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THE EQUINE SKELETON

HOW DOES BONE GROW AND HOW DO ABNORMALITIES IN THE DEVELOPMENTAL PROCESS AFFECT SOUNDNESS?

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Normal long bone development

Long bones develop from cartilage by a process of endochondral (within cartilage) ossification (Fig 1). In the fetus, the bone anlages (early bone templates) are composed entirely of cartilage. Centers of ossification (bone formation) develop in the center of the future long bone (diaphysis) and also at the ends of these long bones (epiphysis). As ossification proceeds, a bony epiphysis develops at each end and a bony diaphysis develops in the center. Between these two centers of ossification is a metaphyseal growth plate (also called physis) and this is what enables the limb to continue to lengthen after birth as the foal grows. Eventually, the epiphyseal center of ossification and the diaphyseal center of ossification will unite and this results in bony closure of the physis.
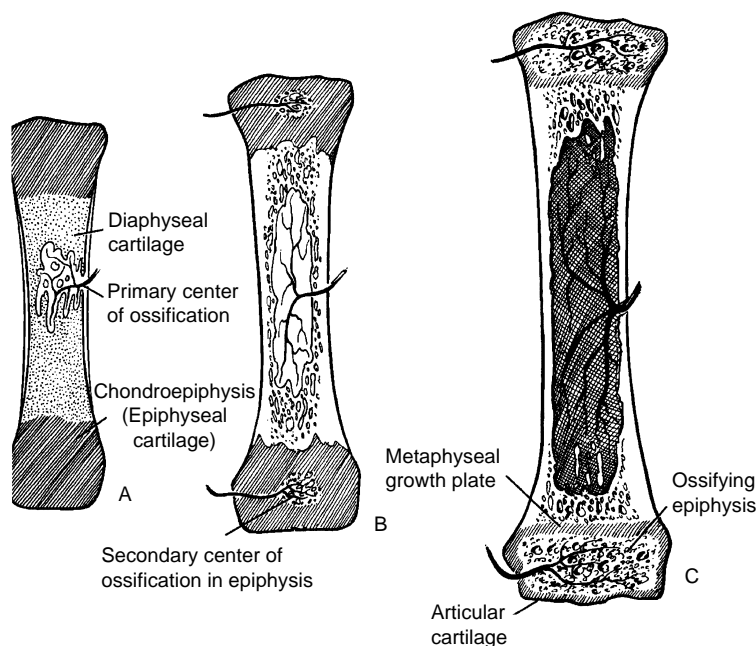

Figure 1. Diagram of ossification of a developing bone

The physes close at various stages. As a generalization, the ones nearest the foot (distal) close before the ones further up the leg (proximal). There is variation within each growth plate as well and knowledge of this growth pattern and closure pattern is critical to manipulation of deformities in the limb.

Ossification of the epiphyses at the ends of the long bones is also critical for an understanding of both joint development as well as defects in this ossification, such as osteochondrosis, the most important condition of which is osteochondritis dissecans (OCD). A diagrammatic representation of the development of the joint is in Figure 2. As can be seen in the diagram, epiphyseal ossification gradually proceeds out and stops at a certain stage. When it ceases, there is a layer of cartilage left at the joint surface called articular cartilage. This should never ossify. Obviously there is physeal cartilage retained as well, up to a certain stage, but this eventually ossifies. Ossification of the epiphysis requires blood vessels and these are provided through cartilage canals. Prior to ossification, the cartilage that contains cartilage canals and blood vessels is called epiphyseal cartilage. Cartilage that remains at the end of ossification and lines the joint is called articular cartilage and is devoid of blood vessels. It also has a very specific structure to provide motion at the joint surface. These aspects will be discussed in more detail when considering OCD.
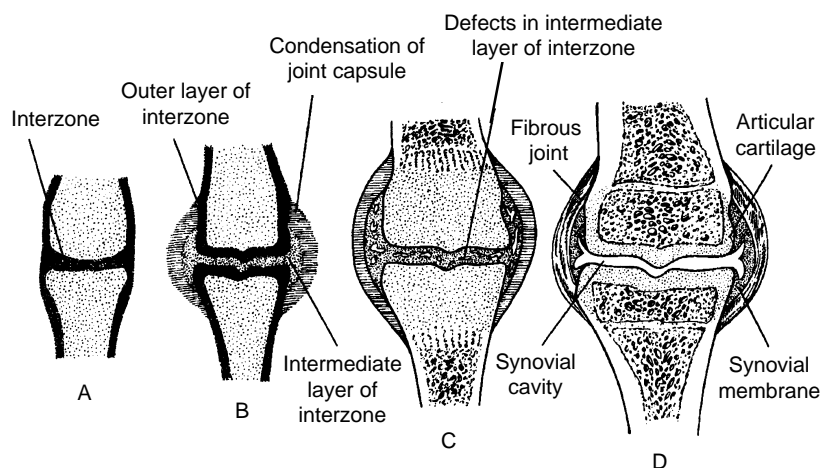

Figure 2. Diagrammatic representation of the development of a joint.

The anatomy of the metaphyseal growth plate or physis is also quite complicated and is illustrated in Figure 3.

Abnormalities of bone and joint development

There are various abnormalities involved in the developmental process. They are generally classified under the term developmental orthopedic disease (see below). These abnormalities may involve defects in endochondral ossification by which the

bone is formed, abnormalities in bone lengthening or metabolic changes within the bone after it is formed, and as the horse goes into training many of these have yet to be identified.
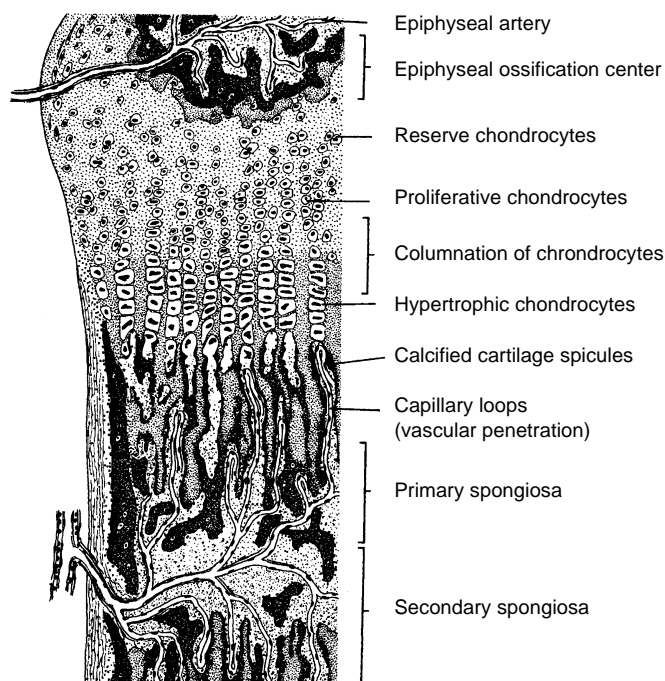

Epiphyseal artery

Epiphyseal ossification center

Reserve chondrocytes

Proliferative chondrocytes

Columnation of chrondrocytes

Hypertrophic chondrocytes

Calcified cartilage spicules

Capillary loops
(vascular penetration)

Primary spongiosa

Secondary spongiosa

Figure 3.

In general, we look at two sets of major orthopedic problems in the horse:

1) developmental problems, discussed under the umbrella of developmental orthopedic disease, and

2) traumatically induced or degenerative problems.

Many of these conditions are only seen at an end stage. For instance, we now recognize that chip fractures in the carpus of the racehorse are secondary to long term degenerative conditions in the subchondral bone. Similarly, stress fractures when they occur in the racing athlete are probably not an acute event but rather a final breakdown in bone structure due to localized osteoporosis associated with bone remodeling.

Developmental orthopedic disease

DEFINITION

The term developmental orthopedic disease (DOD) was coined in 1986 to encompass all orthopedic problems seen in the growing foal. It came out of a blue ribbon panel sponsored by the American Quarter Horse Association. It is a term that encompasses all general growth disturbances of horses and is therefore nonspecific. It is felt that one has to be nonspecific when talking about the various limb anomalies of young horses because previous terms such as metabolic bone disease and osteochondrosis implied that they all had a common cause and pathogenesis (mechanism of disease development). It is still to be determined how closely related the various forms of DOD may be but it is important that the term not be used synonymously with osteochondrosis. It is considered inappropriate for all subchondral cystic lesions, physitis, angular limb deformities and cervical vertebral malformations to be presumed to be manifestations of osteochondrosis (a condition defined below). The spectrum of conditions currently classified as DOD were previously designated as metabolic bone disease. However, it is felt that this term is misleading because it refers specifically to bone, whereas many of these problems are seen essentially as joint and growth plate problems. Severe forms of DOD are seen sometimes when there is little or no aberration in bone histomorphometry, implying questionable change in bone metabolism. Developmental orthopedic disease has become the generally accepted term.

When the term developmental orthopedic disease was first coined, it was categorized to include the following.

1.   *Osteochondrosis* - Osteochondrosis is a defect in endochondral ossification (definition above) that can result in a number of different manifestations, depending on the site of the endochondral ossification defect. These manifestations include osteochondritis dissecans, which is a disease process of the articular surface of joints. As mentioned previously, the epiphyseal ossification center advances out until ossification ceases, leaving a layer of cartilage. This layer of cartilage becomes the articular cartilage. If there is a disturbance in endochondral ossification, an area of retained cartilage can be formed with a consequent defect in the bone. Cracking can then proceed in this retained cartilage to give a flap or fragment of cartilage that may contain bone. These flaps and fragments on the surface of the joint result in osteochondritis dissecans (detailed in another paper).

Another manifestation of osteochondrosis may include some subchondral cystic lesions (Figure 4). These are radiolucent cyst-like structures that occur in epiphyseal bone. Not all subchondral cystic lesions or bone cysts are necessarily the result of osteochondrosis. Recently in a project funded by the American Quarter Horse Association, we demonstrated that subchondral cystic lesions

can develop from defects in the joint surface and in mature animals. However, there is no question that some subchondral bone cysts occur as a result of endochondral ossification. These cysts are also discussed in another paper. Osteochondrosis can also result in lesions in the physis or metaphyseal growth plate. Cervical vertebral malformations (wobblers) are also included. The exact role of osteochondrosis and the pathogenesis of cervical vertebral malformation are still uncertain. In some instances, angular limb deformities will be associated with retained cartilage associated with the physis and this would be evident on radiographs. Such cases are a small minority of angular limb deformities.
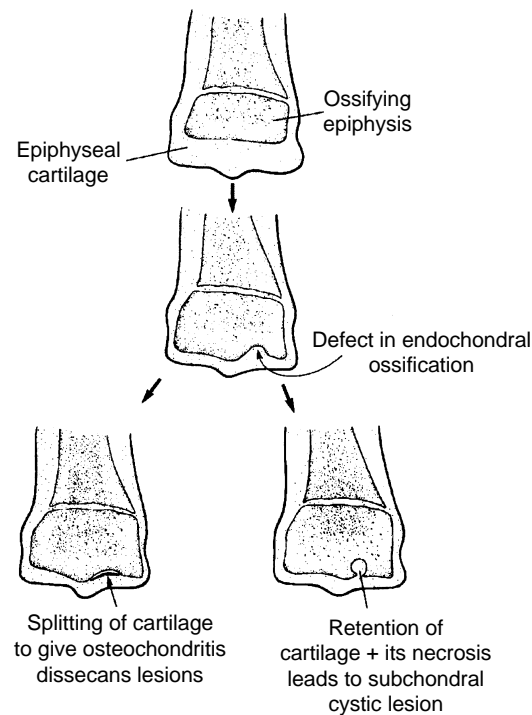

Ossifying epiphysis

Epiphyseal cartilage

Defect in endochondral ossification

Splitting of cartilage to give osteochondritis dissecans lesions

Retention of cartilage + its necrosis leads to subchondral cystic lesion

Figure 4. Diagram of pathogenesis of osteochondritis dissecans and subchondral cystic lesions in relation to the generalized condition of osteochondrosis.

2. *Acquired angular limb deformities*

3. *Physitis*

4. *Subchondral cystic lesions*

5. *Flexural deformities* (may be secondary to osteochondrosis or physitis)

6. *Cuboidal bone abnormalities*

7. *Juvenile osteoarthritis*

The above problems are considered DODs. Individual instances may or may not be associated with osteochondrosis.

CLINICAL DIAGNOSIS OF DOD VERSUS RADIOGRAPHIC SURVEYS

There has been a problem or a point of confusion between reports of clinical instances of DOD and radiographic surveys of horses not necessarily showing clinical signs. This has led to some questionable conclusions, not only regarding the causative factors of DOD but also the effect of treatment or the significance of various problems. There are many instances where radiographs on a prepurchase examination will show OCD, for instance, but that OCD may not be causing clinical problems because the fragment has not separated. That is why it is important to distinguish between the two. Most of the major radiographic studies have involved Standardbred horses. In one study in Quebec, each horse's racing performance at two years of age was related to the radiographic lesions diagnosed at approximately 17 months of age and before training (there were two generations of 41 and 32 yearlings, respectively; five stallions and 46 mares were also radiographed). No complete clinical examinations or lameness diagnoses were made. Radiographic lesions were found in 31 (25%) of the horses (8 adults and 23 yearlings), of which 60% had a single problem, and 40% had between two and four radiographic problems. Subchondral bone cysts were detected in 14 (11.3%) horses (6 carpus, 5 fetlock, 4 pastern, 2 hock, and 2 stifle). Juvenile osteoarthritis lesions were diagnosed 78 times in 35 (47.9%) of the yearlings (40 pastern, 13 fetlock, 11 carpus, 8 coffin, 6 hock) based primarily on the basis of osteophytes. Sesamoiditis was also diagnosed in yearlings. The average winnings and number of starts were compared between radiographically normal horses, the OCD or subchondral bone cyst horses, and the juvenile OA horses; no significant differences were found. Although the radiographic lesions did not seem to be associated with poor racing performance, the authors of the study noted the lack of clinical data and the relatively small numbers. What we need are improved studies in which clinical signs are correlated with radiographs and possibly more important, all horses are radiographed and then followed so we know how many of those horses with x-ray changes develop clinical problems. This would solve many of the current issues at the yearling sales.

   The high incidence of clinically apparent physitis and flexural deformities was emphasized in another study in Canada. Mild to moderate physitis and flexural deformities (concurrent with physitis in most cases) occurred in 88% of 42 weanlings between weeks 6 and 8 of a study looking at the effect of dietary energy and phosphorus on blood chemistry and development of growing horses. In these instances, the clinical signs largely resolved on their own by five weeks. Dietary treatment did not influence the incidence, nor was it related to daily weight gain. There has been another study defining incidence of DOD in Thoroughbred horses in Ireland over a period of 18 months. It was found that angular limb deformities and physitis together

constituted 72.9% of the cases treated. The peak incidence of DOD problems occurred between weaning and the end of December. In a retrospective study, 193 of 1,711 (11.3%) were treated for DOD (21 had more than one type) and are detailed as follows: angular limb deformities - 92, physitis - 64, flexural deformities - 18, wobblers - 7, and osteochondritis dissecans, juvenile arthritis or other joint problems - 28. More than half the animals treated (53.9%) recovered completely, that is they achieved expected sale value as yearlings; 27.5% showed incomplete recovery and mild to moderate loss of sale value; and 18.7% were either killed or lost much of their sale value. It was also noted that 67.7% of the animals showed some evidence of DOD, but only 11.3% were deemed to need treatment. This study was a good start and points out the need to have definition of what disease process we have. I think it can be seen from these studies that when "developmental orthopedic disease" occurs, it commonly involves angular limb deformities and physitis problems that spontaneously self-correct. It is the ones that often do not self-correct, such as OCD subchondral bone cysts, that we need to investigate further.

OSTEOCHONDROSIS

Osteochondrosis was initially defined as a disturbance of cellular differentiation in the growing cartilage. It is considered to be a result of the failure of endochondral ossification and may affect either the articular epiphyseal cartilage complex (previously diagramed and illustrated in Figure 4) or the metaphyseal growth plate (also in Figure 4). It was considered that the loss of normal differentiation of the cartilage cells means the transitional calcification of the matrix, which is important for bone formation, did not take place. Therefore, ossification ceases and cartilage is retained. This retained cartilage then undergoes cellular death (necrosis) in the basal layers and it is proposed that subsequent stresses give rise to fissures in the damaged cartilage and that progressive breakdown of the cartilage can then lead to the syndrome designated as osteochondritis dissecans or subchondral bone cysts.

That these entities are part of the generalized condition of osteochondrosis has resulted from studies in other species and extrapolation to the condition in the horse. There is still considerable argument about the exact association and with more experience, one comes up with more cases that don't quite fit the pattern. For example, we see cases of osteochondritis dissecans in the stifle that do not have thickened cartilage or a defect in the bone and that do not quite fit with the retained cartilage phenomenon. However, they could still fit with some developmental abnormality. At this stage, we therefore like to say that osteochondrosis may lead to osteochondritis dissecans or subchondral cystic lesions. It also may occur in the physis and lead to retained cartilage in that area. It is currently presumed that many instances of subchondral bone cysts result from osteochondrosis. On the other hand, we have demonstrated the development of bone cysts secondary to a defect. In early work, osteochondrosis was suggested to be a generalized condition and

therefore occurred in multiple sites. However, based on our clinical material, it was found that osteochondritis dissecans will be found in a particular joint and a lesion will commonly occur in the same joint but will not occur elsewhere. We feel that certain biomechanical factors and perhaps insults at certain stages of development may also be important factors. Causative factors associated with osteochondrosis are discussed further below.

Osteochondritis dissecans

There is general agreement that this condition involves a dissecting lesion with the formation of a chondral or osteochondral flap. Flaps may become detached and form joint mice. Blood vessels from the periphery of the joint frequently remain in communication with cartilage flaps or detached flakes, leading to calcification or ossification of the avulsed cartilage. It is commonly considered that release of debris from under the flap causes synovitis and pain, but we don't think it is that simple now because we find instances where the cartilage is intact but the horses still have lameness and synovial effusion.

Subchondral cystic lesions

Subchondral cystic lesions have also been proposed as a manifestation of osteochondrosis by a number of authors and there is pathologic evidence to support this. When we have a yearling horse with a large subchondral cystic lesion in one stifle and a defect in the other, we feel comfortable assuming the cause to be osteochondrosis. However, we also get subchondral cystic lesions that develop in all the horses and in some instances, we have noted them developing after a cartilage surface defect. We have also experimentally produced them by creating a defect into the subchondral bone.

Physitis

Physitis has also been described as epiphysitis and physeal dysplasia. It has even been suggested earlier that epiphysitis was the same as osteochondrosis but this is incorrect. In many instances, we have pain and swelling at the growth plate without any evidence of retained cartilage. Most cases of physitis do not have any radiographic lesions that are particularly significant and they resolve with time.

Angular limb deformities

As with these other syndromes, they will be discussed in more detail. Angular limb deformities involve deviations in the limbs as looked at from the front or back, such that the deviation is excessive from side to side from the horse's point of view. Many angular limb deformities cause no problems and resolve themselves.

Cuboidal bone malformation

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.

CAUSATIVE FACTORS OF OSTEOCHONDROSIS

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 than 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.

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 a significantly high proportion of osteochondrosis in the progeny of one of eight stallions, even though the stallion itself did not show radiographic signs of osteochondrosis, was seen. Since that time, there have been two more studies on the heritability of osteochondrosis in the hock of Standardbred Trotters.

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.

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 and 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, 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, height, and cannon bone circumference and it was part of a study on the effect of dietary copper.

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.

Nutrition

As discussed previously, the idea of overnutrition 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.

Many generalizations have been made about excessive growth causing OCD and this is based on the early work done in Sweden in 1978 which was primarily extrapolated from pigs. However, even when the pig work is carefully looked at, a direct association is not demonstrated in some studies. Unfortunately, the correlation of growth and OCD has led to the practice of virtually starving horses (onto grass hay and water) in an attempt to reduce the incidence (which it has not). This creates a whole new malnutrition situation. It may be that lowering energy and protein is a good part of the protocol in some instances to prevent OCD but the possibility of inducing other nutritional imbalances needs to be carefully considered and compensated for.

It has also been pointed out that overfeeding can induce endocrine imbalances (see *Endocrine factors* below).

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 suppresses copper levels) is still being elucidated.

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.

Site vulnerability

Because the lesions of equine osteochondrosis occur at specific anatomic sites, this obviously suggests 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 nonarticulating surfaces is different.

OCD lesions are frequently bilateral in the stifle and 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 was 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 these data to ascertain different periods of onset of the disease process in different joints.

Exercise

As has been discussed under traumatic arthritis, adequate exercise in foals would logically be important for the maintenance of cartilage and bone quality. There are some data suggesting a "protective" effect of exercise. This particular study was done on early weaned Warmblood foals and 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.

The various developmental orthopedic diseases will now be considered in more detail.

OSTEOCHONDRITIS DISSECANS

Osteochondritis dissecans (OCD) is a disease subset of osteochondrosis. This condition affects the articular (joint) cartilage and also often involves the subchondral bone just beneath the cartilage surface. The cause of OCD has generally been considered a defect in endochondral ossification at the joint surface (this process was previously mentioned with regard to ossification of this secondary epiphysis). The paradigm has been that for some reason endochondral ossification does not

occur correctly at a site, leaving an area of thickened retained cartilage which then is secondarily displaced as a flap or fragment. This end result is certainly attained, but more recent data from studies we have done suggest that it is not as simple as this. For instance, we have had OCD lesions that have no thickening of the cartilage and no defects in the bone. We have also had lesions develop after endochondral ossification is complete and when we had a normally appearing surface. There is still a lot to learn about this complicated disease.

The important situation is, however, that these lesions do occur in the joint and if they result in flaps or fragments, lead to an arthritis situation. We will now go through the clinical features of this disease in each of these joints.

OSTEOCHONDRITIS DISSECANS OF THE FEMOROPATELLAR (STIFLE) JOINT

The stifle joint is one of the principal joints affected with OCD. Stifle OCD can be diagnosed in almost any breed but it is more common in Thoroughbreds. Approximately 60% of affected horses will be one year of age or less at the time the condition becomes symptomatic. However we also see the disease show up clinically even after horses have won stakes races. The difference is when the retained cartilage fragment or flap becomes displaced. As a generalization, when animals show up at a younger age with OCD, they tend to have more severe lesions.

Clinically the animals present with joint swelling due to synovial effusion and varying degrees of lameness. Severe cases can be very lame and be confused with wobblers because of the difficulty they have flexing the stifles in getting up and down. In older animals, an increase in the level of exercise may be part of the history. Horses will often have a bunny-hop gait behind and also could be confused with a neurologic problem. Some horses will be very subtle in their lameness. A joint distention (when the disease is clinically significant) is a regular feature. Careful palpation of the joint may identify free bodies or the surface irregularity associated with the damage within the joint. Bilateral involvement is common in the stifle so careful examination of both stifles should be completed. In one major study of cases we operated, 57% of animals had bilateral involvement.

Table 1. BREED DISTRIBUTION OF 161 HORSES PRESENTED FOR FEMOROPATELLAR OCD

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

Table 2. AGE DISTRIBUTION OF 161 HORSES PRESENTED FOR FEMOROPATELLAR OCD

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

(From Foland, McIlwraith and Trotter. Equine Vet J, 1994.)

Lateral to medial radiographs provide the best means of diagnosis regarding specific location of the lesion and its size. The most common location is on the lateral trochlear ridge of the femur and shows up as an area of flattening, irregularity or concavity. The area of the trochlear ridge adjacent to the bottom portion of the patella is most commonly involved. Various degrees of mineralization may be present within the flap tissue, affecting the radiographic signs, and free bodies may also be identified. OCD can also primarily affect the patella or the medial trochlear ridge of the femur. Generally the extent of damage to the joint identified at surgery is more extensive than would be predicted from radiographs. Although other joints can be involved concurrently, this is uncommon. In one study of 161 horses with stifle OCD, 5 also had OCD affecting the rear fetlocks, 4 had hock OCD and 1 had OCD of a shoulder joint.

Treatment

In general, arthroscopic surgery is recommended for the treatment of most of these cases. However, it has been recently identified that in low grade lesions detected very early, stall confinement could allow healing with presumably reattachment of any separated cartilage. When there is a significant concave defect or flaps or fragments identified, arthroscopic surgery is recommended. The joint is thoroughly explored and this usually gives a better assessment of all damage. Suspicious lesions are probed and loose or detached tissue is elevated and removed. Loose bodies are also removed. The defect site is then debrided down to healthy tissue. Animals are usually stall rested for two weeks after surgery at which time hand walking is started. Restricted exercise is continued for two to three months after surgery, when training is started or the horse is turned out (the total period of convalescence depends on the amount of damage).

In a recent study of 252 stifle joints in 161 horses, followup information was available for 134 horses. Of these 134 horses, 64% returned to their previous use or anticipated use (racing), 7% were in training, 16% were unsuccessful, and 13% were unsuccessful due to reasons unrelated to the stifle. The success rate was higher in horses having smaller lesions.

OSTEOCHONDRITIS DISSECANS OF THE TARSOCRURAL (HOCK) JOINT

OCD occurs in a number of locations in the hock, including the intermediate ridge of the tibia (most common), the lateral trochlear ridge of the talus, the medial malleolus of the tibia and the medial trochlear ridge of the talus (descending incidence). It is a very common disease in Standardbreds but is also very common in Quarter Horses and Arabians.

The most common clinical sign of hock OCD is joint distention of the tarsocrural joint. This manifests clinically as a "bog spavin," which simply refers to the prominent swelling seen along the medial or inside aspect of the joint. Lameness can also be seen but it is not common and it is rarely prominent. All ages of horse can be affected. Often in racehorses the disease does not show up until the horses are in training. In nonracehorses, the cases that are going to be clinical commonly show up as yearlings prior to going into training. The disease is confirmed with radiographs.

The location of OCD lesions in 318 tarsocrural joints is shown in Table 3 (taken from a study by McIlwraith, Foerner and Davis published in 1988).

Table 3. LOCATION OF OCD LESIONS IN 318 TARSOCRURAL JOINTS

No. Joints	Location
244	Intermediate ridge (dorsal aspect) of distal tibia
37	Lateral trochlear ridge of talus
12	Medial malleolus (dorsal aspect) of tibia
11	Intermediate ridge of tibia plus lateral trochlear ridge of talus
4	Intermediate ridge plus medial malleolus of tibia
3	Intermediate ridge plus medial trochlear ridge of talus
3	Medial trochlear ridge of talus
3	Lateral trochlear ridge of talus plus medial malleolus of tibia
1	Lateral and medial trochlear ridge of talus
318	Total

Treatment

When clinical signs are present in association with OCD lesions in the hock, surgery is the recommended treatment. It is to be noted that the disease is often diagnosed as an incidental finding on radiographs of horses prior to sale. If there is no joint effusion or lameness, we do not normally recommend surgery. In some instances in racehorses, lameness may be the only problem seen and it is only seen at racing speed or upper levels of performance. Certainly resolution of the joint effusion can only be expected with removal of abnormal tissue. The horses are treated arthroscopically with removal

of fragments and debridement of any damage. The postoperative management is similar to OCD of the stifle but the convalescence time may be faster as many of the lesions are localized and not involving a major articulating surface.

In a study involving 183 horses mentioned previously, 76% raced successfully or performed at their intended use after surgery. If secondary osteoarthritic change is identified at surgery in the cartilage, the prognosis is less favorable. Resolution of the joint distention so there is a normal hock with no fluid is a critical criterion of success for nonracehorse owners. It was found in this study that the resolution of effusion was inferior for lesions involving the lateral trochlear ridge of the talus compared to the intermediate ridge of the tibia, so we take this into account when giving a realistic prognosis to owners.

OSTEOCHONDRITIS DISSECANS OF THE FEMOROPATELLAR JOINT

The most common manifestation of OCD in the fetlock joint is fragmentation and irregularity on the distodorsal (front) aspect of the sagittal ridge and condyles at the bottom of the cannon bones, which is an intra-articular part of the fetlock. This condition affects all breeds but we have seen many cases in Thoroughbreds and Arabians. A second condition that some people have described as OCD is fragmentation at the back of the fetlock off the proximal palmar or plantar aspect of the first phalanx or long pastern bone. Debate continues as to whether these fragments are truly OCD or whether they represent small avulsion fragments. A third disease that has been classified as OCD of the fetlock occurs on the distal back part of the end of the cannon bone condyles and this appears to be a trauma-related condition of racehorses and should not be considered as OCD. The first two entities will be considered separately below.

OCD OF THE DORSAL ASPECT OF THE DISTAL METACARPUS OR METATARSUS IN THE FETLOCK JOINT

Joint filling is the most common clinical sign with lameness being variable in both appearance and severity. Fetlock flexion tests are usually positive. It is not unusual for all four fetlocks to be involved and it is quite common for bilateral forelimb or hindlimb involvement. The condition is diagnosed on radiographs. It is another one of the problems that cause controversy at yearling sales.

We divided the lesions into three types because it does affect the prognosis as well as treatment.

Type I OCD

This involves flattening or a defect in the sagittal ridge or condyles without any fragment. This is obviously diagnosed by x-ray. Most of these cases get better with

conservative treatment and do not need surgery. Not only will they get better clinically with loss of the joint filling but also remodel radiographically.

Type II OCD

This involves a defect as in Type I but also there is a fragment present within it. Based on followup with conservative treatment, these cases don't generally get better with conservative treatment and require arthroscopic surgery. The prognosis for surgery in turn is related to a number of factors. The most favorable cases are hind fetlocks versus front fetlocks and ones with no spurs on radiographs and no secondary osteoarthritic damage.

Type III OCD

These have a defect in the primary area but also free all loose bodies within the joint. They have the same need for surgery as well as the same result as Type II lesions.

With regard to the overall prognosis, it is not as good as with stifle or hock OCD because commonly secondary articular cartilage erosion or wear lines develop in the joint by the time they are operated.

Proximal Palmar/Plantar fragments of the first phalanx

Two types of fragments have been identified in this location. Type I fragments usually involve the hind fetlocks and are located between the middle of the bone and its caudomedial (most common) or caudolateral (less common) borders. Type II fragments are also called ununited proximoplantar tuberosities of the proximal phalanx and these lesions occur almost exclusively in the hind limb. These are much less frequent. Both of these entities have been identified frequently in radiographic surveys completed in yearling Standardbreds, supporting a developmental concept. However, more recently it has been advocated (based on dissections and examination of the fragments) they are traumatic avulsions due to a pull on the short distal sesamoid ligament. However, it is agreed by all parties that they are present before a year of age.

   With the common Type I fragment, joint filling is uncommon and a vague lameness is usually the main presenting sign. Most times cases are not diagnosed clinically until they are in training. Flexion tests are often positive and block of the fetlock area confirms the site of the problem. These cases are treated by arthroscopic surgery and have a high success rate.

   With Type II fragments, they are often unassociated with any clinical signs and are incidental findings at radiography. If they are associated with clinical signs, most will still ossify on their own and surgery is not usually necessary.

Osteochondritis dissecans of the shoulder joint

This a severe problem and represents the worst type of OCD affecting horses. Generally large areas of the joint surface are involved and secondary osteoarthritis is common. It is fortunately less common than other sites and seems to affect Quarter Horses and Thoroughbreds with a similar incidence.

Most cases with shoulder OCD present at one year of age or younger with a history of forelimb lameness of variable severity. Many of these horses will have prominent lameness and if lameness has been present for many weeks, muscle atrophy will also be seen. Because of the altered gait and use of the limb, many cases also develop an upright or clubfooted appearance to the foot and the foot may appear smaller on the affected limb. Deep pressure over the shoulder joint will often cause discomfort. The diagnosis is confirmed with x-rays.

We have never seen conservative treatment solve the problem of OCD in the shoulder. Arthroscopic surgery has been used as a treatment with 50% of the cases becoming sound and the horses going on to do what they are supposed to do. If extensive degenerative arthritic changes are present on radiographs at the time of initial examination, the prognosis for an athletic career is unfavorable. With more localized lesions, the prognosis is much more favorable. The shoulder is a difficult area for surgery due to the depth of the joint below the muscles in the area. Surgery is easier on younger animals due to smaller muscle mass and the only fortunate aspect of OCD in the shoulder is that the clinical signs of lameness show before they are a year of age.

Subchondral cystic lesions
(also called bone cysts or osseous cyst-like lesions)

These are commonly recognized abnormalities of bones and joints that may or may not cause lameness. The ones that concern us the most in terms of soundness are articular cystic lesions that occur in the subchondral bone or epiphyseal bone and communicate with the joint.

Subchondral cystic lesions can occur in multiple sites in horses (see Table 4).

Controversy exists as to whether these lesions are a manifestation of osteochondrosis secondary to joint trauma, or a combination of both. Currently we feel that they have a multifactorial etiology and some are associated with retention of cartilage (a defect in endochondral ossification) but others occur secondary to a defect in the subchondral bone. This defect is probably most commonly a traumatically induced fracture or erosion. Because many of them occur in young growing animals, they are certainly considered to be one of the developmental orthopedic diseases. When seen in yearlings (and they are quite common in this situation in Quarter Horses and Arabians and to a lesser extent, Thoroughbreds), and particularly when they

occur bilaterally, they are certainly considered to be a manifestation of osteochondrosis (discussed at the beginning of these notes as well). When they are due to osteochondrosis, it is felt that cartilage gets retained deeply, undergoes necrosis and leads to the problem. In other instances when seen in older horses, it is felt that they probably result from a defect. It has also been recently proven experimentally that a defect can produce such lesions.

Table 4. SITES OF OCCURRENCE OF SUBCHONDRAL CYSTIC LESIONS IN HORSES

Joint	*Specific location*
Stifle	Medial femoral condyle
	Lateral femoral condyle
	Proximal tibia
Carpus	Distal medial radius
	Cuboidal carpal bones
Elbow	Proximal medial radius
	Distal medial or lateral humerus
Fetlock	Distal metacarpus/metatarsus
	Proximal first phalanx
	Sesamoids
Pastern	Distal first phalanx
	Proximal second phalanx
Coffin	Third phalanx
	Navicular bone
Shoulder	Glenoid
Hock	Trochlear ridge of talus
	Tarsal bones
Hip	Acetabulum
	Proximal femur

The most common location of subchondral cystic lesions in horses is the medial femoral condyle within the stifle. Less common sites include the proximal tibia, distal aspect of the metacarpus and metatarsus (cannon bones), distal aspects of the radius and in the carpal bones, the proximal radius within the elbow, in the phalangeal bones associated with the pastern and coffin joints, in the shoulder and in the acetabulum of the hip.

SUBCHONDRAL CYSTIC LESIONS OF THE STIFLE

In the stifle, most horses present with a clinical problem between one and three years of age and they usually present to a veterinarian because of a unilateral lameness. A small percentage of horses may be lame in both hind limbs. The severity of the lameness is variable and usually ranges grade 1-3/5. Periods of work usually worsen the lameness and improvement occurs with rest.

Obvious signs of swelling (joint effusion) within affected joints are usually absent or minimal. Mild effusion of the stifle joint was reported in 15/41 cases we recently published. The diagnosis is confirmed with x-rays and the clinical significance of the x-ray lesion confirmed with intra-articular analgesia. In general, if clinical signs have become apparent in association with a cyst in the stifle, the recommendation is for surgical enucleation of the lesion. That is because with conservative management our success rate has been 20% or less, whereas our success with surgery is approximately 70%. The cases are operated arthroscopically.

SUBCHONDRAL CYSTIC LESIONS OF THE FETLOCK

These cases present with obvious lameness and usually have some filling in the fetlock joint. The lameness can be made worse by flexing the fetlock. These horses will respond to intra-articular blocks of the fetlock. Radiographs confirm the diagnosis. The cystic lesion either occurs in the bone under the articular surface of the condyle or the central sagittal ridge of the distal metacarpus or metatarsus (cannon bone). Cystic lesions are occasionally seen on the opposing surface of proximal P1 but are commonly insignificant clinically. We have also seen a very poor response to conservative management of these cases and arthroscopic surgery is recommended. A recent paper by Hogan, McIlwraith, Honnas, Watkins and Bramlage reported very good results with surgical treatment.

SUBCHONDRAL CYSTIC LESIONS OF THE CARPUS

Subchondral cystic lesions occur within the carpus (knee) but quite a few of these were incidental findings. If they persist and cause clinical signs, then we recommend surgery.

SUBCHONDRAL CYSTIC LESIONS OF THE PASTERN JOINT

When these occur singly, they are often incidental or cause only temporary lameness. More commonly they are multiple, involving the distal surface of the first phalanx. When they are multiple, they generally have severe secondary osteoarthritis (the only

subchondral entity that shows this) and the only treatment available is fusion of the pastern.

SUBCHONDRAL CYSTIC LESIONS OF THE ELBOW

It is a relatively uncommon but a significant cause of lameness in the elbow. The diagnosis is based on upper limb lameness (usually after eliminating lower limb lameness), intra-articular anesthesia to prove the condition is the problem, and radiographic confirmation. We have had equal results in treating these cases with conservative versus surgical treatment. Conservative management is tried initially.

Angular limb deformities

Angular limb deformities in the young horse can take the form of a lateral or medial deviation of the limb. The angulation can arise in association with uneven elongation from the growth plate (physis) or alternatively can be involved in abnormalities of the cuboidal bones of the carpus and tarsus. Most commonly the problems are associated with uneven physeal growth and involve the physes of the distal radius, metacarpus and metatarsus or tibia (in that order). When a deviation results in the lower part of the limb going out (lateral), it is termed *valgus* while a deviation to the inside (medial) is termed *varus*. The total nomenclature for angular deformities is derived by combining the name of the involved joint or the joint immediately distal to the affected growth plate and the type of deviation. For example, a lateral deviation of the distal limb due to an affected distal radial growth plate or abnormal cuboidal bones of the carpus would be termed carpal valgus.

The etiology of angular limb deformities is complex and thought to be multifactorial. In Figure 5 it can be seen that the two main categories of factors include perinatal and developmental. Perinatal factors are usually involved when a foal is born with an angular limb deformity, whereas foals that are normal at birth but develop an angular limb deformity are more likely to suffer from one or more of the developmental factors.

It is to be noted that carpal valgus is a normal deformity in the young foal and that most of these correct naturally. There have been no nutritional factors consistently associated with this disease.

In managing these cases, one must first realize that depending on the location there is a typical growth curve for each of the physes. In the carpus, the growth plate stays open for two years and there is diminishing rate of lengthening that continues for over a year. On the other hand, when the deformity involves the fetlock, the time available for manipulation of the growth curve is much lower. All effective elongation in the physis at the distal metacarpus or metatarsus ceases around three months of age.
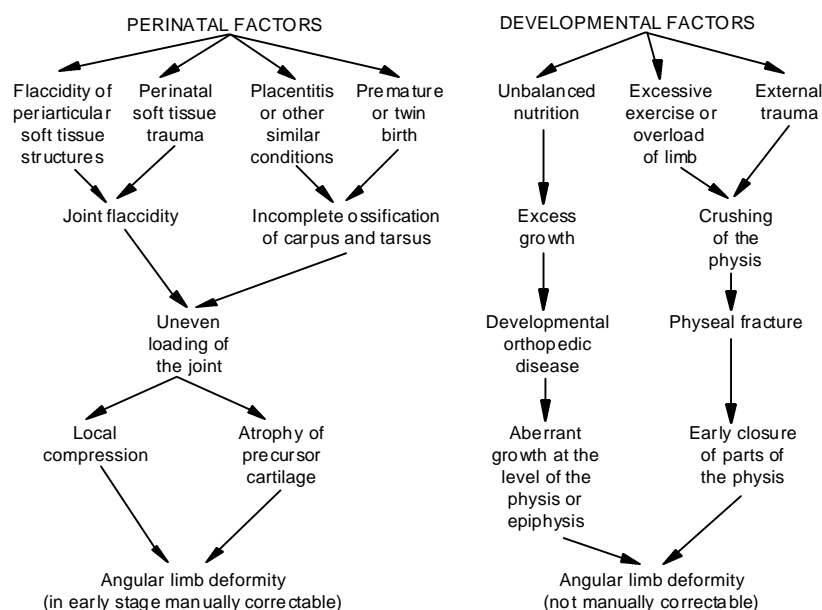
Figure

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      PERINATAL FACTORS                         DEVELOPMENTAL FACTORS

Flaccidity of   Perinatal   Placentitis  Premature    Unbalanced   Excessive    External
periarticular   soft tissue  or other    or twin      nutrition    exercise or  trauma
soft tissue     trauma       similar     birth                     overload
structures                   conditions                            of limb

        Joint flaccidity      Incomplete ossification   Excess        Crushing
                              of carpus and tarsus       growth        of the
                                                                       physis

                    Uneven                        Developmental   Physeal fracture
                    loading of                    orthopedic
                    the joint                     disease

         Local              Atrophy of            Aberrant        Early closure
         compression        precursor             growth at the   of parts of
                            cartilage             level of the    the physis
                                                  physis or
                                                  epiphysis

         Angular limb deformity                   Angular limb deformity
      (in early stage manually correctable)     (not manually correctable)
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Figure 5. The etiology of angular limb deformities (adapted from *Equine Surgery*, Auer, 1992)

When an angular limb deformity involves the joint's bones itself, such as the carpus or tarsus, this needs to be recognized radiographically and is treated with braces or casts to maintain the limb in alignment while the cuboidal bones reconstitute. This problem occurs relatively infrequently. When it does, it is often associated with a premature foal or a hypothyroid foal.

When the more common situation of growth imbalance associated with the physis occurs, the management depends on the location. In the fetlock, it is generally considered an emergency and periosteal stripping is done at one to two months. The ideal time is one month and it should be done by two months. At three months, the amount of correction that can be attained is much lower and the blemish following periosteal elevation is more obvious. With carpal valgus, there is much more time for manipulation and periosteal strippings between two and six months are quite common. The principle of periosteal stripping is to transect periosteum (which acts as a normal restraining device to physeal lengthening on that side) and allow a speed-up of cartilage growth in the physis (hence, lengthening) on that side. It is a simple complication-free procedure. If the problem is not diagnosed until it is too late to obtain sufficient benefit from periosteal stripping, a second more drastic option is stapling or screw and wire fixation of the physis. The principle here is to halt growth on the fastest growing side to allow continued growth on the opposite side of the physis and straightening of the limb.

Physitis (Epiphysitis)

Physitis is also known as epiphysitis but because the problem is associated with the growth plate, the first term is more correct. The term has also been called physeal dysplasia, although not all the factors involved in what goes wrong are understood. In some instances, obvious osteochondrosis is present and radiographically retained cartilage and lipping at the edges of the physis are seen. In other instances, there is lameness and swelling associated with the physis but no abnormalities on radiographs. Microfractures may be involved. The cause of physitis is still uncertain. People have associated it with hard ground and there is no question that there are "bad seasons" for it.

In most instances, the prognosis for natural resolution of the problem is good. The foals are confined to some extent and the amount of confinement versus exercise titrated with careful observation. Horses usually present clinically between four and eight months of age. However, occasionally horses up to two years of age can be affected. The signs are usually seen in the distal radius and distal metacarpus or metatarsus. Less commonly they are seen in the first phalanx and distal tibia. The lameness varies from slight to overt.

TREATMENT

The treatment of physitis is aimed at correcting any possible nutritional deficiencies, excesses, or imbalances but in many instances none of these are recognized. Restriction of exercise is indicated when moderate to severe lameness is present and nonsteroidal anti-inflammatory drugs may relieve some of the pain associated with this condition. As mentioned previously, physitis is normally a self-limiting disease with resolution occurring when skeletal maturity is reached and growth at the affected physis ceases.

Physeal fractures

More drastic injuries to the growth plate may occur. As depicted in Figure 4, there is vulnerable tissue when cartilage cells die and undergo calcification. Fractures can occur along this plane and sometimes propagate through bone as well. These are traumatic events unrelated to any known factors in management or nutrition.

Bone disease in the athletic horse

Obviously, athletic horses sustain many forms of bone injury including chip fractures in the joint, slab fractures in the joint, stress fractures in the cannon bone, and stress fractures in the upper bone, such as the humerus, tibia and pelvis. The questions are always asked as to whether these lesions are associated with poor bone development (low calcium), or various other trace mineral imbalances.

Very little definitive data associating these problems with "bad bone" exist. There