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Few equine diseases have received as much attention recently from the research community as laminitis. In a quick search in the Pub Med using the words “laminitis” and “horse” from the currently available papers published since 1965, approximately 25% (103/405) have been added within the last five years. Additionally, the Equine Veterinary Journal and Veterinary Immunology Immunopathology, two of the highest impact-factor journals that publish in veterinary medicine, recently have dedicated special issues to equine laminitis.

Due to these recent studies, our understanding regarding the pathophysiology of this devastating disease has greatly improved, bringing the hope and possibility of newer and more effective therapeutic approaches to the treatment and prevention of equine laminitis closer to reality. In this new phase, the somewhat controversial traditional theories and dogma about equine laminitis have been replaced by a sustained and cooperative science that considers laminitis in the realm of the vast, comprehensive and complex inflammatory, vascular, and endocrine knowledge derived from human and other animal research.

What Role Does Inflammation Play in the Developmental and Acute Clinical Stages of Laminitis?

Although the laminar tissue from horses with naturally acquired clinical and experimentally induced laminitis has been studied for several decades, the first evidence of inflammatory mediator production and inflammatory cell accumulation during the developmental phase in laminitis was just recently characterized. This advance was certainly made possible with the development and refinement of techniques in molecular biologic and immunohistochemical techniques that allowed for the detection of markers for inflammatory cells in equine laminar tissues.
This new focus on the inflammatory pathway has led to several laboratories studying different causes of inflammatory injury in laminitis. More than clues about the pathophysiology, this new approach has permitted us to collectively align our findings with the vast knowledge regarding local and systemic inflammatory processes acquired in human and laboratory animals. For example, studies performed at Auburn University, the Ohio State University, and the University of Georgia showed that the increases in tissue inflammatory mediators and inflammatory cells were not limited to the laminae, but also occurred in other tissues and organs including the skin, liver, and lungs. This is clear evidence that changes occurring in the laminae represent a local manifestation of a systemic inflammatory response initially caused by a remote insult. In other words, laminitis concomitant with other tissue inflammation may be a good example of a “remote organ dysfunction” after a systemic inflammatory response. These are two events extensively documented and studied in humans and rodents in which remote organ injury occurs secondary to a systemic inflammatory response to a focus of infections elsewhere in the body, commonly referred to as multiple organ dysfunction syndrome (MODS) after a systemic inflammatory response syndrome (SIRS).

Obviously there are important differences among species. While digital laminae are the primary “target organ” in horses (kidneys appear to be second in line to sustain injury in some equine sepsis cases), lungs followed by the liver and kidneys are clinically the organs most affected in humans and rodents. It is difficult to know the reason that the digital laminae are the “remote target organ” most likely to fail in horses with sepsis, but a critical role of the laminae in suspending the distal phalanx in the hoof wall and the unique microvasculature should be considered as main factors.

Unfortunately, the exact mechanisms involved in remote organ dysfunction after SIRS is not yet clearly understood even in rodents and humans. However, leukocyte accumulation and infiltration in the tissue is a consistent finding in affected tissues. Recruitment of neutrophils and macrophages are identified as key players in organ dysfunction. They can perpetuate local inflammation releasing a second burst of cytokines and chemokines that activates local cells and attract new ones into the affected tissues. More importantly, these extravasated leukocytes release a plethora of substances (cytokines, proteases, elastases, free radicals and hypochlorous acid) potentially destructive to normal host cells in affected tissues. Using numerous models (from laboratory rodents to primates) of organ injury in sepsis and SIRS, studies from several laboratories have shown that the blockage of leukocyte extravasation into tissues during SIRS can protect organs from dysfunction.

**What Role Does Blood Flow Play in the Developmental and Acute Clinical Stages of Laminitis?**
Blood flow alterations in equine laminitis may be one of the most controversial issues in the history of veterinary science. Until the beginning of the 1980’s an initial laminar vasodilatation possibly caused by bacterial conversion of the amino acid histidine to histamine would be the key factor in laminitis development. At that time, most veterinary practitioners treated the affected horses with bleeding, cooling of the distal limb and even adrenaline injections. However, experimental injections of histamine failed to promote laminitis and many experimental studies started to demonstrate evidence of decreased digital blood flow during the prodromal stage of laminitis related to possible venconstriction. At that point, some authors started associating laminitis with Reynaud’s phenomenon, a human condition that causes digital vasoconstriction related to increased blood levels of cortisol and adrenaline. The ischemic theory quickly gained popularity based upon several studies and the use of adrenaline injections and cooling therapy were replaced by alpha adrenergic antagonists (acepromazine) injections and use of vasodilators like nitric oxide donors.

Unfortunately, the majority of vasodilator agents haven’t been proven to be clinically effective, in contrast with the cooling therapy that recently was reintroduced. Laminar cooling (“cryotherapy”) has been demonstrated to be clinically and experimentally beneficial when initiated prior to or at the beginning of the prodromal stage of laminitis (i.e. administered to the horse with a disease which put it at risk of laminitis). The success of this therapy may be an argument against the ischemic theory. However, the effects of the cold therapy have not being studied in laminar microvasculature, and the effects may be only due to a likely decrease in metabolic rate of the cooled laminae, and possibly a decrease in laminar inflammation induced by cooling (discussed below).

Another finding that may corroborate blood flow reduction is platelet (and also neutrophil) aggregation forming microthrombi within the veins of the dermis, which are consistently reported in horses with carbohydrate overload (CHO) induced laminitis. Those microthrombi occur concurrently with increase in post-capillary resistance and are believed to be an importance source of endogenous vasoconstrictor agents such as serotonin and thromboxane.

Because studies involving the serial evaluation of microvascular perfusion of the laminae of horses during the prodromal and development of laminitis, it is unclear what role, if any, decreased blood flow, perfusion and ischemia/hypoxia might play in the pathophysiology of laminitis. Additional studies are needed to determine the exact role of pathophysiology of acute laminitis.

Separation of the Dermal and Epidermal Laminae - Is the Attachment Being Degraded by MMPS or Are the Epithelial Cells Just “Letting Go”?

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The role of matrix metalloproteinases (MMPs) in basal membrane breakdown and separation of dermal and epidermal laminae has been extensively stressed in the last ten years. *In vitro* experiments characterized the action of endogenous MMPs in the laminar interface demonstrating that the separation of dermal and epidermal laminae can be promoted by MMP activator aminophenylmercuric acetate (APMA), and prevented by the addition of the MMP inhibitor batimastat (BB-94). Additionally, *in vivo* studies reported degradation of collagens IV and VII laminin and possibly other structural components of the basal membrane in laminae from horses with CHO-induced laminitis; increased serum concentration of collagen IV was reported in horses with naturally acquired laminitis. Recent studies from different laboratories have shown that MMP-9 and MMP-2 concentrations are increased and some of the natural inhibitors of MMPs (tissue inhibitors of metalloproteinases [TIMPs]) are possibly decreased during the progression of laminitis. While only MMP-9 is consistently induced in the BWE model at Obel grade 1 lameness both MMPs are increased in the CHO model. MMP accumulation can be linked to different factors reported to be present in equine laminitis. Neutrophil accumulation in the laminae is associated with increasing concentration of the zymogen form of MMP-9; this is not surprising because of a large concentration of MMP-9 in neutrophils. Although it has been suggested that there are numerous factors possibly present to activate the zymogen form of MMP-2 and -9 (i.e. cytokines, bacterial products, gut amines, ischemia/reperfusion), many investigations have only found the MMPs present in the inactive/zymogen form.

Adding further question to the role MMPs play in laminar breakdown, a 2009 paper (Loftus et al, Vet Immunol Immunopathol, 2009) reports that both MMPs-2 and -9 are not present in the active form in clinical cases of laminitis due to either a lack of activation/cleavage of the zymogen, or because of interaction of MMPs with inhibitors such as TIMPs. Thus, the presence of the MMPs-2 and -9 does not necessarily mean that they are playing an active role in the disease process. Recent work on other matrix proteases, including MMP-14 and ADAM TS-4 (breaks down matrix proteoglycans), indicates their up-regulation in laminar tissue during the early stages of laminitis; thus, it certainly possibly that other matrix proteases may be involved in the disease process.

**Evidence-Based Treatment of the Inflammatory Component of Laminitis**

The evolution of effective therapies is difficult in a disease such as laminitis where the therapeutic targets—the pathologic mechanisms that lead to laminar injury and failure—have been so controversial. Thus, a large number of laminitis treatments have appeared only to be discontinued owing to either the proposed pathologic mechanism being discounted or the drug being proven not to be efficacious for the desired effect. Interestingly, anti-inflammatory drugs are one of the few classes of drugs to stand the test...
of time and have remained as a cornerstone of laminitis therapy despite the fact that early on there was dogma (actually incorrect) stating that inflammation did not exist in laminitis. During the first AAEP Foundation Equine Laminitis Research Panel and Meeting in 2004 a survey of 60 veterinary practitioners revealed that the most commonly used drugs for the treatment of laminitis is the non-steroidal anti-inflammatory drugs (NSAIDs or AINEs) phenylbutazone (58/60) and flunixin meglumine (40/60). Although there is no direct scientific evidence that laminar injury can be ameliorated by AINEs at this time, there has been a large number of reports the past few years detailing inflammatory events in the laminae and in endotoxic horse that may be addressed by AINEs. These reports include data indicating increased cyclooxygenase-2 expression in the affected laminae, greatly increased concentrations of some pro-inflammatory cytokines in the developmental (prodromal) and acute stages of laminitis. Interestingly although AINEs are used mainly to inhibit cyclooxygenase activity, there is evidence from human-related research that at high doses AINEs can also block other inflammatory pathways, including those involved in cytokine expression (i.e. NFkB signaling). In support of this concept in equids, in the study introducing the clinical community to “low-dose” flunixin meglumine in 1987, the investigators found that although “low-dose treatment” (0.25 mg/kg) had a similar efficacy as the recommended regular “high dose” (1 mg/kg) of flunixin with regard to decreasing prostanoid concentration, the ponies with low-dose flunixin exhibited more severe clinical signs of endotoxemia than the animals given high dose flunixin. This indicates that the higher dose of flunixin is blocking more than prostanoid production. Thus, at least one of the authors (JKB) uses high dose flunixin (1.1 mg/kg TID for 2-3 days) on animals at risk of laminitis (i.e. exhibiting signs of sepsis/endotoxemia) unless there is ongoing renal compromise or GI ulceration.

Controversy exists about the use of heparin in horse laminitis. Whereas heparin was initially used in medicine only as an anticoagulant, it is now realized that this class of drugs also has anti-inflammatory properties (somewhat due to the fact that platelets and factors involved in coagulation can have pro-inflammatory properties). Recently, heparin was reported to have potential anti-inflammatory effects on equine endothelium exposed to the deleterious activity of neutrophil-derived myeloperoxidase (MPO). However, there is confounding data from retrospective clinical studies on the efficacy of heparin as prophylaxis in horses at risk of laminitis. Furthermore, experimental treatment with heparin 24 hours after CHO administration did not ameliorate signs of laminitis or laminar lesions. One problem with previous heparin studies is that unfractionated heparin was used, which induces autoagglutination of equine red blood cells and become lodged in capillaries.
(including laminar capillaries); this event may further compromise affected laminar capillaries in laminitis. Low molecular weight heparin (LMWH) may be a valuable alternative as it does not cause equine RBC autoagglutination and has recently been reported to reduce the incidence and severity of laminitis in postoperative colic cases (these data also obtained from a retrospective study).

A constant rate infusion (CRI) of intravenous lidocaine is frequently used in horses with colic to improve peristalsis and is thought to have anti-inflammatory properties. Due to these purported properties, clinicians have also used lidocaine CRI in other inflammatory diseases including laminitis. However, recent findings using the BWE model of laminitis indicate that not only is a lidocaine CRI not effective in inhibiting inflammatory events in affected laminae, endothelial activation appears to be exacerbated by lidocaine.

**Evidence-Based Treatment of the Vascular Component of Laminitis**

Limited scientific information can be retrieved regarding the use of vasoactive drugs in laminitic horses. In the archives of veterinary medicine, there are reports about the use of vasoconstrictor strategies. More recently, when the goal became to inhibit vasoconstriction and improve blood flow, the main drugs used were vasodilators and rheological agents (increase flow by increasing malleability of blood cells), including acepromazine, nitroglycerin, isoxsuprine and pentoxiphylline. Both isoxsuprine and pentoxiphylline have been somewhat discounted as useful therapeutic agents for laminitis as neither increased digital flow when administered to normal horses; to our knowledge, the same studies have not been performed in affected horses, although there are apparently ongoing studies evaluating pentoxifylline as a potential therapeutic in experimentally-induced CHO laminitis. To our knowledge, acepromazine is the only drug demonstrated to increase digital blood flow in normal horses. The increase appears somewhat transient as intramuscular acepromazine administration significantly increased digital blood flow for approximately 30-40 minutes. Treatment with an endothelin-1 receptor antagonist (ET1RA) and nitroglycerin resulted in significant decreases in vascular resistance in isolated perfused digits of anesthetized horses with CHO-induced laminitis. However, nitroglycerin applied topically (as described to treat laminitis) was found to have no effect on digital blood flow. The administration of an endothelin receptor antagonist has been studied in the conscious horse subjected to experimental laminitis, including the effect on digital blood flow and laminar perfusion, but these results are not yet published.
Distal limb cold therapy has been proved to be a reliable method of preventing acute laminitis. This therapy is based on maintaining the distal limb at or below 5°C for the entire prodromal phase (48 to 72h) after administration of CHO. Initially, the authors that proposed such therapy believed that the cooling should be uninterrupted to promote constant vasoconstriction that would reduce the influx of laminitis “trigger factors” like bacterial-derived compounds or even endogenous inflammatory mediators. However, it was not established if the protective effect of cold therapy is due to decreased digital perfusion, the reduction of metabolic and thus enzymatic activity caused by lower tissue temperatures, or, as reported in human medicine, due to inhibition of inflammatory events.

**Possible Therapeutic Targets for Laminitis Therapy on the Horizon**

This scientific burst characterized by the exponential increase of publications regarding different aspects of equine laminitis is like fertile soil for the development of new strategies in controlling and treating this devastating disease. Currently the Barbaro Memorial Fund from the National Thoroughbred Racing Association is granting three exciting proposals testing new treatments, each one directed toward a specific aspect of the pathophysiology of laminitis.

In 2007, two proposals were funded. "Targeting 5-HT in Equine Laminitis," by Dr. Douglas Allen, et al at the University of Georgia is a study based on the vascular nuances and is testing an available serotonin blocker. Serotonin is a potent vasoconstrictor mediator, which can be release by microthrombi formed by platelet deposition within the veins of laminar dermis during the prodromal stage. It is certainly an important study since previous studies demonstrated that laminitis can be prevented by administration of a competitive inhibitor of platelet aggregation. The other was studied funded by the Barbaro Memorial Fund was the "Treatment of Equine Laminitis with Doxycycline," by Dr. Susan Eades, et al at Louisiana State University and is based on the use of MMPs inhibitors such as doxycycline since there has been substantial evidence regarding the involvement of these enzymes in the basal membrane degradation.

In 2008, the proposal "Effect of Digital Hypothermia on Inflammatory Injury in Laminitis," by Dr. James Belknap, et al at the Ohio State University was funded. In this multicenter project, the effects of cold therapy and the relevance of laminar inflammation in the development of laminitis will hopefully be clarified and further detailed.
Moreover, there are many potential therapeutic targets based on extensive research about SIRS and MODs in rodent model. One point of particular interest should be blocking leukocyte accumulation in the developmental phase. More than a therapeutic option, this strategy could be useful to determine the role of those cells on epidermal detachment and basal membrane destruction. Currently there is project involving the Universidade Federal de Minas Gerais and the Ohio State University in the development and testing of chemokine and adhesion molecule blockers in CHO-induced laminitis.

Corticoids, which have been used in the past for the treatment of horses with laminitis, are currently contraindicated based on reported cases of laminitis after treatment of normal horses with long-acting corticosteroid agents. However, Brazilian studies have shown that hydrocortisone, a short-acting glucocorticoid, is effective in reducing local lesions observed during experimental intestinal ischemia-reperfusion in different models, and also prevents lung leukocyte accumulation in horses subjected to small colon distention. Recent studies conducted at Faculdade de Ciências Agrárias e Veterinárias (UNESP Jaboticabal) using a model of jejunum distention demonstrated horses treated with hydrocortisone had decreased laminar indices of MMP and markers of neutrophil accumulation compared with untreated control horses. Although these findings are still preliminary, a therapeutic and/or preventive effect of hydrocortisone on horses with or predisposed to laminitis should be considered in upcoming studies.

Another interesting concept is using regional intravenous perfusion (RIP) as a method of delivering therapies to the affected digits in order to obtain therapeutic levels in the digits while avoiding systemic effects. This has the possibility of being a great advantage to equine clinicians treating laminitis versus physicians treating visceral organ injury in human septic patients where their only avenue is the systemic use of potent anti-inflammatory drugs to counteract the inflammatory damage to the visceral organs. This has resulted in severe immunosuppression leading to fatality from expansion of the septic focus which originally initiated the systemic inflammatory response. Thus, our advantage of having the “target organ” located more peripherally may allow us to deliver high doses of drugs to the affected laminae without devastating systemic consequences. One of the first concerns that need to be addressed is whether the short time that a tourniquet needs to be applied to the distal limb for RIP will exacerbate laminar injury owing to increased venous and thus capillary hydrostatic pressure and whether this could contribute to or exacerbate laminar
edema and further pathology.

**Addressing the Foot: Clinical Assessment and Therapy for Laminar Instability in the Acute Case of Equine Laminitis**

**Clinical Assessment:** The diagnostic approach should include a combination of digital palpation, lameness examination and radiography or other imaging modality. Palpation of the coronary band may provide initial information regarding distal displacement of the third phalanx (P3) with a furrow commonly occurring immediately proximal to the coronary band in those cases. The coronary band is also assessed for any cracking or hemorrhage. In most cases, an abaxial sesamoid nerve block will be needed on both forelimbs for thorough assessment of the solar surface, for radiographs, and importantly for the assessment of any hindlimb involvement. Lidocaine is the preferred local anesthetic due to its rapid onset and short duration of action, allowing time for digital examination and radiographs, but not an extended time of analgesia for the animal to further traumatize the laminae due to excessive ambulation.

Both lateral and D-P radiographs of affected limbs should be taken. It is important to place a marker at the center of the dorsal aspect of the coronary band (i.e. a BB, Fig. 1), and in the absence of digital radiography, to place some type of radiographic marker down the center of the dorsal hoof wall. To diagnose rotation, intersecting lines can be drawn parallel to the dorsal hoof wall and dorsal P3. For distal displacement, a line parallel to the ground should be drawn through the distal aspect of the BB (line a, Fig. 1). Measurement is then made from the proximal aspect of the P3 extensor process to that line (line b, Fig. 1). This measurement should be less than 10 mm in the digit without distal displacement of P3. The other measurement made is the distance between the dorsal hoof wall and dorsal P3 immediately distal to the extensor process (line c, Fig. 1); this distance is normally 13-18 mm in most breeds of horses, and up to 20 mm in larger breeds. Some radiologists prefer to make a similar measurement between the dorsal hoof wall and dorsal P3 at the
dorsodistal tip of P3 (line d, Fig. 1), and compare that measurement to the more proximal one (line c) to again assess whether a rotational component exists to the displacement. The DP radiograph is assessed for any “gas lines” present in the medial and/or lateral quarter, and to assess for the rare instance in which P3 sinks unilaterally (medial or lateral).

**Therapy for the Foot:** The primary concern in the acute case of laminitis is support of the entire sole caudal to the point of the frog to counteract the downward forces on P3. This support needs to be obtained via a somewhat stiff but resilient substance that will provide support but not cause excessive compression of the subsolar corium. Another concern is to decrease the stress on the dorsal hoof wall, most commonly performed by bringing the breakover point caudal to the dorsal hoof wall. The final concern is decreasing the pull of the deep digital flexor tendon, which is more of a concern in animals undergoing rotation of P3. It is of immense importance that the veterinarian approaching the acute laminitis case is open to trying multiple options if the animal does not respond within 24-48 hours to the initial choice of foot support. If P3 is undergoing distal displacement (“sinking”) with minimal rotation, most horses will respond better to a type of pad that does not or only minimally raises the heel; most “sinkers” worsen if the heel is extensively raised as occurs with the Redden Ultimate (described below). As some horses undergo rotation concomitant with or after distal displacement, it may be best to allow a slight amount of wedging in the digital support. In some respects, each horse is different and thus foot support is tailored according to their response, and thus this is often a “trial and error” approach to finding what works best for an individual patient.

For animals in which the pain is primarily in the forelimbs with no P3 displacement, two inch high density (blue) industrial Styrofoam (for construction, Fig. 2a) can be taped to the bottom of the feet. Over the next 1-2 days, the Styrofoam will compress to approximately ¾” of the total thickness (Fig. 2a, arrow). At this point, a second layer of 2-inch Styrofoam can be taped over the first. If there is pain in the toe on hoof testers, it may be helpful to trim the compressed layer of Styrofoam back to a point immediately caudal to the painful area in the toe before applying the second layer. A third layer may be applied over the second layer in 1-2 days if needed. This technique can also be used on all four limbs in animals in which...
distal displacement of P3 is a concern. This can be effective for small horses, but does not appear to be as effective for larger horses. A better product which is being used more frequently now and can be purchased from multiple building and marine companies is closed cell foam (Fig. 2b, 1.5-2 inch, 4 lb density, similar to the type of matting used in gymnasiums). This appears to not crush as much as Styrofoam, while still providing a resilient substance for support. One author (JKB) has also added a second layer of closed foam, slightly smaller than the first layer, to give a “clog” effect to the pad. Finally, in very heavy horses, it seems best to make pads out of the commercial putties available to the equine clinician (Fig. 2c&d).

A second option for digital support in the acute phase include commercial shoes such as the, the Redden Modified Ultimate (Fig. 3), which consists of two five degree wedges adhered together with screws and a cuff attached for purchase against the hoof wall. The front of this shoe is beveled (arrow) to bring the breakover point more caudal. These shoes come in
multiple sizes. It is critical to realize that these shoes must be used in combination with a commercial resilient putty (usually a 2 part system, Fig. 2c,) supporting the entire sole caudal to the point of the frog to support the sole (Fig. 3b). A pour-in pad will not work with these shoes. If this shoe (or any shoe) is used without the putty supporting the caudal aspect of the sole, it worsens the stress on the laminae by making the digit support the entire weight of the horse on the wall and laminae, increasing the chances of P3 displacement. Many horses respond favorably to this system; however, some animals become more painful. If this occurs, one reason may be that the heel elevation is too high for that animal. It may be helpful to remove the bottom wedge, thus leaving the horse with a five degree heel elevation (JKB does this more commonly in animals where there is a concern about distal displacement of P3 than rotation). In some animals with severe rotation, the attachment of an additional wedge to the shoe may be indicated in the case of P3 rotation which does not respond satisfactorily to application of the shoe (F. Nickels, Michigan State University, personal communication). With this system, the commercial putty is mixed, applied in ample quantity to the sole caudal to the point of the frog (Fig. 3b) to provide a continuous layer between the sole and shoe, and the shoe rapidly applied followed by full weight bearing by the horse. Once the putty has hardened (usually approx. 5 minutes), it is important to remove the shoe and cut out any putty that pushed ahead of the point of the frog in order to ensure that there is no pressure on the toe of the sole. A cotton leg wrap is usually applied that covers the coronary band (Fig. 3c), and the shoes are typically taped to the hoof and bandage rather than gluing the shoes. This gives the option of removing the shoe periodically to examine or treat the foot, and to change shoeing rapidly without damaging the wall if the animal does not respond favorably to this shoeing technique. Another commercial option, especially for the “sinker”, is the Soft-Ride boot (black arrow, Fig. 4a). These boots provide good solar and frog support, and can be easily taken off and placed back on by owners. The only concern is that there is minimal beveling of the toe to decrease pressure on the dorsal hoof wall, so these are less likely to be used if there is active rotation concomitant with the sinking. Otherwise, these are good options which allows for the horse to be rather easily managed at home by the owners until the time that shoeing is able to be done. Another option is to apply Steward Clogs temporarily by placing the screws in the clog, and using either tape (temporary) or cast material around the clog and foot to attach the clog to the foot (A Parks, Univ. of Georgia, personal communication). Putty material is usually placed between the sole and the clog. Dr. Andrew Parks (University of Georgia) started this technique, which is now being used by many others.

A final option in the animal in which the pain is unrelenting or the P3 displacement continues despite the aforementioned techniques is the application of open-sole casts (Fig. 5).
4). This technique is best used unilaterally in a case where one foot is rapidly deteriorating. I have placed casts on both forelimbs (Fig. 4c), but owners should be warned of the increased incidence of cast sores with bilateral casts. Although some individuals use foot casts, a half limb cast is preferred by one author (JKB) in order to allow as much of the horse’s weight as possible to be borne at the flare of the proximal metacarpus. JKB has recently made the cast by continuing a thick rim of cast material approximately 1.5-2 cm. distal to the hoof, but leaving the sole exposed. The rim is strengthened with the application of acrylic to the exterior of the cast material (Fig. 4b, open arrow). The commercial putty is then placed in the caudal 2/3 of the sole (arrow, Fig. 4b) even with the bottom of the rim of the cast. Therefore, this provides access to the bottom of the foot so that resilient putty can be applied to the sole, while still having the support advantages of a half limb cast. It is believed that this technique is most indicated in horses with distal displacement of P3 where none of the aforementioned techniques offer a great deal of benefit.

Other supportive methods include applying deep bedding to encourage the horse to lie down as much as possible, with a thick layer of peat moss or tanbark as a base for the bedding (tanbark is beneficial as it absorbs a great deal of moisture which is critical.