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Advances in Equine Nutrition

Volume I

Edited by

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INVESTIGATION OF FARM WIDE INCIDENCE OF BONE FORMATION PROBLEMS IN THE HORSE

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The first step in identification of a herd problem is to determine if the individual occurrences of disturbed bone formation have anything in common. The clinical signs may be different, but the problem may be the same because the same disturbance will affect different sites at different ages under different circumstances.

The investigation of a herd problem begins by examination of the individual problems. Identification of any common factors among individual cases, and retrospective identification of where in the growth process the bone formation problems are occurring, is most likely to result in help with a farm wide problem. Concurrent ration analysis should also occur and be reviewed by a competent nutritionist. Marginal deficiencies can often lend clues as to where the bone formation disturbance is occurring. Integration of ration analysis information, bone formation disturbance data and management schedules are the best clues to the cause of a herd problem, if one exists.

Normal bone formation

The normal development of equine bone is a multi-phase two-step process occurring as a continuum of activity resulting in calcification of degenerating physeal cartilage, reabsorption, and redeposition as trabecular bone. The process takes place at the metaphyseal side of the physis and circumferentially around the ossification fronts of all epiphyses and cuboidal bones simultaneously. The growth process requires the formation of cartilage which causes the increase in size or length. The cartilage degenerates in an orderly fashion, as its inter-cellular matrix is calcified (step 1). This calcified cartilage must be reabsorbed and bone must be redeposited as trabeculae, oriented against the lines of stress and deposited in sufficient numbers and size, to handle the load applied to the bone (step 2). It is not until trabeculae are deposited that "ossification" has occurred. If either of these two steps is disturbed or interrupted, one of the syndromes we generally refer to as developmental orthopedic disease results.

Developmental orthopedic disease (DOD)

The conditions referred to as developmental orthopedic disease are those which result from a disturbance in the change from the cartilage precursor of the skeleton into

functional bone. Clinical manifestations include, but are not limited to, physitis, osteochondritis dissecans (OCD), and some angular limb deformities.

Since acquired contracted flexor tendons can develop as a sequela to the pain resulting from these diseases, they are generally included as a part of the syndrome (Knight, *et al.*, 1985) . Histologic changes in the cervical vertebrae of horses diagnosed as having cervical vertebral malformation have been reported and recently incriminated as a form of developmental orthopedic disease (Stewart, Reed and Weisbrode, 1991). Some articular bone cysts and juvenile arthritis, due to malformation of articular surfaces and cuboidal bones, especially of the distal tarsal joints and interphalangeal joints are probably manifestations of this same disease complex.

Physeal disturbances

Defective or slowed conversion of the calcified cartilage into mature (trabecular) bone results in a weakened metaphyseal component of the growth plate complex. This weakened bone leads to structural overload, microfracture and inflammation. The response to the microfractures is inflammation and callus formation leading to the enlargement of the physis. Physitis in the long bones, which results from a disturbance in the ossification of the calcified cartilage (step 2) on the metaphyseal side of the physis, is largely reversible because the physeal plate is a temporary structure, which is remodeling continuously and which disappears at skeletal maturity. A disturbance in ossification (step 2) of calcified physeal growth cartilage is seen most commonly clinically. A disturbance in the physeal cartilage degeneration and calcification (step 1) results in retention of cartilage and a much more severe structural deficit, which is more difficult to overcome but is fortunately less frequently seen clinically. The initial clinical signs are similar but the cause is a different process.

Epiphyseal disturbances

When either of these two disturbances involves the subarticular epiphyseal growth surfaces, the articular surface becomes involved and may permanently affect the pain free function of the joint. In the epiphysis, defective formation of the subchondral bone results in a poorly supported articular surface. Increasing weight and activity levels which result from advancing maturity overload the poorly formed joint surfaces. The articular cartilage, undermined by abnormal bone formation, fractures, becomes detached or loosened (osteochondritis dissecans), and results in signs of arthritis. Osteochondritis dissecans is often the underlying cause of secondary degenerative joint disease (osteoarthritis) in the young horse. Though bone formation problems and their clinical manifestations differ depending on the site of the disturbance, similar types of problems can yield a clue as to where to look in the process of bone formation for the cause of the disturbance.

Evaluation of a herd problem

Some horses have more than one manifestation of developmental orthopedic disease simultaneously and often many horses are affected on the same farm. On some farms, the problem recurs annually. Though developmental orthopedic disease is multifactorial, clinical impression suggests that one variable needing investigation in most large scale multi-horse manifestations of the disease is the diet. The diet is also the only easily manipulated factor. Though diet changes are often helpful, they can not prevent the disease completely, because it is caused by many factors. Genetics and management are two additional major factors (Knight, *et al*., 1985). Since genetics is a major contributor, elimination of all genetically predisposed animals would solve much of the problem. However, even if we could manage to identify all carrier animals it is doubtful if a purge would occur since many of the producers of offspring with bone formation problems are also producers of talented athletes, and therefore we persist in breeding them in spite of their possible genetic flaws.

Management factors

Management of the growth of a foal consists primarily of controlling diet and exercise. Therefore, we will consider them together in the evaluation of possible sites of bone formation disturbance. The formation of functional bone requires the delivery of 1) the right material; 2) in the right proportions; 3) with the right cofactors; 4) at the proper rate; 5) for the right stimulus. If any of the factors are disturbed, the possibility of poorly formed or malformed bone exists.

Delivery of "the right materials" indicates that the three major components of bone, calcium, phosphorus, and protein must be made available at the site of bone formation. A severe or prolonged Ca++ or PO_4 deficiency can limit the availability of these mineral substrates of bone. The bone protein, osteoid, is manufactured at the site of bone formation, so unless deprivation is severe, it is rarely a cause of depression of osteoid formation. A more frequent cause of malformation of bone due to Ca++, $P0_4$, or protein deficiency at the site of bone formation, is an interruption of the delivery of these nutrients due to disruption of the blood supply. This interruption may result from traumatic, infectious, and possibly even metabolic causes. It is usually localized and therefore creates a localized disruption of bone formation. Any obstruction of the tenuous vascular supply to the growing bone will result in the lack of transformation of the degenerating cartilage to calcified cartilage and eventually to bone. Vascular obstruction can result in the interruption of bone formation locally even though the bodywide supplies of Ca++, PO_4 and protein may be plentiful. The most common localized vascular obstruction is fibrous tissue left behind after resolution of an infectious process. A hematogenous shower of bacteria, as is frequent in a foal, can create multiple potential sites of disturbed bone formation simultaneously. Trauma can also strike at any site or in multiple sites.

The delivery of the Ca++ and PO_4 in reasonably "the right proportions" is necessary for the hydroxyapatite crystal to be formed and deposited within the osteoid matrix. The crystal Ca (10){PO_4}(6){OH}(2) requires the delivery of calcium and phosphorus at about 1.7 to 1 to form bone. The body can fine tune needs as long as reasonable proportions and quantities are fed. The most well known disparity in Ca++ and PO_4 balance is secondary nutritional hyperparathyroidism caused by feeding excess phosphorus and deficiencies of calcium. The resultant robbing of the skeleton of the calcium necessary for the more critical metabolic needs results in an inability to reform the hydroxyapatite crystal after routine bone remodeling. This gradually reduces skeletal content of hydroxyapatite leaving the osteoid without mineral to stiffen its structure. The more rapid remodeling in the flat bones make the clinical signs apparent in the jaw ("rubber jaw") first, but in the growing animal OCD can also result. Due to current owner awareness, nutritional secondary hyperparathyroidism is unusual.

What is occasionally seen in the current management systems is absolute calcium deficiency due to a relatively low level of both nutrients. Absolute deficiencies in calcium in the presence of normal phosphorus in foals is most common and will result in poor bone strength seen as an increase in fractures of the more trabecular sesamoid and third phalangeal bones as well as physitis at the most active, most vulnerable physes at the time. Therefore, clinical signs referable to deficiencies in structural integrity of cancellous bone should cause the available levels of Ca++ and PO_4 to be examined.

"The correct cofactors" for bone formation are still largely speculative. Vitamins A, C and D, copper and zinc are proven cofactors, but manganese and others may be needed in small amounts. Horsemen are very aware of the need for vitamins, but are unaware, in most instances, of the horse's ability to provide these vitamins when supplied with sunshine and pigmented roughage.

Relatively recently, the trace minerals copper and zinc have seen increased attention (Knight, *et al*., 1985; Knight, *et al*., 1991).[1,3] Their supplementation appears to help protect the quality of newly formed bone in the foal. In our experience, copper supplementation is most effective when trying to prevent the formation of poor quality bone (step 2) such as with physitis and less effective with retarded cartilage mineralization (step 1).

"At the correct rate" recognizes the spectacular coordination, among the many physeal functions, which must result in growth to a body size of 5 times the human in about 1/10th the time, must result in limb straightening of any angular limb deformity, and must bear a body weight increase of 2 to 3 lbs per day. Slowed bone formation, resulting in inferior bone quality, can occur if deficiencies or imbalances exist. However, the most frequent bone formation rate derangement encountered is the addition of body weight faster than the natural or even accelerated bone growth rate can accommodate. The juvenile physis is wide, made up of a thick layer of the growth cartilage. It gradually thins to decrease the vulnerability to trauma as the stress, a

result of body size and activity, increases with age. The more distal the growth plate the less the soft tissue protection by muscles and tendons, and therefore the more vulnerable the physis. As a result, the distal physes close earlier than the more proximal physes.

Human intervention to alter development and form of an appealing halter horse often results in an oversized body on normal sized limbs. Since the maturation of the physis is primarily time dependent, acceleration of the body growth can overload the physes which are normally mature and closed prior to attainment of a particular body size. Even though bone formation may be normal, the signs are the same as poor quality bone. Normal activity of the horse then becomes traumatic to the growing bone due to the body size. Injury to the growing bone can result in pain, structural damage and OCD if the traumatic insult results in interruption of vascular ingrowth at a localized epiphyseal bone formation site.

Pharmacologic alterations in bone formation rates may also be possible. We have correlated extended Dexamethasone use with the occurrence of OCD in our practice. Though speculative, the catabolic effects of corticosteroids may affect the anabolic process of new bone formation. These effects are generally body wide but clinical signs become apparent only in the rapid bone formation sites.

The "correct stimulus" for bone formation is the exercise which the newly formed bone is expected to withstand. The gradual increase in body size combined with the gradual increase in strength and activity causes the bone to be gradually "trained" to accept the load to be applied. Danger appears when this gradually increasing load, which results in gradual adaptation, is interrupted.

Stress, a product of body load and activity, dictates the size and number of trabeculae produced to withstand the stress applied. Since growth occurs at such a rapid rate in the foal, stress is a continual necessity to dictate proper bone formation. Deprivation of exercise due to illness of the mare or foal, or other influences such as weather or management practices, can leave the foal's newly formed bone inadequate when normal exercise is resumed. A few days is generally of little consequence, but a few weeks can result in a significant amount of structurally inferior bone. When exercise is resumed the now larger foal's normal activity can change stress to trauma on the poorly prepared bone. The injury to the bone can disturb bone formation to the point that physitis and OCD can be created. If exercise is curtailed for a prolonged period, it must be resumed gradually to circumvent the risk of disturbed bone formation. If the return to exercise is accompanied by signs of cancellous bone overload (most evident at the rapidly growing physes as inflammation and lameness) then the quantity of daily exercise must be reduced until the inflammation subsides and then exercise can be gradually increased. Conformation flaws are another way of increasing the load (stress) on a physis. Angular deformities have a very marked propensity for increasing the load in a localized area of the physis. If this load magnification, combined with exercise, creates physitis, either the angular deformity must be corrected or the exercise curtailed until the physis matures or compensates for the overload.

Summary

There is little doubt that multiple factors are involved in the development of developmental orthopedic disease in young horses. Some are identifiable, some are as yet unknown. Genetic predisposition, conformation, lack of exercise and nutrition have been said to contribute to the etiology of these disease states. Of all the factors, nutrition has received the most attention in recent years because it is the only factor under direct control of the management.

Results of the recent study at Ohio State (Knight, *et al*., 1985; Gabel, et al., 1987) indicate that there is a strong inverse relationship between ration and farm problems. It appeared likely in that study that calcium, phosphorus, copper and zinc deficiencies were involved in the development of epiphysitis, contracted tendons and OCD lesions in yearlings. Since protein and energy levels were similar on all farms in the study, it is unlikely that they are directly involved in this disease process. In fact, farms with the most problems (highest scores) had the lowest protein levels in the study. From the data collected in the survey, it is apparent that the quality of nutrition, rather than the quantity, plays a significant role in the development of metabolic bone disease.

In addition to proper nutrition, proper management practices are part of the development of a normal foal. Any variation from the original ecological niche of the horse presents a potential for disruption. Extreme variations from normal management practices should be looked at most closely. The integration of nutrition and management is the art of raising a horse.

In the investigation of a "herd" problem the bone formation disturbances of the individual must be identified and investigated. The same herd wide insult causes different clinical signs when different anatomic sites are involved and vary in severity and signs when different aged individuals are involved. It is only by identifying the commonalities of a herd situation that causes and eventually therapy can be identified.

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