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Charles W. Raker  
*University of Pennsylvania*

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## Clinical Observations of Bone and Joint Diseases in Horses

### Abstract

Bone and joint diseases in the horse, as observed by the veterinary clinician, assume many forms frequently producing signs of lameness. The bones or joints of the limbs are most commonly affected and many factors which operate either singly or collectively produce the lesions observed.

### Disciplines

Large or Food Animal and Equine Medicine | Medicine and Health Sciences | Veterinary Medicine

# CLINICAL OBSERVATIONS OF BONE AND JOINT DISEASES IN HORSES

By C. W. RAKER

*Lawrence Baker Sheppard Professor of Surgery  
School of Veterinary Medicine  
University of Pennsylvania  
Philadelphia, Pennsylvania*

**B**ONE and joint diseases in the horse, as observed by the veterinary clinician, assume many forms frequently producing signs of lameness. The bones or joints of the limbs are most commonly affected and many factors which operate either singly or collectively produce the lesions observed.

While diagnosis of the lesion resulting in lameness and the treatment of lameness are challenging problems, we first require a deeper insight into the causation and pathogenesis of bone and joint disease. A brief review of the more recent literature reveals how serious are our deficiencies in this sphere.

Compared to investigation on degenerative joint disease in man, that in the horse has not been studied in very great depth. Sippel<sup>7</sup> has published on the pathogenesis of equine joint disease, and Van Pelt<sup>9,10</sup> has reviewed the anatomy and physiology of the diarthrodial joints and presented data on the properties of equine synovial fluid. A summary of our current knowledge in the area of nutrition and endocrine disorders and certain other etiological factors has been presented by Rooney.<sup>5</sup> Adams<sup>1</sup> has made a substantial contribution to the field of lameness in the horse, particularly in the areas of etiology, diagnosis, and therapy of the more common lesions.

Primary disease of soft tissues and fracture of long bones are subjects unto themselves and will not be discussed at this time, except as they may relate to diseases of the diarthrodial joints of the limbs.

## ETIOLOGY

The most common lesions observed in bone and joint disease are thought to be the result of a *wear-and-tear* syndrome. The work a horse is made to perform is often extremely stressful, and racing breeds are particularly susceptible to stretching or tearing of collateral ligament attachments and to injury of the joint capsule and joint surfaces. Weight bearing and concussion at high rates of motion increase the loading of the joints, resulting in an increased rate of wear and trauma to the articular surfaces and underlying subchondral bone.

Direct trauma, such as might result from being kicked or falling, may also result in injury to these tissues.

Many other factors may also be important, but unfortunately, we know very little about them. Endocrine disorders may well affect normal bone development as shown by the work of Krook and Lowe<sup>3</sup> on nutritional secondary hyperparathyroidism.

Inadequate nutrition may also be of importance in respect to the development of bone and joint disease in the horse. Kelsner and Callender<sup>2</sup> and more recently Trum<sup>8</sup> have suggested that nutritional deficiencies are of importance.

Infection of a joint as a result of systemic disease, trauma, or intra-articular therapy leads to septic arthritis and degenerative disease. Alteration of the synovial fluid and enzymatic dissolution of the articular cartilage leads to erosion of the joint surface, exposure of the subchondral bone, eburnation, and possibly ankylosis.

Improper care of the feet and improper shoeing can lead to disease of the bone and joints of the limb and, most particularly, to diseases of the foot.

Deviation from the normal conformation of the limb is of particular significance as a predisposing cause of injury to ligaments and joints and frequently leads to degenerative disease or fractures. Certain defects in conformation are thought to be hereditary.

Muscle fatigue resulting from overwork, inadequate physical conditioning, or both can lead to overflexion and overextension of joints. Also, stress to supporting soft tissue is increased at work and results in fractures within a joint, long bone fractures, and signs of degenerative joint disease.

Additional predisposing causes are believed to include age and maturity of the appendicular skeleton, type of surface over which the horse is worked, and type of work performed.

Clinical observations suggest that the more common forms of bone and joint disease have a multifactorial causation; for example, a nutritional or conformational defect, or both, when added to the wear-and-tear stress on a joint, result in signs of disease.

Thus, though many etiological factors are worthy of consideration, it is usually impossible to explain fully the cause of a specific bone or joint lesion.

#### PATHOPHYSIOLOGY AND CLINICAL FINDINGS

The course of events which follows injury to bones and joints varies depending upon the area and tissues affected, but the first signs are those associated with inflammation. Trauma to the periosteum results in a painful lesion, which may, with appropriate therapy, be repaired so that no permanent signs or effects remain. However, nature usually responds to the injury in a manner which leads to a permanent thickening of the cortical bone.

Stretching or incomplete tear of ligamentous attachments results in a periosteal reaction and the proliferation of new bone in the affected area. As repair proceeds, this new bone production can be observed both clinically and radiographically. It is important to note that the new bone may not become evident on a radiograph until several weeks following the injury, the reaction being comparable to the formation of a fracture callus.

Splints result from a tearing of the interosseous ligament between the third metacarpal bone and the second and fourth metacarpal bones. Splints in the hind limbs occur less frequently but result from the same cause. The splint bones are weight-bearing bones without a distal opposing joint surface. Thus, weight applied to the proximal joint surface, especially when accompanied by defects in conformation, leads to a tear of the interosseous ligament. Anatomical deviations in the equine carpus, as noted by Rooney,<sup>6</sup> may explain the high incidence of splints known to affect the second metacarpal bone.

Degenerative joint disease and fractures of the carpus

occur frequently, especially in breeds used for racing. The initial signs are those of a synovitis of the radiocarpal or intercarpal joints, more frequently the latter. The increased production of synovial fluid leads to a distension of the joint capsule. Changes in some of the chemical components and an increase in the leukocytes of the fluid follow. Depending upon the therapeutic measures employed, the disease may or may not progress. The changes which occur in the synovial fluid affect the nutrition of the articular cartilages. The lowered viscosity and less efficient lubricant qualities enhance wear of the articular cartilage and reduce its effectiveness as a shock absorber. The cartilage loses its translucence and becomes discolored. The articular surface may show signs of scoring or grooving, and the eventual loss of cartilage leads to eburnation and exposure of the subchondral bone. Concurrently there occurs a proliferation of the cartilage and bone at the margins of the joint surface with the formation of osteophytes or spurs leading to a remodeling of the joint.

In chronic cases the joint capsule is thickened and the synovial membrane hypertrophied, and these changes result in so-called villous synovitis.

Damage to the attachments of the joint capsule and interosseous ligaments is observed. In time, this trauma is reflected radiographically and clinically by a firm fibrous proliferation. These lesions are observed most frequently on the central cranial surface of the radial carpal bone and on the distal end of the radius.

Lesions within the joint space are often very difficult to recognize. Many joints explored surgically reveal the presence of relatively extensive degenerative disease, which was not detected on clinical or radiographic evaluation before surgery.

Fractures of the carpus are common and three basic types are recognized. Chip fractures are the most common. In order of frequency of occurrence, they affect the distal margin of the radial carpal bone and the proximal margin of the third carpal bone on the craniomedial surface of the intercarpal joint. Chip fractures in the radiocarpal joint usually occur at the distal surface of the radius on the craniolateral aspect of the

joint. Chip fractures of other carpal bones and at other locations occur less frequently.

Slab fractures usually involve the third carpal bone. Slab fractures of the radial and fourth carpal bones have been observed less frequently.

Multiple fractures of the carpal bones are less common, usually affect two or more of the bones, and are of various types.

Osteophytes or bone spurs, originating from the cranial borders of the carpal bones, are frequently observed. Continual trauma results in proliferative lesions within the joint which may be enhanced by overextension of the carpus. A *calf-kneed* conformation of the front limb predisposes the carpal joints to trauma at the anterior joint margins. The distal margin of the radial carpal bone and the proximal margin of the opposing third carpal bone are the most common sites for the development of spurs. Use and motion of the affected joint is altered and at times is quite limited. Progression of these lesions leads to a partial or complete loss of flexion and a bridging over of the cranial surface of the joint. The distal end of the radius and opposing proximal margin of the radial carpal bone may show similar lesions. The proximal and distal margins of the intermediate carpal bone are affected less frequently.

Viable chip fractures, exhibiting little or no displacement, will heal but frequently result in a spurring from the margin of the joint. In these cases, the fracture may recur and additional proliferation from the margin of the bone may take place.

Lesions which involve the carpal joints are observed most frequently on the cranial surface of the carpal bones. While these may result from striking the carpus on a stall door, falling, and similar types of trauma, the majority are related to stress, concussion with weight bearing, and muscle fatigue during fast work with overextension and overflexion of the joints. The added handicap of poor conformation of the carpus increases the incidence of carpal disease.

The proximal sesamoid bones are subject to the same

forces of stress as the carpus. A short, upright pastern results in decreased angulation of the metacarpophalangeal joint which increases concussion and loading of the joint. A long sloping pastern, while providing a longer fulcrum and decreasing joint loading and concussion, increases stress to the collateral ligaments of the joint and to the suspensory ligament. Conformational defects of this type greatly increase the rate of wear and tear to the soft tissues, sesamoid bones, and to the metacarpophalangeal joint. Improper trimming of the foot or poor shoeing, especially a high heel, increases the tension on the suspensory ligament which may lead to a tearing of the attachments of the suspensory ligament to the abaxial surface of the sesamoid bones. A desmitis of the suspensory ligament and/or inferior sesamoidean ligaments results and, at times, there may be a stress fracture of the sesamoid bone. Degenerative sesamoiditis, reflected radiographically by a demineralization and proliferative deformity of the bone, occurs frequently. While trauma applied directly to the sesamoid bones may result in similar lesions, it is believed that indirect trauma is the most important cause.

Stress fractures of the apex or base of the sesamoid bone occur frequently in the racing breeds. Comminuted fractures of the sesamoid bones are less common, as are fractures through the middle of these bones. It is noteworthy that the incidence of apex fractures of the sesamoid bones is highest in the hind leg, especially in the Standardbred horse. Fractures of the base of the sesamoid bones are observed most frequently in the front leg and the incidence is higher in the Thoroughbred horse. Fractures of the base of the sesamoid bone are usually T-fractures with two large fragments; apex fractures are primarily of a simple type.

Fractures of the sesamoid bones are known to heal slowly without the formation of a good bone callus. Occasionally, even after 6 to 12 months or longer, no osseous repair is evident and signs of lameness recur if the horse is worked. Recurrence of the fracture in those cases in which radiographs show apparent healing is common. This course of events could be explained by the poor quality of the fibroosseous or osseous union. However, the statement that failure of bone union is



the result of inadequate immobilization of the joint is questionable. Inadequate osseous repair may be associated with differences in circulation to and the structure of the sesamoid bone.

Disease of the metacarpophalangeal and metatarsophalangeal joints result from the same basic causes previously noted. However, the range of motion in these joints is greater than in the carpus or interphalangeal joints. Lesions involving the attachments of the joint capsule, collateral ligaments, and extensor tendon occur with great frequency. Many breeds are affected as are horses performing many different kinds of work. Observations on gross specimens, especially in older horses, reveal the extreme degree of wear to which these joints are subjected. Grooving or scoring and thinning of the articular cartilages are nearly always present. Osteochondrosis and eburnation are common findings combined with remodeling of the joint surfaces and the occurrence of spurs on the cranial margin of the proximal end of the first phalanx. Chip fractures of the proximal end of the first phalanx result from extreme overextension of the joint and at times become *free bodies* within the joint.

Degenerative disease of the proximal and distal interphalangeal joints (high and low ringbone) is observed more frequently in the older horse and once again reflects trauma to the attachments of the joint capsule and associated ligaments and tendons. It is seen most commonly in pleasure and cutting horses, and the etiological factors are similar to the one affecting other joints. The statement that ringbone is hereditary is open to question. While defects in conformation which predispose to the development of the disease might be inherited, it is unlikely that the disease itself can be transmitted.

Concussion with loading of the leg accompanied by speed, poor footing, improper shoeing, improper care of the foot, and possibly underlying disease of the bone are thought to account for fractures of the third phalanx and navicular bone. Fractures of the third phalanx occur most frequently in the front feet. The fact that approximately 60 percent of the body weight, plus the weight of the rider, is carried by the front legs may be an important factor in this disease. With respect

to healing, fractures of the third phalanx and less frequently of the navicular bone, are similar to fractures of the sesamoid bone. Union of fractures of the third phalanx is slow and often inadequate to meet the stress of work. Thus, a digital neurectomy may be indicated in cases of inadequate or absent fracture repair. A neurectomy relieves discomfort to the affected area of the hoof, but does not correct the lesion and leads to even more rapid breakdown. Also, painful neuromas occur in approximately 25 percent of operated cases, a complication which should always be considered.

Ossification of the collateral cartilages of the third phalanx is said to be a disease of the aged horse. However, this statement is not entirely accurate as the disease has also been diagnosed in 2- and 3-year-old horses. A consideration of the mechanics of the hoof indicates that the collateral cartilages serve as shock absorbers. Thus, concussion occurring almost continuously can lead to degrees of ossification of the cartilages, the incidence increasing with age. Many factors may influence the physiological function of the hoof, leading to ossification early in the life of the horse. A hoof which is small in proportion to the weight carried by the leg increases both weight bearing and concussion. Incorrect trimming or shoeing and an absence of frog pressure can limit the normal function of the built-in shock absorber system. A flat sole, contracted feet or heels, and hard surfaces increase the intensity of concussive forces applied to the hooves, all of which influence the age at which ossification of the collateral cartilages may develop.

The degree of lameness is dependent upon the relative activity of the ossifying process in relation to the character of the feet and the work performed. Rapid ossification is accompanied by signs of lameness, whereas, in a slow degenerative process clinical signs may be absent. It is of interest to note that while a fracture of an ossified cartilage will heal with a firm bony union, there is often more than one center of ossification within the cartilage. These random bony lesions should not be confused with a fracture of the cartilage.

Navicular disease is a chronic, usually progressive, degenerative disease of the navicular bone and its associated

bursa and flexor tendon, which may also extend into the pedal joint. Being a disease in which the incidence increases with age, one suspects that wear and tear, combined with concussion and weight bearing, are the most important causes. Undoubtedly, improper care of the foot is also important. A relatively high incidence of navicular disease also occurs in young horses, especially in Quarter horses.

The type of work performed by a Quarter horse frequently involves fast starts, quick stops, and turning. However, from the standpoint of concussive forces, it would appear unlikely that the incidence of navicular disease should be higher in this breed than in the racing Thoroughbred or Standardbred horse.

The usual course in a case of navicular disease is one of progression until irreversible degenerative disease has developed. In a few cases, clinical recovery has been observed. It is dangerous to base a positive diagnosis of navicular disease on radiographic findings alone. The clinical signs are more diagnostic. The absence of positive radiographic findings should not negate a positive clinical diagnosis.

Bone and joint diseases of the vertebral column and pelvis present a difficult diagnostic problem, except when the most overt signs of disease are present. It is anticipated that newly available radiographic equipment will aid materially in making a definitive diagnosis.

Diseases of the femerotibial and femeropatellar joints remain challenging and somewhat difficult problems. Upward fixation and subluxation of the patella are associated with the conformation of the hind leg. A horse with less than normal angulation of the stifle joint and an upright tibia is predisposed to upward fixation and subluxation of the patella. The problem occurs frequently in ponies and in Standardbred horses. It seems reasonable to assume that the driving force of the hind legs of the Standardbred horse, at gaits not normally used for speed, may result in stress and stretching of the patellar ligaments leading to subluxation.

Several horses exhibiting fractures of the patella have been treated. Repair of a fracture of the patella is frequently unsatisfactory and leads to degenerative disease of the joint

and to chronic lameness. Chip fractures of the distal margin of the patella have been removed successfully in a few horses. Removal of chip fractures from the proximal aspect (base) of the patella has not been highly successful, while complete removal of the patella has been entirely unsatisfactory. The extensive insertion of the quadriceps femoris muscle onto the base and cranial surfaces of the patella probably accounts, in part, for the poor natural and surgical repair.

Instability of the femorotibial joint resulting from a *loose* lateral collateral ligament results in the legs being carried forward in abduction.<sup>4</sup> This gait is frequently observed in the Standardbred horse and less often in the Thoroughbred. Radiographs of most, if not all, of these joints show evidence of an incomplete ossification of the proximal one-third of the fibula. It is assumed that the developmental failure of the fibula aids in the development of an instability of the lateral femorotibial ligament because of its insertion onto the head of the fibula. However, racing on a banked racetrack and maneuvering turns at high speed cannot be discounted as a cause of this syndrome.

Disease of the equine tarsus is similar to the bone and joint disease affecting other joints. Chip fractures of the fibular and tibial tarsal bones and distal end of the tibia are common. Most fractures of the tibia and of the fibular tarsal bone appear to result from kicks or falls. Chip fractures of the tibial tarsal bone usually affect that portion of the trochlea which lies adjacent to the central tarsal bone. Both the lateral and medial trochlea may be involved. Chip fractures of the distal anterior margin of the tibia have been observed less frequently. These fractures probably result from overflexion of the tibiotarsal joint with compression of the opposing surfaces of the bones.

Slab fractures of the central and third tarsal bones are observed less frequently. They probably result from severe compression of the bone on its anterior margin.

Degenerative joint disease in the tarsus occurs most commonly in the proximal and distal intertarsal joints and in the tarsometatarsal joint (bone spavins). Poor conformation of the tarsus predisposes these joints to excess stress and to an

increased rate of wear and tear. A horse, standing under behind or having a sickle-shaped hock, is likely to develop a desmitis of the plantar ligament (curb) and also bone disease. A tarsus with too straight a conformation or a horse which stands with his hind leg behind him is likewise predisposed to degenerative bone and joint disease of the tarsus.

Degenerative disease of the tarsus is common in many breeds. Horses which apply great stress to the tarsus are especially prone to disease. The Quarter horse at work may start fast, turn quickly, and stop within a very short distance. All of these acts result in extreme stress to the tarsus. Horses used for jumping stress the tarsus at both the take off and the completion of the jump. Harness horses stress the tarsus to its maximum while racing at gaits not normally considered as speed gaits and in association with extreme concussive forces.

While all of the marginal surfaces of the intertarsal and tarsometatarsal joints are subject to proliferative bone disease, the medial surface of these joints is the most common site for the development of lesions. A proliferative periostitis, which may involve the cunean bursa and tendon, is the usual clinical and radiographic finding. Lameness results, accompanied by a shortened stride and a tendency to wear the toe; if unshod, the heel will grow high.

Degenerative disease extending into or originating within the intertarsal and tarsometatarsal joints (occult spavins) can be difficult to diagnose. Typical signs related to a lameness in the tarsus are evident. Diagnostic anesthesia is usually unsatisfactory; however, good quality, properly positioned radiographs will usually enable the clinician to confirm the clinical diagnosis. The prognosis is guarded, as signs of lameness will persist until ankylosis of the joint and inactivation of the degenerative disease occurs.

#### THERAPY

Treatment of lesions resulting in lameness is often non-specific and unsatisfactory. Fractures of long bones require the use of orthopedic measures and devices, where applicable, to reduce and stabilize the fracture during repair.

Chip fractures of many long bones and in several joints

can be removed surgically. This seems to be indicated when the fragment is displaced and repair by natural processes is unlikely.

Spurs or osteophytes of bones along the margin of a joint are in some cases managed best by surgical removal of the proliferative bone.

Fractures of the distal one-half, or less, of a splint bone can be operated upon and the distal fragment removed. Surgical removal of the distal segment is indicated in fractures in which the distal segment is displaced or when a marked separation at the fracture site exists.

Fractures which affect one-third or less of the apex of the proximal sesamoid bone are treated by surgical removal of that portion of the bone and the prognosis is favorable. The prognosis on fractures of the base of the sesamoid bone, while they can be removed surgically, is guarded to unfavorable. It has been estimated that approximately 25 percent of the cases exhibiting fractures of the base of the sesamoid bone will race soundly following surgical removal of the fragments. This figure compares unfavorably to the estimated 75 percent of horses which race soundly following surgical removal of a fracture at the apex of the sesamoid bone.

Selected cases exhibiting chip or slab fractures of the femorotibial and tarsal joints are operated upon with removal of the chip of bone or reduction and stabilization of the fracture.

Rest and time are two of the most important and least appreciated therapeutic measures. Physical therapy, including radiotherapy, hydrotherapy, diathermy, and electrical stimulation and similar modalities also have a place in the therapy of lameness.

Antiinflammatory therapy with corticosteroids, phenylbutazone, and similar drugs is useful, providing one recognizes that their administration can sometimes be detrimental to the future soundness of the horse. Use of these agents alone, in the presence of fractures and infection, is contraindicated.

Vesication and cautery, while they still may have a place in treating certain specific lesions of bones and joints, appear to be of little value. Hopefully, the use of the agents will continue to decline.

Degenerative joint disease, with involvement of the articular surfaces, is difficult to treat, and signs of lameness persist until ankylosis of the joint is relatively complete. Measures to hasten ankylosis, such as surgical arthrodesis, and other measures to destroy the articular cartilage have been developed. Although wear and tear or use of a joint will hasten the degenerative changes, allowing a horse to use the affected joint seems to be more beneficial than a program of complete rest or immobilization. The clinical signs of lameness and associated pain can often be controlled by administration of antiinflammatory drugs.

#### CONCLUSIONS

Bone and joint disease in the horse is one of the most common problems facing the veterinarian and the horse industry. It results in enormous economic losses. Diagnosis and therapy of lesions producing lameness is challenging. We still know relatively very little concerning the etiology and prevention of bone and joint diseases. Studies are needed in the areas of endocrinology, nutrition, genetics, and pathology. Also, we need more information on the relationship between bone maturity and stress, on the importance of the various types of racing surfaces, and on improved methods both to condition a horse and to evaluate its condition.

Intensive studies on the biomechanics of the joint, its nutrition, and its underlying congenital or structural weaknesses are needed.

Until more basic information is available, equine bone and joint disease will continue to be a major problem confronting the veterinary profession.

#### REFERENCES

1. Adams, O. R. Lameness in horses. 2d. ed., Philadelphia: Lea and Febiger, 1966.
2. Kelsner, R. A., and G. R. Callender. Equine degenerative arthritis. *Vet. Med.*, *33*, 307, 1938.
3. Krook, L., and J. E. Lowe. Nutritional secondary hyperparathyroidism in the horse. *Path. Vet.*, *1*, Sup. 1, 1964.
4. Mackay-Smith, M. P., and C. W. Raker. Mechanical defects of the equine stifle-diagnosis and treatment. *Proc. Amer. Vet. Med. Assoc.*, 1963, pp. 82-85.
5. Rooney, J. R. Equine medicine and surgery. 1st. ed. Santa Barbara, Calif.: American Veterinary Publications, 1963.

6. Rooney, J. R. Foreleg splints in horses. *Cornell Vet.*, 56, 259, 1966.
7. Sippel, W. L. Equine degenerative arthritis. *Cornell Univ. Thesis*, 1942.
8. Trum, B. F. Pathogenesis of osteoarthritis in the horse. *Lab. Invest.*, 8, 1197, 1959.
9. Van Pelt, R. W. Anatomy and physiology of articular structures. *Vet. Med.*, 57, 135, 1962.
10. Van Pelt, R. W. Properties of equine synovial fluid. *Jour. Amer. Vet. Med. Assoc.*, 141, 1051, 1962.

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## RADIOGRAPHIC DIAGNOSIS OF BONE AND JOINT DISEASES IN THE HORSE

By JOE P. MORGAN

*Assistant Professor, Department of Radiology and Radiation Biology  
College of Veterinary Medicine and Biomedical Sciences  
Colorado State University  
Fort Collins, Colorado*

USE OF radiography for diagnoses of bone and joint diseases in horses has largely been limited to examinations of extremities. New equipment is currently in use that permits consistently adequate evaluation of the pelvis, vertebral column, shoulder joints, and hip joints. Because of limited experience in these areas, they will not be discussed in this paper.

There are specific problems in performing radiographic procedures in the horse. Because of the size of the animal, it is difficult to obtain the exact positioning required. Because of the animal's nature, motion during exposure is a constant problem. Cleanliness of the examined part can be difficult to obtain. An example of a problem of this type is radiography of the third phalanx.

A high quality radiograph may be considered to be one with maximum detail, good contrast, good radiographic density, and minimum distortion. To obtain the most from a ra-