Dr. Ric Redden revealed a new shoe, and new ideas in the treatment of horses suffering from laminitis

RAPID RELIEF

BY LES SELLNOW

A radically different approach to the treatment of laminitis was unveiled for more than 1,000 attendees—some 200 of them from other countries—at the Fourth Annual Bluegrass Laminitis Symposium held Jan. 25-27 in Louisville, Ky.

Dr. Ric Redden, whose International Equine Podiatry Center of Versailles, Ky., sponsored the symposium, told veterinarians and farriers from across the United States and other countries that he had made a "180-degree turn" in his approach to treatment of most cases of laminitis between last year's symposium and the one held this year.

His treatment approach today involves raising the heels of afflicted feet either with wedge pads or a specially designed shoe. In very severe cases that do not respond to the heel-raising approach, he surgically severs the deep flexor tendon.

An oversimplified definition of laminitis is an inflammation of the horse's sensitive laminae, which are located between the coffin bone and the hoof wall. The laminae are tiny interlocking projections that connect the coffin bone to the hoof wall. The laminae are folded, accordion-like, in about 500 places around the hoof. The laminae of the hoof wall are insensitive, but those arising from the surface of the coffin bone are sensitive and contain blood vessels and nerves.

During acute laminitis, the sensitive laminae become stretched and torn, thus losing their ability to hold the coffin bone in place. As a result, the coffin bone often rotates downward, with the point angling toward the sole of the foot. If this rotation is severe, the coffin bone may actually penetrate the sole of the hoof. The hoof sole of the horse afflicted with laminitis becomes flat rather than concave and eventually heavy rings may form on the wall of the hoof because of abnormal hoof wall growth. The toe may curl up at the end if not trimmed and the white line separates.

In some severe cases the afflicted horse may become a "sinker," a situation where the entire bony column of the foot drops.

An inflammation or tearing of the sensitive laminae is extremely painful and could be compared with tearing a fingernail to the quick. The pain is caused to a large degree by pressure resulting from congestion of blood in the sensitive laminae.

The amount of rotation or sinking of the coffin bone is determined by how much damage has been done to the sensitive laminae. The greater the damage to the laminae, the greater will be the rotation or sinking and the more grave the prognosis. Any rotation at all of the coffin bone in the first five to 10 days of the affliction makes for a bleak prognosis.

Treatment of the disease by practitioners and farriers through the years has involved injection of anti-inflammatory and pain-killing drugs, lowering the heels of affected hooves, attaching heart bar shoes to shift the weight-bearing pressure to the frog of the foot, and soaking the affected feet in water laced with Epsom salt. In severe cases, the front wall of the affected hoof or hooves has been removed in a procedure called anterior wall resection. The purpose is to open the way to the afflicted laminae, so that the foot can be treated with topical applications of medication and to clean out debris created by the disease. In all cases, stall rest is advocated. Frequent radiographs are utilized to monitor the degree of rotation or sinking.

Under his new approach, Dr. Redden told the group, he no longer lowers the heels, no longer does anterior wall resections, and no longer soaks the foot. He continues to take a dim view of using the heart bar shoe in acute cases of laminitis.

While Dr. Redden's change in position concerning treatment of laminitis is dramatic, the new approach is far less involved and traumatic than some of the procedures that had become commonplace through the years, such as anterior wall resection.

Focal point of the new approach, according to Dr. Redden, is reducing the tension or pull on the coffin bone that is exerted by the deep flexor tendon. The pulling pressure of the deep flexor, he said, is the prime force involved in causing rotation of the coffin bone. With the laminae inflamed and torn, the firm anchor between the coffin bone and hoof wall is gone. The deep flexor, which is attached to the coffin bone, literally pulls it from its normal position and causes it to rotate downward. To negate this force, under Dr. Redden's new approach, the heel of a horse suffering from laminitis is raised via wedge pads at the heel or by applying a specially constructed shoe. This relieves the pressure or tension exerted by the deep flexor tendon. In more serious cases, where salvage of the animal's life is the prime goal remaining, the deep flexor tendon itself is severed in a quick and simple surgical procedure called a tenotomy that brings about an immediate release of the pressure.

"Since January of last year," Dr. Redden told the group, "I have consistently raised the heel on every laminitis patient I've seen. I have either seen or consulted with approximately 1,500 cases since I saw you this time last year and I have found dramatic clinical differences in this particular technique."

Dr. Redden said he was led to the raised heel approach by observing dramatic changes in horses on which tenotomies have been performed, some of them with such severe afflictions that tenotomy was a last resort before euthanasia.

"We've cut in excess of 205 tendons in the last three years," he said. "The effect of absolutely stopping rotating forces or tension (on the coffin bone) has been tremendous."

Normally, Dr. Redden said, he has been raising the heels with three Number 4 wedge pads, an elevation of about two inches. He added that he and his research partner in Germany, Helmut Dallmer, have developed a shoe that will raise the heel and will fit over whatever shoe the horse might be wearing.

Raising the heel, Dr. Redden said, is aimed at reducing the pull of the deep flexor tendon on the coffin bone in much the same manner as a tenotomy.

He underlined the role of the deep flexor tendon by relating to the group an experience with a Standardbred race horse more than 15 years ago which suffered such a severe case of laminitis in the front feet following a race that it eventually had to be destroyed. The horse was in such severe pain, he said, that it hopped about its stall like a rabbit, never putting its front feet on the floor. Yet, he said, radiographs showed that the coffin bone had not rotated.

"At that time I had no idea what effect the deep flexor tendon had on rotation," he said, "but looking back over my observations of the past few years I would have to say the reason he never rotated is because he never touched the ground with his front feet. The deep flexor did not have the opportunity to actually pull the laminae apart, causing rotation."

The relief brought to a horse suffering from laminitis that has its heels raised, Dr. Redden said, comes quickly: "Within two to three hours he will be improved considerably. In three to four days he may be comfortable."

Raising the heel appears either to prevent rotation or stop it once it has started.

"To this date," Dr. Redden said, "I have yet to have a clinical case that has continued to change radiographically when the heel has been raised to this height (about two inches)."

Dr. Redden recognized that the new approach he is advocating must sound strange to the ears of veterinarians and farriers who for years have been lowering the heels of horses afflicted with laminitis.

"That's what I've been doing for 20 years, too," he said, "lowering the heel, trying to make the coffin bone parallel to the ground so I could reduce the forces destroying that foot. And I've had some great luck and a lot of you have, too. But now I'm seeing a better response with a less dramatic, more economic approach, and my prognosis is

(Continued on next page)

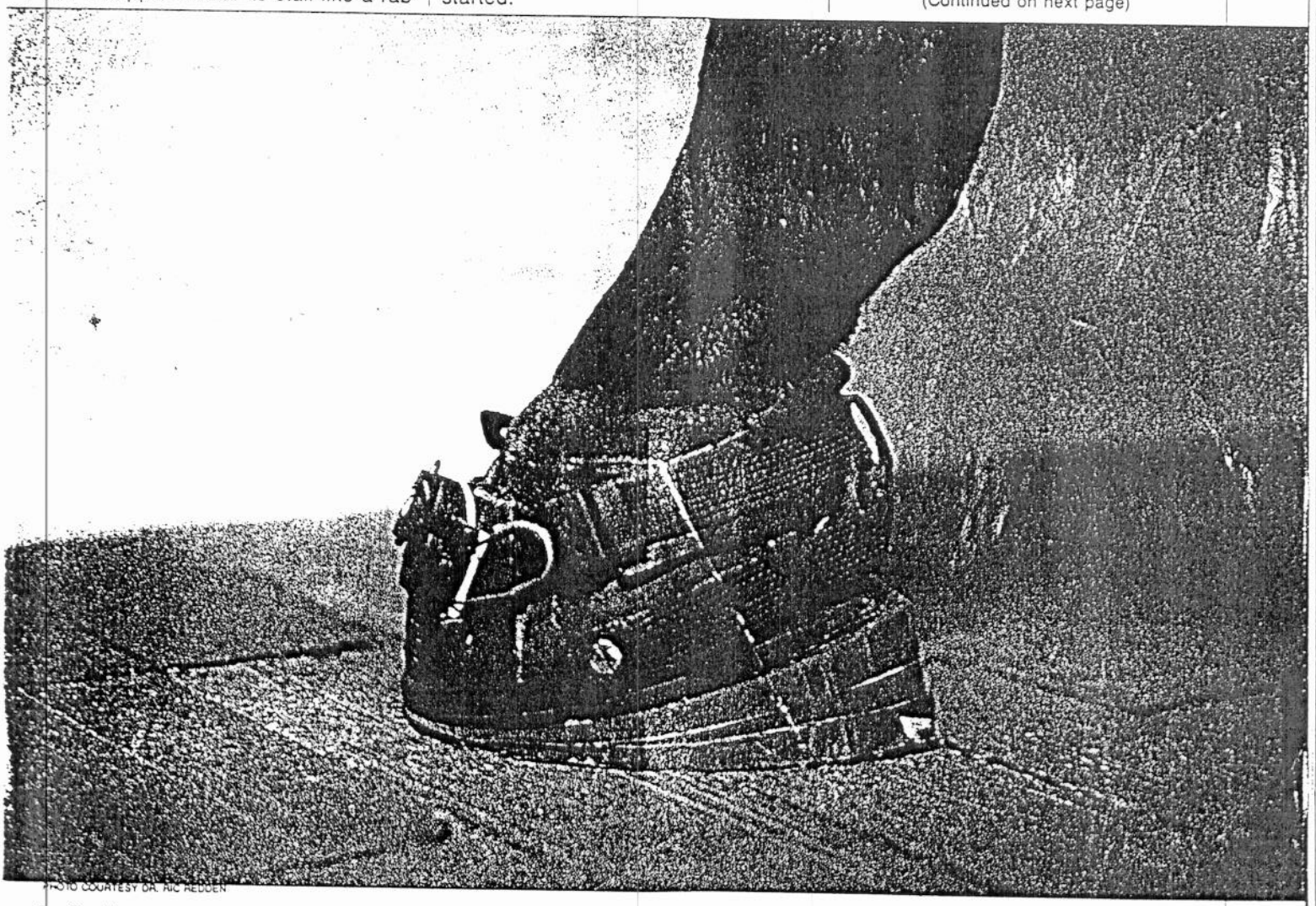


PHOTO COURTESY DR. RIC REDDEN

Dr. Redden now advocates raising the heel of a horse suffering from laminitis in order to reduce the pull of the deep flexor tendon on the coffin bone. This shoe was developed by Dr. Redden and Helmut Dallmer to fit over the regular shoe.

LAMINITIS

(Continued from page 833)

getting better as we go along . . . It's a whole new ball game for everyone."

Gone, too, from his repertoire in treating laminitis cases, he said, is anterior wall resection:

"I do not do anterior wall resections any more. I think they are totally uncalled for. I don't clean these things up like I've lectured to you before because it's totally uncalled for, and the reason is that by raising the heel or cutting the tendon, for some reason, increases the ciculatory pattern to that digit so that I have a better chance of normal physiologic healing."

Gone, too, is the time-honored approach of soaking the afflicted foot or feet on a long-term basis, although he still uses soaking early on in the treatment:

"I do not soak these long-term any more because soaking softens the foot and a lot of strength is lost in the hoof capsule. It begins to flare out and the bottom falls out. I want to keep the capsule nice and hard."

Once the rotating or sinking action is brought to a standstill, one must then consider lowering the heel back toward what had been normal. Dr. Redden suggests easing the heels down gradually.

"When lowering them, even five degrees can make a difference, so I play it by ear," he said.

Dr. Redden discussed the heart bar shoe in both a paper presented and in his talk, explaining why he does not use it in acute cases:

"Laminitis is not simply a mechanical disease that can be cured by use of a heart bar shoe. The heart bar shoe is only a supportive aid that is beneficial to cases with mild to moderate lamellar necrosis. It does not stop rotation or sinking of the coffin bone in cases of massive lamellar necrosis, and its application often offers a false sense of security due to clinical improvement of the patient . . . it is impossible to push the coffin bone back into place with the heart bar shoe."

In severe cases of laminitis, raising the heel might not produce the desired or necessary results, and when that is the case, Dr. Redden said, the only alternative is a tenotomy—surgically severing the deep flexor tendon at a point about midway down the cannon bone:

"Tenotomy is a practical, inexpensive approach to salvaging these types of horses, but it is a salvage procedure only. The client must understand that you are trying to save this horse's life and not make a riding animal. This is a salvage procedure for brood animals. It's not for the gelding unless he is a professional pet. It is done as a last resort. You raise the heel as a conservative approach to accomplish the same thing, in hopes of receiving the same clinical response. Please understand that."

Placed in the category of a likely candidate for a tenotomy by Dr. Redden is "an animal that has rotated rapidly, more than 10 degrees within 30 days, or is a sinker that has sunk as much as half a centimeter in the first five days of the syndrome, is past the point of being an athlete ever, but has breeding potential. I routinely will cut the deep flexor tendon on such an animal and won't bat an eye in doing so . . . The effect of absolutely stopping the rotating forces or tension has been tremendous. We have lost animals after we have cut the deep flexor, but the only ones that we have lost we cut very late in the necrotic stage of the syndrome when they were ready to slough the digit."

While the horse on which a tenotomy has been performed normally is not usable as a riding animal, it often can still play a reproductive role. Mares may be able to bear foals, and, Dr. Redden said, the procedure also has made it possible for a number of valuable stallions which suffered laminitis to continue a breeding career.

One of the secrets to success when performing a tenotomy, Dr. Redden told his listeners, is to do it before advanced bone disease sets in.

"If you wait until you have advanced bone disease or osteomyelitis," he said, "it can still be effective, but it will only last approximately six months to one year, and then you are right back to where you were. If you do the tenotomy prior to advanced bone disease, you have a relatively decent prognosis for two or three years."

Eventually, he said, in most cases the deep flexor tendon will contract again at some time in the future following the tenotomy, and the pulling tension will be applied to the coffin bone again.

"Some cases may require a second tenotomy within months of the first in order to keep the heel of the frog in contact with the ground," Dr. Redden said. "It is recommended to use (as a surgical site) the mid-cannon area but once. A second tenotomy should be performed in the pastern area. However, even by removing a piece of the deep flexor tendon below the ankle, the fibrosing effect of the tendon causes the favorable results of the surgery to be short-lived."

The tenotomy is normally performed with local anesthesia blocking nerves in the leg from the knee down. Once the surgery has been performed, there will be little outward indication that it has been done, Dr. Redden said:

"They don't look any different after you cut them than before surgery. They will have a slight flip of the toe, and they'll walk with a slight limp."

The first line treatment of laminitis, for cases ranging from mild to very severe, however, involves raising the heel, Dr. Redden emphasized. In a number of cases, he said, the procedure has allowed horses to return to a useful function after the heel has been lowered and appropriate new foot growth has occurred.

No matter what the procedure, he said, one of the most important elements in treating laminitis is stall rest:

"I can't emphasize this too much. If a horse is even mildly or moderately involved, he should be in the stall for months, not days. That's what saves his life because he is not destroying the laminae. Though a horse looks like he's clinically healed, he may be a long way from going outside. That's the biggest mistake made in the treatment of a majority of laminitis cases, especially with brood stock—turning them out too soon." ■

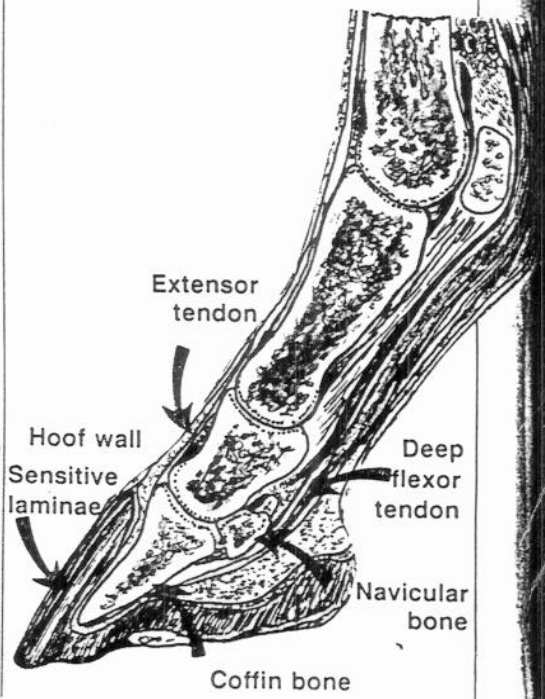
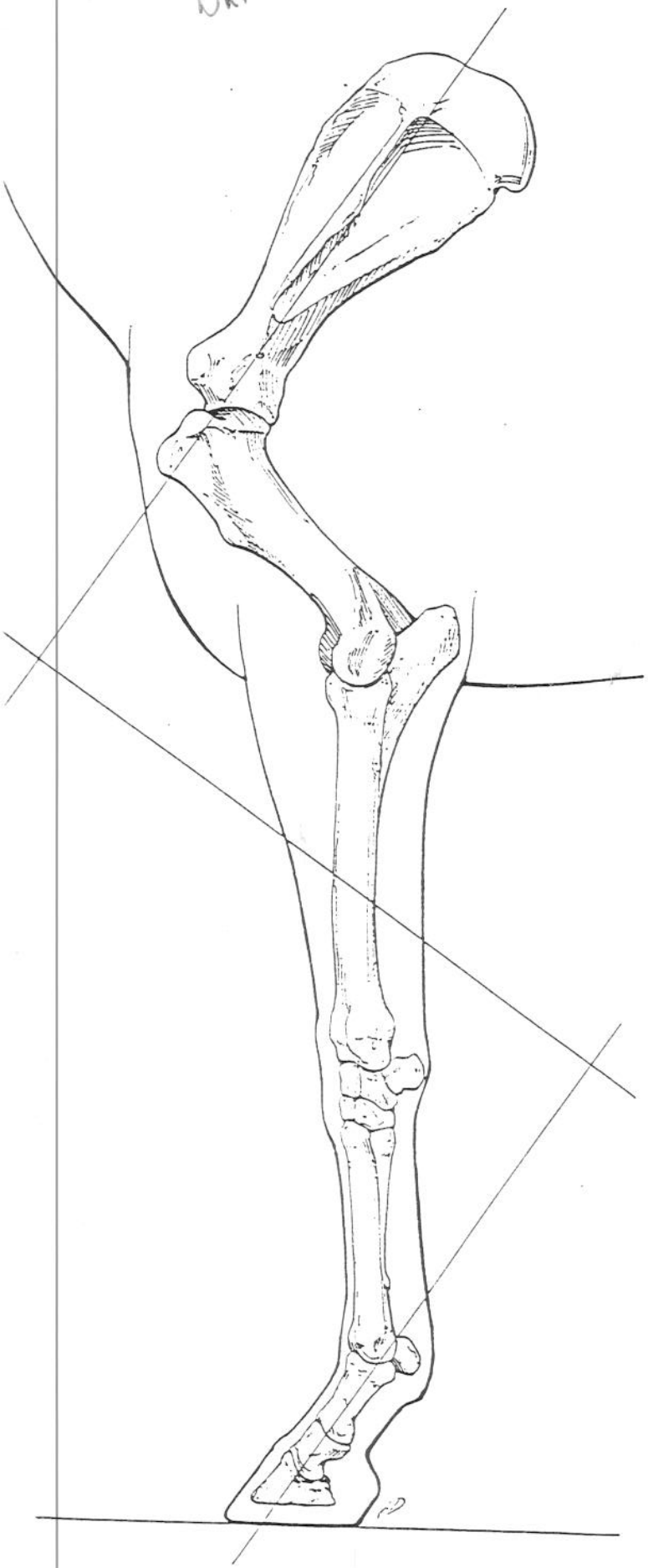


ILLUSTRATION BY LINDA WITTEN MEARNS

Docket No. 55-052

Russ Thompson

Dr. C.



Lateral View (see Fig. 2–1; 2–4*B*)

The shoulder should be sloping. A line dropped from the tuber spinae, on the spine of the scapula, should bisect the limb to the fetlock joint and then carry to a point just behind the heel. The carpus should not deviate forward or backward. The musculature of the forearm should be well developed and balance the limb. The region just distal to the carpus should not be cut in on the dorsal or palmar surface (cut out under the knees; tied-in knees). The hoof wall should slope at the same angle as the pastern.

The length and angle of the scapula and humerus in relation to the body should be such that it allows for maximum elevation, advancement of the limb, and absorption of concussion. The scapula and humerus should be long enough to allow for proper alignment of the forelimbs distal to it yet short enough to provide a rapid synchronous movement. In general, the straighter the shoulder the more frequently the forefeet will hit the ground during exercise and the less concussion will be absorbed. Straight conformation leads to increased stress, strain, and concussion of the distal limb. Whereas a more sloping shoulder will cause less stress and strain and concussive forces on the forelimbs distal to it (Fig. 2–5). Another feature of the scapulohumeral (shoulder) joint worth mentioning is that it is supported entirely by the muscles and tendons surrounding it. This purely muscular support allows the joint freedom of movement during flight which is in keeping with its ball-and-socket arrangement. Because this muscle support is so important, a horse should have well developed muscles in this region. The angle formed by the humerus and radius and ulna at the elbow joint should be between 120 to 150°. Straighter conformation (lesser angulation) at this joint results in a short, choppy gait and increased concussion on the distal limb. The radius and ulna should be of sufficient length to provide good muscular function.

Ideally the limb should form a straight column from the elbow joint to the fetlock. This conformation will disseminate the axial compression forces

FIG. 2–5. The slope of the shoulder usually influences the slope of the pastern. The straighter the shoulder, the straighter the pastern.

to all bony surfaces equally. If the bones are out of alignment, axial compressive forces become focused on one side and tensional forces are created opposite to it, resulting in increased stress and strain.

The carpus (knee) is a compound joint that is interposed between two long bones. Its major functions include: 1) flexion, 2) absorption of concussion, and 3) extension. Flexion primarily occurs at the antebrachiocarpal (radio-) carpal and midcarpal joint spaces. Concussion is absorbed by all three carpal joints and extension is developed by a locking mechanism that occurs while the horse is weight-bearing and in the extension phase of the stride. Because of this requirement it is important that the carpal bones are in good axial alignment with the radius and ulna and third metacarpal bone (cannon bone). They also should be of sufficient size to support the force brought to bear on them. Since flexion and extension are an important function of the carpus, the muscles of the forearm should be well-developed to support these functions.

The angle between the third metacarpal (cannon) bone and the proximal (first) phalanx is about 125 to 135°. The angle between the ground surface and the foot and the pastern foot axis is about 45 to 50°. The angle of the proximal phalanx with the third metacarpal bone should be sufficient to provide good shock absorption. In general, the shorter and straighter the pasterns (proximal and middle phalanges), the greater the axial compression (concussive) forces that are distributed to the foot.

The foot should be large enough, shaped properly, and made of the proper consistency to support the body's weight and absorb concussion. The reason for this is that it must propel the animal, resist wear and tear of continual exercise, and pump the blood from the foot upward.

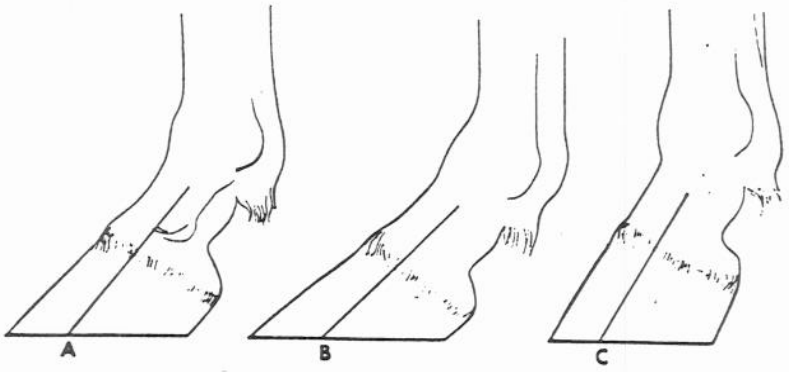


FIG. 2–36. Side view of foot and pastern axis. *A*. Normal front foot and pastern axes (approximately 47°). *B*. Foot and pastern axes less than normal (less than 45° in front or less than 50° behind). *C*. Foot and pastern axes greater than normal (greater than 50° in front or greater than 55° behind).

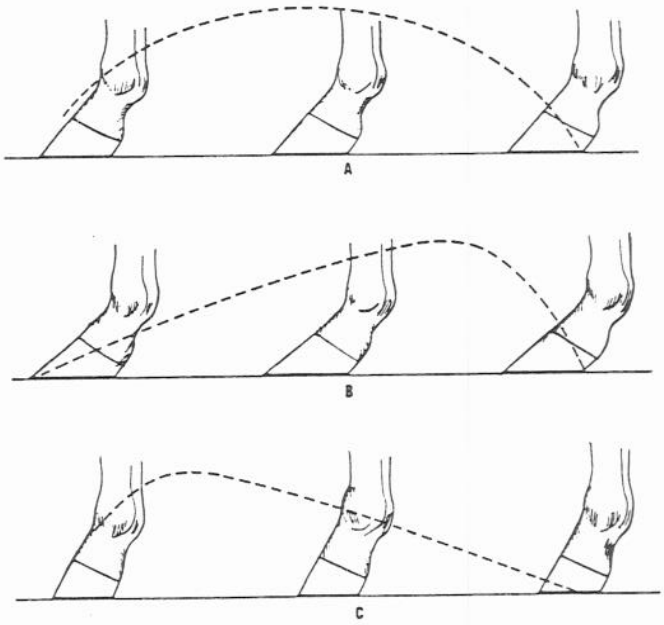


FIG. 2–41. Examples of foot flight. *A*. Flight of a foot with normal foot and pastern axis. The peak of the arc occurs as the foot passes the opposite supporting foot. *B*. Flight of a foot with foot and pastern axis less than normal: long toe, low heel. The peak of the arc occurs before the foot reaches the opposite supporting foot. *C*. Flight of foot with foot axis greater than normal: short toe and high heel. The peak of the arc occurs after the foot passes the opposite supporting foot.

1.

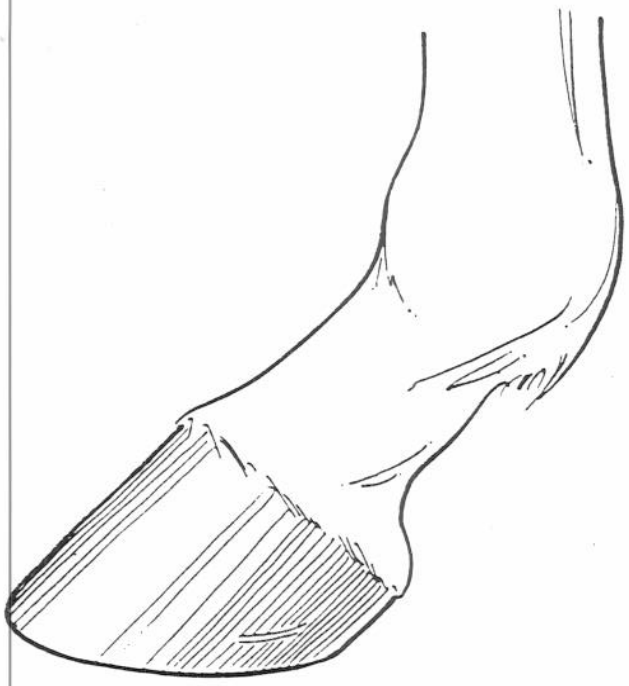


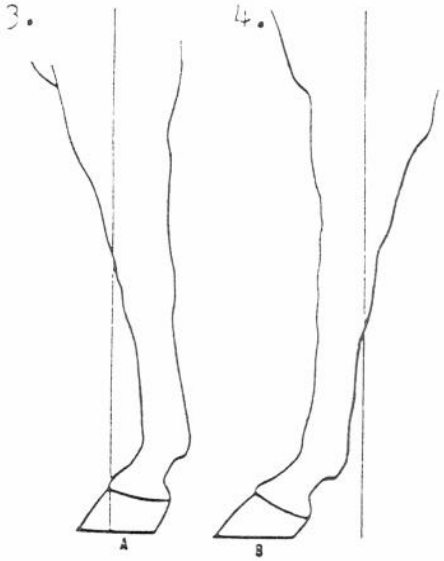
FIG. 2–26. Example of a long sloping pastern. The foot and pastern axes are less than normal (less than 45° in front or less than 50° behind).

2.

FIG. 2–27. A long upright pastern with a broken foot and pastern axis caused by lowering of the heels in an attempt to produce normal angulation of the hoof wall. This puts even greater stress on the navicular region by forcing the deep flexor tendon tighter against the navicular bone.

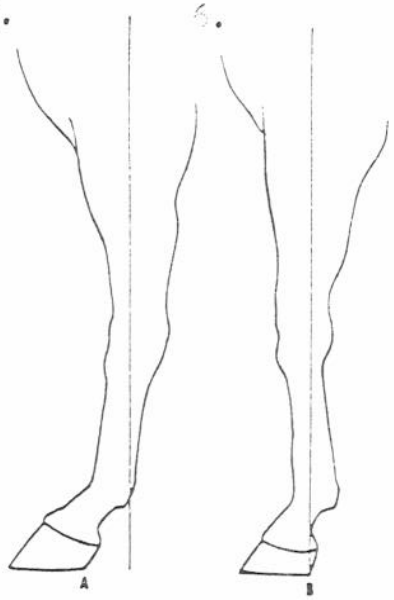
All six of these horses can travel in a normal manner by putting the correction of their incorrect conformation into a build-up, but this is not possible using only 1" of pad. But by using 3" to 4" of pad, these horses would be serviceable sound.

3.



4.

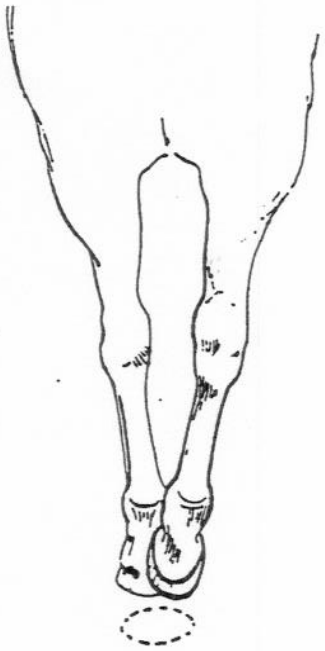
5.



6.

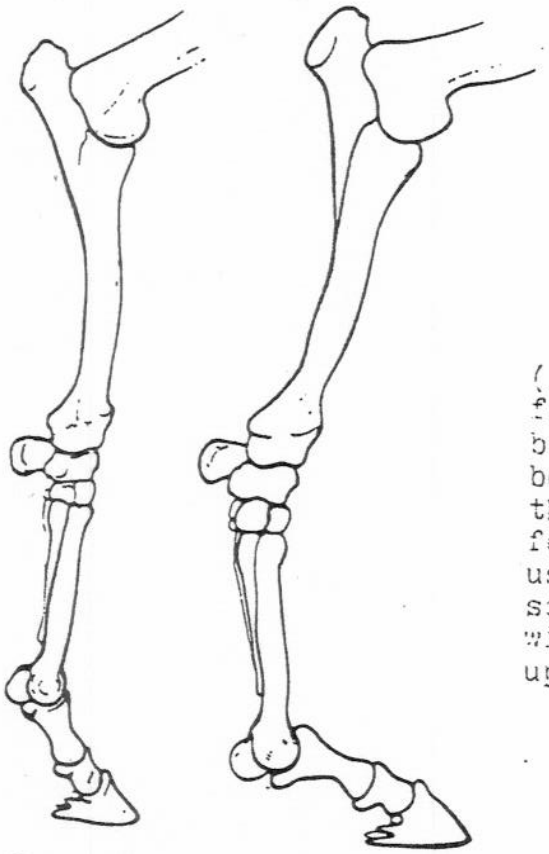
. 2–24. Examples of poor conformation. *A*. Standing under in it. *B*. Camped in front.

FIG. 2–16. Examples of poor conformation. *A*. Calf knees—a backward deviation of the carpus. *B*. Bucked knees—a forward deviation of the carpus.



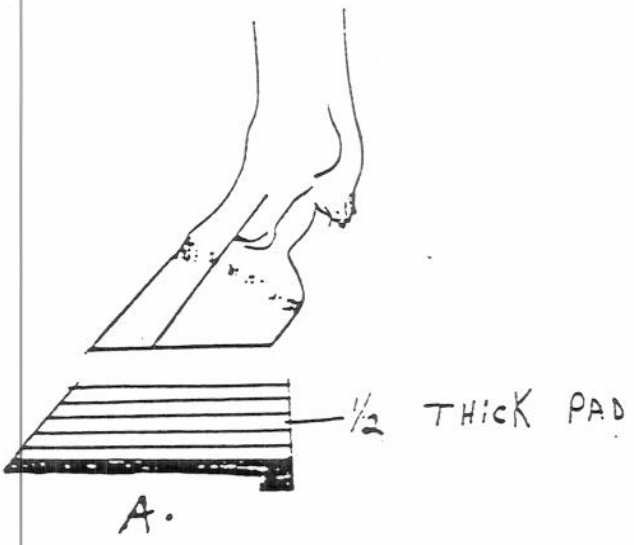
This horse cannot travel sound without corrective shoeing and build-up with pads, and the correction cannot be done with only 1" of pad.

FIG. 2–15. A. Plaiting. Plaiting is most often found in a horse with base-narrow, toe-out conformation. After the foot trav arc it lands more or less directly in front of the opposite forefoot. In some cases this leads to stumbling as a result of a degree of plaiting.

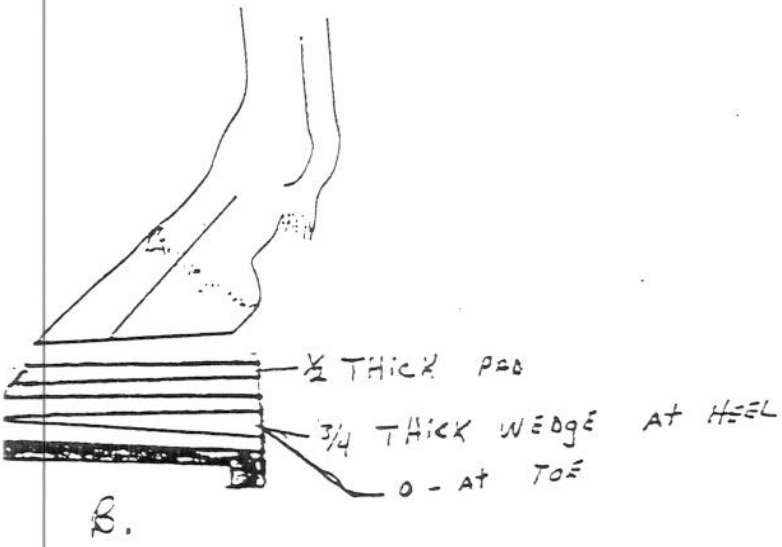


(Right) This horse cannot travel for any length of time without being raised in the heel and support behind the heel, due t the stress that the calf knee puts on the fetlock, and cannot be corrected using 1" of pa., but can travel sound and be shown successfully with 3" to 4" of corrective build-up.

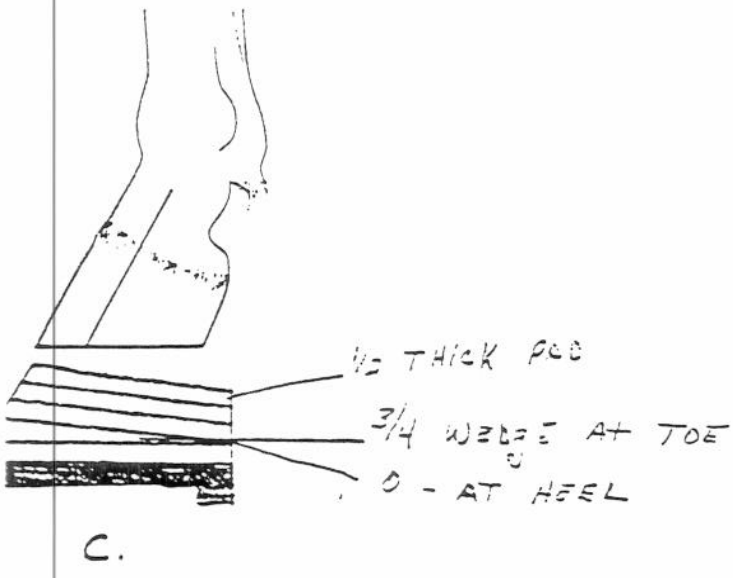
FIG. 2–17. Example of normal (left) and calf-kneed (right) position of the forelimb. (Courtesy of Dr. W. Berkley.)



A. This horse has a normal foot, so he is easy to shoe with or without pads. But as you can see in the field of horses, very few are normal.



B. This horse with an abnormal foot and pastern axes, can be shown successfully with no extra stress or strain on foot or pastern axes. You do need two or three pads next to the foot for a foundation, then a wedge for correction, a flat pad to come in contact with the ground, then the shoe.



C. This horse with an abnormal foot and pastern axes, can be shown successfully with no extra stress or strain on foot or pastern axes. You do need two or three pads next to the foot for a foundation, then a wedge for correction, a flat pad to come in contact with the ground, then the shoe.

FIG. 2–36. Side view of foot and pastern axis. *A*. Normal front foot and pastern axes (approximately 47°). *B*. Foot and pastern axes less than normal (less than 45° in front or less than 50° behind). *C*. Foot and pastern axes greater than normal (greater than 50° in front or greater than 55° behind).

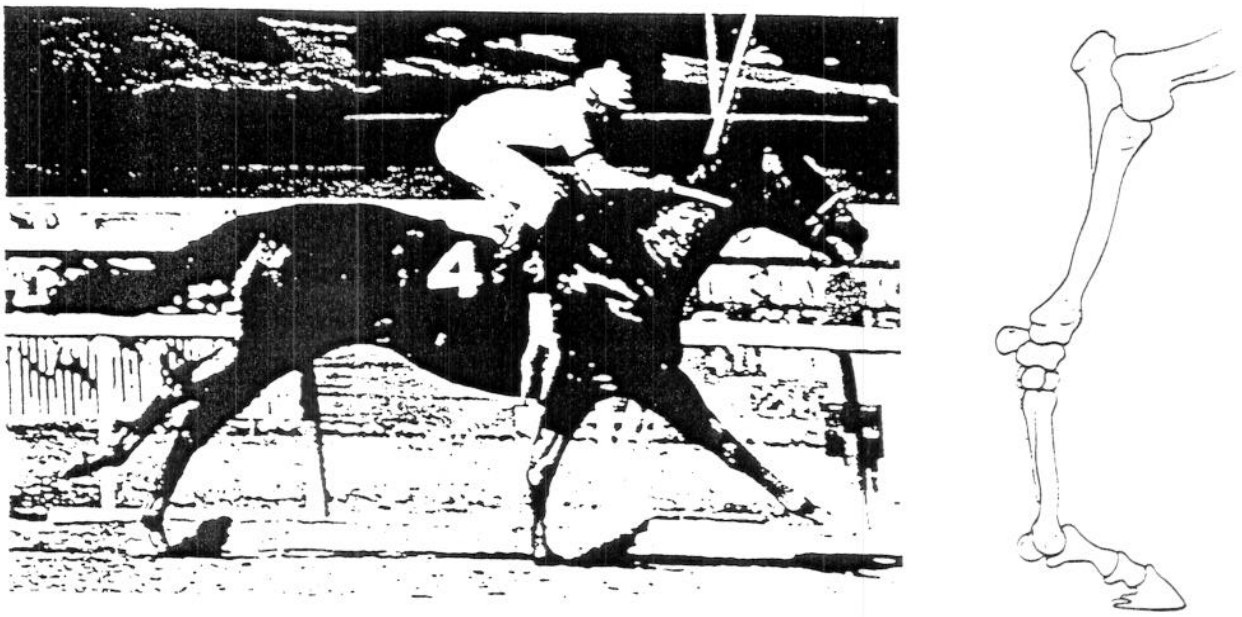


FIG. 2–18. Photograph of a Thoroughbred near the finish of a race. Note the backward deviation of the carpus, predisposing to chip fracture of the carpal bones. If a horse has a backward deviation of the carpus before limb fatigue forces it into this position, there is a greater possibility of carpal fracture. Courtesy of Dr. W. Berkley.[1]

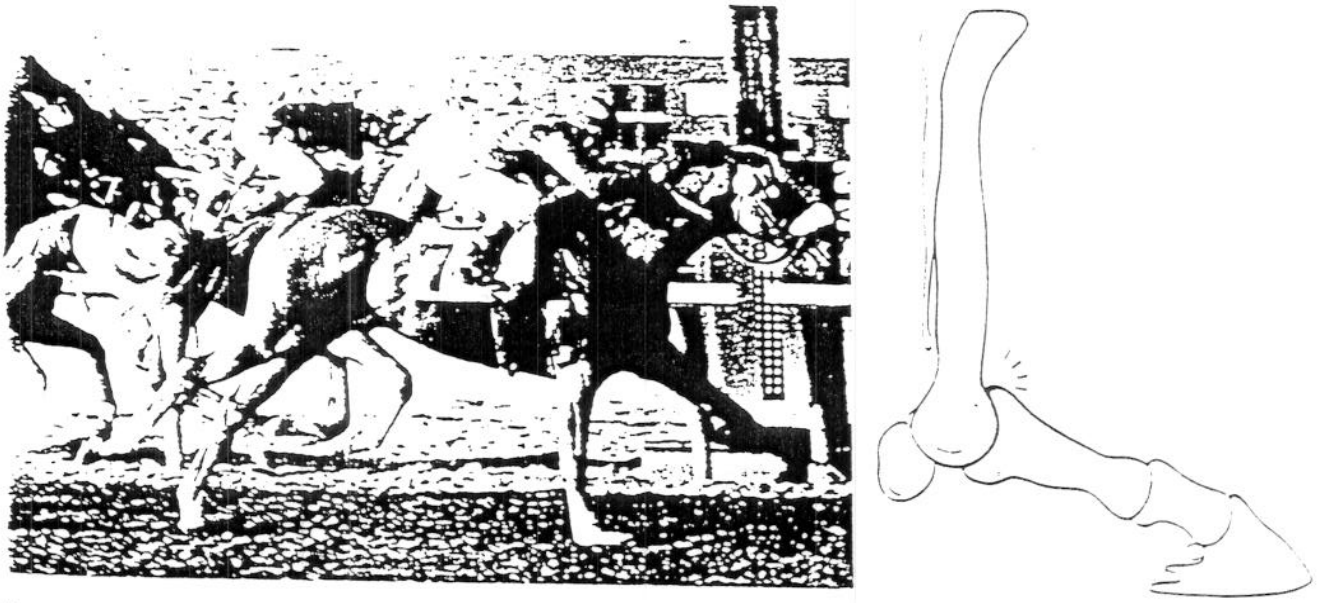


FIG. 2–19. Photograph of a Thoroughbred near the finish of a race. Note the backward deviation of the carpus and the [illegible] at the fetlock. When the fetlock is in this position there is a possibility of chip fracture of the proximal [illegible]. Courtesy of Dr. W. Berkley.

...sion. This condition causes increased tension ... the lateral surface of the limb, particularly, the lateral collateral ligament of the carpus. An increased compression force is distributed to the medial surface of all joints with an increased compression force being brought to bear on the medial carpal bones (see Chapter 8).

Open Knees

The term refers to an irregular profile of the carpal joints when viewed from the side (Fig. 2–21). This irregularity gives the impression that the carpal joints are not fully closed. This conformation is usually found in young horses (1 to 3 years of age) be-