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LAMINITIS IN THE HORSE: A REVIEW

M.M. Sloet van Oldruitenborgh-Oosterbaan

SUMMARY
Laminitis has been a recognized disease since early Greek and Roman times, but it is still bothering both practitioners and scientists. In the last decade a lot of new fundamental research has been done to elucidate the pathogenesis of laminitis. New insights into the pathogenesis, the predisposing factors (including nutritional overload, endotoxaemia, shock, management, etcetera), clinical and radiological signs, differential diagnosis, therapy, and prognosis of the disease are described. The data, however, are not always in agreement with each other, giving further proof of the complexity of the syndrome.

Keywords: equine, horse, laminitis, radiological evaluation, founder, hoof, pedal bone.

INTRODUCTION
Acute laminitis is a common, painful, and potentially disastrous sequela of many equine diseases such as grain overload, colitis, endotoxaemia, septic metritis, etcetera (53). A horse has laminitis when the lamellae of the inner hoofwall, which normally suspend the pedal bone from the inner surface of the hoof capsule, fail (44). Without the pedal bone properly attached to the inside of the hoof, the weight of the horse and the forces of locomotion drive the bone down into the hoof capsule, shearing and damaging arteries and veins, crushing the corium of the sole and coronet, and causing unremitting pain and a characteristic lameness (44). Although laminitis has been studied periodically for nearly 50 years, its pathogenesis has still not been elucidated completely. In the last decade, however, pioneering research has been published by several research groups (4,23,26,29,36,40,41,42). For this reason a review of these new perceptions from a practical point of view seemed appropriate.

PATHOGENESIS
The blood supply of the foot is based on a delicate microvascular system deep in the corium (38,43). Disturbances in this system have a direct influence on the dermal laminae that interdigitate with the horny laminae on the deep surface of the hoof wall. These interdigitating laminae normally provide a firm bond between the corium and the hoof wall. The corium is on the other side also strongly attached to the pedal bone, thus maintaining the normal anatomical alignment of the foot (43). Disturbances of the blood supply to the laminar region, for example by vasoconstriction, microthrombosis, perivascular oedema, or arteriovenous shunting, cause degeneration of the laminae, resulting in separation of the pedal bone from the hoof wall (6,25,32,44). If this occurs, the pedal bone will rotate and/or sink (17,21,44). The factors that provoke disturbances of the blood supply and their mechanisms of action are not yet fully understood, but numerous predisposing factors have been described.

These predisposing factors can be divided into five groups (6):
- carbohydrate overload
  - excess grain intake
  - lush pasture
  - feed change to high-energy legumes
- endotoxaemia, sepsis, shock
  - colitis
  - proximal enteritis
  - intestinal strangulation/obstruction
  - retained placenta, metritis, abortion
  - septicemia or endotoxaemia from any cause, such as pleuritis
- excessive unilateral weight-bearing (support laminitis)
- severe lameness
- rehabilitation of fracture repair
- management
  - ingestion of cold water by overheated horse
  - unconditioned horse worked on hard surface
  - overweight horses or ponies
  - trimming hooves too short
  - black walnut wood shavings used for bedding
- miscellaneous
  - treatment with corticosteroids
  - Cushing disease
  - hypothyroidism
  - diet of plants containing oestrogens
  - continuous oestrous in mares
  - allergic-type reactions to certain medications

In experiments to study the pathogenesis and the therapeutic effect of medications and treatments, laminitis has been induced by an overload of carbohydrate or an aqueous extract of black walnut (5,19,20,31). Some authors, however, query whether experimentally induced laminitis is completely comparable to clinical laminitis (25,33). Clinical cases are often the result of a combination of factors (50).

In the last few decades, the shunting of blood away from the nutrient capillaries of the hoof lamellae via inappropriately dilated arteriovenous anastomoses (AVAs) has been proposed as a pathophysiological mechanism for the development of equine laminitis (24,38,48). The digital arteries have been shown to be much more sensitive to the vasoconstrictor 5-hydroxytryptamine (5-HT) than the facial, tail, and coronary band arteries (4). This suggests a role for 5-HT in the developmental phase of acute laminitis. Endothelin-1 (ET-1) is another endogenous vasoconstrictor that may play a role in the pathophysiology of laminitis. In the future, antagonists of
these mediators may eventually play a role in the prevention of laminitis.

Pollitt (39) showed that, in acute laminitis, the tissue suspending the pedal bone from the inner hoof wall fails specifically at the junction between the connective tissue of the dermis (corium) and the basal cell layer of the epidermal lamellae. This appears to be a weak link in an otherwise robust structure. There is widespread epidermal detachment from and lysis of the lamellar basement membrane, which leads to failure of the lamellar anatomy and ultimately to failure of the suspensory attachment between hoof and pedal bone. In addition Pollitt and coworkers demonstrated the role of metalloproteinases (MMP) (40). These enzymes are capable of lysing key components of normal lamellar hoof basement membrane. It seems likely that uncontrolled MMP activity is responsible for the loss and disorganization of the lamellar basement membrane, resulting in widespread separation of the secondary epidermal lamellae from their basement membrane (42). There is no agreement whether there is vasoconstriction (26) or vasodilation (41) during the prodromal phase of laminitis. This has serious consequences for the choice of preventive measures in horses prone to developing laminitis.

CLINICAL SIGNS

Equine laminitis is a systemic disease which is divisible into three phases: developmental, acute, and chronic (25,44). The developmental phase, however, is only observable in experimental studies. The clinical signs of laminitis are similar irrespective of the cause of the disease, and can be divided into acute and chronic.

Acute laminitis

Symptoms of acute laminitis are (6,44,51,53,58):

* severe lameness, reluctance to move, or even recumbency
  Most commonly, both fore feet are involved, sometimes all four feet or a single digit.
* typical stance when both fore limbs are involved (Figure 1)
  The horse will role back on the heels of the fore feet and take more weight onto the hind limbs by shifting the hind limbs forward under its body. This stance is more obvious when the horse has to walk or to turn in a circle.
* shifting weight (paddling)
  Affected horses may shift their weight from one foot to another.
* increased digital pulses and heat over the hoof wall
  Exaggerated digital pulse ('bounding') and warmth in the hoof area are nearly always present.
* pain in the hoof
  Percussion of the hoof or pressure with hoof testers over the sole at the toe reveals sensitivity.
* cavitation or depression along the coronary band
  This is often the first clinical sign of sinking and with time, blood or serum may ooze from the coronary band. In this phase the laminitis starts to become chronic.
* systemic changes
  The systemic manifestations of laminitis include alterations in the cardiovascular, endocrine, and renal systems, acid-base imbalance, and coagulation. Horses with laminitis may show anorexia, anxiety, trembling of the musculature, increased respiration and pulse rates, and variable elevation of body temperature.

Obel (34) described the gradations of lameness associated with acute laminitis:

* grade 1 At rest the horse will alternately and incessantly lift the feet. Lameness is not evident at walk but a short stiffened gait is noted at trot.
* grade 2 The horse moves willingly at a walk, but the gait is characteristic for laminitis. A hoof can be lifted off the ground without difficulty.
* grade 3 The horse moves very reluctantly and vigorously resists attempts to lift a forefoot.
* grade 4 The horse must be forced to move and may be recumbent.

Recently, a good correlation between Obel grade lameness and severity of lamellar histopathology was established (39). Apparently, in acute laminitis the lameness may vary from being very mild to severe (grades 1 to 4). Frequently misdiagnosis/non-diagnosis of acute laminitis arises from waiting for the development of severe signs for a diagnosis to be made. However, treatment should be initiated as early as possible and waiting for classical, severe symptomatology is detrimental to good medical management (44).

Chronic laminitis

Laminitis is considered chronic after 48 hours of lameness or after rotation and/or sinking of the pedal bone (51). This displacement may occur as early as 3 hours after disease onset and may vary from mild to severe (25). Rotation may result in separation of the hoof wall and bulging of the sole dorsal to the apex of the frog, the latter indicating that the pedal bone is beginning to penetrate the sole. Chronic rotation causes tipping of the tip of the pedal bone which becomes obvious after 4 to 6 weeks. Sinking causes separation and infolding of the coronary band over the extensor process region. The laminar damage resulting from laminitis causes

Figure 1. Typical stance of a horse with laminitis of both forelimbs.
abnormal hoof growth. This is seen as diverging rings on the hoof wall, wider at the heel than at the toe, a flattened or convex sole, and the widening of the white line (51). If trimming is not regular and corrective in nature, the toe becomes abnormally long. At walk, horses with chronic laminitis tend to land on the heel first, followed by an exaggerated toe slap.

**RADIOLOGICAL EXAMINATION**

Radiographic examination demonstrates the degree of pedal bone rotation and/or sinking (Figure 2) and associated complications such as infolding of the coronary band, separation of the dorsal hoof wall, deformation of the tip of the pedal bone or the tendency of the pedal bone to perforate the sole (15, 17, 51, 52). Different techniques are used to measure the degree of rotation and/or sinking.

### Bone rotation

Stick and coworkers (52) used the degree of bone rotation to predict the prognosis for return of function. The hoof angle is determined from the intersection of a line drawn parallel to the palmar or plantar aspect of the hoof (= the sole) and a line drawn parallel to the cranial aspect of the hoof wall. The angle of the pedal bone is formed by the intersection of lines drawn parallel to the anterior aspect of the pedal bone and the palmar or plantar aspect of the hoof (= the sole). The degree of rotation is obtained by subtraction of the hoof angle from the angle of the pedal bone. Horses with less than 5.5 degrees rotation may return to athletic function with corrective trimming and shoeing. Horses with more than 11.5 degrees rotation are lost as performance animals (52). Eustace and Caldwell (15), however, had encouraging results in horses with more than 11.5 degrees of rotation, using the dorsal hoof wall resection technique and a heart bar shoe.

### Vertical distal displacement and true bone rotation

The vertical distal displacement and the true rotation of the pedal bone are also used as parameters of the degree of rotation or sinking (5, 14, 15, 17). These parameters are determined using the following technique. First, the frog of the unshod foot is trimmed. The hoof wall is gently rasped to remove loose horn and create a flat dorsal surface. A straight stiff wire marker of known length is Sellotaped to the dorsal hoof wall with the proximal end of this wire on the coronary band. The horse is radiographed standing squarely on a flat wooden block incorporating a wire marker as a ground line. The cent of the radiographic beam should be parallel to the long axis of the navicular bone and the top of the wooden block. The vertical distal displacement of the pedal bone is measured by drawing two lines on the radiograph, both parallel to the ground line, one through the top of the extensor process and one through the top of the dorsal wall wire. The mean value in a normal digit varies in Thoroughbreds from 0 - 10 mm, average 3.5 mm (17). True rotation of the pedal bone is measured by comparing the angle subtended by the line connecting centres of curvature of the proximal and distal inter-phalangeal joints with the ground surface and the angle subtended by a line extending from the dorsal surface of the pedal bone to the ground surface. Determination of the true rotation of the pedal bone has the advantage above the rotation measurement technique of Stick and coworkers (52) that malformation of the hoof does not influence the outcome.

### Practical measurement of bone rotation and/or vertical displacement

An alternative, less complex technique, used in Utrecht, is measurement of the thickness of the dorsal hoof wall distal to the extensor process and at the tip of the pedal bone on a lateral radiograph of the foot. The mean value of each measurement in a normal digit of Warmblood horses is 19 mm ± 1.5 mm (K.J. Dik, personal communication). With rotation the proximal measurement remains normal but the distal measurement is enlarged. With sinking both measurements are similar but enlarged. With sinking and rotation both measurements are enlarged, but the distal value is higher than the proximal value (Figure 3).
THERAPY
The treatment of horses that develop acute laminitis should be considered an emergency (6,30,44,58). Even a few hours’ delay in treatment can mean the difference between a successful outcome and a failure (30). However, a therapeutic regimen, using biological or chemotherapeutical agents able to arrest or block the triggering of laminitis, does not exist (44). Since laminitis usually develops as a sequel to a disease process in a body compartment other than the foot, it is of paramount importance that the primary disease is treated urgently and effectively. So, under circumstances that increase the risk that a horse will develop laminitis (i.e., ingestion of large quantities of grain or concentrates, retention of the placenta, or diseases producing Gram-negative sepsis and endotoxaemia), therapy should be started before the clinical signs become visible.

The treatment of laminitis should be focussed on the following aims (6,26,30,44):
* elimination of cause or triggering factor
* block pain - hypertension cycle
* improve digital circulation (not during developmental phase?)
* prevent further rotation or sinking of the pedal bone
* promote keratinization and healing of hoof and sole defects
* maintain systemic health.

Therapy can be divided into several aspects (22):

**Dietary management**
The most important measure is to withhold grain and concentrates from the diet and to supply sufficient good-quality hay. Laminitis may be accompanied by hypokalaemia and, if necessary, potassium chloride can be given orally in a dosage of 15 gram tid for a 500-kg horse (13). Supplementation with biotin and methionine might be useful to optimize horn production. Biotin is given to a 500-kg horse in a dosage of 15-100 mg per day orally (13) and methionine is administered once a day orally in a dosage of 22 mg/kg during the first week, 11 mg/kg during the second week, and 5.5 mg/kg during the third week (53). Vitamin supplementation seems useful as laminitis is a debilitating and stressful disease. Virginiamycin, in the correct formulation, is considered a useful laminitis prophylactic for horses and ponies with a high carbohydrate intake, but has little value as a therapeutic agent (49).

**Medical treatments**
Medical treatments can be divided into four groups (the dosages are given for a 500-kg horse):
* medication to decrease or prevent the absorption of toxin
  Both mineral oil (2-4 litre bid) and activated charcoal (0.2-1 g/kg in water bid) can be administered by nasogastric tube for several days (44). However, these medications should not be given at the same time, because mineral oil decreases the adsorptive capacity of activated charcoal (21). Retained placenta should be treated appropriately, if the placenta has not been expelled within 3-4 hours following parturition. Horses with laminitis resulting from exhaustion, dehydration, and hypovolaemia should be given balanced electrolyte solutions (44,58).

* drugs to improve the circulation of the foot
  Both vasodilators and anticoagulants may improve the circulation of the foot. Administration of heparin has been advocated to inhibit the formation of thrombi in the digital vasculature, and it is most efficacious when used prophylactically, 40-100 units/kg bid SC for several days (22), or 100 units/kg qid for several days (32). Although the clinical effectiveness of heparin has been questioned (7), recent publications seem more promising (11,57). Acepromazine (40 mg IV or IM 4-6 times daily) may be beneficial in the treatment of the vasoconstriction that is thought to accompany acute laminitis (12,22). Although there is some discussion whether it acts as a vasodilator in the foot, it seems to benefit the laminitic horse (26). Peripheral vasodilator therapy should only be used in horses with a stable cardiovascular system (6,9,58). Other vasodilator or anticoagulants mentioned in literature are isoxsuprine, phenoxymazine, dimethyl sulfoxide and warfarin, but objective data concerning their effectiveness are not available at this time (6,22,44,51,53). Recently, the use of nitroglycerin (gyceryl trinitrate) as a topical ointment or a metered-release transdermal patch has been advocated to resolve affected hooves (45).

* breaking the pain - hypertension cycle
  Nonsteroidal anti-inflammatory drugs (NSAIDS), such as acetylsalicylate, flunixin meglumine, phenylbutazone, have analgesic, antipyretic, and anti-inflammatory effects due to their ability to inhibit the synthesis of prostaglandins. The disadvantage of these drugs is that partial abatement of pain may lead to increased ambulation and weight bearing on compromised laminae, which could cause further tissue destruction in the feet. Pain abatement also may obscure the clinical detection of abscesses that require debridement and drainage. However, the benefits of NSAIDS outweigh their risks. As long as affected horses are stall rested and exercise is restricted, NSAIDS are beneficial in the treatment of laminitis (22). Acetylsalicylate (10-25 mg/kg bid PO) may have an analgesic and, as some believe, a vasodilator effect, but it has a proven anticoagulant effect mediated through its antiplatelet activity. Flunixin meglumine (1.1 mg/kg bid IV) may be an analgesic and, as some believe, a vasodilator, but objective data concerning its effectiveness are not available at this time (6,22,44,51,53). Recently, the use of nitroglycerin (glyceryl trinitrate) as a topical ointment or a metered-release transdermal patch has been advocated to resolve affected hooves (45).

Perineural anaesthesia of the digital nerves at the base of the sesamoids (35), although advocated by some authors (58), is not recommended nowadays because complete abatement of pain can lead to an increase in ambulation and weight bearing sufficient to cause further distal phalangeal rotation, sinking, and tissue damage (8,9,22,44). In the last few years, platelets were proven to be involved in the pathogenesis of equine alimentary laminitis (55). Recent research has revealed that platelet aggregation inhibitors may be useful for the prevention and/or treatment of laminitis (56,57).
However, the frequent occurrence of hypothyroidism in horses with laminitis has not been confirmed by other authors.

Corticosteroids are known to augment the vasoactive effect of catecholamines and are contraindicated in laminitis (16,17,18,22,30).

Antihistamines are thought to be of little value after histamine is released (51), although they might be effective in the developmental stage of the disease (22).

**Therapeutic trimming and shoeing**

Therapeutic trimming is essential for the re-establishment of normal vascular perfusion and of normal spatial orientation among the pedal bone, hoof wall, and sole, normalization of hoof growth, and exposure of submural and subsole sepsis, allowing local treatment (22). These facts are supported by all authors. However, whether acute cases of laminitis should be trimmed and/or shod and the way in which this should be done, is controversial (12,58). The choice of the best approach is also influenced by the stage of the disease (acute or chronic) and by whether the pedal bone is beginning to rotate, in the process of rotation, or already rotated.

Radical trimming of the feet of horses with acute laminitis should be avoided, but shoes should be pulled, and too long toes should be shortened (6). Adair (1), however, does not recommend that shoes be applied or removed during the acute phase of laminitis. If seroma formation exists between the hoof wall and the pedal bone, several techniques are available. Some horses develop seroma within 5 days, while in other cases seroma formation takes 6 weeks. Advocated techniques are partial or total hoof wall resection (22,46), dorsal hoof wall stripping (22), or drilling several holes in the dorsal hoof wall (47). These procedures, however, demand not only experience, but also great care to prevent infection. In Utrecht, thinning of the dorsal hoof wall is advocated for acute cases of laminitis (3). The horn of the dorsal hoof wall is removed gradually by rasping until the laminar dermis shines through. This laminar dermis, however, is still covered by a very thin layer of horn preventing infection.

Mechanical support of the apical one-third of the frog, while avoiding sole pressure, might be helpful and is advocated by several authors, although the techniques used and the moment of application are different (9,15,21,22). Coffman (9) describes that in acute cases frog support is best achieved by placing a 5-cm gauze roll longitudinally on the frog, just behind the apex, holding it in place with elastic tape. He believes that the use of a heart bar shoe is contraindicated and questions the advantages of anterior wall resection in acute laminitis. Yelle (58) describes that thick cotton or rubber paddlingatraumatically applied can support and protect the sole sufficiently. Chapman and Platt (8) and Eustace and Caldwell (14) described the use of a heart bar shoe to support the skeletal column of the horse, but also warned against its improper use, as it can cause much damage when not applied in the proper way. Eustace and Caldwell (15) and Goetz (22) advocated both the dorsal hoof wall resection technique and the use of the heart bar shoe in horses suffering from acute laminitis, but these ‘acute cases’ already showed symptoms for several weeks. Huskamp (28), in contrast, advocated elevating the heel to decrease the tension of the deep digital flexor tendon. For this he used a plaster cast that takes the stress off the anterior part of the hoof.

In acute cases of laminitis, Stashak (51) abandoned all the other approaches and used a stall bedded with ample soft sand. Soft sand provides good physiological support for the sole and allows the horse to stand in the position of choice. In chronic cases, he advocated techniques to re-establish the normal alignment of the pedal bone and to provide good support (51). The choice between different shoes, pads and/or silicone rubber is dependent on the reaction of the horse and the personal preferences of the veterinarian and farrier.

**Other supportive care**

Hydrotherapy of the affected hooves seems essential during the first days in horses with developing laminitis. Eustace and Redden stated that hot water soaks tend to provide quicker and longer-lasting clinical benefit than do cold or ice water soaks (17,47). There is, however, insufficient data to suggest whether cold (via reduction of metabolic activity and decreased perfusion ectotera) or hot (via vasodilatation and increased metabolic activity) water hydrotherapy is more effective. Cold water hydrotherapy is, however, in most cases readily available.

Exercise is recommended by many authors, but it is a ‘double-edged sword’ (51). Exercise on one hand improves the circulation in the foot, but on the other hand it also increases the mechanical forces that are thought to contribute to pedal bone rotation and the pain-related positive feedback cycle that precipitates and perpetuates hypertension and vasoconstriction (25). Because of these factors, limited exercise is only recommended early in the acute phase, and only if it does not result in increased pain (6,9,51). Recently, Wattle and coworkers discussed the possibility of using short-term forced recumbency as adjunctive treatment in an acute attack of equine laminitis (54). Carpal check ligament desmotomy and deep flexor tenotomy, to decrease the tension on the deep digital flexor tendon, are advocated by some authors (1,2,6,47) and rejected by others (51). These techniques, however, are primarily indicated for breeding animals and should only be considered if conventional therapy fails (6).

Blood letting, although used frequently in the past, is no longer advocated (46).

A comfortable surface to lie on will invite the horse to lie down frequently, taking the weight off its feet (10,37) and prevents bed sores (58). The ideal ‘laminitis stall’ should have a wet sand area near the crib for the horse to stand in during eating, while the rest of the stall is covered with a thick layer of straw and/or shavings for the horse to lie down on.

**Prognosis**

The treatment of laminitis is often costly and time-consuming...
and has an uncertain prognosis. The result of treatment of laminitis is probably mainly determined by the severity of the initial damage and the possibilities to treat or remove the initial cause or predisposing factors. Redden (47) stated that ‘approximately 80 per cent of all laminitis cases respond favourably, regardless of the therapy; the remaining 20 per cent fall into the complicated category and their prognosis is guarded at best from the onset’.

A major determinant of outcome may be the commitment of the owner and his or her resolve to follow the advice of veterinarian and farrier. It is critical to identify any underlying disease and to treat this accordingly (1, 30). In cases of acute laminitis it will take at least 8 weeks before any prognosis can be given. The prognosis depends on the response to treatment clinically and the progression of the radiographic changes (27). Hunt (27) found in a retrospective evaluation of 202 horses that the presence or absence of pedal bone displacement and the degree of pedal bone rotation could not be used to predict the outcome of a horse with laminitis. In this study, the clinical assessment was a more reliable means of determining the final outcome and should be given precedence over radiographic findings (27). In the long-term follow-up, the prognosis for laminitic horses was poor: only about 25% of affected animals returned to an athletic career, about 25% were intermittently or permanently lame, and about 50% had died (27).

In chronic cases it will take at least 4 months to 1 year before the future performance abilities can be estimated because horses that respond favourably to therapy often show remission (22). However, horses with more than 15 degrees of rotation accompanied by downward displacement of the pedal bone into the hoof capsule within 4-6 weeks of the initial episode of laminitis have a poor prognosis (44). Considering the enormous pain for the horse, it seems not humane to treat a horse with prolapse of the pedal bone through the sole (Figure 4).

CONCLUSIONS

At the moment, no known therapeutic regimen, involving either biological or chemotherapeutic agents, is able to arrest or block the triggering of laminitis (44). Many authors state that, despite this imperfection, treatment should be started as soon as possible after the onset of symptoms (9, 51) and systemic treatment is essential as trimming and shoeing cannot cure a systemic disease (42). No one therapeutic regimen has been proven to be superior to another for the treatment of severely affected horses (33) and many current treatments have not been critically evaluated (12, 25, 44). However, there are numerous remedies that, used empirically, may give symptomatic relief to the horse after it has developed laminitis (44).

Because laminitis usually develops as a sequel to a disease process in a part of the body other than the foot, the treatment of horses ‘prone to laminitis’ at Utrecht is also focussed on treating the primary disease (mostly septicaemic/endotoxaemic problems) urgently and effectively. In recent years, flunixin meglumine (0.25 mg/kg IV tid), acebutolol (5-10 mg/kg PO bid-qid), acepromazine (0.05-0.1 mg/kg IV or IM qid), and cold water hydrotherapy (qid) were added in most cases to the treatment of the primary disease in these horses. After the publications by Pollitt et al. (41, 44), there is some hesitation about using acepromazine in the prodromal phase. Further evaluation of the effect of different treatment protocols is necessary. However, many of the horses ‘prone to laminitis’ fortunately do not get the disease, and the ones showing the acute phase have a large variety of primary diseases. As result of these limitations, evaluation of different treatment protocols is very difficult. In protracted and/or severe cases of laminitis, euthanasia should be considered, because treatment may be prolonged, costly, and not beneficial to the welfare of the horse.

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