

Developmental Orthopedic Disease

Steve Adair MS, DVM, DACVS

Associate Professor Equine Surgery

I. GENERAL

- A. The phrase developmental orthopedic disease is a nonspecific term that includes:
 1. Osteochondritis dissecans (Osteochondrosis)
 2. Subchondral cystic lesions (Osteochondrosis)
 3. Physitis
 4. Flexural deformities
 5. Angular deformities
 6. Cuboidal bone disease
 7. Juvenile osteoarthritis
- B. Long bones develop from cartilage by a process of endochondral (within cartilage) ossification. The centers of ossification (bone formation) develop in the center of the future long bone (diaphysis) and at the ends of these long bones (epiphysis). As ossification proceeds and a bony epiphysis develop, as does a bony diaphysis. Between these two centers of ossification is a metaphyseal growth plate and this is what enables the limb to lengthen after birth as the foal grows. There is a second growth plate called the epiphyseal growth plate that forms as the epiphyseal ossification center advances toward the ends of the bone and what is destined to be the articular surface of the joints.
- C. Causative Factors of Developmental Orthopedic Disease
 1. General - Certain etiologic factors have been recognized that contribute to the development of these lesions. These factors have been varied, but the idea that there is a multifactorial etiology (a number of causes contributing to it) has generally been accepted. Much of the information is from clinical and pathologic reports, as well as experimental studies in the horse. The experimental studies in the horse have given some answers but have also created confusion. The primary problem has been that the lesions are often somewhat different from what we most

commonly encounter as veterinarians looking at clinical cases. However, we have identified major factors that seem to predispose the growing animal to osteochondrosis type problems, including rapid growth, genetic predisposition, nutritional excesses or imbalances and superimposed trauma on the cartilage. In pigs, it has been demonstrated that a high growth rate is the main reason for high incidence of osteochondrosis and that the high growth rate is the result of both genetic selection and caloric intake. Some genetic predispositions have been demonstrated in the horse and increased energy will increase the incidence of osteochondrosis. However, it is not a simple cause/effect relationship. We will now discuss the various factors that have been implicated in contributing to this complex disease.

2. Genetic predisposition - Radiographic studies in Swedish Trotters and Warmbloods have shown progeny of one stallion from each breed having a significantly high frequency of OCD amongst his progeny, compared with the progeny of the other stallions. In another study in Denmark, radiographic evidence of osteochondrosis was seen in a significantly high proportion of the progeny of one of eight stallions, although the stallion itself did not show radiographic signs of osteochondrosis. There has been little work done in the United States with regard to heredity and we certainly haven't been able to develop any type of screening program for osteochondrosis in stallions and mares that will ensure freedom from that condition. However, it would appear very likely that there are genetic components to this disease. Individual instances of certain stallions and mares producing these individuals have been seen.
3. Growth and body size - Fast growth was implicated with a high incidence of osteochondrosis in dogs and pigs. There have been anecdotal reports of this in the horse. However, the controlled studies that have been done in the horse question whether growth rate is indeed a factor. It has been pointed out

that the most intense phase of growth occurs in the first three months of life. If growth was a big factor, we expect this would be the time that most lesions would occur, but this is not when we see them clinically. At this stage, the evidence implicating growth rate and body size in the pathogenesis is largely unsubstantiated, at least with any definitive studies. Growth rate is obviously associated with a number of factors. In one study, done at Ohio State, foals with a higher number of lesions had similar growth rates to those with fewer or no lesions, suggesting that rapid growth may not be a necessary predisposing factor in the development of cartilage lesions. Growth was based on measurements of body weight with a height in cannon bone circumference and it was part of a study of the effect of dietary copper.

4. Mechanical stress and trauma - It has certainly been recognized that mechanical stresses often precipitate clinical signs with OCD and it is presumed that this is by separating the OCD flap or fragment from the parent bone. Whether trauma or physical stress is involved in the primary induction of an OCD lesion is controversial. However, some people do tend to feel this is the case and we do recognize there are certain predisposing sites for the occurrence of OCD, suggesting possible mechanical factors. A notable veterinary bone and joint pathologist, Dr. Roy Pool, feels that shear forces may disrupt capillaries in the subchondral bone (bone under the cartilage) and give rise to chondrocyte or cartilage cell damage. This is based on histologic observations of various lesions.
5. Nutrition - As discussed previously, the idea of over nutrition as a cause of OCD has been extrapolated from work in dogs and pigs. There has been an increased incidence of OCD lesions noted in horses fed 130% of what the National Research Council (NRC) recommends for carbohydrate and protein. A second study in Australia by Dr. Kate Savage, which was very well controlled, showed that high-energy diets (120% NRC requirements) consistently produced

lesions of osteochondrosis in weanling foals compared to a control diet based on 100% NRC requirements. Some people have focused on "high protein" being a problem but this has not been demonstrated.

6. Mineral imbalances - Various mineral imbalances have been implicated as causative factors with OCD, including high calcium, high phosphorus, low copper and high zinc. Although high calcium levels have been implicated, experimental research in the horse with three times the NRC level of calcium in the diet, failed to produce lesions of osteochondrosis. High phosphorus diets (five times NRC) did produce lesions of OCD in young foals. Low copper has been implicated as a cause. An epidemiologic study on clinical cases of DOD implicated low copper levels as the most consistent factor. In experimental studies, it has been noted that a marked copper deficiency (1.7 ppm - a very artificially low level) produced OCD-like lesions and flexural deformities. In another study in Thoroughbred foals in which osteochondrosis developed before weaning, seven had serum copper and ceruloplasmin concentrations below normal. In a third controlled experiment in Canada with high (30 ppm) and low (7 ppm) copper diets, there was a much higher incidence of lesions seen in the foals fed the low copper diet. However, it is to be noted that most of the changes were present in the cervical vertebrae rather than the limbs where we commonly see clinical problems. Excessive zinc intake has been related to equine osteochondrosis. Generalized osteochondrosis has been seen in foals raised near a zinc smelter. The relationship between zinc and copper (it has been suggested that high zinc suppress copper levels) is still being elucidated.
7. Endocrine Factors - It has been postulated by one investigator that the production of osteochondrosis lesions in association with overfeeding is mediated by the endocrine system. Certainly the long-term administration of dexamethasone has produced osteochondrosis-type lesions and it is considered that

glucocorticoids induced a parathyroid hormone resistance at the level of the osteocyte causing an inhibition of normal remodeling. Glucocorticoids also induced decreased GAG levels and this decrease in turn inhibits capillary penetration of the cartilage, which is a very important step in forming bone from cartilage. The failure of ossification could also be mediated through induced defects in vitamin D metabolism. Corticosteroids are also a potent inhibitor of lysyl oxidase, which is involved in cross-linking of collagen in cartilage and bone. It is felt this could be a way of inducing lesions.

8. Site Vulnerability - Because the lesions of equine osteochondrosis occur at specific anatomic sites, there is a suggestion of site vulnerability. This predilection could be related to an ossification defect or trauma caused by excessive stress in that region. In nearly all instances, the sites of occurrence of OCD are very close to the limits of articulation and we know from basic research that the makeup of the cartilage between articulating and non-articulating surfaces is different. OCD lesions are frequently bilateral in the stifle, hock, and quadrilateral in the fetlock joint, although they frequently involve different joints in the same animal. It is felt this may suggest a "window of vulnerability" in the endochondral ossification of that specific joint when an environmental insult may have occurred. If the causative factor were present intermittently or for a transient period during the foal's growth period, this would explain the development of the disease in only one pair of joints. It is not possible from this data to ascertain different periods of onset of the disease process in different joints.
9. Exercise - Adequate exercise in foals would logically be important for the maintenance of cartilage and bone quality. There is some data suggesting a "protective" effect of exercise. This particular study was done on early, weaned Warmblood foals. There was a dramatic reduction in the incidence of OCD in foals,

subjected to forced exercise and a high-energy diet, compared with foals fed the same diet but with limited exercise.

II. TREATMENT

- A. Medical - Medical therapy consist of nutritional management, pain relief, corrective trimming, exercise modifications, external coaptation and physical therapy.
 - 1. Either one or a combination of the above may be tried.
- B. Surgical - varies with the disease. Arthroscopy, periosteal stripping, transphyseal bridging and check ligament desmotomy are a few examples.

III. DISEASES

- A. Osteochondrosis
 - 1. This is a defect in the normal, endochondral ossification process that can result in a number of different manifestations dependent upon the site of the defect. The prevalence is anywhere from 5-20%. It is commonly seen bilaterally in the contralateral joint but infrequently seen in more than one type of joint (e.g. fetlocks and hocks). Large studies overseas have documented at least a genetic predisposition. Nutritional studies have been able to reproduce forms of the disease in growing foals, but in most cases experimentally produced osteochondrosis is somewhat different from the clinically seen form.
 - 2. Osteochondrosis is most simply thought to cause either cysts or flaps. A cyst can be considered a retained core of cartilage as the bone continues to grow out around it. A flap may start out similarly, but it is more likely a smaller linear area that is affected and a flap result, as the rest of the bone grows. When most people refer to flaps, they will use the term osteochondrosis dissecans (OCD). The inflammation and synovitis often lead to joint distension (effusion), the most common clinical sign of OCD. Subchondral cystic lesions that are bilateral in a young animal

are probably a form of osteochondrosis. However, it has been shown that cysts that develop in adult horses are most likely a result of trauma to the articular cartilage that leads to cyst development.

3. Lameness is a less frequently observed sign of osteochondrosis, and is more often associated with the cyst like lesions. These are also more commonly found on the weight bearing portion of bones, such as the distal medial femoral condyle of the stifle. Osteochondrosis may also be asymptomatic, and the only sign may radiographic abnormalities.
4. Treatment of osteochondrosis is greatly dependent upon the site and type of lesion, the size of the lesion, the clinical effect, the intended use of the horse, and the age discovered. There is rarely one right way to treat a single lesion. Below is an overview of some of the more common forms.
5. Hock Joint
 - a) This is the most common site of osteochondrosis seen. It usually presents with joint distension of the tibiotarsal joint without significant lameness. Clinical signs are usually noted in weanlings or yearlings. The most common site is the distal intermediate ridge of the tibia, and as with many forms of OCD it can often be bilateral. Therefore, if it is found in one hock, the other hock should also be evaluated radiographically whether there is effusion or not. Other sites in the hock include the lateral trochlear ridge, the medial malleolus, and the medial trochlear ridge.
 - b) Surgical treatment is strongly recommended. In most cases, the animal will be able to perform its intended use. This is even more likely than complete resolution of the effusion. The site of the lesion does not have a significant effect on outcome but it does affect the likelihood of resolution of the synovial effusion. Complete resolution of effusion is significantly inferior for lateral trochlear ridge lesions.

6. Stifle Joint

- a) Lateral trochlear ridge flap lesions are commonly seen to cause effusion without significant lameness. Often the radiographic size underestimates the actual degree of bone affected. Arthroscopic surgery allows a more accurate assessment of the bone involved and debridement. Larger lesions generally have a worse prognosis for performance.
- b) The second most common site in the stifle is cyst formation in the medial femoral condyle. These are more likely to be first noticed because of lameness. The size of the lesion may dictate either conservative or surgical management. The prognosis for surgical management is reported to be from 60-75%.

7. Fetlock Joint

- a) Sagittal ridge flap lesions can be seen bilaterally, or in all four fetlocks. Effusion of the joint is most commonly observed. Arthroscopic debridement is usually met with success. Osteochondrosis can also manifest itself as small fragments of the back of the first phalanx. It is very frequently noted in only the hind limbs. These may not even present for effusion, but usually cause mild lameness with a high level of work or racing. Again arthroscopic removal is recommended and met with significant success.

8. Shoulder Joint

- a) This is the most debilitating form of osteochondrosis. It is almost invariably found because lameness develops. The animal will have the classic signs of swinging leg lameness including a significant head nod and shortened stride. It is usually much worse than the radiographs indicate. While conservative management the horse has a guarded prognosis, surgical debridement only improves the prognosis to 50-60% for high level performance.

B. Wobbler

1. A type of osteochondrosis
2. This refers to young, growing horses that demonstrate signs of neurologic disease secondary to spinal cord compression. The horses demonstrate signs of ataxia or "wobbliness." The spinal cord compression is a result of impingement from a narrow or constricted bony canal. The compression can either be considered static (present at all times) or dynamic (occurring only when the animal bends their neck). The disorder is often termed cervical vertebral malformation (CVM).
3. Significant strides have been made in diagnosing the problem early with standing lateral radiographs and adjusting the animal's diet and management to encourage remodeling and greater luminal diameter to the canal. Results are very encouraging, but if the problem is not diagnosed until later in life, diet changes will have little to no effect.
4. Older animals (1-2 yr. of age), that cannot be managed successfully with management changes can consider surgical options. Since many of the areas of compression are dynamic, stabilization of these areas will prevent repeated trauma. Yet, if the damage to the nervous tissue is beyond complete repair, the animal will have residual deficits. Again, early recognition is imperative to increase the odds of success.

C. Physitis

1. This can be considered an overloading of a growth plate that results in pain and inflammation. It has also been inappropriately called epiphysitis, but the physis (growth plate) is the problem area, not the end of the bone (epiphysis). Management includes restriction of exercise, judicious use of anti-inflammatories, and nutritional changes. It may not be very apparent to the casual observer that an animal is suffering from low-grade physitis and it is these cases which may result in other secondary abnormalities.

2. Angular and flexural deformities can be the result.
3. This is usually treated medically.
 - a) Decrease inflammation with the judicious use of NSAID's.
 - b) Slow rate of growth by changing diet
 - c) Insure proper Ca: P ratio and provide source of trace minerals.
 - d) Prevent trauma with controlled exercise.

D. Angular Deformities

1. The most common deformity is carpal valgus. Some valgus is expected in the normal newborn foal. There are multiple factors which result in angular deformities and determination of the cause is pertinent in determining treatment options. With time and exercise, there is a certain amount of self-correction. The amount of remaining self-correction should be considered in the equation when determining if intervention is required. A normal foal should correct to within 5-7 degrees by four months of age, and should be almost straight by 8-10 months of age. Those that are not improving fast enough for growth potential remaining should be considered surgical candidates.
2. The second most common angular deformity is fetlock varus. Because the distal cannon bone physis stops growing much sooner than the distal radius, action must be taken earlier. There is relatively rapid growth in the distal radius physis for 6 months (knee); the distal cannon bone (fetlock) has little growth after three months. It is the remaining growth that we take advantage of with surgical intervention.
3. Surgical options include increasing the growth on the slow side with periosteal transection or slowing the growth on the side that is growing too fast, transphyseal bridging. Periosteal transection is a short, minimally invasive procedure that cannot overcorrect the deformity. However, recently its efficacy has come into question. Transphyseal bridging employs implants to inhibit the physis from growing and requires a second surgical

procedure to remove the implants. If the implants are not removed, over correction may occur.

4. Medical options include corrective hoof trimming, dietary changes, splints and physical therapy.

E. Flexural Deformities

1. These can be congenital or acquired. They also can be categorized as contractual or flexor laxity. Laxity is most often a neonatal problem from soft tissue laxity that will resolve with maturity. Contracture can occur at any age and often worsens if not appropriately addressed.
2. Contracture in the growing animal is often the result of subclinical physitis and the flexors overpowering the extensors. Therefore, the same management changes recommended for physitis can apply in the mild uncomplicated situations of contracture. Oxytetracycline can also be employed to provide relaxation in the younger animals. Splinting and bandaging is a mainstay to management but must be performed carefully to prevent soft tissue injury. If conservative and medical management is not successful, surgical correction should be considered before the deformity worsens. Again, early recognition is critical. A prolonged problem will be more refractory to treatment.
3. Transacting the appropriate check ligaments and allowing the muscle belly to stretch performs surgical correction. Shoeing changes are also made at this time to take advantage of the mechanical release associated with transacting the check ligament.

F. Cuboidal Bone Malformation

1. Cuboidal bone malformation certainly represents a delay in endochondral ossification. Usually it is a result of either prematurity or a delay of ossification caused by hyperthyroidism. Usually the condition manifests as a carpal deformity or a hock deformity because of a collapse of the cuboidal bones in this area. They have collapsed because of insufficient ossification of the bones by the time they are bearing

weight on them. They are treated by casting in a position that takes weight off the bones and the cuboidal nature of the bone can return if treatment is initiated sufficiently early.