

Tutorial Article

Treatment of acute laminitis

A. H. PARKS

Department of Large Animal Medicine, College of Veterinary Medicine, University of Georgia, Athens, Georgia 30602, USA.

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Introduction

The acute phase of laminitis has been defined as the first 72 h following the onset of clinical signs or until displacement of the distal phalanx occurs, whichever is the sooner (Hood 1999). As such, acute laminitis is the **transition between** the developmental phase and either the subacute or chronic phase of the disease. The **subacute phase** follows the **acute phase** when no displacement of the distal phalanx has occurred within the first 72 h, and the **chronic phase** follows the acute if the distal phalanx has displaced. Of course, a horse may initially enter the subacute phase, only to become chronic later on.

Displacement of the distal phalanx occurs when the stresses imposed on the lamellae exceed their functional capacity to maintain the distal phalanx in its normal relationship to the hoof capsule. The functional capacity of the lamellae in a healthy horse greatly exceeds the stresses they normally encounter. However, in the horse with laminitis, the strength of the lamellae may drastically be reduced. The overall stresses on the lamellae and the distribution of the stresses within the lamellae may then become the limiting factor that determines the displacement of the distal phalanx. Stability of the distal phalanx in the laminitic horse becomes a delicate balance.

The most commonly seen patterns of displacement of the distal phalanx are **capsular rotation** and **distal displacement**. In capsular rotation, the dorsal parietal surface of the distal phalanx diverges from the dorsal hoof capsule. In distal displacement, the distal phalanx descends distally within the hoof capsule but maintains its orientation to the distal interphalangeal joint and the ground. In most instances, the displacement is a combination of these 2 patterns, but asymmetrical mediolateral displacement may also occur.

The **sources of pain** in laminitic horses have not been precisely defined, but intuitively would appear to involve inflammation or ischaemia of the lamellae. Secondly, mechanical stresses on weakened lamellae and inflammation or ischaemia of the solar dermis as it is compressed by the descending distal phalanx are likely to cause pain.

The **objectives of treatment** in horses with acute laminitis are to contain the pathophysiological processes that cause the initial weakening of the lamellae, and reduce or redistribute stresses that cause the damaged lamellae to separate. As such, **treatment of acute laminitis is divided into medical therapy and supportive care.**

Medical therapy

Medical therapy is based on understanding the pathophysiology of the disease. Laminitis has been investigated by studying both the pathophysiological changes and the effects of different pharmacological interventions. The pathophysiology of laminitis has been investigated by observation of morphology and measurement of physiological parameters. The study of laminitis by pharmacological intervention in experimentally induced disease has focused almost entirely on treatment started prior to or at the same time as the initiating insult. This research has focused on the developmental and acute stages of the disease but, from the standpoint of pathophysiology, the division between developmental and acute laminitis is based arbitrarily on the point at which a clinician can identify the disease because of the appearance of recognisable signs. A detailed description of the pathophysiology of laminitis is beyond the scope of this discussion.

The theories regarding the pathogenesis of laminitis can be summarised as:

- a vascular derangement;
- an inflammatory response;
- a coagulopathy.

The evidence for each of these mechanisms is sufficient to suggest that they are all important, either as part of a common pathway or as an entrance to a common pathway.

There are many diseases associated with the onset of laminitis. If all horses with each of these diseases are considered to be at risk, the pool of horses at risk for developing laminitis is very large. The overall likelihood of a horse from this pool developing laminitis is low. However, the precise incidence varies with the associated condition and the

severity of that condition, which for most of these diseases is unknown. Additionally, the predictability of whether, and if so when, a horse will develop laminitis is poor. Therefore, **it is frequently not possible to target aggressive pharmacological therapy at horses that are going to develop laminitis**; and it is unknown for how long to continue such therapy among those that are subjectively considered to be at very high risk when such therapy is warranted. Consequently, while there is little room for doubt that aggressive prophylactic therapy is optimal for those horses that will develop laminitis, most medical therapy is started in the acute phase of laminitis. To date, clinicians have considered acute laminitis to be a continuation of developmental laminitis and, as such, they will be discussed together. Therefore, this discussion briefly considers each of the theories of pathogenesis, and the related evidence for medial therapy.

Blood flow

Measurements of total blood flow to the foot and lamellar blood flow during the developmental and acute phases of laminitis have varied widely with the model used to induce laminitis and the methods used to measure blood flow (Galey *et al.* 1990; Trout *et al.* 1990; Pollitt and Davies 1998b; Adair *et al.* 2000; Hood *et al.* 2001a). However, the preponderance of evidence supports vasoconstriction, the opening of arteriovenous anastomoses (Hood *et al.* 1978) and, in the isolated digit, capillary collapse secondary to increased interstitial pressure (Allen *et al.* 1990).

Therefore, therapy has been directed at enhancing the lamellar blood supply with vasodilators and rheologic agents. Of the **available vasodilators, α -adrenergic antagonists, isoxsuprine and nitrovasodilators** have received most attention. The effects of the α -adrenergic antagonist phenothiazine derivative acepromazine has been both investigated experimentally and observed clinically. Phenoxybenzamine has been used to a limited extent clinically. Experimentally, **acepromazine** increases digital and lamellar blood flow in normal horses (Adair *et al.* 1994, 1997; Ingle-Fehr and Baxter 1999), but it has not been tested in horses with induced laminitis. However, due to its availability, widely accepted safety and subjective clinical impressions of efficacy, it is the most widely used vasodilator in the treatment of clinical laminitis.

Isoxsuprine is an effective dilator of isolated palmar digital vessels *in vitro* (Baxter *et al.* 1989), but conflicting data regarding its ability to improve peripheral blood flow (Rose *et al.* 1983; Harkins *et al.* 1996; Adair *et al.* 1997; Ingle-Fehr and Baxter 1999) and the inability to detect isoxsuprine in the plasma after oral dosing raise questions about its efficacy (Matthews *et al.* 1986; Harkins *et al.* 1998). However, some clinicians have subjectively observed improvements in horses with clinical laminitis. Alternative explanations for its apparent efficacy have been suggested, including potential rheologic properties. More data are needed to make a scientifically informed decision about the role of isoxsuprine in the treatment of acute laminitis.

The use of nitrovasodilators, such as **nitroglycerin**, is similarly controversial. Different studies have reported an increase or no change in the lamellar blood flow (Hinckley *et al.* 1996a; Hoff *et al.* 2002). One clinical trial has reported improvement in lameness in horses with naturally occurring acute laminitis following treatment with nitroglycerin (Hinckley *et al.* 1996b). However, it did not improve lamellar blood flow in an experimental model (Adair *et al.* 2000), and this author has not observed any clinical benefit from its use.

Drugs with rheologic properties that increase the deformability of red blood cells potentially increase lamellar capillary blood flow. **Pentoxifylline** is the principal drug that has been investigated, although isoxuprine is reported to have similar properties. However, experimentally, neither drug has been shown to increase lamellar blood flow (Adair *et al.* 1997; Ingle-Fehr and Baxter 1999).

The presence of microthrombi in the feet of horses with acute laminitis (Weiss *et al.* 1994), and the prophylactic protection identified in horses treated with heparin before experimental laminitis is induced (Hood *et al.* 1982), suggest that thrombosis and a hypercoagulable state are present in developmental and/or acute laminitis (Weiss *et al.* 1997). However, the microscopic evidence of thrombi in the digital microvasculature is an inconsistent finding, and the preponderance of evidence from studies that have examined coagulation during developmental and acute laminitis indicates no changes in coagulation or fibrinolysis (Prasse *et al.* 1990). Yet decreased platelet survival, hyperaggregation of platelets and an increase in the number of platelet/neutrophil aggregates in the plasma in the developmental phase of laminitis suggest that platelets are involved in alimentary laminitis (Weiss and Evanson 1997); additionally, a novel platelet aggregation inhibitor reduces these changes and the incidence of acute laminitis (Weiss *et al.* 1998).

Aspirin irreversibly inhibits platelet cyclo-oxygenase, decreases platelet aggregation in response to arachidonic acid or collagen stimulation, decreases thromboxane production, and increases bleeding time (Cambridge *et al.* 1991). However, it does not affect platelet aggregation induced by various other stimuli including endotoxin (Heath *et al.* 1994; Jarvis and Evans 1994). Therefore, without determining how platelet agglutination associated with developmental laminitis is stimulated, the benefits of aspirin therapy are unknown. Its efficacy has not been tested in an experimental model of laminitis.

Heparin has been administered to horses both prophylactically and to treat acute laminitis. In the previously cited experiment, heparin administered prophylactically reduced the severity of laminitis, but two published clinical reports provide conflicting evidence regarding its benefits (Belknap and Moore 1989; Cohen *et al.* 1994). Additionally, concern has been voiced that the red cell agglutination observed after treatment with heparin (Moore *et al.* 1987) may actually impede microvascular blood flow.

Inflammation

Laminitis has long been considered an inflammatory disease based on the clinical signs of heat and pain that are cardinal signs of inflammation. Histological features compatible with inflammation include epidermal cell oedema, epidermal cell necrosis, leucocyte infiltration, and separation and loss of the basement membrane (Obel 1948; Pollitt 1996; Pollitt and Daradka 1998a). Recently, the expression of inflammatory mediators in the lamellae has been demonstrated in developmental laminitis (Fontaine *et al.* 2001).

The exact causal relationships between the events in developmental laminitis are undetermined. For example, **basement membrane separation** can be demonstrated in the lamellae (Pollitt 1996), metalloproteinases cause separation of the lamellae *in vitro* (Pollitt *et al.* 1998c) and **activated metalloproteinases** can be demonstrated in the lamellae of acutely laminitic horses (Mungall and Pollitt 1999). This evidence points towards activated metalloproteinases as a likely cause of basement membrane separation in the carbohydrate overload model of laminitis, but it is still uncertain. Additionally, if the basement membrane separation is caused by the metalloproteinases, the metalloproteinase activation may be stimulated by one specific factor or a variety of such factors. For example, supernatant from a culture of *Streptococcus bovis* isolated from the equine caecum activates metalloproteinases (Mungall *et al.* 2001); absorption of such a factor *in vivo* might, therefore, precipitate laminitis. However, laminitis occurs naturally in horses that do not appear to have a gastrointestinal dysfunction, for example those with pleuritis or renal failure, which suggests that other mechanisms might also be involved.

Endotoxaemia

Endotoxaemia is implicated in the pathogenesis of laminitis because horses that clinically appear endotoxic are more susceptible to development of laminitis. Increased endotoxin has been found in the intestinal tract and plasma of horses that

developed laminitis after carbohydrate overload (Moore *et al.* 1979; Sprouse *et al.* 1987). However, not all or even most endotoxic horses develop laminitis and increased plasma endotoxin has not been identified in other horses that have developed laminitis (Weiss *et al.* 1994; Eaton *et al.* 1995). Rapid removal of endotoxin from the portal circulation by the liver following absorption of endotoxin from the gastrointestinal tract may, in part, explain the latter. Additionally, infusion of endotoxin into the peripheral vasculature has never been shown to induce laminitis, although clinical signs indicative of laminitis have been reported after infusion of endotoxin into the portal circulation (Duncan *et al.* 1985).

Nonsteroidal anti-inflammatory drugs are used routinely in the treatment of acute laminitis based on the clinical, histological and histochemical evidence of an inflammatory response and because of their analgesic properties. Currently, no metalloproteinase inhibitors have been employed in horses with either experimental or clinical laminitis. **DMSO**, an oxygen free radical scavenger, is similarly widely used based on clinical impressions, but there is little scientific evidence to support its use. *In vitro*, endotoxin antiserum is effective in binding lipopolysaccharide (Wells *et al.* 1987) and is bactericidal (Gaffin and Wells 1987), but it was ineffective in improving clinical signs or the expression of inflammatory mediators in 2 experimental models of endotoxaemia (Morris *et al.* 1986; Durando *et al.* 1994).

In a single clinical trial, endotoxin antiserum increased survival of horses with abdominal disease associated with endotoxaemia (Spier *et al.* 1989), and it is this author's impression that it improves clinical signs of horses that appear endotoxic, but its effect on the development of laminitis is unknown. It is probably justifiable to administer endotoxin endoserum prophylactically to endotoxic horses that are considered to be at high risk of imminently developing laminitis, because it is expensive and only likely to protect for a limited amount of time. Polymixin B is more commonly used to treat endotoxaemia because it binds endotoxin. Polymixin B inhibits tumour necrosis factor induction *ex vivo* (Parviainen *et al.* 2001); and both improved clinical parameters and reduced

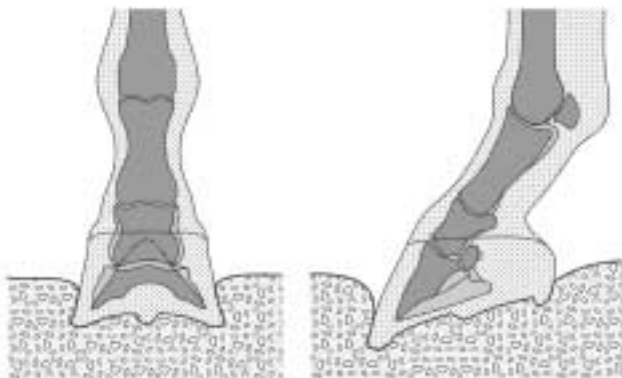


Fig 1: A schematic diagram of a horse's distal limb standing on sand, showing broad distribution of weight across the ground surface of the foot as the sand conforms to the surface of the sole. Additionally, the horse may adjust the angle of its feet to the ground for increased comfort.



Fig 2: A schematic diagram showing the optimal area of the ground surface of the foot that can be recruited to bear weight (hatched area) without applying pressure to the dorsal margin of the distal phalanx or the adjacent wall.

cytokine production in experimentally induced endotoxaemia (Durando *et al.* 1994; MacKay *et al.* 1999), but produced only marginal delay in onset of clinical signs of laminitis in a carbohydrate overload model (Raisbeck *et al.* 1989). Pentoxifylline, in addition to its rheologic properties, inhibits cytokine synthesis *in vitro* (Barton and Moore 1994; Barton *et al.* 1997a; Baskett *et al.* 1997) but is of limited benefit after experimental administration of endotoxin (Barton *et al.* 1997b). It appears that both polymixin B and pentoxifylline are more effectively used in conjunction with nonsteroidal anti-inflammatory drugs (Baskett *et al.* 1997; MacKay *et al.* 1999).

Supportive care

The principal goals of supportive therapy are to stabilise the distal phalanx and to control pain. This necessitates some understanding of normal foot function. The principal stresses imposed upon the foot are caused by weightbearing and movement. The traditional view of weightbearing holds that the weight of the horse is transmitted from the axial skeleton through the distal phalanx, the lamellae and the wall to the ground. This pattern of weightbearing is applicable when a horse is standing on a flat, firm surface. However, **when the**

horse is standing on a yielding surface, the sole and frog become significant, if not the principal, weightbearing structures instead of the wall (Hood *et al.* 2001b). The centre of the weightbearing, i.e. the position of the ground reaction force, is in approximately the same location, near the dorsal third of the frog, in either circumstance. Therefore, the stresses applied are approximately symmetrical about the centre of the ground surface of the foot, **but in the foot on a firm surface they are concentrated on the wall whereas they are more broadly distributed across the ground surface of the foot on a yielding surface.**

As the animal moves about, however, the location and magnitude of the ground reaction force change during the stride. When a horse walks in a straight line, weight is initially borne at the heels, is centred in the middle of the foot during most of weightbearing, and shifts to the toe as the horse breaks over. The magnitude of the ground reaction force is greater during the stance phase of the stride and less at initial weightbearing and breakover. However, because the force is applied over a small surface area at the beginning and end of the stride, the stresses are concentrated. The stresses within the dorsal lamellae change with the moment about the distal interphalangeal joint, which is determined by the force on the



Fig 3: A frog pad made from rolled gauze.



Fig 5: Silicone putty filling the concavity of the sole.



Fig 4: Commercial frog pad (Lily Pad)¹.



Fig 6: Styrofoam sole support.

dorsal lamellae and hoof wall, and the opposing tension in the deep digital flexor tendon. Just as the stresses are concentrated at the heel and toe as an animal moves in a straight line, so they are concentrated at the quarters as a horse turns around.

Because pain is associated with lamellar injury, these goals are intrinsically related so that most treatments that improve one will improve the other. The specific objectives are:

- decrease overall stresses on the foot;
- reduce the load on the most severely affected part of the wall;
- transfer load to the less affected part of the wall;
- recruit additional ground surface of foot to bear weight;
- decrease the moment about the distal interphalangeal joint;
- avoid pressure on the sole distal to the dorsal weightbearing surface of the distal phalanx;
- avoid harming the digital vasculature.

The greatest overall stresses on the foot are associated with weightbearing. The weight of the animal cannot be changed abruptly, but the animal can be encouraged to lie down. Additionally, a horse may be partially suspended in a sling or partially floated in a pool but, for practical reasons, these options are limited. To control the focally increased stresses associated with ambulation, **it is imperative that an acutely laminitic horse is strictly confined to a stable.**

Conventional shoes concentrate the stress around the perimeter of the foot, i.e. on the wall. Therefore, the **shoes should be removed**. Some clinicians advocate leaving the shoes on the horse, because shoe removal risks further injury to the lamellae. Rasping the clenches and removing the nails with crease nail pullers can minimise this risk. If necessary, this author performs a nerve block of the feet to remove shoes. **Optimally**, the shoes should be removed in the developmental phase, i.e. routinely in horses considered at risk of laminitis, or early in the acute phase of the disease. If the shoes are left on, the sole may prolapse between the branches of the shoe; **the likelihood of this occurring may be reduced** by packing the concavity of the sole and the space

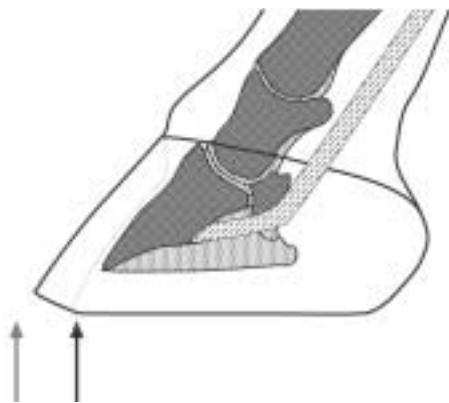


Fig 7: A schematic diagram illustrating how the toe may be bevelled. The grey arrow identifies the original breakover point, and the black arrow the new breakover point.

between the branches of the shoe. **However, leaving the shoes on limits other hoof care that can be performed.**

Reducing the load on the most severely affected part of the wall, loading less affected wall and recruiting additional ground surface of the foot to bear weight are achieved by optimising the footing and use of supportive appliances. **Bedding materials that conform to the ground surface of the foot** partially redistribute the load from the walls to the sole. In this regard, **sand is significantly superior to shavings (Fig 1)**; peat moss is probably similar to sand but this author has no experience with it. Additionally, a horse standing on a deep yielding surface can position its feet for optimal comfort. Although the immediate benefits of sand stables are unquestionable for the acutely laminitic horse, in the longer term they are harder to maintain than shavings stables, the **sand abrades the surface of the hoof**, and they do not prevent abscess formation.

All of the ground surface of the foot may be recruited to bear weight, but **caution must be used** when employing the dorsal area of the sole distal to the dorsal margin of the distal phalanx and the adjacent wall (**Fig 2**). The simplest devices used are frog pads, either made from rolled-up gauze (**Fig 3**) or a commercial equivalent¹ (**Fig 4**). This author's experience with frog pads is highly variable; some horses improve, others don't change, and yet others become more lame. No doubt there are subtleties in adjusting the pressure applied to the frog. Regardless, this author uses them infrequently.

Weight can be redistributed towards part or all of the ground surface of the foot by filling the concavity of the foot with a packing material. A wide variety of products have been tried with success, ranging from rigid materials, such as methylmethacrylate or casting tape, to more yielding compounds, such as silicone putty (**Fig 5**) or high density Styrofoam (**Fig 6**). **Legitimate concerns exist** regarding the impact of rigid sole casts applying pressure to the sole distal to the margin of the distal phalanx and subsequently impeding the vascular supply to the sole and distal lamellae. Therefore, despite instances where they have been used, successfully, **it is advisable to avoid the use of rigid materials to pack the sole** until more is known about the



Fig 8: A commercial cuff/wedge pad combination (modified Redden Ultimate)².

physiological relationship between weightbearing and the blood supply to the lamellar and solar dermis.

The moments about the distal interphalangeal joint may be reduced by moving the breakover point palmarly or elevating the heels; frequently, one is done in conjunction with the other. The simplest way to move the breakover point palmarly is to bevel the dorsal aspect of the ground surface of the foot until it is at approximately 25–30° to the ground. How far the breakover may be moved palmarly depends on the thickness of the sole, but it can almost always be moved back to the white line and frequently several millimetres palmar to that (**Fig 7**). Additionally, bevelling the toe in this manner reduces weightbearing by the dorsal hoof wall at rest. Any shoe or pad taped or glued to the foot that is set back from the toe also moves the breakover palmarly.

The heels may be elevated with wedge pads. It is most convenient to use a commercially available combination of wedge pads and cuff² that also have a bevelled toe to move the breakover palmarly (**Fig 8**), but regular wedge pads can be customised and taped to the foot. The wedge pads are usually used in conjunction with a packing material to fill the gap between the sole and the pads. As an alternative to wedge pads, **Styrofoam** can be built up into a wedge by retaining the palmar half of an already compressed Styrofoam pad and adding a new full Styrofoam pad to the foot. Concern has been voiced that heel elevation increases the shear stress in the dorsal part of the hoof wall and cause the distal phalanx to displace (Chapman and Platt 1984). However, the effects of heel elevation on the stresses in the dorsal lamellae have not been systematically investigated. Although it is likely that dorsal shearing stresses do increase, the reduction in lameness indicates that the lamellae are less painful, suggesting that they are under less mechanical stress which, in turn, infers that the distal phalanx is more stable. This is presumably due to stress redistribution as the moments about the distal interphalangeal joint are decreased.

There are several other more minor considerations in the supportive care of acute laminitis. **Support bandages** may be necessary to control limb oedema. The merits of hot and cold therapy on the limb have been debated, but this author considers soaking the foot to be potentially harmful to the hoof capsule and so uses neither. **Walking the acutely laminitic horse is contraindicated, as is the use of perineural anaesthesia**, with the occasional exception such as when removing the shoes. **Transporting the horse is undesirable** unless absolutely necessary to take it to a treatment facility. Surgery and hoof wall treatment, except bevelling of the toe, are seldom indicated in horses with acute laminitis.

Applying treatment principles

Clinical decision-making is necessarily a balance between risk of a disease process occurring or deteriorating against the expense and effort incurred, and the potential side effects of the treatment itself. In this regard, preventing and treating laminitis is particularly difficult, because its occurrence, progression and time course are difficult to predict.

Additionally, no medication has been proven to be of unquestionable benefit in the treatment of acute laminitis, yet no clinician would contemplate discounting medical therapy. Therefore, prevention and treatment strategies are balanced against clinical signs, largely based on experience; and **must be adaptable should the circumstances change.**

Treatment of primary disorders such as colitis, pleuropneumonia, colic and metritis frequently include measures such as nonsteroidal anti-inflammatory drugs, DMSO, polymixin B and endotoxin antiserum, that would also be used prophylactically to limit the development of laminitis. Horses with many of these primary disorders are already on restricted diets and, although there is no rationale for starving horses at risk of developing laminitis, restricting or removing grain from the diet is probably warranted. If not already in use, administration of phenylbutazone (2.2 mg/kg bwt, i.v. or *per os*, b.i.d.) and/or flunixin meglumine (0.25 mg/kg bwt, i.v., t.i.d. to 1 mg/kg bwt, i.v., b.i.d.), in conjunction with removing the shoes and adding frog or sole support, may be sufficient if the risk of developing laminitis is perceived to be low. As the perceived risk increases, DMSO (0.1–0.2 g/kg bwt, i.v., b.i.d.–q.i.d.) and acepromazine (0.01–0.02 mg/kg bwt, i.m or subcut. b.i.d.–q.i.d.) may be added to the regimen, followed by isoxsuprine (0.6–1.2 mg/kg bwt, *per os*, b.i.d.) and pentoxifylline (4.4 mg/kg bwt, *per os*, t.i.d.). **Acepromazine should be used with caution in stallions.**

The initiating cause should be removed if it can be identified; the treatment of acute laminitis may be as simple as removal from the pasture, rest and phenylbutazone for the pony that develops mild laminitis after overindulging on spring grass. However, for the more severely affected horse, the most commonly used drugs are DMSO and acepromazine, in addition to phenylbutazone (up to 4.4 mg/kg bwt, i.v. or *per os*, b.i.d.), which is frequently used at a higher dose therapeutically than it is prophylactically. Of the other drugs available, pentoxifylline and topical nitroglycerine (15 mg applied over each palmar digital artery b.i.d.) are the most likely to be added to the treatment regimen. Heparin (25–100 iu/kg bwt, subcut. t.i.d.) and aspirin (5–10 mg/kg bwt, *per os*, s.i.d.–every other day) are less frequently employed and are not currently used by this author.

Initial supportive therapy should include absolute box rest, removal of shoes and support of the frog and/or sole, and observing the response. This author prefers to support the sole first with silicone putty. This is also frequently the most appropriate time to bevel the toe. If the lameness does not improve or becomes worse, the silicone putty is replaced with 5 cm thick Styrofoam. Similarly, if the response is inadequate, the author uses a commercial cuff/wedge pad combination in conjunction with silicone putty. There are no hard and fast rules about the time lines to be followed. **If the disease is mild**, the response may be monitored over days and treatment adjusted accordingly. However, **if the disease is severe**, the response to a treatment may be observed for only a few hours before changing it. Alternatively, a step may be bypassed completely, e.g. attaching the cuff/wedge pad as a first line of treatment, or moving the horse immediately to a sand stable.

After 72 h, laminitis is either considered subacute or it has become chronic. The therapy initiated in the acute phase of the disease is continued into the next phase. If the laminitis becomes subacute, the initial therapy is frequently continued for several days after the onset of signs. **The time course for discontinuing therapy depends on the severity and progression of the disease.** However, some drugs, such as DMSO, probably have limited benefit after the first few days and can be discontinued relatively rapidly, whereas phenylbutazone treatment may be prolonged because of its analgesic effects as well as its anti-inflammatory properties. If the heels have been elevated, their height should be reduced gradually, and sole support removed some time later. **Above all, one change should be made at a time and the effect observed for long enough before making another to identify the effects of each change and be able to reinstate a treatment if necessary.** The more severe the disease, the slower the convalescence. Severely affected horses should be given time if they do not become chronic, because many will improve in 2–3 weeks, although total recovery frequently takes months. Should the laminitis become chronic, treatment assumes a different course, which is beyond the scope of this discussion.

Manufacturers' addresses

¹Therapeutic Equine Products Inc., Shelbyville, Kentucky, USA.

²Advance Equine, Versailles, Kentucky, USA.

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