Volume 46 | Issue 1 Article 5

1984

Navicular Disease: New Developments With an Old Dilemma

Dean E. Christianson
Iowa State University

Eric L. Reinertson
Iowa State University

Navicular Disease: New Developments With an Old Dilemma

Dean E. Christianson, BS*
Eric L. Reinertson, DVM**

Navicular disease (distal sesamoiditis, podotrochilitis, podotrochleosis, bursitis podotrochlearis) affects the distal sesamoid (navicular) bone and its surrounding structures in the equine limb. The disease is characterized by lameness of varied degrees, usually in the forelimbs. It is one of the most common causes of equine lameness seen by veterinarians. It is a difficult disease for clients to deal with because there is no known cure for navicular disease, and most treatments are only palliative.

Navicular disease has been recognized almost as long as the horse has been domesticated. The veterinary profession has devoted much time and effort to the study of this disease. However, much controversy remains concerning its etiology, pathogenesis, diagnosis, and treatment. Through the years, many theories have been proposed, but none have been proven. There are no black and white answers. Those who study navicular disease must approach it with an open mind and use all the information available to draw their conclusions.

Anatomy

In order to understand navicular disease, the relevant anatomy must first be understood. The navicular bone is a small, shuttle-shaped bone over which rides the deep flexor tendon. It provides an articular surface for the second phalanx, permitting distribution of the force of that bone over a larger area—the third phalanx and the navicular bone—rather than the third phalanx alone. The major function of this bone, however, appears to be in providing a constant angle of insertion for the deep flexor tendon on the third phalanx.[1]

Three ligaments are clinically important. There are two suspensory ligaments of the navicular bone. They originate on either side of the distal end of the first phalanx and cross obliquely downward and backward over the sides of the distal phalanx. They attach chiefly on the ends of the proximal border of the navicular bone.[2] The distal ligament of the navicular bone attaches to the distal border of the navicular bone and to the third phalanx near the insertion of the deep flexor tendon.[3]

The navicular bursa is found between the deep flexor tendon and the navicular bone; it extends about 1 to 1.5 cm proximal to the navicular bone and distally to the insertion of the deep flexor tendon.[4]

The vascular supply of the navicular bone has become an area of increased interest in recent years because of the possible role of the blood supply in the pathogenesis of navicular disease.

The palmar arterial branches of the second and third phalanx originate from the medial and lateral digital arteries and give rise to the major blood supply to the navicular bone. The proximal and distal borders of the navicular bone receive arteries from a complex network arising from the palmar branches of the second and third phalanx, respectively. These anastomotic networks provide collateral support to help ensure that the navicular bone gets adequate blood supply.[5]

Some differences between the thoracic and pelvic limbs' vascular supply should be noted. In the pelvic limb, it is possible that the concavity of the plantar aspect of the second phalanx protects the arteries of the proximal network from compression and transient occlusion by pressure from the deep flexor tendon.[5] This protection may be one contributing factor in the explanation of why navicular disease is so much more common in the forelimbs. Also, the distal supply of the pelvic limb gives rise to

*Mr. Christianson is a fourth-year student in the College of Veterinary Medicine at Iowa State University.
**Dr. Reinertsen is an associate professor in Veterinary Clinical Sciences at Iowa State University.

a greater average number of primary (3.4) and secondary (9.4) arteries than does the thoracic limb (2.2 primary and 7.7 secondary). If occlusion is significant in the case of navicular disease, the thoracic limb would appear more susceptible to ischemia.[5]

The nerve supply to the navicular area is furnished by the medial and lateral palmar nerves. These nerves bifurcate above the fetlock joint, forming anterior and palmar digital nerves with the palmar branch supplying the navicular area. Many anatomical variations of the artery and nerve supply are common. There may be small subcutaneous branches arising high on the palmar digital nerve that travel with the ligament of the ergot. Other minor variations are common.[3] This nerve supply variation is significant when considering the success of a palmar digital neurectomy to alleviate the pain of navicular disease.

Clinical Signs

Horses with navicular disease usually have intermittent, bilateral forelimb lameness with an insidious onset. Navicular disease has been described in the hind limbs, but it is rare.[6] The lameness is usually aggravated by work and alleviated with rest in its early stages. The owner may report that the horse does not extend its forelimbs fully, and often a shoulder lameness is suspected.[7]

As the lameness progresses, a horse may refuse to take a particular lead at the canter, want to make square turns, or fall to the inside on sharp turns. These actions are an attempt to alleviate the irritation of the lameness. Trainers may feel the horse is being sloppy and work it harder to improve performance, thus exacerbating the condition.[2]

An important factor in signs of navicular disease is the horse's temperament. Bold, stoic horses may allow the disease to progress further before showing clinical signs of lameness. Distractions (i.e. roping, jumping) may make the horse appear less lame by taking his mind off the pain.[2]

As the disease progresses, the hoof begins to change its shape. In reaction to pain, the horse begins to bear more weight on his toe and less on his heels and quarters ("kicking dust"). This allows contraction of the heels. Some horses with wide, flat hooves that are too weak to contract will have little or no heel because the hoof does not produce the necessary horn. Thus, many horses with navicular disease do not develop the classical tall, narrow, boxy hoof.[2]

Further progression may lead to a pointing stance in which the horse rests one foot intermittently and places it out in front. The horse may switch feet periodically since pain is often bilateral. If one foot is more severely affected, he may consistently point with one foot.

Necropsy Findings

The pathological findings in navicular disease vary tremendously and are inconsistent. Yellow discoloration of the navicular bone's flexor surface and the adjacent deep flexor tendon is a common finding, but it is probably due to normal "wear and tear" and is not significant.[8,9]

Cartilage erosions on the flexor surface are also a consistent finding. Some believe these lesions to be significant[8] while other feel these erosions are also present in normal horses.[9] Sometimes, the fibrocartilage is eroded to the extent that subchondral bone is exposed.[8]

Often, in the later stages of the disease, adhesions develop between the navicular bone and the deep flexor tendon. The adhesions are often composed of circular areas of granulation tissue entering the bone through the area of erosion. There is also necrosis and partial hyalination of the deep flexor tendon. This finding is not confined to the superficial portions of the tendon, but extends into the tendon core.[8] No neovascularization of the injured tendon occurs. The granulation tissue that adheres the bone and tendon is derived from the marrow spaces of the bone.

Also associated with navicular disease are sclerotic osteitis, osseous metaplasia of the proximal and distal suspensory ligament,[8] and increases in the number and diameter of vascular channels traversing the flexor surface.[10] Bone necrosis and arterial thrombosis have been found by some investigators to be a consistent finding.[9]

Predisposition

All horses are candidates to develop navicular disease, but some horses seem more prone to the condition. It is generally considered that quarter horses are more susceptible than other breeds. This is believed to be due to genetic selection for large muscle mass and small feet. This combination increases both concussion and compression on the front feet. In practices involving racing animals, both thoroughbreds and standardbreds are commonly affected. In

one study, a larger percentage of navicular cases involved standardbreds than thoroughbreds.[11] This probably reflects the older average age of racing standardbreds. The peak age of incidence is from four to nine years,[7] but the disease has been reported in horses ranging from 14 months to 18 years.[12]

Arabians and ponies seem less susceptible to navicular disease than other breeds.[13] One explanation may be that most Arabians are light on their feet and have a short stride, thus spending less time on each foot. They are short-backed and their hindquarters carry a larger portion of the weight. This decreases the compression on the feet.[2] If this theory of explanation were entirely true, it would also be expected to see less navicular disease in trotters, which is not the case.

Unnatural hoof shape and improper trimming are commonly seen in horses diagnosed as having navicular disease. Adams wrote:

> Pressure of the deep digital flexor tendon against the navicular bone is commonly increased by improper trimming and shoeing. It is common to trim the heel too low on a horse that has upright pasterns. This breaks the pastern and foot axis and produces greater pressure of the flexor tendon against the navicular bone.[3]

Forward deviation of the anterior-posterior axis is often thought to be damaging because it increases concussion. This is probably not a predisposing factor for navicular disease, as it is compression of the navicular apparatus with which we should be concerned, not concussion. The navicular bone is well-protected from concussion by the fibro-fatty frog below and the lateral cartilages laterally. As weight is placed on the limb, the navicular region is compressed against the deep flexor tendon. The blood vessels supplying this bone must experience some of this compression; it is conceivable that the nutrition of the bone, its bursa and the adjacent tissues is thereby disturbed.[14]

Activities that increase stress in the navicular region, such as polo, cutting, barrel-racing and jumping, along with co-existing inflammatory disease (laminitis, chronic infections, etc.) also predispose an individual to degeneration of the navicular bone.[2,3,12]

Etiology and Pathogenesis

The most controversial subject regarding navicular disease is its etiology and pathogenesis. We know what kind of post-mortem lesions are generally found, and we know what type of confrontation predisposes to the disease. We do not know what the primary cause of these lesions is. Not knowing the cause makes prevention and treatment much more difficult. After much study, there is still considerable disagreement among veterinarians. The three main theories of the pathogenesis of navicular disease are: (1) third-order acceleration, (2) arthrosis, and (3) thrombosis (ischemic necrosis).

Third-order acceleration theory

The oldest theory of navicular disease pathogenesis is third-order acceleration and friction. This theory states that vibration, which is third-order acceleration, causes tendon and navicular cartilage degeneration. The tendon/cartilage damage could be compared to that of automobile bearings or any other damage due to repetitive vector changes and starting and stopping.[1]

A study by a Danish group indicated the most striking feature of navicular disease was a defect in the cortical bone. The underlying trabecular bone was filled with highly vascularized connective tissue and was surrounded by active remodeling in the bone. There was increased osteoclastic activity along with areas of new bone formation. This group used tetracycline labeling and fluorescent microscopy to indicate that signs of bone vitality were present. They indicated bone necrosis was not present. This study found thickened arterial walls, but they did not find any thrombosis of the vessels. It is felt that the increased vascularity is secondary to the high rate of bone turnover and increased cellular activity.[11] How the defect in the cortical and trabecular bone came about in the first place is not explained, but they do indicate that increased pressure on the deep flexor tendon is important in the etiology.

In recent years, microangiography has been used to study in detail the vascular changes associated with navicular disease.[15,16,17] Two new theories of the pathogenesis of navicular disease have resulted.

Arthrosis theory

The supporters of the arthrosis theory of pathogenesis found significant differences in the vascular patterns of normal horses and those with navicular disease. Central and peripheral navicular vessels were dilated and more numerous. In some cases, pooling of con-

trast media occurred. These changes were more dramatic in the vessels underlying the flexor surface of the bone than in the vessels underlying the articular surface. The vessels often had a spiral, tortuous course in those horses with navicular disease.[17] Horses with clinical navicular disease do have an increased passage time of contrast media—about three times that of a normal horse.[18,19] The vascular pattern of the navicular bone in clinically affected horses is similar to the pattern in humans with hip arthrosis. It is believed that ischemia is not important in the pathogenesis of human arthrosis.[20]

Other studies in human medicine have shown a relationship between pain of arthrosis and juxta-articular bone marrow pressure.[21] Subchondral pressure measurements in the horse are consistent with studies done in human medicine.[18] This evidence is highly suggestive that higher bone marrow pressure in the diseased navicular bone must be a consequence of or a contributing factor to the development of navicular disease in the horse. It seems reasonable to assume that the increased duration of pressure drop to a stable state is due to a change in the blood flow of the bone marrow.[18] It has been shown that a reduced arterial supply results in decreased subchondral bone pressure.[22] These observations imply that the functional changes registered must be due, at least in part, to changes in the venous drainage.[18] This evidence thus contradicts the theory of thrombosis. Thrombosis would cause lowered subchondral pressures, not increased pressures.

Another similarity betwen human arthrosis and navicular disease is the development of resting pain. The exact cause of this pain is now known, but it could be due to the increased pressure, stretching of nerve endings, temperature changes or any combination of circumstances.

Arthrosis in humans is also characterized by changes in the synovial membrane.[23] Similar changes occur in the navicular bursa of horses affected with navicular disease.[24]

Supporters of the arthrosis theory believe that arterial hyperemia and venous congestion can lead to a state of venous hypoxia in navicular disease.[17] This venous congestion also contributes to the increased subchondral bone pressure.[18] Therefore, they believe the cause of this congestion is probably very significant in the pathogenesis of navicular disease.[17]

Ischemic necrosis theory

Another theory of navicular disease pathogenesis which came about through angiography studies is the theory of ischemic necrosis. Examination of specimens from horses with navicular disease showed that the distal arteries were occluded in the distal part of the bone. This occlusion was shown to be due to thrombosis.[9,15,25]

There was also a marked increase in the degree of anastamosis between arteries within the bone and an increase in the number of arterioles leaving the nutrient foramina. Many of these arterioles were also occluded. Occlusions were also found in some arteries of horses with no signs of navicular disease. In these cases, only one or two arteries were involved, and there was no occlusion of the compensating supply.[9,15,25]

It is felt that occlusion of these arteries leads to the development of an area of painful necrosis, and that the disease can progress in one of three ways.[15] These three possibilities are diagrammed in Figure 1. Supporters of the ischemic necrosis theory feel that for chronic lameness to occur, at least two of the distal arteries must be occluded, together with some involvement of the compensating supply.[9,16]

When all the different theories of the etiology and pathogenesis of navicular disease are considered, it is easy to become frustrated, because at this time there are still no clear answers. It seems obvious that how hard a horse is worked

Figure 1. Possible sequellae to ischemia in the equine navicular bone

A. Mild ischemia

↓

Enlarged secondary nutrient foramina and extensive arterial anastomosis

↓

Transient lameness only

B. Ischemia with several primary arteries and some secondary arterioles affected in a progressive thrombosis

↓

Slowing of the revascularization process

↓

Development of slowly developing ischemia and progressive clinical signs of navicular disease

C. Ischemia with vascular occlusion so severe that necrosis occurs

↓

If lesion is near flexor surface, it could break through and involve the fibrocartilage

and its conformation do contribute to an increase in friction and the disease process, but exactly what that process is remains in question. The disease process does involve the vasculature to the bone, as is shown by the discovery of increased contrast passage time by proponents of both the arthrosis and ischemic necrosis theories. The advent of anticoagulants and vasodilators in the treatment of navicular disease also supports this.[26,27] The cause of the slowed blood flow is still questionable. One group found thrombi in a significant number of cases,[15] while another study using similar techniques could not find any thrombi.[17] Venous pooling was found in one study, but its cause could not be explained.[17] Tetracycline labeling indicated that there was no true necrosis in the navicular bone, but this tetracycline labeling was not compared to results in clinically normal horses' navicular bones.[11]

In none of the new theories of etiology is there any mention of an inflammatory process. In order for adhesions to be formed between the bone and tendon, there must be some inflammation occurring somewhere in the disease process.

Recent research has helped us to understand this disease, but further studies are needed if we are to develop satisfactory methods of prevention and treatment. Those individuals studying navicular disease must approach the subject with an open mind and refrain from devising their methods to try and prove something they are already convinced of.

Diagnosis

The clinical signs of navicular disease are fairly characteristic, but at the same time, there is considerable variation from horse to horse as the disease progresses. Thus, navicular disease cannot be diagnosed on clinical signs alone.

Considerable pressure with a good set of hoof testers over the center third of the frog and across the heels elicits a response with most horses that have navicular lameness. The reaction of one foot should be compared to the other forefoot and the hind feet. The disease is usually at least partially bilateral in the forefeet. It must be remembered that there may be considerable sole bruising at the toe, due to the altered gait.[3]

Other simple tests can be done, which are not specific for navicular disease, but which will give positive results. 1) Forced flexion of the pastern will cause increased lameness when the horse is subsequently trotted, but lesions in the phalangeal and fetlock joints give more severe results, 2) Forced extension of the coffin joint (by standing the horse with its toe on a plank or trotting it using toe grabs) also accentuates the lameness. Lesions of the pastern joint and the flexor tendons may also give positive responses.[25]

Blocking of the palmar digital nerves is often used as an aid in the diagnosis of navicular disease. The landmarks for this block are well described.[3] Most horses with navicular disease will go sound after both palmar digital nerves of that foot are blocked. Often, the lameness in the less severely affected foot will not be apparent until the more severely affected foot has gone temporarily sound due to the nerve block. If the anesthetic is deposited too close to the cranial branch of the palmar digital nerve, the anesthetic can spread and block the entire foot. This could lead to an improper diagnosis if the limits of the block are not checked thoroughly.[28]

There are several reasons why a properly done palmar digital nerve block will not alleviate the lameness caused by navicular disease. These include fibrous adhesions, arthritis of the coffin joint, accessory nerve supply, sole bruising at the toe, concurrent traumatic arthritis of the fetlock, and incomplete anesthesia.[3]

Radiography has been used extensively as a diagnostic aid for navicular disease. Several authors[3,7] believe radiographic changes are absent in up to forty percent of horses with navicular disease. Others believe that all horses with navicular disease will have some radiographic changes.[25,29]

It is necessary that good hoof preparation be done and several radiographic views be taken to fully evaluate a suspected case of navicular disease. The shoes should be removed, the sole, frog and sulci cleaned and scrubbed, and the sulci packed with a soft soap or equivalent material.[a]

The number of views varies from one to several via serial tomography,[10] but four views are commonly used. These views include a lateral-medial, caudal-tangential, 45-degree dorsopalmar and either a 65-degree dorsopalmar or an 80-degree upright pedal view.

The classical radiographic changes in navicular disease are: (1) marginal osteophyte formation, (2) cystic formation within the bone,

[a]Play Doh, Kenner Products, a division of General Mills Fun Group, Inc., Cincinnati, Ohio.

(3) enlarged vascular foramina (roughened "saw-toothed" appearance along ventral border), (4) thinning of the posterior cortex, and (5) marked remodeling of the cross-section of the bone resulting in dorso-palmar collapse of the normal shuttle shape.[30]

Spur formation can occur naturally in aged, hard-working horses. Therefore, their significance must be evaluated along with history, clinical signs, and other radiographic changes.[10]

Cysts and enlarged vascular foramina are regarded as undeniable evidence of navicular disease when accompanied by clinical lameness.[10] A cystic lesion in the trabecular portion of the bone can theoretically heal, but cystic lesions in the cortex of the flexor surface may collapse from pressure from the deep flexor tendon.[9] The caudal tangential view can differentiate the location of the cyst. The foramina are considered enlarged when they change from a cone-shape to mushroom-shaped. Again, age, workload, and other signs must be considered here since minimal roughening may not be significant in a ten-year-old roping horse, but it may be clinically significant in a three-year-old thoroughbred.

Thinning of the palmar cortex is best seen with the caudal tangential view. The degree of thinning may vary from minimal to almost complete dissolution.[30]

The remodeling of the navicular bone is best seen on the lateromedial view. Early, the bone loses its square appearance. Next, the distal margin seems to point and the bone assumes a narrow and longer appearance. Finally, spurs can sometimes be seen on the proximal border.[30]

Fractures of the navicular bone are considered rare, but are usually vertically directed[3] and follow a transverse plane through the bone.[30] Chip fractures are occasionally found. These are considered to be avulsion-type fractures, not pathologic, and originate from the prominent edge on the distal border of the navicular bone. The relationship of chip fractures to other more common radiographic changes is not known.[31]

Structural alteration in the navicular bursa and the deep flexor tendon cannot be seen radiographically.[32]

The lesions of navicular disease and the views in which they are best seen are summarized in Figure 2.

Other diagnostic techniques which have been proposed include angiography[33] and thermography.[34] Both of these techniques show evidence that the disease is vascular in origin.

When clinical signs of navicular disease are present but no changes can be seen radiographically, clinicians must use all the diagnostic tools available to them to eliminate other possible causes of the lameness before "backing into" a diagnosis of navicular disease without radiographic changes. Differential diagnoses should include puncture wounds of the sole and frog, fractured navicular bone, fractured third phalanx, laminitis, sole bruising, corns, pedal osteitis and ringbone.

Treatment

Treatment of navicular disease has been a

Figure 2. Summary of Radiographic Findings in the Navicular Bone Visualized on Different Radiographic Projections.[32]

Projection	Anatomical Area Optimally Seen	Structural Alteration in Navicular Bone
45° Dorsopalmar	Proximal Border	Exostosis at ligament attachment
		Increased size and number of vascular channels
		Navicular bone fractures
65° Dorsopalmar or 80° upright Pedal	Distal Border	Exostosis at ligament attachment
		Increased size, number and irregularity of vascular channels
		Distal extent of vascular channels
		Avulsion fractures from distal border
Lateromedial	Not optimal for any area	Exostosis on promixal and distal margin
		Increased medullary density and cortical defects in flexor surface
		Remodeling of general shape
Caudal tangential	Medullary Cavity	Loss of trabecullar pattern
		Increased size, number and irregularity of vascular channels
	Cortical bone of flexor surface	Localized radiolucent regions
	Flexor Surface	Flattening or depression of the surface
	Miscellaneous	Fracture of navicular bone or angle of third phalanx

challenge to veterinarians for decades. Corrective shoeing is often done, but more aggressive therapy including neurectomies, juxtabursal orgotein, systemic warfarin and systemic isoxoprine are now being used in conjunction with corrective shoeing.

The configuration of the feet of horses with navicular disease varies greatly. As discussed before, it is only after a long time that these horses develop the "navicular look" of contracted heels and an upright axis. Most cases have a long toe and an underslung heel.[35] The principles of corrective shoeing are to raise the posterior aspect of the foot to decrease the tension produced by the deep flexor tendon, shorten the toe to hasten breakover, and to allow heel expansion at impact.[12]

The thickening of the heel should begin at the bend of the quarters and increase toward the end of the shoe. The amount of lift should be adequate to bring the digital bones into alignment and make them relatively parallel to the angle of the shoulder. A device to measure the angle of the shoulder can be made. This information should then be passed on to the farrier.[2]

Methods of raising the heel vary tremendously among farriers. It is generally considered that hand-made shoes with lay-back lifts or stacked heels are best, but there are commercially available shoes with raised heels.[b]

There are basically three ways to roll the toe: rocker toe, rolled toe, and chamfered toe. Rocker toed shoes are made by turning up the full width of the web of the shoe. Rolled toes turn up about half the width of the web. Chamfered toes are made by rasping off steel from the ground surface of the toe and leaving the hoof surface flat.[2] The individual farrier and the horse's conformation will determine which method is used.

The shoe must be fitted full enough to allow for expansion and to prevent the hoof from overgrowing the shoe and collapsing around it. The shoe should protrude 1/16–1/8 inch beyond the wall at the rear of the quarters and heels. The shoe should be set with six nails when possible, and the last nail should not be beyond the bend of the quarters.[2] In cases of severe contraction, slippered shoes may be necessary.

Palmar digital neurectomy has been an accepted treatment for navicular disease for many years. This method does not correct any pathological changes; it only masks the symptoms of the condition. Adams used the palmar digital nerve block with 1.5–2 cc of lidocaine to determine the degree of relief that would be obtained with the neurectomy. There are two accepted methods of doing the neurectomy: guillotine and epineural capping. The technique of epineural capping has been suggested as a means of reducing the incidence of painful neuroma,[36] but controlled studies with an adequate number of cases are lacking.[37] An inexperienced surgeon's use of the epineural capping technique with undue trauma may lead to more complications than the faster guillotine method performed atraumatically.[37] Both techniques are adequately described.[3,37]

A cryoneurectomy technique has been described that combines the effects of extreme cold on nervous tissue and epineural capping.[38] The authors claim a significant decrease in the incidence of painful neuroma formation with this technique. Further studies are needed, but this technique may hold promise for the future.

Possible complications of neurectomies are numerous and should be discussed with the owners prior to the surgery. Complications include painful neuroma formation, regeneration of nerve stumps leading to return of lameness, rupture of the deep flexor tendon, loss of the hoof wall, subluxation of the coffin joint, fractured navicular bone and complete bilateral luxation of the coffin joint.[3,39]

The use of orgotein,[c] a non-steroidal anti-inflammatory metalloprotein of bovine liver origin, has been suggested for use in soft tissue inflammation in horses. Orgotein has been injected both juxtabursally and systemically in cases of navicular disease. Authors claim a good percentage of favorable responses, especially if the horses had been lame less than two months.[40] The anti-inflammatory action of orgotein is probably helping to decrease the amount of adhesion formation between the navicular bone and the deep flexor tendon. In early cases, orgotein may be justified in conjunction with other means of therapy.

With the development of the possibility that the etiology of navicular disease is vascular in nature, two new methods of treatment are being tried. Warfarin,[d] an anti-coagulant, and

[b]Dutchtown Forge, Ltd., R.D. 2 Belle Mead, N.J., Walter J. Kopek, Operator.

[c]Palosein, Diagnostic Data, Inc., Mountain View, California.

[d]Panwarfin Abbot Pharmaceuticals, Inc., N. Chicago, Illinois 60064.

isoxoprine, a peripheral vasodilator, both have shown promise as potential treatments for navicular disease.

Warfarin is the anti-coagulant of choice because it is a cheap drug (current cost is less than $25/year), which can be given orally, and it has a specific antidote in vitamin K_1. Used in correct doses, side-effects are minimal although fetal death has been reported in humans.[26] Warfarin should probably not be used in pregnant mares.

The dosage requirements for warfarin vary among individuals with changes in feeding and management. For this reason, management must be carefully controlled ten days prior to and throughout treatment. Two blood samples should be taken to establish the horse's normal one stage prothrombin time (OSPT). A good starting dose is 1 mg/50 kg body weight given orally. The OSPT should be monitored twice weekly. The object of the therapy is to increase the OSPT by 20 percent of the pre-treatment level. If no clinical improvement is seen in six to eight weeks, the starting dose is increased by 20 percent. Prolongation of the OSPT may be increased up to 50 percent. It is essential that monitoring be carried out twice weekly during the stabilizing period. Once stable, the horse should continue to be monitored at least monthly.[25] Claims for successful warfarin therapy are as high as 77 percent of treated horses becoming sound and remaining sound for at least 12 months.[25]

Little is known about the pharmacokinetics of warfarin in horses. In man, 97 percent of it is bound to plasma protein. This means concurrent use of drugs which compete for protein binding sites (e.g. phenylbutazone) may release a quantity of "active" warfarin and cause a marked increase in OSPT.[26] A list of drugs believed to be safe are listed in Figure 3.

Owners should be thoroughly educated to beware of signs of hemorrhage, edema, hematomas, painful joints, colic, lassitude and pale mucous membranes. If mild warfarin toxicity is suspected, hematomas can be bandaged and the dose of warfarin decreased.[26] If the toxicity is severe, vitamin K_1 should be administered and transfusion therapy may be necessary if the condition is life-threatening. A response should be seen within 30 minutes of treatment with vitamin K_1. After administering vitamin K_1, it will take two to three weeks before a normal response is again seen to warfarin, so treatment should be withheld for that time.[25]

Figure 3. Drugs considered to be compatible with warfarin therapy in the horse.[25]

Drugs tested for compatibility	Comments
Pyromtel 43.9 percent	No effect on OSPT
Fenbendazole	No effect on OSPT
Thiabendazole	Slight lengthening of OSPT occasionally
Procaine Penicillin-dihydrostreptomycin	No effect on OSPT
Dipyrone	No effect on OSPT (but coliz will lengthen)
Acepromazine	Slight lengthening of OSPT
Drugs found to be compatible in practice	
Nitrofurantoin	Controlled experimental testing has not been carried out.
Orgotein	

Isoxoprine hydrochloride is a beta-blocking peripheral vasodilating agent used to treat ischemic disorders in man. If arteriosclerosis and thrombosis are significant in the pathogenesis of navicular disease, it seems logical that a vasodilating agent would be helpful in its treatment.

Initial studies in Australia indicate improvement occurred in 24 of 27 horses treated with isoxoprene. This was very significant when compared to horses treated with placebos.[27]

The dosage of isoxoprine is 0.6 mg/kg body weight given orally twice daily for six to 14 weeks. If no improvement is noted, the dose may be increased 50 percent. At the above dose, there are no significant effects on the complete blood counts, plasma biochemical parameters, cardinal signs or systemic blood pressure. Infra-red thermography of the lower limb showed a significant increase in lower limb temperature within eight hours of administration.[27]

It appears that the optimal length of treatment is nine to 12 weeks, then a decrease to once daily treatments in the later stages of therapy. Eight of nine horses remained sound for two months after ceasing therapy. The duration of treatment and extent of long-term improvement may become clearer with further use of the drug.[27] Further studies are planned in the United States by Baker at Illinois and Turner at Colorado State and in Europe by Colles.

If isoxoprene is proven to be effective in the treatment of navicular disease, it would appear to be superior to warfarin because of fewer possible side-effects and the elimination of the need to hospitalize and monitor all patients.

The only disadvantage would be the necessity of two treatments per day. Cost of isoxoprine therapy at today's prices would be approximately $1.00/day.

Treatment of navicular disease is still not usually a rewarding experience for veterinarians. The ultimate decision must be made by the owners, but it is the responsibility of the veterinarian to be aware of all the possibilities and their advantages and disadvantages. Whatever method of treatment is chosen, corrective shoeing should be done in conjunction with the therapy, in order to slow the ongoing disease process.

Conclusions

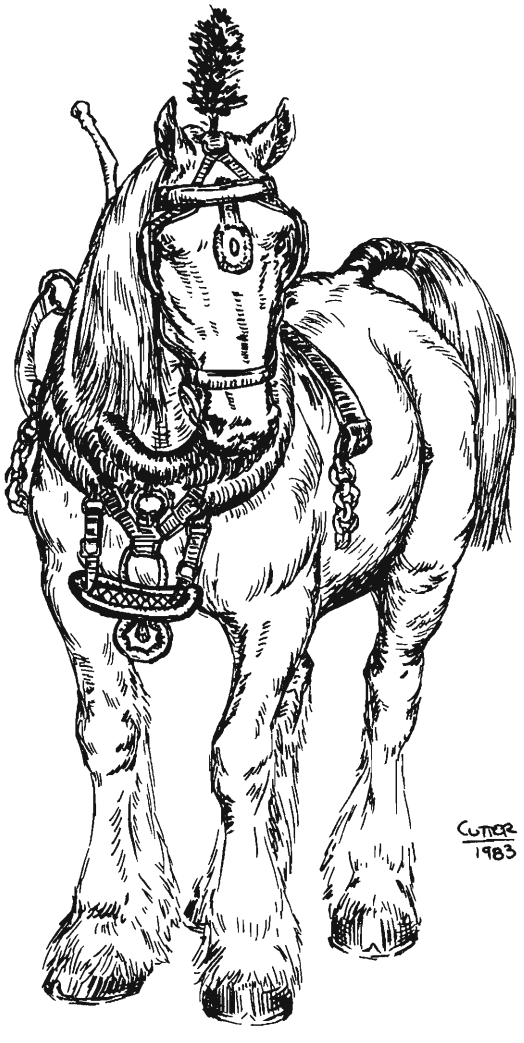
Navicular disease is very common, and veterinarians should be aware of its possible causes in order to answer the numerous questions owners may ask. A good working relationship should be established with local farriers because they are instrumental in correction of navicular lameness. If monitoring possibilities exist, warfarin therapy may be attempted. Isoxoprine shows promise as a potential treatment and warrants further investigation.

With continuing research on the etiology and pathogenesis of navicular disease, we may be able to more successfully control this lameness in the future.

References

1. Rooney JR: Navicular Diseases, *Proc. AAEP,* 1967, pp 263-772.
2. Emery L, Miller J, Van Hoosen N: *Horseshoeing Theory and Hoof Care.* Philadelphia, Lea and Febiger, 1977.
3. Adams OR: *Lameness in Horses.* Philadelphia, Lea and Febiger, 1974.
4. Sisson S, Grossman JD: *The Anatomy of Domestic Animals.* Philadelphia, W. B. Saunders, Co., 1975.
5. James PT, Kemler AG, Smallwood JE: The Arterial Supply to the Distal Sesamoid Bones of the Equine Thoracic and Pelvic Limbs, *J of Vet Orthopedics* 2:38-46, 1980.
6. Valdez H, Adams OR, Peyton LC: Navicular Disese in the Hindlimbs of the Horse, *JAVMA* 172:pp. 291-292, 1978.
7. Ackerman N, Johnson J, Dorn R: Navicular Disease in the Horse: Risk Factors, Radiographic Changes, and Response to Therapy, *JAVMA* 170:pp. 183-187, 1977.
8. Wilkinson GT: The Pathology of Navicular Disease, *Bristish Vet* 109:38-42, 55-59, 1963.
9. Colles CM: Ischemic Necrosis of the Navicular Bone and Its Treatment, *Vet Record* 104:133-137, 1979.
10. Turner TA, Fessler JF: The Anatomic, Pathologic, and Radiographic Aspects of Navicular Disease, *The Comp on Cont Ed for the Pract Vet* 4:S350-S355, 1982.
11. Ostblom L, Lund C, Melsen F: Histological Study of Navicular Bone Disease, *Equine Vet J* 14:199-202, 1982.
12. Moyer W, Diseases of the Equine Heel, *Proc of the AAEP,* pp. 21-29, 1979.
13. Lowe JE: Sex, Breed and Age Incidence of Navicular Disease, *Proc of the AAEP,* pp. 37-46, 1973 (1974).
14. Richardson C: *Practical Farriery.* London, Sir Isaac Pitman and Sons, Ltd., 1950.
15. Colles CM, Hickman J: The Arterial Supply of the Navicular Bone and its Variations in Navicular Disease, *Equine Vet J* 9:150-154, 1977.
16. Fricker C, Riek W, Hugelshofer J: Occlusion of the Digital Arteries—A Model for Pathogenesis of Navicular Disease, *Equine Vet J* 14:203-207, 1982.
17. Svalastoga E: Navicular Disease in the Horse. A Microangiographic Investigation, *Nord. Vet. Med.* 35:131-139, 1983.
18. Svalastoga E, Smith M: Navicular Disease in the Horse. The Subchondral Bone Pressure, *Nord Vet Med* 35:31-37, 1983.
19. Colles CM: Navicular Disease, *Equine Vet Data 2:*50-51, 1981.
20. Harrison MH: Osteoarthritis of the Hip: A Study of the Nature and Evolution of the Disease, *J Bone Jt Surg* 35:498-626, 1953.
21. Arnoodi C, Leinperg RL, Linderholm H: Immediate effect of Osteotomy on the Intramedullery Pressure of the Femoral Head and Neck in Patients with Degenerative Osteoarthritis, *Acta Orthop Scand* 42:357-365, 1971.
22. Stein AH, Variations in Normal Bone Marrow Pressures, *J Bone Jt Surg* 39:1129, 1957.
23. Arnoldi C, Reiman I, Bretlau B, The Synovial Membrane in Human Coxarthrosis: Light and Electron Microscopic Studies. *Clin Orthopaed* 148:213-220, 1980.
24. Svalastoga E, Nielsen K: Navicular Disease in the Horse. The Synovial Membrane of Bursa Podotrochlearis, *Nord Vet Med* 35:28-30, 1983.
25. Colles CM: Navicular Disease and Its Treatment, In *Practice 4:*29-36, 1982.
26. Colles CM: A Preliminary Report on the Use of Warfarin in the Treatment of Navicular Disease, *Equine Vet J* 11:187-190, 1979.
27. Rose R, Jennifer R, Hodgson D, Hohnke J: Studies on Isoxoprine for the Treatment of Navicular Disease, *Equine Vet J.* 15:238-243, 1983.
28. Mansman RA, McAllister ES: *Equine Medicine and Surgery.* Santa Barbara, California, American Veterinary Publications, pp. 1045, 1982.
29. Rose RJ, Taylor BJ, Steel JD: Navicular Disease in the Horse: An Analysis of Seventy Cases and Assessment of a Special Radiographic View, *J Equine Med Surg* 2:492-497, 1978.
30. Morgan JP: *Radiology in Veterinary Orthopedics.* Philadelphia, Lea and Febiger, 1972.
31. Van De Watering CC, Morgan JP: Chip Fractures as a Radiographic Finding in Navicular Disease of the Horse, *J Am Vet Radiol Soc* 16:206-210, 1975.
32. O'Brien TR, Millman TM, Pool RR, Suter PF: Navicular Disease in the Horse: A Morphologic Investigation Relative to a New Radiographic Projection, *J Am Vet Radiol Soc* 16:39-47, 1975.
33. Scott EA, Sandler GA, Shires MH: Angiography as a Diagnostic Technique in the Equine, *J Equine Med Surg* 2:270-278, 1978.

34. Turner et al.: Thermographic Evaluation of Horses with Podotrochlosis, *Am J Vet Res* 44:535–539, 1983.
35. Mayer W, Anderson J: Lameness Caused by Improper Shoeing, *JAVMA* 166:47–52, 1975.
36. Evans LH: Procedures Used to Prevent Painful Neuromas, *Proc of AAEP,* p. 103, 1970 (1971).
37. Turner SA, McIlwraith CW: *Techniques in Large Animal Surgery.* Philadelphia, Lea and Febiger, pp. 133–135, 1982.
38. Tate, LP, Evans, LH: Cryoneurectomy in the Horse, *JAVMA* 177:423–426, 1980.
39. Rose, RJ: Bilateral Luxation of the Coffin Joints in a Horse as a Sequel to Palmar Neurectomy, *Vet Record* 100:198–199, 1977.
40. Coffman et al.: Orgotein in Equine Navicular Disease: A Double Blind Study, *JAVMA* 174:261–264, 1979.



New Publication From the AVMA

Pet Loss and Human Emotion: When The Question Is Euthanasia, is the newest client information brochure published by the American Veterinary Medical Association (AVMA). The eight-page brochure is intended to help veterinarians handle the subjects of euthanasia and pet loss by offering special information to the client. The client can read about the decision-making process, sharing that decision with family members, saying good-bye to the pet, the euthanasia procedure, the grieving process, and other aspects of coping with the loss of an animal. A suggested reading list is also included.

Accompanying each brochure order is material specially prepared for the veterinarian and the hospital staff offering a deeper insight into client emotions.

This brochure was produced at the suggestion of the AVMA's Committee on the Human/Animal Bond. Many veterinarians, psychiatrists, psychologists, sociologists, and educators furnished background information and reviewed the brochure before it was printed.

The brochure is available at a cost of $10 per one hundred copies. To order, send name and address along with a check made payable to AVMA to: American Veterinary Medical Association, 930 North Meacham Road, Schaumburg, IL, 60196 or contact Tim Donovan (312) 885–8070.