SHOEING FOR LAMENESS OF THE FOOT

by Andy Weitzman '87
with special thanks to Buster Conklin,
Farrier, Large Animal Hospital

When considering the soundness of horses, proper foot care is of great importance. A significant percentage of forelimb lamenesses are in the foot. This differs from the hind limb where most lameness occurs in and above the hock, in the stifle, or in the hip. This difference reflects the fact that the forelimbs bear most of the body weight (60-65%) and are subject to greater concussion forces, whereas the hindlimbs are involved in propelling the animal.

Foot care is not the only consideration in lameness, of course. Many factors such as conformation, type of work, diet, environmental conditions and injuries are related to the soundness of the horse. But as the saying goes, "no foot-no horse" and, in this respect, without proper foot care you won't have a sound horse. The essential ingredients of good foot care include clean bedding, frequent cleaning and picking of the feet, and regular trimming and shoeing by a skilled farrier.

Corrective shoeing is often a part of foot care and can be divided into corrections for conformation and gait deficits and shoeing for lameness due to injury or disease (also called pathological shoeing). Corrective shoeing for conformation and gait usually involves redistributing the horse's weight over the foot ("balancing the foot") and leg or redirecting the force acting on the foot as it takes off from or lands on the ground. This article will discuss pathological shoeing for lameness in the foot.

As with any case of lameness, a thorough examination of the horse both at rest and in motion is a necessary prerequisite to treatment. In the case of foot lameness, close examination of the foot would include comparing sound and un­sound feet with respect to size and shape (an inflamed foot grows faster), feeling for heat, squeezing with hoof testers to determine sensitive spots and paring the foot to examine the sole and frog for evidence of abscesses, punctures or thrush. To definitively isolate the cause of lameness, nerve blocks and radiography are often necessary.

Just as the causes of lameness vary greatly, there are often many ways to shoe a lame horse that will achieve the desired results. Different farriers may have different techniques, but what counts the most is the end result of providing relief to the horse and preventing the problem from growing worse. Shoeing for lameness generally involves a shift in weight distribution by trimming or use of a particular type of shoe, protection of the foot with pads and cushions and support of the foot.
Three case histories of horses that were treated by Buster Conklin, the farrier here at Cornell, will serve to illustrate some of the techniques used in shoeing the lame horse.

Case #1

This horse came in very lame on the left foreleg. Radiographs showed that he had sidebone on the outside of the foot. Sidebone is an ossification of the lateral cartilages of the coffin bone (the distal phalanx) causing loss of flexibility and frequently lameness. Compounding the problem was the fact that this horse was low on the inside of the foot, causing the outside to hit the ground first and increase the pressure and irritation of the sidebone.

Sidebones don't go away, they can only be removed surgically (removal of the lateral cartilages). Therefore, relief of the chronic irritation is the aim of corrective shoeing. To treat this horse, the weight bearing surface was shifted away from the outside by trimming the outside down and putting a wedge pad sideways, elevating the inside wall. A bar shoe was used to give full support all around the foot.

In three days time this horse was walking considerably better. As his feet grew out, they were trimmed level and either shod normally or with a wedge pad, depending on the degree of lameness.

Case #2

This horse arrived extremely lame in the right foreleg. He had a history of navicular disease, but radiographs didn't show any further deterioration of the navicular bone that supported the severe lameness observed. Examination with hoof testers showed the medial quarter to be very painful, indicating a bruise. This horse also had very long toes and short shoes which created excess strain on the back of foot (the quarters) and on the flexor tendons of the leg.

Corrective shoeing for this horse aimed at relieving the weight-bearing strain on the bruised medial quarter and on the flexor tendons. This was accomplished by shortening and rolling the toe which facilitated easier breakover of the foot in motion. The bruised inside quarter was trimmed more than the rest of the foot so that it didn't support weight. Full pads were used to decrease concussion and protect the foot and an egg bar shoe was used to create full support with a longer platform behind the heel.

This treatment was very effective and, mainly due to the relief of pressure on the bruised medial quarter, this horse walked off 80-90% sound.

Case #3

This horse was lame in both front feet due to chronic laminitis. Both front feet were severely affected with rotations of the distal phalanx of 13° and 17° respectively. Any rotation greater than 4-5° is bad and becomes very painful as the front edge of the coffin bone presses into the sole.

To treat this horse a fairly new technique was used; one previously developed in Texas. The method consisted of cutting and removing the wall in the front part of the foot and covering it with a protective wrapping. Then a heart-bar shoe was placed on both feet. This shoe is heart-shaped with the inside point of the heart pressing on the point of the frog. This puts pressure on the coffin bone to stabilize and prevent further rotation and reduces the pain caused by the coffin bone digging into the sole. This added pressure on the point of the frog is matched by the absence of pressure on the front of the hoof wall.

This technique, though certainly not a "cure all" for chronic laminitis, significantly improved the condition of this horse and is an effective way to shoe severely foundered horses.

These are but a few of the corrective techniques used in shoeing the lame horse.

Note: ANDY WEITZMAN is a 2nd year vet student - class of 1987, and is interested in all aspects of equine medicine.
Subsection (g) of the American Horse Show Association's Drugs and Medications Rule has been designed to provide for the legitimate treatment of illness and injury of a horse in competition within the confines of the Rule. Subsection (g) states that a horse receiving any medication which contains a substance forbidden by the Rule may be eligible for competition if certain requirements have been met.

First of all, the medication, administered by a licensed veterinarian or the trainer, must be "therapeutic and necessary" for the treatment of an existing illness or injury. This does not include the administration of a tranquilizer or like substance for clipping or shipping. Secondly, after administration of the forbidden substance the horse must be withdrawn from competition for at least 24 hours. Forbidden drugs and medications may be detected in a horse's blood for longer than this period of time, however if the horse is tested at the show and a forbidden substance shows up in the chemical analysis, compliance with subsection (g) by the trainer will be entered as evidence in the AHSA's investigation of the matter.

Compliance with subsection (g) also requires the filing of a written report with a Steward/Technical Delegate of the show or event. Information to be included in this report, as stated in the Rule, includes identification of the medication, diagnosis and reason for administration and identification of the horse. The report must be signed by the person administering the medication and the Steward/Technical Delegate, within certain time restrictions. Dr. John G. Lengel, Administrator of Drugs and Medications for the AHSA, offers this seven day advice:

Any time a horse is administered any forbidden substance in the seven days preceeding the time of competition, file a medication report and otherwise comply with all the requirements of subsection (g). Nontherapeutic administrations excluded.

While seven days prior to a show may seem like a long time, some drugs (e.g. procaine) may be detected by laboratory chemical analysis six to seven days after administration.

The AHSA Drugs and Medications Rule is one good reason to train your horse to be clipped or shipped without tranquilizers. If you absolutely must tranquilize your horse to ship him, note that subsection (g) does not apply (nontherapeutic use of medications) and filing a medication report is not indicated. Ship the horse seven days prior to the competition to avoid having a blood sample from your horse turn up positive for a forbidden substance should you be tested. If a crisis should arise while shipping and medications are administered to protect the horse's health, file a medication report when you reach the show and comply with all other provisions of subsection (g). Be aware that all medication reports are kept as a permanent record by the AHSA in an effort to minimize abuses of these provisions.

The Drugs and Medications Rule applies to all entrants into AHSA-recognized competitions, thus insuring the fairness of competition for all involved.

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Dr. Susan Fubini grew up in Maryland, Connecticut and Virginia. She spent her first undergraduate year at Tufts University and the last two at Virginia Polytechnic Institute. She received her D.V.M. at the University of Georgia College of Veterinary Medicine and then came here to the New York State College of Veterinary Medicine at Cornell for an Internship in Large Animal Medicine and Surgery. She continued here as a Resident in Large Animal Surgery and was recently appointed as Instructor in Large Animal Surgery.

Equine Rounds:
Tell us about your equine background.

Dr. Fubini:
I have a hunter-jumper background. I worked with them from age thirteen to eighteen, showing and training. My family had always kept horses for the kids and I began with the Pony Club and local horse show routine. My career in equitation began when the local stable was taken over by a professional horseman. I ended up training with Ronnie Mutch for years.

Equine Rounds:
Did you always dream of being a veterinarian?

Dr. Fubini:
No, I thought horses were my life. But at 16, the glamour of showing was wearing off. I was at my first year at Tufts when the university shut down for three months during the energy crunch. So I worked three months as an independent study with the vet at my stables. I had a great time.

Dr. Fubini:
Not entirely. My sister is a veterinarian in a small animal practice, so the thought of becoming a veterinarian was always in the back of my mind. Gradually my time working with veterinarians became more meaningful to me than my time at horse shows. I started to gradually phase out of the horse shows and concentrate on studying.

Equine Rounds:
While in vet school you helped establish University of Georgia's Colic Team. Did you have a part in starting Cornell's Large Animal Emergency Crew Team?

Dr. Fubini:
Yes. The team worked so well in Georgia that it made sense to do it here.

Equine Rounds:
This issue is featuring lower limb lameness. Time to switch gears - find out what Cornell is doing. There is a lot of talk about a new vasodilator drug, isoxoprine, used for navicular disease. How do you feel about it?

Dr. Fubini:
Isonoprine is a new drug and we are just starting to use it. There have been very few clinical trials, but many practitioners feel it has efficacy in treating navicular disease. Some clinicians here are seeking grant money to study its vasodilatatory effects, and be sure that there are no systemic side effects.
Equine Rounds:
What does Cornell recommend for a horse with navicular disease?

Dr. Fubini:
Clinicians at Cornell prescribe corrective shoeing and analgesics. If this regime has been tried religiously we may, as a last choice, recommend posterior digital neurectomies. However, there are a lot of problems with this surgery such as nerve regrowth and neuroma formation. Dr. Cummings, Dr. Todhunter, Dr. Erb and I are investigating a method to prevent neuronal proliferation. You must have an intact cell body for nerve proliferation. We will be injecting a neurotoxic drug into the cut end of the nerve. The drug travels up the nerve and kills the ganglia and therefore prevents axonal proliferation. What makes this even more interesting is that this technique (if successful) has a human application in amputee neuromas.

Equine Rounds:
Tell us about the new procedure of periosteal tripping to correct angular limb deformities.

Dr. Fubini:
There are three major types of angular limb deformities of the carpus which must be differentiated. The first is a laxity of the ligamentous structures surrounding the carpus. This is straightened manually with casts or splints. The second is a rotational problem, which is a true conformational defect. Corrective trimming may be of some value for this. The third type is due to abnormal growth at the distal radial physis (growth plate just above the knee) and can be surgically corrected. Traditionally, a screw and wire or stapling technique has been used which stops the growth of the medial (inner) radius and allows the lateral (outer) side to catch up. However, this procedure leaves a scar and a second surgery is required to remove the implant. The new procedure of periosteal stripping seems to work. It was first used in the horse by Dr. Aver at Texas A&M. The periosteum is cut on the distal lateral radius (just above the outside of the knee). This acts as a releasing mechanism for physeal growth. It essentially allows bone growth on the outer aspect of the growth-plate (physis) to catch up with the inner aspect of the growth plate.

Equine Rounds:
At what age do you use this technique of periosteal stripping?

Dr. Fubini:
We like to use it on foals at 1-2 months of age. A lot of "crooked foals" will straighten by one month of age on their own. This is still a relatively new procedure in horses and long term data and followup is minimal. However, it seems to work and practices that see many foals report good success.

Equine Rounds:
How do you feel about being here at Cornell?

Dr. Fubini:
The strongest point for me is the people I work with. There are a lot of young faculty members. We work well together. The atmosphere is excellent; between the cases, the clients, the students, and the administration, it is a constant challenge.

KATHLEEN O'BRIEN '86 is interested in equine practice after graduation.
LAMINITIS - WHAT IS IT?

by: Pamela Livesay-Wilkins '86

Special thanks to Dr. Prabin Mishra
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No foot, no horse. This saying has been repeated by horsemen for centuries, and in reference to laminitis - also known as founder - it becomes a truism. Although laminitis has plagued horses for centuries and affects thousands of them yearly in the United States alone, no one yet knows exactly what causes the disease or how to prevent it. Laminitis is defined as an inflammation of the dermal laminae, over which the horny hoof slides. The pedal laminae also serve as one of the mechanisms supporting the coffin bone (the third phalanx) within the hoof. Various theories exist as to why damage is restricted to the hoof in laminitis. Suggested mechanisms by which damage occurs are endotoxemia, vasodilation/stasis and vasoconstriction.

All four feet may be afflicted in laminitis and lameness develops rapidly. The signs of acute founder are quite characteristic and include a reluctance to move forward and a pronounced heel-toe landing when the horse does move, a so-called "walking on eggs" gait. The affected foot will be warm or hot to the touch and the digital arterial pulse will be strong. The horse will also be sensitive to hoof testers applied to the sole of the hoof. Some horses may show signs of endotoxic shock and/or diarrhea. A dropped sole, bilateral lameness and a diverging growth line in the hoof wall are all indicators that the laminitis is chronic. Most therapy is aimed at preventing or halting rotation of the coffin bone through the sole of the hoof. (See article on shoeing for lameness in this issue.)

Laminitis has been associated with several conditions, among them acute intestinal disease (colic), primary hepatic disease, bacterial septicemia, stress from surgery, prolonged exercise, carbohydrate or grain overload, retained placenta, metritis, excessive water consumption following exercise and excessive trimming of all four feet. Horses, particularly ponies, may develop "pasture founder" in the spring when the grass is lush, but overeating grain appears to be the most common cause of laminitis. During grain overload, lactic acid-producing bacteria proliferate in the cecum and intestine and release large amounts of lactic acid causing a rapid drop in the pH in the cecum. This pH drop (or increase in acidity) in turn kills bacteria known as Enterobacteria spp., liberating endotoxin in the cecum that eventually enters the bloodstream of the animal.

Lactic acid, endotoxin and histamine are all potent vasoactive substances that have been associated with the development of laminitis in many of the conditions described above. As these substances are produced, they enter the bloodstream and affect blood vessels by stimulating cell proliferation, inducing damage to the endothelial cells lining the blood vessels, thereby increasing the permeability of the vessels and constricting arterioles. These compounds also decrease the oxygen-carrying ability of the blood and increase blood coagulation, with ischemia (oxygen deprivation), exudation and cellular proliferation being the final results in the hoof.

Here at Cornell, Dr. Prabin C. Mishra of the Veterinary Anatomy Department is currently engaged in research supported by the Zweig Fund, aimed specifically at determining the compounds responsible for the lesions of laminitis and the mechanisms by which these lesions develop. It is only when these aspects of the disease are clearly defined that a rational treatment for laminitis can evolve.
Using electron microscopy, Dr. Mishra is examining the lesions found in horses experimentally exposed to histamine to determine if it produces lesions similar or identical to those found in naturally occurring laminitis cases. He will be repeating this experiment using lactic acid and endotoxin as well as various combinations of all three compounds to determine their roles in the pathogenesis of laminitis. The ischemia, exudation and cellular proliferation caused by histamine, endotoxin and lactic acid damage the hoof in different ways. In ischemia, the epidermal cells of the hoof receive fewer nutrients and less oxygen. This results in destruction of the uppermost layer of cells, concurrent with a proliferation of subepithelial capillaries in an attempt to get more blood, and thus more nutrients and oxygen, to the area. The basal cells of the epidermis also rapidly increase in number, and all these factors together result in separation of the hoof wall and coffin bone from one another. In the normal hoof the coffin bone is always under tension to rotate away from the hoof at the toe; in laminitis this is allowed to occur as the link between these two structures is weakened.

Treatment of acute laminitis is always an emergency. Critical to its treatment is the time that is allowed to elapse between the commencement of signs of the disease and the initiation of treatment. Any delay beyond 24 hours could be fatal and, if the animal goes beyond 48 hours without treatment, the prognosis is poor. The first consideration in treatment is to remove or control the primary cause of disease, such as endotoxemia following grain overload. Arterial blood pressure is elevated in acute laminitis and it may be lowered by administration of small doses of phenothiazine tranquilizers, diuretics or removal of salt from the diet. The general treatment program for laminitis usually includes phenylbutazone, mineral oil and a reduced salt diet. Antihistamine drugs have been used and are helpful if given early in the course of the disease.

Phenylbutazone is used for its antiinflammatory and analgesic properties. Corticosteroids may be used in the initial anti-shock therapy, but treatment with this drug beyond the first 24 hours may worsen the condition by further compromising protein synthesis in the hoof. A mild purgative such as mineral oil given by stomach tube has a laxative effect that hastens the elimination of toxic ingesta and helps to prevent further absorption of endotoxins.

If the animal will walk, controlled exercise for short periods at frequent intervals is beneficial, as it increases the movement of blood through the foot. Walking the horse remains one of the important features of a good recovery. Butazolidin can be used to relieve the pain so the animal will walk. A palmar nerve block can also be done for the same purpose and has the additional benefit of re-establishing blood flow to the toe of the hoof in many cases. One word of warning however: a horse that has already suffered a rotation of the coffin bone will be very reluctant to walk and should not be encouraged to do so.

Acutely affected horses may be stood in cold water to decrease the oxygen requirements of the tissues of the hoof. This treatment may be alternated with short periods of hand walking. The soles of the affected hooves may be filled with plaster of Paris or some similar compound to help prevent rotation of the coffin bone or, if a blacksmith or farrier is available, a special shoe such as the heart bar shoe can be placed on the feet for the same purpose. The feet should be radiographed to monitor any rotation of the coffin bone and to determine the extent of permanent damage to the hoof.

Medical treatment of chronic laminitis is seldom satisfactory. Old-time horsemen say to stand a horse with chronic laminitis in a stream,
and certainly this will alleviate some of the pain. Corrective trimming, both of the feet and of body weight, is the best approach to managing horses with chronic laminitis and keeping them usable. Corrective shoeing can improve the angle of rotation of the coffin bone, usually using a reverse wedge pad and a wide bar shoe, but care should be taken that the wide bar does not compress the sole, as this can result in sole necrosis and rupture.

Some horses with acute laminitis respond to therapy and don't develop coffin bone rotation; others develop complications that may lead to recurrent problems. As stated before, the exact nature of the causes of laminitis are not known and most current therapy is aimed at treating the symptoms and not the underlying cause. Researchers such as Dr. Mishra are working on the problem and someday will have a rational treatment and prevention scheme worked out, but this ideal situation still is in the future.

The horse owner can do a few things to lessen the chance that his horse will be one of the thousands who develop this disease each year. Heavily fed or fat horses should be given some exercise when they are not working, such as being turned out or hand walked or longed. If possible, horses being shipped over long distances should be periodically removed from the trailer or van and given light exercise. They should also be rested off the trailer for several hours each day if possible. Mares that foal should be monitored carefully for signs of a retained placenta; if the mare has not completely expelled the placenta within 24 hours of foaling a veterinarian should be contacted. Carbohydrate overloading should be avoided at all costs; all horses that get into the grain bin and eat their fill should be seen by a veterinarian as a matter of course. Fat ponies that tend to founder each spring should be slowly introduced to their pastures and closely observed for signs of founder or colic.

Laminitis is a frightening disease for horse owners, but research is going on that should lead to practical rules for prevention and rational standards of therapy when the disease occurs. Horse owners will be among the first to find out when this happens, but common sense and a quick eye for recognizing the problem when it does occur will help horses over a crisis of laminitis right now. Remember, no foot, no horse, and laminitis is an emergency situation.

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