnosed (5 horses had no radiographic lesions initially but subsequently developed them, and 1 never had a radiographic lesion but had persistent synovial effusion). Of these 16, 4 horses were sold as yearlings, reportedly without synovial effusion or lameness, 1 died of unrelated causes, 10 are currently racing or training, and 1 subsequently developed OCD of the metatarsophalangeal joints. There was a trend in these data to indicate that lesser lesions healed with conservative treatment and more severe ones persisted in having clinical signs. There were two exceptions (successful results when the lesion was large) to this rule.

A case can be made for arthroscopy in all cases in that unsuspected intra-articular pathologic changes are often found, and early management of these problems can be instituted. It has been stated that once a radiographic diagnosis of OCD has been made in humans, arthroscopic staging of the disease and treatment are the next logical steps and that conservative treatment by casting, immobilization, and limitations of activities (particularly in children) should be tried initially before resorting to surgery (Ewing and Voto, 1988). It was noted that it is still difficult to predict when a lesion will heal and when a lesion will persist, particularly if arthroscopy is not done (Ewing and Voto, 1988). If conservative treatment is to be attempted, restriction of exercise with confinement is the critical factor. Restricted activity is the basis for the conservative management of human juvenile OCD but is not consistently successful (Aglietti et al., 1994).

The treatment of femoropatellar OCD with arthroscopic surgery has been extensively described elsewhere (Martin and McIlwraith, 1985; McIlwraith and Martin, 1985; McIlwraith, 1990; Foland et al., 1992).

Evaluation of the lesion in the joint is carried out thoroughly as not all lesions are detected radiographically, and each lesion is assessed with a probe. Elevators are used to separate OCD flaps. The flaps are generally removed with rongeurs, and the underlying lesions are debrided with curets, motorized equipment, or both. Loose bodies are also removed. Debridement of all pathologic tissue is necessary.

The results of arthroscopic surgery for the treatment of OCD of the femoropatellar joint have been reported in 252 femoropatellar joints in 161 horses (Foland et al., 1992). Follow-up information was obtained on 134 horses, including 79 racehorses and 55 nonracehorses. Eighty-six (64%) of these 134 horses returned to their intended use, 9 (7%) were in training, 21 (16%) were unsuccessful, and 18 (13%) were unsuccessful because of other defined reasons. Of the 18 horses that were unsuccessful because of other reasons, 14 developed unassociated lameness (10 forelimb and 4 hindlimb), 1 died of colic, 1 became a wobbler, 1 developed a fatal case of enteritis, and 1 died under anesthesia during surgery for an unrelated problem in another clinic. The time from surgery until the horses started training was dictated in many cases by the age of the horse. Those horses that had already performed or trained before surgery returned to training 4 to 6 months after surgery. Sufficient follow-up was available for 11 of the 12 horses that had surgery for OCD in other joints at the time of femoropatellar surgery. Six of these horses (55%) were successful, 3 (27%) were unsuccessful, and 2 (18%) were unsuccessful because of other reasons.

There was a significant effect of lesion size on prognosis. Horses with grade 1 lesions (<2 cm in length) had a significantly higher success rate (78%) than horses with grade 2 (2 to 4 cm) or grade 3 (>4 cm) lesions (63 and 54% success rates, respectively) (Foland et al., 1992). A significantly higher success was also noted for horses operated on at 3 years compared with the remainder of the study population. A significantly lower success rate was noted for yearlings than for the remainder of the population. It was felt that the lowest success rate in horses operated on as yearlings is probably associated with the fact that more severe lesions occurred in the yearling horses (16% grade I, 13% grade II, 46% grade III) and less severe lesions occurred in the 3-year-old group (52% grade I, 38% grade II, 10% grade III). There was no significant difference in outcome related to the sex of the animal involved; racehorse versus nonracehorse; the lesion location; unilateral versus bilateral involvement; the presence or absence of patellar or trochlear groove lesions; or the presence or absence of loose bodies.

Although the results of this study may at first seem somewhat discouraging in that only 64% of the horses returned to or achieved the intended level of performance, many of these horses were operated on at relatively young ages and consequently had not yet proved themselves as athletes (over 50% were 1 year of age or younger). A study on racing performance in Thoroughbreds born in 1984 indicated that approximately 60% of all named foals should start a race (Dink, 1990). The inclusion of only named foals (registered with The Jockey Club) would exclude those that showed no potential or developed other problems at a young age and were consequently not registered. In our study, many of the horses had surgery before they were named, and this led us to believe that a 64% success rate is comparable with successful performance in the normal population of racing Thoroughbreds (Foland et al., 1992).

Although a permanent clinical cure can often be associated with surgery, limited data show that the nature of the healing tissue is different from that of normal osteochondral tissue (McIlwraith, 1990) on the basis of long-term follow-up radiographs of sound horses operated on by the author. Irregular subchondral contours frequently persist, and this suggests that subchondral bone remodeling does not take place in the femoral trochlear ridges. In humans, OCD may affect the trochlear ridges but more commonly affects the femoral condyles (Smith, 1990). Surgical treatments include the removal of flaps, drilling through the lesion into bone, and fixation of the flap (Ewing and Voto, 1988; Smith, 1990; Aglietti et al., 1994).

## Osteochondritis Dissecans of the Femorotibial Joints

Subchondral cystic lesions are the most common developmental orthopedic disease involving the femoral condyles. However, cases of OCD of the femoral condyles have been encountered. Lesions of OCD may accompany subchondral cystic lesions or occur on their own (most commonly in Thoroughbreds). The lesions manifest radiographically as an irregular defect in the subchondral bone, best seen on a flexed lateral view. They appear arthroscopically as typical OCD lesions and have responded to surgical treatment. A series of lesions of the femoral condyle, some of which may be lesions of osteochondrosis, was also recently reported. Because these cases have been recognized infrequently, figures for prognosis do not exist.

## Osteochondritis Dissecans of the Tibiotarsal (Tarsocrural) Joint

OCD of the tarsocrural joint was first described in the horse in 1972 (DeMoor, 1972). Before this, however, seven cases of a condition that appears identical to OCD were described by Birkeland and Haakenstad as intracapsular bony fragments of the distal tibia (1968). These authors later described the lesions as OCD (Birkeland and Haakenstad, 1974). In 1978 Stromberg and Rejno also reported OCD lesions in 61 tarsocrural joints in 49 horses (Stromberg and Rejno, 1978). There have been a number of recent reports documenting the incidence, clinical signs, and results of treatment of OCD of the tarsocrural joint (Hoppe, 1984a,b; Sandgren, 1988; Alvarado, 1989; Grondahl, 1991; McIlwraith et al., 1991; Carlsten et al., 1993; Laws et al., 1993; Beard et al., 1994).

### *INCIDENCE, CLINICAL SIGNS, AND DIAGNOSIS*

OCD of the tarsocrural joint is seen most frequently in Standardbred horses (Birkeland and Haakenstad, 1968, 1974; DeMoor, 1972; Stromberg and Rejno, 1978; Hoppe, 1984a,b; Sandgren, 1988; Alvarado, 1989; Grondahl, 1991; McIlwraith et al., 1991; Carlsten et al., 1993). In the largest series of clinical cases published to date, 154 of 225 horses were intended for racing (106 Standardbreds, 30 Thoroughbreds, 18 Quarter Horses), and the remaining 71 comprised 20 Arabians, 18 Quarter Horses, 13 warmbloods, 4 American Saddlebreds, 4 Appaloosas, 4 Thoroughbreds, 3 draft horses (1 Clydesdale, 1 Percheron, 1 Shire), 2 American Paint Horses, 1 Morgan, 1 National Show Horse, and 1 Lipizzaner (McIlwraith, 1991).

The presenting clinical signs were synovial effusion of the tarsocrural (tibiotarsal) joint and lameness. Synovial effusion of the joint is the most common reason for cases being presented, particularly in animals presented before being put into training. Obvious lameness is often not observed. Older horses (>2 years) or racehorses may be presented for lameness. Of 303 joints with tarsocrural OCD in which the presence or absence of synovial effusion was recorded, synovial effusion was the presenting clinical sign in 261 joints (86.1%) (McIlwraith, 1991). In racehorses, effusion was present in 166 joints (81%) and absent in 39 joints. In nonracehorses, effusion was present in 95 joints (96.9%) and absent in 3 joints. The degree of lameness was not recorded consistently but was usually designated as mild. The exception was when severe lesions were present on the lateral trochlear ridge of the talus (lesions involving the entire visible portion of the lateral trochlear ridge of the talus when viewed arthroscopically in the flexed position). Racehorses presented most often at 2 years of age, having trained or raced, whereas nonracehorses presented most often as yearlings before training (Table 4) (McIlwraith, 1991). The age range was from yearling or less up to 14 years of age. Lesions of OCD of the distal articular surface of the tibia have been reported at postmortem in a 3-day-old foal euthanized for neonatal maladjustment syndrome (Rejno and Stromberg, 1978).

**Table 4.** Age of racehorses and nonracehorses at time of presentation with tarsocrural osteochondritis dissecans (McIlwraith et al., 1991).

| *Type* | *Age (years)* | *Number* | *Percentage* |
|---|---|---|---|
| Racehorse | 1 | 34 | 22.1 |
| | 2 | 68 | 44.2 |
| | 3 | 36 | 23.4 |
| | 4 | 8 | 5.2 |
| | 5 | 4 | 2.6 |
| | 6 | 1 | 0.6 |
| | 7 | 2 | 1.3 |
| | 9 | 1 | 0.6 |
| Nonracehorse | 1 | 33 | 46.5 |
| | 2 | 18 | 25.3 |
| | 3 | 6 | 8.5 |
| | 4 | 6 | 8.5 |
| | 5 | 1 | 1.4 |
| | 7 | 1 | 1.4 |
| | 8 | 1 | 1.4 |
| | 9 | 1 | 1.4 |
| | 10 | 1 | 1.4 |
| | 13 | 2 | 2.8 |
| | 14 | 1 | 1.4 |

The radiographic manifestations depend on the location of the lesions. In a series of 318 joints, lesions were seen most frequently in the intermediate ridge of the distal tibia, followed by the lateral trochlear ridge of the talus and the medial malleolus, respectively (Table 5) (McIlwraith, 1991). Lesions were also seen in multiple sites in 22 joints, and loose bodies were present in eight joints. Five of these had separated from intermediate ridge lesions, and three had separated from lateral trochlear ridge lesions. The lesions on the distal intermediate ridge of the tibia commonly consist of separation of a bony fragment from the dorsal aspect of the intermediate ridge and are best demonstrated on the dorsomedial-plantarolateral oblique radiograph. OCD lesions of the intermediate ridge of the tibia have been rated on a scale of 0 to 5, according to the defects and the presence and size of the fragments within them (Hoppe, 1984b). Most of the author's surgical cases are either grade 4 or grade 5 (some grade 3) with this classification, and Hoppe grade 1 and 2 lesions (defect but no fragment) are rare, at least in cases with clinical signs. In a separate study, intermediate ridge lesions were classified into three sizes to evaluate the possibility that the size of the fragment affects the prognosis (McIlwraith, 1991). Fragment size did not influence prognosis, and the usefulness of such a grading system is questionable. Lesions on the lateral trochlear ridge are best demonstrated with dorsomedial-plantarolateral oblique radiographs.

**Table 5.** Location of osteochondritis dissecans lesions in 318 tarsocrural joints (McIlwraith et al., 1991).

| *No. 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 |
| Total 318 | |

The lesions may consist of areas of lucency in the bone with or without osseous flaps or fragments visible on the radiographs. Loose bodies may be quite remote and on the medial side of the joint. Radiographs may not accurately depict the amount of articular cartilage dissection extending beyond the subchondral bone defect in some lateral trochlear ridge lesions (McIlwraith, 1991). Lesions of the medial malleolus of the tibia may be demonstrated with a dorsoplantar or dorsolateral-plantaromedial oblique radiograph. These lesions are depicted relatively accurately by radiographs. Lesions of the trochlear ridge of the talus may be demonstrated with dorsolateral-plantaromedial oblique or lateromedial radiographs.

A longitudinal study of 77 Standardbred foals examined and radiographed six times from birth to the age of 16 months provides information on the timing of the development of radiographic lesions (Sandgren, 1988). Eight horses (10.4%) showed lesions of OCD in the tarsocrural joints at the age of 12 months (considered to have permanent OCD). These eight horses all showed abnormal ossification and/or OCD before 3 months of age, and in four of these the lesions were present before 1 month of age. At the sites of predilection for hock OCD the authors also recognized abnormal endochondral ossification of the subchondral bone that reverted to normal in 11 other horses. All of these were radiographically normal after the examination at 7 or 8 months, and there were no other lesions at examination at 16 months. In another study in Norway, radiographs were taken of the tarsocrural joints in 753 Norwegian Standardbred trotters, all yearlings (Grondahl, 1991). OCD lesions of the intermediate ridge of the distal tibia and/or the lateral trochlear ridge of the talus were diagnosed in 108 (14.3%) horses. The lesional changes were bilateral in 49 (45.4%) affected horses. Radiographs were repeated in 79 horses after 6 to 18 months and revealed OCD in only one additional joint. No clinical evaluation was reported in this latter study.

Lesions that were not apparent on radiographs may also be identified during arthroscopy. In one study, in 13 joints OCD lesions were present at arthroscopy without being identified by radiographic examination (McIlwraith, 1991). In four of these cases, there was synovial effusion without radiographic change in the joint contralateral to the one with the radiographic lesion (three on the distal intermediate ridge and one on the medial malleolus). In nine other cases, the lesions (four medial malleolus, three lateral trochlear ridge, and two medial trochlear ridge) were found during arthroscopy of a joint with other radiographically apparent lesions. Loose bodies were detected by different radiographic views, depending on their location. It is also important to recognize that OCD can be diagnosed frequently on radiographs when no clinical signs are present (Hoppe, 1984a; Sandgren, 1988; Alvarado et al., 1989; Grondahl, 1990; Carlsten et al., 1993). Distinction of these cases from ones with clinical signs is important when assessing the need for surgery or the results of conservative treatment (Hoppe, 1984a; Alvarado et al., 1989; Carlsten et al., 1993; Laws et al., 1993; Beard et al., 1994).

In hocks, observable radiographic changes that are not lesions of OCD include spurs or fragments (dewdrop lesions) of the distal end of the medial trochlear ridge of the talus, an irregularly shaped depression (synovial fossa) in the central region of the intertrochlear groove of the talus, and a degree of flattening of the medial trochlear ridge centrally that may be seen particularly in heavy horses (Shelley and Dyson, 1984). Separated OCD fragments can occasionally lodge in the proximal intertarsal joint.

*TREATMENT*

The need for surgery on individual cases of OCD of the tarsocrural joint is still questioned by some, but the literature supports a surgical approach (Birkeland and Haakenstad, 1968; DeMoor et al., 1972; Stromberg and Rejno, 1978; McIlwraith, 1991). In a study comparing 25 horses treated conservatively with 23 horses operated on with arthrotomy, it was concluded that lesions of the hock were of clinical significance and that surgical removal of the fragment seemed to give a better result than conservative treatment (Stromberg and Rejno, 1978). Hoppe found that horses affected with OCD seemed to have a poorer performance capacity than normal horses, but their performance was improved by surgical treatment (1984a). One reason for a discrepancy in opinions is that some radiographic surveys are without any clinical data (Alvarado et al., 1989; Grondahl, 1991).

When clinical signs are present, surgical treatment is preferred, particularly if an athletic career is planned (McIlwraith, 1991). Arthroscopic surgery is used, and follow-up results support its value (McIlwraith, 1991; Beard et al., 1994). It is recognized that some horses have had full athletic careers despite lesions being present radiographically, and it is presumed that the lack of clinical signs is associated with some form of stability between the lesion and parent bone. In contrast, horses often develop problems when in training, and lameness is a factor in many of these cases. Resolution of synovial effusion is also of particular importance to nonracehorse owners. Case selection is important, however. The presence of radiographic changes in the distal tarsal joints (such changes are seen quite often) should be noted when prognosis is discussed. As mentioned above, dewdrop lesions or the presence of calcified fragments at the distal end of the medial trochlear ridge of the talus are not indications for surgery, as they are usually extra-articular. If a free OCD fragment is present in the proximal intertarsal joint, then removal is indicated. Lateral malleolus fragments are usually traumatic in origin and are rarely a manifestation of OCD.

Arthroscopic surgery provides definite advantages over arthrotomy, and techniques have been described extensively elsewhere (Martin and McIlwraith, 1985; McIlwraith, 1991). The overall functional ability and cosmetic appearance of the limbs are excellent. In a study in which postsurgical follow-up was obtained for 183 horses, 140 (76.5%) raced successfully or performed their intended use after surgery (McIlwraith, 1991). Of the remaining 43, 11 were considered to still have a tarsocrural joint problem. Nineteen developed other problems precluding successful performance. Eight were considered poor racehorses without any lameness problems identified, three were killed because of septic arthritis, and two died from other causes. There was no effect of age, sex, or limb involvement on the outcome. The success rate relative to location of the lesion was 139 of 177 (78.5%) for the distal intermediate ridge of the tibia, 24 of 31 (77.4%) for the lateral trochlear ridge of the talus, 7 of 9 (77.8%) for the medial malleolus of the tibia, 3 of 3 (100%) for the medial trochlear ridge of the talus, and 17 of 22 (77.3%) pooled for multiple lesions (no significant differences). The success rate relative to the three size groups for intermediate ridge lesions was 27 of 33 (81.8%) for lesions 1 to 9 mm in width, 86 of 116 (74.1%) for lesions 10 to 19 mm in width, and 41 of 47 (87.2%) for lesions 20 mm or greater in width.

When the success rate was considered relative to the findings of additional lesions at arthroscopy, 16 of 19 (84.2%) with articular cartilage fibrillation, 5 of 10 (50%) with articular cartilage degeneration or erosion, 3 of 5 (60%) with loose fragments, 0 of 2 with proliferative synovitis, and 0 of 1 with joint capsule mineralization were successful. There was a significantly inferior outcome in racehorses with articular cartilage degeneration or erosion ($p < 0.05$). The presence of articular cartilage fibrillation did not affect the prognosis. The results with proliferative synovitis and joint capsule mineralization were poor, but there were insufficient numbers to determine the significance.

Follow-up data on the degree of synovial effusion resolution were obtained for 217 joints that had effusion preoperatively (McIlwraith, 1991). The synovial effusion resolved in 117 of 131 racehorse joints (89.3%) and 64 of 86 nonracehorse joints (74.4%). Of the 22 nonracehorse joints in which resolution did not occur, the owner calculated that 75% resolution had occurred in 12 and 50% resolution had occurred in another four. The resolution of synovial effusion was also documented relative to the location of the lesion. The outcome for synovial fluid resolution was significantly inferior ($p < 0.05$) for lesions of the lateral trochlear ridge of the talus or medial malleolus of the tibia compared with lesions of the distal intermediate ridge of the tibia.

There was no relationship between postoperative performance and the resolution of effusion (McIlwraith, 1991). In 165 horses in which effusion was resolved, 141 (85.4%) raced or performed successfully. Of the 30 horses in which effusion was not resolved, 25 (83.3%) raced or performed successfully. Five horses that had OCD in the tarsocrural joint also had proximal plantar lesions of the first phalanx (four had successful results and one was lost to follow-up). Two horses had lesions of the lateral trochlear ridge of the femur (one was successful and one was lost to follow-up). One horse had a proximodorsal lesion of the distal sagittal ridge, and no follow-up was available.

Recently the results of treatment of 64 Thoroughbreds and 45 Standardbred horses treated for OCD of the tarsocrural joint with arthroscopic surgery before 2 years of age were reported, and the results were compared with those of other foals from the dams of the surgically treated horses (siblings) (Bears et al., 1994). Racing data, including the number of starts and money won during the 2 and 3-year-old racing years, were obtained for affected horses and their siblings. Statistical analysis was performed to test the hypothesis that there is no difference between the racing performance of horses with OCD of the tarsocrural joint that have been surgically treated by means of arthroscopic removal of the fragments before racing and that of their siblings. In 109 horses, 174 lesions were recorded. The distribution of lesions was similar to that previously reported (McIlwraith, 1991). For the Standardbreds, 22% of those that had surgery raced as 2-year-olds and 43% raced as 3-year-olds, compared with 42 and 50% of the siblings that raced as 2- and 3-year-olds, respectively. For the Thoroughbreds, 43% of those that had surgery raced as 2-year-olds and 78% raced as 3-year-olds, compared with 48% and 72%

of the siblings that raced as 2- and 3-year-olds, respectively. The median number of starts for surgically treated horses was decreased compared with the median number of starts for siblings for all groups except 3-year-old Thoroughbreds. Median earnings were lower for affected horses than for siblings for both breeds and both age groups. Among affected horses, the ability to start at least one race was not associated with lesion location or unilateral versus bilateral involvement. There was a tendency for horses with multiple lesions to be less likely to start a race than horses with only a single lesion; however, the difference was significant only for 2-year-old Standardbreds. Affected Standardbreds and Thoroughbreds were less likely to race as 2-year-olds than were their siblings (Beard et al., 1994). The authors noted that although the percentage of horses that raced was lower than that previously reported (McIlwraith, 1991), it was inappropriate to compare this study with previous studies because selection criteria and control groups were different and racing performance was not analyzed by year in previous studies. In the previous studies, older horses that had already raced were included. For other performance-limiting injuries such as apical sesamoid fractures, the prognosis after surgical treatment is better for horses that have already proved themselves capable of racing than for horses that have never raced (Spurlock and Gabel, 1983). The authors recognized the stringent definition for outcome in that horses that lived to racing age and did not compete were counted as failures, regardless of whether the reasons the horses did not compete were related or unrelated to the surgery. The authors stated that they currently recommended removal of any osteochondral fragment associated with joint effusion but warned owners that affected foals may already have or may develop other orthopedic conditions that could limit their performance. In another study it was shown that horses treated for osteochondrosis of the cranial intermediate ridge of the tibia performed as well as matched controls (Laws et al., 1993).

## Osteochondritis Dissecans of the Metacarpophalangeal and Metatarsophalangeal Joints

There is some divergence of opinion as to what is considered OCD within the fetlock and also those entities that might be considered appropriate to include within developmental orthopedic disease (Yovich et al., 1985; Barclay et al., 1987; Foerner et al., 1987; McIlwraith and Vorhees, 1990; Grondahl, 1992b; McIlwraith, 1993b) The following conditions should be addressed:

1. *OCD of the dorsal aspect of the distal metacarpus and metatarsus.*

   It is undisputed that this is a manifestation of OCD (Yovich et al., 1985; McIlwraith, 1987; McIlwraith and Vorhees, 1990). The condition was initially described as OCD of the sagittal ridge of the third metacarpal and metatarsal bones (Yovich et al., 1985), but the term has been modified after recognition that the disease process commonly extends onto the condyles of the metacarpus and metatarsus (McIlwraith and Vorhees, 1990). In one radiographic study, OCD changes in the dorsal aspect of the sagittal ridge of the third metacarpus or metatarsus were seen in 118 of 753 yearling Standardbred trotters, with 61 forelimbs and 147 hind limbs affected (Grondahl, 1992b). In a second study in which horses were evaluated and treated on the basis of having clinical signs, the problem was assessed in 65 horses (McIlwraith and Vorhees, 1990). These lesions usually involve the proximal aspect of the distal dorsal metacarpus or metatarsus. In some instances the most distal aspect of the metacarpus or metatarsus is involved (McIlwraith and Vorhees, 1990). When this is the case, the lesion is within the metacarpophalangeal or metatarsophalangeal articulation.

2. *Proximal palmar or plantar first-phalanx fragments.*

Bony fragments associated with the palmar or plantar part of the metacarpo- and metatarsophalangeal joints were first described in 1972 by Birkeland (Birkeland, 1972). Opinions differ as to whether these fragments are the results of fractures (Birkeland, 1972; Pettersson and Ryden, 1982; Bukowiecki et al., 1986) or osteochondrosis (Foerner et al., 1987; Roneus and Carlsten, 1989; Nixon, 1990). Because follow-up radiographic examination showed that such fragments seldom develop in horses beyond 1 year of age, it was considered that this condition is a manifestation of developmental orthopedic disease (Grondahl, 1992b; Carlsten et al., 1993). More recent studies suggest that although these fragments do indeed show up in young horses, they are the results of a traumatic avulsion associated with the short distal sesamoidean ligament (Dalin et al., 1993). Lameness caused by the bony fragments has been reported to be evident only at the horse's maximal performance (Barclay et al., 1987; Foerner et al., 1987; McIlwraith, 1990) and some fragments at this site do not cause lameness (Barclay et al., 1987; Hardy et al., 1987; Grondahl, 1991). In one radiographic study, these fragments were observed in the palmar or plantar aspect of the metacarpo- and metatarsophalangeal joints in 89 of 753 (11.8%) yearling trotters (Grondahl, 1992b). Fragments were recorded in 7 forelimbs and 86 hind limbs, and bilateral occurrence was observed in the hind limbs of 11 horses. Eleven of 77 foals developed palmar or plantar fragments in another study (Carlsten et al., 1993).

3. *Proximodorsal first-phalanx fragments.*

These fragments, at least in racehorses, have long been considered traumatic in origin and to cause lameness (Yovich and McIlwraith, 1986; McIlwraith, 1987). One group has proposed that these fractures in Thoroughbred racehorses are manifestations of osteochondrosis (Krook and Maylin, 1988), but this is not generally accepted, at least in Thoroughbreds. However,

> dorsal bony fragments in the metacarpo- and metatarsophalangeal joints were diagnosed in 36 of 753 (4.8%) yearling Standardbred trotters in a radiographic survey (Grondahl, 1992b); 11 horses had two affected joints, and 1 horse had three affected joints. The condition was seen in 35 forelimbs and 14 hind limbs. The author also considered these to be manifestations of developmental orthopedic disease. Similar fragments may be found in warmblood horses as well, and some of these fragments could be osteochondrosis-related. The majority of clinical conditions, however, are considered to be traumatic in origin.

The fourth condition that has been labeled as OCD is the condition that was initially described as OCD of the palmar metacarpus (Hornof et al., 1981). This condition is now generally accepted to be a traumatic entity and not a syndrome of osteochondrosis (Pool and Meagher, 1990).

## Osteochondritis Dissecans of the Dorsal Aspect of the Distal Metacarpus and Metatarsus

### *INCIDENCE, CLINICAL SIGNS, AND DIAGNOSIS*

Figures on the incidence of this condition are mentioned in the previous section. Synovial effusion is usually the first indication of a problem. The degree of associated lameness varies, but flexion of the fetlock usually provokes lameness (Yovich et al., 1985; McIlwraith and Vorhees, 1990). Confirmation of OCD is made by radiography. If OCD is diagnosed in one fetlock, the other three are radiographed, because clinically silent lesions are commonly found. Although there may be no synovial effusion in these latter joints and lameness is inapparent, a positive response is often induced with flexion.

For purposes of treatment decision and prognosis, the lesions have been divided into three types: Type I is that in which a defect or flattening is the only visible radiographic lesion; Type II is that in which a defect or flattening with fragmentation is associated with the defect; Type III is that in which there is a defect or flattening with or without fragmentation plus one or more loose bodies.

Oblique radiographs should be taken as well as dorsopalmar (-plantar) and lateral radiographs for the purposes of discerning involvement of the medial or lateral condyles of the distal metacarpus or metatarsus (McIlwraith and Vorhees, 1990).

### *TREATMENT*

When this condition was first reported, there were eight horses in the series (Yovich et al., 1985). Two horses with Type II OCD were euthanized, four horses with

Type I OCD were treated conservatively, one horse with Type II OCD was treated conservatively, and one horse with Type II OCD was operated on arthroscopically. Based on these small numbers, a working hypothesis was made that if the defects are without fragmentation (Type I lesion), conservative treatment will generally be successful. In contrast, it was hypothesized that defects with fragmentation need surgery. This hypothesis has turned into our current recommendations for treatment based on follow-up data (McIlwraith and Vorhees, 1990).

Of 15 horses with Type I lesions that were treated conservatively, 12 resolved clinically, and 8 of these showed remodeling of the lesions with improvement on radiographic examination. In 3 horses, the clinical signs persisted. In 2 of these cases, the radiographs showed no change and the horses eventually underwent surgery. In the other case, the clinical and radiographic signs progressed but the horse was not operated on. In 8 horses with Type II lesions in which owners requested conservative management, 2 eventually underwent surgery because of the persistence of clinical signs. Clinical signs persisted in 5 other horses, but surgery was not performed. The clinical signs improved in only 1 horse. In most of the cases in which clinical signs persisted, the fragmentation also progressed radiographically or at least did not resolve. It was also clear in this study that clinical signs of effusion may appear before definitive radiographic changes. Progression of some Type I lesions was noted. Such joints do not develop osseous fragmentation, but the lesions progressed to become larger defects, particularly on the condyles (seen on oblique-view radiographs). A few cases of Type II lesions improved radiographically. These were generally joints with small fragments, and the fragment fused in place, resulting in a bony protuberance at this location. In the above group of conservatively managed horses, most horses were 1988 foals. At that time, the horses on the farm were followed radiographically without any particular management change. In 1989, creep feed was discontinued in foals in which any swelling developed, and this was successful in reducing problems. During 1990, the energy intake was routinely restricted, with an apparent decrease in problems.

Surgery is usually recommended for Type II or Type III lesions. Most of the cases in a series of 42 horses operated on with arthroscopic surgery and previously reported were Type II or Type III lesions (McIlwraith and Vorhees, 1990). Some Type I lesions were operated on if they had not responded to conservative management. In other instances, Type I lesions were operated on in individual joints if a Type II or Type III lesion was present in another fetlock joint in the same horse. This was before our retrospective data with conservative cases recognized that Type I lesions do not usually require surgical treatment. The technique for arthroscopic surgery for the treatment of this condition has been described elsewhere (McIlwraith, 1990). The series of 42 horses previously reported included 20 Thoroughbreds, 8 Quarter Horses, 7 Arabians, 4 warmbloods, 1 Standardbred, 1 Percheron, and 1 Appaloosa (McIlwraith and Vorhees, 1990). There were 18 fillies, 15 colts, and 9 geldings. The forelimbs were involved in 10 horses, the hind limbs in 15, and both forelimbs and hind limbs in 17. Surgery was done on one fetlock in 10 horses, two fetlocks in 17, three fetlocks in 1, and four fetlocks in 14. In 48 joints, the proximal 2 cm of the sagittal ridge was involved, whereas in 11 joints the lesions extended distal for more than 2 cm. In 14 joints, the lesions involved the lateral or medial condyles of the metacarpus or metatarsus with or without lesions of the sagittal ridge.

Of the 42 horses operated on, follow-up was obtained in 28 (McIlwraith and Vorhees, 1990). Eight horses were convalescing, and in six horses follow-up was unavailable. Surgery was successful in 16 horses (57.1 %) and unsuccessful in 12 horses (42.8%). Of the 12 unsuccessful cases, 7 horses were considered still to have a problem in the fetlock (25%); in 3 horses, treatment was unsuccessful because of other reasons; in 1 horse, treatment was unsuccessful for unidentified reasons but the fetlock joint was considered to be normal; and 1 horse died. The success rate was found to be related to certain other factors. There was a trend for the success rate to be higher for surgery in hindlimbs than in forelimbs ($p = 0.09$). The lack of statistical significance in some instances is probably related to low overall numbers. In the forelimbs only 2 cases were successful, whereas 6 were unsuccessful. In the hind limbs 7 cases were successful and 3 were unsuccessful. When both forelimbs and hind limbs were involved, there were 7 successes and 3 failures. Type III lesions had 4 successes and 4 failures, whereas Type II lesions had 10 successes and 4 failures. The difference, however, was not statistically significant ($p = 0.25$). There was no statistical difference between proximal and distal lesions. In contrast, there were statistical differences in the success rate depending on whether there was articular cartilage erosion or wear lines on the articular surfaces. Only 3 of 12 cases with erosions or wear lines were successful, whereas 13 of 16 with no erosions were successful ($p = 0.0029$). There was also a significantly inferior result when a defect was visible on the condyle on oblique radiographs. When a defect was visible, 6 of 13 were successful, whereas if a defect was not visible, 10 of 15 were successful ($p = 0.0274$). Osteophytes were also negative prognostic indicators: 3 of 9 with osteophytes present on the first phalanx were successful, whereas 13 of 19 with no osteophytes were successful ($p = 0.1792$).

It was concluded that surgical management of Type II and Type III lesions will allow athletic activity in most cases, but clinical signs will persist in 25%. Whether surgery will be successful or not will be affected by the extent of the lesions as evident arthroscopically (and in some instances radiographically), as well as the presence of osteophytes and the presence of erosion and wear lines.

## Proximal Palmar or Plantar First-Phalanx Fragments

### *INCIDENCE, CLINICAL SIGNS, AND DIAGNOSIS*

Two types of fragments have been described: (1) Type I osteochondral fragments of the palmar or plantar aspect of the first phalanx (Barclay et al., 1987; Foerner et al., 1987), also called bony fragments of the palmar or plantar part of the metacarpo- and metatarsophalangeal joints (Grondahl, 1992b), and (2) Type II osteochondral fragments of the palmar or plantar aspect of the fetlock joint (Foerner et al., 1987) also called ununited proximoplantar tuberosity (Grondahl, 1992a) or united plantar eminence (Carlsten et al., 1993) of the proximal phalanx. As discussed previously, these fragments have been found frequently on radiographs of yearling trotters (Grondahl, 1992b).

Type I fragments usually occur in the hind fetlock joints, and the consistent complaint is that the horse has a hind limb problem that occurred at the upper level of the horse's performance ability and prevented the horse from competing successfully. Metatarsophalangeal joint distension is uncommon (Barclay et al., 1987; Foerner et al., 1987). There may be a response to flexion tests, and intra-articular anesthesia usually eliminates the existing lameness or response to flexion. Fragments are best demonstrated on lateromedial oblique and dorsal 20° proximal 75° lateral-plantarodistomedial views. Dorsoplantar radiographs may also demonstrate the fragments. In one series of clinical cases the fragments were most commonly seen medially (Table 6).

**Table 6.** Location of proximal palmar (plantar) fragments of first phalanx in a clinical series of 119 horses (146 joints) operated on arthroscopically (Fortier et al., 1995).

| *Limb* | *Medial* | *Lateral* | *Total* |
|---|---|---|---|
| Left rear | 72 | 20 | 92 |
| Left fore | 5 | 1 | 6 |
| Right rear | 42 | 21 | 63 |
| Right fore | 1 | 2 | 3 |
| Total | 120 | 44 | 164 |

Lameness may develop in association with Type II fragments but is uncommon. These fragments are easily recognized on conventional oblique radiographs (Grondahl, 1992a).

In a longitudinal study of 77 Standardbred foals examined and radiographed six times from birth to the age of 16 months, 11 foals (14.3%) showed either palmar (or plantar) fragments (or bony defects greater than 5 mm at the site of attachment of the short sesamoidean ligaments to the proximal phalanx) or ununited palmar (or plantar) eminences of the proximal phalanx (Carlsten et al., 1993). At four or more examinations from birth to 16 months, some were considered to have permanent lesions. All of these 11 foals had the lesions identified before the age of 5 months and six before the age of 3 months. In seven horses, early radiographic changes reverted to a normal appearance before the age of 8 months. It was noted that the extra-articular osteochondral fragments of ununited proximal and united plantar eminences cannot be considered permanent until after the age of 1 to 2 years because these fragments may unite to the proximal eminence of the proximal phalanx after 2 years of age, but in such cases early signs of unification are seen after 12 months of age.

*TREATMENT*

If Type I osteochondral fragments are incidental findings at radiography, treatment is not usually indicated. To be considered a surgical candidate, the patient must have demonstrable lameness referable to the fetlock in addition to a radiographically demonstrable lesion. In these cases, arthroscopic surgery is an effective method of treatment (Foerner et al., 1987; McIlwraith, 1990) In one series of 19 horses, 10 were treated with arthrotomy and all of these returned to full use of the joint (Foerner et al., 1987). Seven horses were treated intra-articularly with corticosteroids, and only one of these horses was able to return to full use of the joint. Successful results have been obtained more recently with arthroscopic surgery (Foerner et al., 1987; McIlwraith, 1990; Fortier et al., 1995). In 55 of 87 (63%) racehorses and in 100% of 9 nonracehorses, performance returned to preoperative levels after surgery (Fortier et al., 1995). Standardbred racehorses constituted 109 of the 119 (92%) horses. At surgery, evidence of full-thickness cartilage fibrillation was noticed in nine metatarsophalangeal joints but was not found in any metacarpophalangeal joints. Synovial proliferation in the area of and immediately adjacent to the fragment was recorded in an additional four metatarsophalangeal joints. A significant ($p < 0.0001$) association between abnormal surgical findings and unsuccessful outcome was found with 10 of 32 (31%) unsuccessful horses with evidence of articular cartilage loss or synovial proliferation. Only 1 of 55 (2%) successful horses had synovial proliferation evident at surgery, and none had evidence of articular cartilage damage (Fortier et al., 1995). All osteochondral fragments removed in this study were Type I fragments.

With Type II osteochondral fragments (or ununited proximoplantar tuberosity [UPT] of the proximal phalanx), surgery is rarely indicated. UPT was seen radiographically in 18 (2.4%) of 753 Standardbred yearlings in one report (Grondahl, 1992a). All fragments were in the pelvic limb. The condition was seen laterally in 16 horses, whereas one horse had a medial and lateral tuberosity affected and another only one medial tuberosity. Lameness was not observed in any horse before the first examination. On follow-up examination, 12 UPTs in 11 horses had united to the proximal phalanx after 6 to 12 months. One horse was unchanged at 7 months, and the remaining four had a radiographic worsening of the condition, with the UPT more dislocated. Three of these four horses also had calcification of the distal sesamoidean ligaments and periosteal proliferation. Two of the horses with the most severe radiographic changes developed lameness and subsequently underwent surgery to remove the fragment. This gives an incidence of clinically significant disease for UPT in 2 of 16 horses (12.5%) diagnosed and followed. It is also to be noted that in 11 of 18 horses, Type I osteochondral fragments of the plantar part of the metacarpophalangeal joint were seen together with UPT in the same pelvic limb. Occurrence of the latter condition may be an indication for surgery. A common etiologic factor could explain the incidence of the simultaneous occurrence of these two conditions. It has been proposed that clinical signs in conjunction with a UPT may have been caused by tension on the distal sesamoidean ligaments with training (Grondahl, 1992a). Wear and tear of the attachment of these ligaments was considered to possibly stimulate dislocation of the fragment, ligamentous calcification, or periosteal proliferation, and the author therefore recommended restricted training of horses with radiographic evidence of the disease (Grondahl, 1992a). The author also recommended that owners of these horses have them radiographed regularly (every 4 months) and consider surgery if radiographic or clinical evidence indicates progression of the condition. Such cases are unusual.

## Osteochondritis Dissecans of the Scapulohumeral Joint

OCD of the shoulder is the most severely debilitating form of OCD seen in the horse. It is, however, less common than the previously discussed entities. Primary lesions of OCD occur on the glenoid as well as the humeral head, and the disease often affects a major part of the joint surfaces. Severe diffuse OCD lesions as well as single or multiple cystic lesions may occur on the glenoid. Secondary degenerative joint disease was recorded in 35 of 54 cases (Nyack et al., 1981).

### *INCIDENCE, CLINICAL SIGNS, AND DIAGNOSIS*

As mentioned above, OCD of the shoulder is less common than that of the femoropatellar, tarsocrural, or fetlock joints. A series of 54 cases has been reported (Nyack et al., 1981). In a series of 59 joints in 48 horses operated on by the author, there were 19 Quarter Horses, 14 Thoroughbreds, 6 crossbreds, 3 Arabians, 3 warmbloods, 2 Morgans, and 1 American Paint Horse (Howard and McIlwraith, unpublished data). The problem was unilateral in 38 horses and bilateral in 10. The humeral head was involved in 12 horses, the glenoid was involved in 11, and both the humeral head and the glenoid were involved in the 26 other joints that had arthroscopic surgery.

Most cases of OCD of the shoulder present as yearlings or younger (it has been reported at 3 months) and manifest with a history of intermittent forelimb lameness of insidious onset. The forelimb lameness often exhibits a swinging component, with reduced limb protraction a common finding. It is common to see muscle atrophy over the shoulder, and pain may be demonstrated by using direct pressure over the joint or by pulling the leg upward and craniad, caudad, or into an adducted position. Stumbling may occur as a result of inadequate foot clearance and the shortened anterior phase of stride. A small foot with a long heel and club-footed appearance often develops in the affected limb because of the altered gait. Synovial effusion cannot usually be detected because of the muscles and tendons overlying the scapulohumeral joint. When chronicity is evidenced by a smaller foot and when there is muscle atrophy over the shoulder, we consider the presentation of a horse 1 year old or younger for forelimb lameness to be sufficient reason for taking standing radiographs of the shoulder.

The problem may be localized to the shoulder using intra-articular analgesia. This diagnostic aid is important, as the condition can often be diagnosed definitively only by taking radiographs with the horse under general anesthesia. A 3-inch, 18-gauge spinal needle is used for intra-articular analgesia of the shoulder. The needle is inserted cranial to the infraspinatus tendon at the level of the greater tuberosity of the humerus. The needle is inserted slightly caudad and ventrad. Mepivacaine or lidocaine 2% (20 ml) is injected. A 100% response to the block is not necessary to consider the test positive. It is relatively common for an OCD lesion in the shoulder to have intact cartilage at the surface and a dissection plane with subchondral cavitation beneath when it is evaluated arthroscopically. A dramatic response to local analgesia cannot be expected in such cases.

When OCD involves the humeral head, the most common radiographic change is flattening or indentation of the caudal aspect of the humeral head. Lesions in the glenoid manifest either as diffuse areas of subchondral lucency or as cystic lesions (usually multiple). Subchondral bone irregularities are a significant sign in either the humeral head or the glenoid. Lesions may occur in both locations in the same joint. Osteophyte formation (caudal humeral head) is reasonably common, and subchondral sclerosis may also be seen. Cystic lesions in the glenoid have been seen as solitary lesions. Free bony fragments are rare.

### *TREATMENT*

Conservative nonsurgical treatment of osteochondrosis of the shoulder has met with minimal success with respect to athletic performance (Meagher et al., 1973; Nyack et al., 1981; Rose et al., 1985). Animals have been treated successfully with arthrotomy (Schmidt et al., 1975; Mason and McLean, 1977; DeBowes et al., 1982; Nixon et al., 1984). Extensive soft tissue dissection is necessary, however, and the craniomedial aspect of the joint may not be visualized (Schmidt et al., 1975). The development of arthroscopic surgery techniques has provided advantages over arthrotomy in both avoiding these complications and providing additional benefits, particularly improved visualization of the whole joint and a lack of surgical morbidity (Bertone and McIlwraith, 1987a,b; Bertone et al., 1987; Nixon, 1987; McIlwraith, 1990); however, the arthroscopic technique is not easy and becomes extremely difficult in an adult horse.

Because of the generalized pathologic changes present in many instances, surgical cases should be selected carefully. However, surgery will benefit some horses even when secondary degenerative changes are present (Bertone et al., 1987). Although the ability of the young equine joint to heal after curettage of major defects is impressive, we still lack sufficient numbers to give realistic percentages. With very severe cases, a poor prognosis is offered and surgery is not recommended.

At arthroscopic surgery the lesions are usually more extensive than could be surmised from the radiographs (Bertone et al., 1987). In most instances, the cartilaginous changes extend beyond the limits of the subchondral bone abnormalities observed on radiographs, particularly in the glenoid. In some horses in which a lesion is limited radiographically to the glenoid or the humeral head, additional lesions are found arthroscopically on the opposing articular surface. The most common arthroscopic abnormalities of the humeral head are cartilage discoloration with undermining or erosion down to subchondral bone on the caudal aspect of the articular surface. In some instances, a lesion is not visible initially and probing is required to ascertain the area of undermined cartilage. The most common arthroscopic abnormality in the glenoid is cracked and undermined articular cartilage with fissure formation and fibrillation. An additional common finding is defective, friable subchondral bone, and these lesions may extend quite deeply (young horses do have subchondral bone of a softer consistency, and it is sometimes difficult to differentiate pathologic from nonpathologic bone). Problems with arthroscopic surgery in the shoulder include difficulty with arthroscopic placement, difficulty establishing triangulation with the instrument portal, extravasation of fluids, difficulty in reaching potential lesions, and damage to instruments (Bertone et al., 1987; Nixon, 1987).

The results of arthroscopic surgery for OCD and subchondral cystic lesions of the shoulder were initially described for 13 shoulders in 11 horses (Bertone et al., 1987). The lameness decreased in all 11 horses after surgery, with 9 of the 11 horses reported as becoming sound, and 2 remaining lame at short-term follow-up. On long-term follow-up, five horses were athletically sound and were being shown, ridden, or raced after 5 to 20 months. A sixth horse was sound when beginning race training. A seventh horse was pasture-sound and was to begin race training in several months at the time of the report. An eighth horse showed well in halter for 12 months, but shoulder lameness returned; this horse was donated and necropsy was performed. The ninth, tenth, and eleventh horses remained lame. Complications included the development of subchondral cyst-like lesions and signs of degenerative joint disease. Follow-up radiographic assessment of 6 of the 9 sound horses revealed improvement in the contour of the humeral head and joint space and more even density of the humeral epiphysis and glenoid of the scapula in 6 horses. One of these horses showed marked improvement in subchondral bone density and the surface contour of the glenoid cavity. In two of the remaining five horses, the caudal border of the glenoid cavity had remodeled to appear more like the contralateral joint. In the fourth of the six sound horses, radiographs obtained 1 year later showed a subchondral cystic lesion in the bone adjacent to the scapula that had definitely not been present previously, but the horse was still sound and remained so. The contour of the glenoid articular surface on its caudal border was smoother postoperatively, the subchondral osteosclerosis was reduced in thickness, and the horse was athletically sound. In the fifth horse in this group, an osteophyte on the humerus had enlarged, but definite improvement was noted in the joint contour of both the humeral head and the glenoid cavity. Radiographs obtained from one of the two horses that improved but was still lame showed no improvement in the glenoid lesion radiographically. In the horse that deteriorated clinically, in which euthanasia was chosen, the humeral epiphysis was severely deformed with a defect in the articular surface contour, a subchondral cystic lesion, and a small intra-articular fracture of the cranial margin of the glenoid cavity.

A larger, long-term follow-up study has recently been completed. Of 49 horses operated on by the author, complete follow-up was obtained in 35. Sixteen operations were successful (45.7%) and 19 unsuccessful (54.3%). Five additional horses were in various stages of convalescence or training, and nine horses were lost to follow-up. An alternative arthroscopic technique has been reported in nine normal horses and two cases of osteochondrosis (Nixon, 1987).

## References

Adams, W.H., and J.P. Thilsted. 1985. Radiographic appearance of the equine stifle from birth to six months. Vet. Radio. 26:126-132.

Aglietti, P., R. Buzzi, P.B. Bassi, and M. Fioriti. 1994. Arthroscopic drilling in juvenile osteochondritis dissecans of the medial femoral condyle. J. Arthroscopy Rel. Surg. 10:286-191.

Alvarado, A.F., M. Marcoux, and L. Breton. 1989. The incidence of osteochondrosis on a Standardbred breeding farm in Quebec. In: Proc. Amer. Vet. Med. Assoc. Equine Practnr. 35:293-307.

Barclay, W.P., J.J. Foerner, and T.N. Phillips. 1987. Lameness attributable to osteochondral fragmentation of the plantar aspect of the proximal phalanx in horses: 19 cases (1981-1985). J. Amer. Vet. Med. Assoc. 191:855-857.

Beard, W.L., L.R. Bramlage, R.K. Scheider, and R.M. Embertson. 1994. Postoperative racing performance in Standardbreds and Thoroughbreds with osteochondrosis of the tarsocrural joint: 109 cases. J. Amer. Vet. Med. Assoc. 204:1655-1659.

Bertone, A.L., and C.W. McIlwraith. 1987a. Arthroscopic approaches in intra-articular anatomy of the equine shoulder joint. Vet. Surg. 16:317-322.

Bertone, A.L., and C.W. McIlwraith. 1987b. Osteochondrosis of the equine shoulder: Treatment with arthroscopic surgery. In: Proc. Amer. Assoc. Equine Practnr. 33:683-686.

Bertone, A.L., C.W. McIlwraith, B.E. Powers, et al. 1987. Arthroscopic surgery for treatment of osteochondrosis in the equine shoulder joint. Vet. Surg. 16:303-311.

Birkeland, R. 1972. Chip fractures of the first phalanx in the metatarsophalangeal joint of a horse. Acta. Radiol. Suppl. 29:73-77.

Birkeland, R., and L.H. Haakenstad. 1968. Intracapsular bony fragments of the distal tibia of the horse. Equine Vet. J. 152:1526-1529.

Birkeland, R., and L.H. Haakenstad. 1974. Osteochondritis dissecans. Ii heseledtet hos hust. Kirurgiskog konservativ behandling. In: Proc. Nord. Vet. Cong. Reykjavik 34.

Bukowiecki, C.F., L.R. Bramlage, and A.A. Gabel. 1986. Palmar/plantar process fractures of the proximal phalanx in 15 horses. Vet. Surg. 15:383-388.

Carlsten, J., B. Sandgren, and G. Dalin. 1993. Development of osteochondrosis in the tarsocrural joint and osteochondral fragments in the fetlock joints of Standardbred trotters: I. A radiological study. Equine Vet. J. Suppl. 16:42-47.

Dalin, G., B. Sandgren, and J. Carlsten. 1993. Plantar osteochondral fragmentation in the fetlock joints of Standardbreds: Results of osteochondrosis or trauma? Equine Vet. J. Suppl. 16:62-65.

DeBowes, R.M., P.C. Wagner, and B.D. Grant. 1982. Surgical approach to the equine scapulohumeral joint through a longitudinal infraspinatus tenotomy. Vet. Surg. 11:125-128.

DeMoor, A., F. Verschooten, P. Desmet, et al. 1972. Osteochondritis dissecans of the tibiotarsal joint of the horse. Equine Vet. J. 4:139-143.

Dink, D. 1990. Advantages of a February foal. Thoroughbred Times 18:28-32.

Ewing, J.W., and S.J. Soto. 1988. Arthroscopic surgical management of osteochondritis dissecans of the knee. J. Arthroscopic Rel. Surg. 4:37-44.

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

Foland, J.W., C.W. McIlwraith, and G.W. Trotter. 1992. Osteochondritis dissecans of the femoropatellar joint: Results of treatment with arthroscopic surgery. 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. Assoc. 206:71-74.

Grondahl, A.M. 1991. The incidence of osteochondrosis in the tibiotarsal joint of Norwegian Standardbred trotters: A radiographic study. J. Equine. Vet. Sci. 11:272-274.

Grondahl, A.M. 1992a. Incidence and development of ununited proximoplantar tuberosity of the proximal phalanx in Standardbred trotters. Vet. Radiol. Ultrasound 33:18-21.

Grondahl, A.M. 1992b. The incidence of bony fragments in osteochondrosis in the metacarpometatarsophalangeal joints of Standardbred trotters: A radiographic study. J. Equine Vet. Sci. 12:81-85.

Hance, S.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,

Hardy, J., M. Marcoux, and L. Breton. 1987. Prevalence et des fragments articulaires retrouves au boulet chez le chevel Standardbred. Med. Vet. Quebec 17:57-61.

Hoppe, F. 1984a. Osteochondrosis in Swedish horses: A radiographic and epidemiological study with special reference to frequency and heredity. Thesis. University at Uppsala, Sweden.

Hoppe, F. 1984b. Radiological investigations of osteochondrosis dissecans in Standardbred trotters and Swedish Warmblood horses. Equine Vet. J. 16:425-429.

Hornof, W.H., T.R. O'Brien, and R.R. Pool. 1981. Osteochondritis dissecans of the distal metacarpus in the adult racing Thoroughbred horse. Vet. Radiol. 22:98-106.

Howard, R.D., and C.W. McIlwraith. Unpublished data.

Krook, L., and G.A. Maylin. 1988. Fractures in Thoroughbred racehorses. Cornell Vet. 78:5-47.

Laws, E.G., D.W. Richardson, M.W. Ross, et al. 1993. Racing performance in Standardbreds following conservative and surgical treatment for tarsocrural osteochondrosis. Equine Vet. J. 25:199-202.

Martin, G.S., and C.W. McIlwraith. 1985. Arthroscopic anatomy of the equine femoropatellar joint and approaches for the treatment of osteochondritis dissecans. Vet. Surg. 14:99-104.

Mason, A., and A.A. McLean. 1977. Osteochondrosis dissecans of the head of the humerus in two foals. Equine. Vet. J. 9:189-191.

McIlwraith, C.W. 1987. Disease of joints, tendons, ligaments and related structures. In: Stashak, T.S. (Ed.) Adams' Lameness in Horses (4th Ed.). p. 339-447. Lee and Febiger, Philadelphia.

McIlwraith, C.W. 1990. Diagnostic and Surgical Arthroscopy in the Horse. p. 113-159. Lee and Febiger, Philadelphia.

McIlwraith, C.W. 1993a. Inferences from referred clinical cases of osteochondritis dissecans. Equine Vet. J. 16:27-30.

McIlwraith, C.W. 1993b. What is developmental orthopedic disease, osteochondrosis, osteochondritis, metabolic bone disease? In: Proc. Amer. Assoc. Equine Practnr. 39:35-44.

McIlwraith, C.W., and G.S. Martin. 1985. Arthroscopic surgery for the treatment of osteochondritis dissecans of 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 M. Davis. 1991. Osteochondritis dissecans of the tarsocrural joint: Results of treatment with arthroscopic surgery. Equine

Vet. J. 23:155-162.

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

Meagher, D.M., R.R. Pool, and T.R. O'Brien. 1973. Osteochondritis dissecans of the shoulder joint in the horse. In: Proc. Amer. Assoc. Equine Practnr. 19:247-256.

Moore, J.N., and C.W. McIlwraith. 1977. Osteochondrosis of the equine stifle. Vet. Rec. 100:133-136.

Nilsson, F. 1947. Hastens goniter. Sven. Vet. Tidskr. 52:1-14.

Nixon, A.J. 1987. Diagnostic and surgical arthroscopy of the equine shoulder joint. Vet. Surg. 16:44-52.

Nixon, A.J. 1990. Osteochondrosis and osteochondritis dissecans of the equine fetlock. Compend. Cont. Ed. Pract. Vet. 12:1463-1475.

Nixon, A.J., T.S. Stashek, C.W. McIlwraith, et al. 1984. A muscle-separating approach to the equine shoulder for the treatment of osteochondritis dissecans. Vet. Surg. 13:247-256.

Nyack, B., M.B. Morgan, R.R. Pool, et al. 1981. Osteochondrosis of the shoulder joint of the horse. Cornell Vet. 71:149-163.

O'Brien, T.R. 1973. Radiology of the equine stifle. In: Proc. Amer. Assoc. Equine Practnr. 19:271-287.

Pascoe, J.R., R.R. Pool, J.D. Wheat, et al. 1984. Osteochondral defects of the lateral trochlear ridge of the distal femur of the horse: Clinical, radiographic and pathologic examination and results of surgical treatment. Vet. Surg. 13:99-110.

Pettersson, H., and G. Ryden. 1982. Avulsion fractures of the caudoproximal extremity of the first phalanx. Equine Vet. J. 14:333-335.

Pool, R.R., and D.M. Meagher. 1990. Pathologic findings and pathogenesis of racetrack injuries. Vet. Clin. N. Amer. 6:1-30.

Poulos, P. 1986. Radiologic manifestations of developmental problems. In: McIlwraith, C.W. (Ed.) AQHA Developmental Orthopedic Disease Symposium. p. 1-2. American Quarter Horse Association, Amarillo, Texas.

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

Roneus, B., and J. Carlsten. 1989. Bone fragments in fetlock and hock joints in young Standardbred trotters. Sven. Vet. Tidskr. 41:417-422.

Rose, J.A., R.D. Sande, and E.M. Rose. 1985. Results of conservative management of osteochondrosis in the horse. In: Proc. Amer. Assoc. Equine Practnr. 31:617-626.

Sandgren, B. 1988. Bony fragments in the tarsocrural and metacarpo-metatarsophalangeal joints in the Standardbred horse: A radiographic study. Equine Vet. J. Suppl. 6:66-70.

Schmidt, G.B., R. Dueland, and J.T. Vaughan. 1975. Osteochondrosis dissecans of the equine shoulder joint. Vet. Clin. North. Amer. (Small Anim. Pract.)

70:542-547.

Shelley, J., and S. Dyson. 1984. Interpreting radiographs: V. Radiology of the equine hock. Equine Vet. J. 16:488-495.

Smith, J.B. 1990. Osteochondritis dissecans of the trochlea of the femur. Arthroscopy 6:11-17.

Spurlock, G.H., and A.A. Gabel. 1983. Apical fractures of the proximal sesamoid bones in Standardbred horses. J. Amer. Vet. Med. Assoc. 183:76-79.

Steenhaut, M., F. Verschooten, and A. DeMoor. 1982. Osteochondritis dissecans of the stifle joint in the horse. Vlaams Dierg. Tigdchrift. 5:173-191.

Steinheimer, D.N., C.W. McIlwraith, R.D. Park, and P.F. Steyn. 1996. Comparison of radiographic subchondral bone changes with arthroscopic findings in the equine femoropatellar and femorotibial joints: A retrospective study of 72 joints (50 horses). Vet. Radiol. 36:478-484.

Stromberg, C., and S. Rejno. 1978. Osteochondrosis in the horse: I. A clinical and radiological investigation of osteochondritis dissecans of the knee and hock joint. Acta. Radiol. Suppl. 358:139-152.

Trotter, G.W., C.W. McIlwraith, and R.W. Norrdin. 1983. Comparison of two surgical approaches to the equine femoropatellar joint for the treatment of osteochondritis dissecans. Vet. Surg. 12:33-40.

Wright, I.M. and A.C. Pickles. 1991. Osteochondritis dissecans (OCD) of the femoropatellar joint. Equine Vet. Educ. 3:86-93.

Wyburn, R.S. 1977. Degenerative joint disease in the horse. NZ Vet. J. 25:321-335.

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. 188:273-279.

Yovich, J.V., C.W. McIlwraith, and T.S. Stashak. 1985. Osteochondritis dissecans of the sagittal ridge of the third metacarpal and metatarsal bones in horses. J. Amer. Vet. Med. Assoc. 186:1186-1191.