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Iowa State University

Eric L. Reinertson
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Theory and Application of the Chapman Heartbar Horseshoe for Laminitis

William J. Skog, BS, DVM*
Eric L. Reinertson, DVM, MS••

INTRODUCTION

In the past fifteen years great advances have been made in understanding the pathogenesis and treatment of laminitis. One of these advances is the use of the heartbar shoe. In theory the Chapman heartbar should work well for the treatment of laminitis, however, in practice favorable results depend on the proper application of the shoe along with supportive care of the foot.

The hoof is analogous to the human fingernail. Its outer surface is smooth and covered with a thin layer of cells called the periople. This layer acts like a coat of varnish and holds moisture inside the hoof. The majority of the thickness of the hoof is composed of tubules that grow down from the coronary corium and are embedded in a matrix of keratin. The inner surface of the hoof is arranged in parallel vertical leaves. These leaves, called the insensitive laminae, interdigitate with the laminar corium, or sensitive laminae of the coffin bone. The interdigitation of these two laminae holds the coffin bone in its proper position inside the hoof.

Encased inside the hoof is the coffin bone, or third phalanx (P3). The peristomeum of the coffin bone and the dermis covering it are collectively known as the corium. The corium is very vascular, contains nerve endings and is divided into several parts (Figure 1). On the dorsal surface of the coffin bone is the laminar corium (sensitive laminae). Located along the coronary band (Figure 2) is the coronary corium. The perioplic corium is a thin band of corium just above the coronary band which produces the periople. The solear corium produces the sole.

The corium is drained by masses of veins called plexi. The plexus that drains the coronary and perioplic corium is called the coronary venous plexus. The venous drainage of the laminar corium is called the dorsal venous plexus. The solear corium is drained by the palmar (plantar) venous plexus.

The coronary plexus is supplied primarily by the dorsal branches to the second phalanx of the medial and lateral palmar (plantar) digital arteries (Figure 3). The digital arteries then pass through the solear foraminae in the coffin bone and anastomose in the arcus terminalis. Vessels of this terminal arch then pass through channels in the coffin bone called semilunar canals and re-emerge at the distal border of the coffin bone to form the circumflex artery. Blood from the terminal arch

* Dr. Skog is a 1985 graduate of the College of Veterinary Medicine at Iowa State University.
** Dr. Reinertson is an associate professor of Veterinary Clinical Sciences at Iowa State University.
and circumflex artery empties into the dorsal venous plexus as well as part of the palmar (plantar) venous plexus. The rest of the palmar (plantar) venous plexus is fed by arterial branches to the digital cushion from the medial and lateral palmar digital arteries. There are vast amounts of vessels, mainly veins, throughout the foot. The normal weight-bearing surfaces of the horse include the wall, the bars, and the frog (Figure 4). Very little weight is borne by the sole. The frog and its deeper counterpart, the digital cushion, are relatively avascular areas of the foot that absorb the initial force of impact when the foot hits the ground. The digital cushion (Figure 2) is composed of fibroelastic cartilage. Much of the energy absorbed by the digital cushion is transferred to the venous plexi which act as hydraulic cushions and help to pump blood up the leg.

Laminitis (founder) is defined as an inflammation of the laminar corium and may be caused by many pathologic states, the most common being grain overload founder.3 Other highly digestible carbohydrates will also cause founder. Grass founder and beet-top founder are good examples.3,4 Severe systemic diseases such as acute enteritis, metritis, retained placentas, pneumonia, pleuritis, or toxemia will cause laminitis.3,4,5,6 Severe stress such as abortion, dystocia, yearling sales, extended shipping, and peak training periods may also cause laminitis.5,7 Stress associated with hormonal changes may lead to a recurrent founder during parturition and breeding season in certain mares.3,4 Steroid treatment may predispose horses to laminitis.8 Hot horses that drink a lot of cold water may cause water founder.3,4 The opposite limb supporting weight due to a chronically lame limb may become foundered.9 Local trauma from working too long on a hard surface may cause road founder.3,4

The signs of laminitis may be variable. Classically, the horse will show pain in the af-
affected feet and take short choppy steps or refuse to walk. The horse will attempt to take weight off of the toes and place it on the heels. There will be a bounding digital pulse and the feet will be hot. If only the front feet are affected the horse will bear more weight on his back feet and stand camped under behind. Laminitis may affect only one foot as in opposite leg lameness, or it may affect all four feet and the horse will be down. The severity of the laminitis may lead to varying degrees of coffin bone rotation. In a mild case the coffin bone may not rotate at all. If the coffin bone has rotated a depression behind the coronary band at the toe can be felt. If the laminitis is very severe, the coffin bone will sink instead of rotate. In this case there will be a depression behind the coronary band at the heels as well as at the toe.

With the possible exception of road founder and laminitis due to opposite leg lameness, all causes of laminitis probably follow one final pathogenic pathway.

PATHOGENESIS OF LAMINITIS

Grain overload is the only reliable model used to study laminitis. The easily digested grain causes an overgrowth of lactic acid producing bacteria. This produces an acidic environment in the gut that kills off the normal flora of Enterobacteriaceae. When these bacteria die they release endotoxins which damage the intestinal mucosa and enter the systemic circulation. The horse develops diarrhea and the end result is a dehydrated, hypovolemic horse with systemic endotoxemia.

The natural response to shock is to maintain blood flow to the heart and brain. Blood pressure to these organs is maintained by intrinsic release of cortisol and norepinephrine resulting in stimulation of alpha-adrenergic receptors in the pre-capillary sphinctors of peripheral arterioles and general peripheral vasocostriction. Arterio-venous (A-V) shunts develop so that blood is returned to the heart to maintain central venous pressure. Research has shown that during the onset of laminitis, A-V shunts develop in the vascular foot. The arterioles in the semilunar canals constrict and shunt blood through the arcus terminalis into larger vessels. This explains the bounding digital pulse characteristic of laminitis. Constriction of these arterioles feeding the dorsal plexus and the laminar corium causes ischemic necrosis of the laminar corium. When the laminar corium dies the coffin bone rotates because it is no longer held in place by the interlacing of the laminae. In severe cases of laminitis, blood doesn’t reach the terminal arch and all of the laminae may die in this case. Instead of rotating, the coffin bone sinks in the hoof which may eventually slough.

Histamine release may or may not cause the vasocostriction. In a study of the vascular response of the equine foot to exogenous drugs, catecholamines such as epinephrine did cause vasocostriction of precapillary arterioles. Cortisol potentiates this affect of epinephrine, as well as potentiating the ability for blood to spontaneously coagulate and plug capillaries. Endotoxins worsen the condition by damaging blood cells and vascular endothelium. Reflex vasocostriction due to pain may also play a role in the ischemic process.

Since horses evolved as herbivorous animals subject to predation, their fight or flight system is highly developed. They release massive amounts of catecholamines and steroids in response to stress. These substances interact in an exaggerated physiological response that results in laminitis. Any combination of stress, shock, or increased coagulation of the blood may lead to laminitis. This does not completely explain road founder or opposite leg lameness founder. Direct trauma and

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venous congestion may play important roles in these types of founder.

The laminae near the toe have the least collateral circulation and are the most common laminae to undergo necrosis. The laminae at the quarter and heel often remain viable. The horse's weight acts as a force on the front of the coffin bone, and the deep digital flexor tendon exerts upward pull that allows the coffin bone to rotate. The viable laminae at the heels act as a pivot point for this rotation (Figure 5). Horses severely affected in all four feet due to some severe systemic disease usually die. If only one foot is affected some horses can be saved using thermoplastic devices to support the frog and act as an artificial wall. However, if more than one hoof sloughs the best recommendation may be euthanasia.

The horse will exhibit signs of pain with any degree of founder. Pain associated with laminitis keeps the horse from increasing the damage by walking. However, pain also increases stress which leads to more vasoconstriction and ischemia. The horse may be down and unable to move leading to muscle necrosis and decubitis ulcers. Most treatments for laminitis deal with reducing the pain in the foot. Pain associated with laminitis results when the tip of the coffin bone puts pressure on the sole after it has rotated. Another type of pain is the tearing pain that the horse feels when it breaks over at the toe. The ground exerts a force on the hoof that tears it away from the coffin bone. Nerve endings in the sensitive laminae exposed and irritated will also cause pain.

Diagnosis of laminitis is made by the history and physical signs. Radiographs are useful in detecting rotation; however, they may be misleading if the coffin bones sinks rather than rotates.

**TREATMENT OF LAMINITIS**

The treatment of laminitis depends on the underlying cause, the severity of ischemia, and the duration since onset. The underlying cause must be treated first. This article will deal only with specific treatment of laminitis. Acute laminitis is treated as a medical emergency before damage becomes irreversible. Fluid therapy should be instituted to correct any deficits. Ideally one should estimate ongoing losses and daily maintenance requirements and correct for these also.

Alpha-blocking drugs can be used to counteract the peripheral vasoconstriction. Acetylpromazine may be useful, but has a short duration. Promazine hydrochloride has a longer action. Phenoxylbenzamine may be the drug of choice because of its long action and lack of tranquilization. Care must be used when administering alpha-blockers if fluid deficits have not been corrected. Cerebral blood flow may be decreased by the peripheral vasodilation induced by alpha-blockers and cause syncope.

Phenylbutazone or flunixin meglumine at high doses have several good effects. Besides their analgesic and anti-inflammatory properties, both drugs inhibit prostaglandins and will prevent platelet aggregation. Therefore thrombosis may be inhibited. Although aspirin has little anti-inflammatory action in the horse, it too acts to inhibit platelet aggregation and may be useful. Heparin can also be used to inhibit clotting, but is more likely to cause hemorrhage and is generally not recommended.

Local nerve blocks may also be helpful to decrease reflex vasoconstriction due to pain in the feet. Care should be taken to discontinue nerve blocks if rotation occurs, as continued walking will worsen rotation.

Several authors feel that cooling the feet with cold water for ten or fifteen minutes twice a day is beneficial. This reduces pain and some of the inflammation. It may...
also decrease the metabolic requirements of ischemic cells and allow them to survive without oxygen for a longer period of time.\textsuperscript{13}

The coffin bone must be supported to prevent stress on the laminae. Theoretically this is easy to do by supporting the sole with a bar shoe (Figure 6) or cast.\textsuperscript{4,18,19} In practice this rarely is satisfactory because of the pressure necrosis and abscesses that develop on the sole. A convenient way to support the coffin bone is to stand the horse in a sandy stall.

Many attempts have been made to correct $P_3$ rotation by application of special hardware and rasping techniques. The most effective methods appear to be the pad support and the Chapman heartbar shoe.

Several types of frog support pads have recently come on the market. The Poly-Hoof pad (Figure 7)\textsuperscript{24} effectively supports the coffin bone by putting pressure on the frog and concentrating that pressure at the apex of the frog. It is relatively easy to apply and minimizes the chance of pressure necrosis of the sole. It can be used prophylactically to minimize rotation that may occur in high risk patients, such as post-surgical colic cases and horses with acute enteritis and diarrhea. It gives support to horses with acute laminitis and can be used on horses with rotation in absence of severe abscesses. The pad's drawback is that it interferes with treating active abscesses.

The Chapman heartbar is most useful in treating chronic cases of laminitis with rotation and draining abscesses.\textsuperscript{13,22} The Chapman heartbar (Figure 8) consists of an elevated bar, shaped like the frog, which concentrates pressure at the apex of the frog and pushes the coffin bone into a normal pattern angle. The toe of the shoe is intact in order to protect exposed sensitive laminae, and it is rolled to decrease the force required by the deep digital flexor tendon to break the foot over. The inner edge of the hoof surface is bevelled to decrease the chance of pressure necrosis in cases of dropped sole. The nail holes are fullered so that a nail puller can be used to remove each nail separately, thus reducing trauma to the hoof during shoe removal. The heartbar must originate from the caudal aspect of the web of the shoe to prevent pressure necrosis in the buttress area of the sole.\textsuperscript{22}

A shoe similar to this was used in the 1800's to prevent road founder in carriage horses.\textsuperscript{23} Chapman modified this shoe so that it put increased pressures at the apex of the frog, stabilizing the coffin bone. By putting the pressure on the relatively avascular frog and digital cushion and by keeping pressure off the sole he has been able to minimize necrosis of the sole.

To apply the Chapman heartbar, the feet should not be blocked because the horses's reactions will indicate that the shoe is being applied correctly. The most severely affected foot should be handled first. A gauze pad can be taped under the frog of the opposite foot to establish temporary frog pressure and relieve pain.\textsuperscript{18} Abscesses and necrotic material

\begin{figure}[h]
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\includegraphics[width=\textwidth]{figure6.png}
\caption{The Frog Apex Bar Shoe}
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\includegraphics[width=\textwidth]{figure7.png}
\caption{Poly-Hoof pad A. hoof surface B. ground surface}
\end{figure}
should be trimmed from the sole to allow better drainage. The heels should not be lowered at this time, as this will decrease flexor tendon tension on the coffin bone. The hoof wall is resected with a dremel tool, rasp, or sharp hoof knife (Figure 9). This serves four functions: it relieves pressure constricting vessels in the coronary plexus; it debrides necrotic laminae, establishes drainage, and allows better regrowth of new hoof matrix; it relieves pressure pain from swollen laminae; and it decreases resistance on the coffin bone when it is pushed into its normal position by the heartbar. 25

The shoe and the heartbar are shaped with the contour of the foot. The apex of the bar should be % inch behind the apex of the frog, and no part of the bar should overlap the frog or touch the sole, bars, or sulci. If the bar touches sole outside the frog it will cause necrosis.18,22

The most delicate judgment is deciding how much pressure to apply to the frog and where to apply it. Radiographs will give an idea, but too much pressure over too small of an area will cause pain. If too much pressure is applied an abscess may form under the frog. If the horse pulls away with the shoe seated on the hoof without nails, too much pressure is being applied by the heartbar and it should be adjusted. With only the heel nails in place, allow the horse to walk. If it won't, there is too much pressure on the frog.18,22 If the horse does walk but becomes increasingly lame over a few days, there is probably not enough pressure on the frog. The shoe must be removed and the heartbar adjusted until the correct amount of pressure is established. The shoe can then be affixed using all the nails.

Contact between the heartbar and the frog increases the surface area supporting the horse's weight. It is suggested that at least the cranial third of the frog be supported. Two common technical faults in applying the heartbar include; allowing only the tip of the bar to touch the frog, and using too steep of an angle for the heartbar.26 Mechanical pressure used to force the coffin bone into a normal angle will cause some degree of necrosis of the frog.18 However, if properly applied, this necrosis can be minimized.

Rim pads are often used to elevate dropped soles from pressing on the ground. This will decrease much pain and pathology.

Another asset of the heartbar shoe is that a boiler plate can be added to the bottom of the shoe. It can be removed for easy treatment of abscesses and when in place can protect the bottom of the sole from trauma. The biggest
Figure 9. Hoofwall Resection. A. dubbing the toe B. true hoofwall resection

drawback in using the heartbar is the technical difficulty in applying the shoe and the lack of farriers experienced in the technique.

If correctly applied the heartbar will reduce the pain associated with laminitis. The horse may immediately walk and stand better. If abscesses are present, the feet should be soaked once or twice a day in warm water. Povidone iodine can be alternated with epsom salts in the soaking solution. After the feet are dried they should be kept clean and can be packed with antibacterial ointments to soothe exposed tissues and inhibit sepsis. An abscess may need care for 30 to 90 days before clearing up.

It is also important to keep the feet from becoming too dry or too wet. An optimum humidity will aid the fibrotic bonding of hoof to corium. Excessive moisture may lead to the development of proud flesh. Hoof growth is promoted through proper nutrition. Supplementing the feed with 20 grams of d-methionine per thousand pounds of body weight will increase sulfur-bridging in the keratin of the hoof. Under optimum conditions a new hoof can be grown in six to seven months.

The heartbar should be reset approximately every four weeks because growth of the hoof will decrease the pressure of the bar on the frog. The horse may begin to show pain in his feet at this time because of the lack of coffin bone support. Depending on the severity of laminar necrosis, the architecture of the laminae will be lost to varying degrees. Coffin bone rotation may reoccur in some horses if not shod with the heartbar. Other horses may return to riding soundness. The bonding between the hoof lamina and the laminar corium will not regenerate to its pre-laminitis condition.

CONCLUSION

Economics may preclude the use of the heartbar. The lack of a competent farrier may also be a limiting factor. Successful treatment depends on following several basic principles: Treating the underlying cause; transferring weight from the bearing surface of the wall to the bearing surface of the frog; avoiding pressure necrosis of the sole; debriding abscesses; treating for extended periods; and monitoring the patient frequently. Effective means are available for saving animals that would have been destroyed only a few years ago.

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