Equine Laminitis: Practical Clinical Considerations

Robert J. Hunt, DVM, MS, Diplomate ACVS

Author's address: Hagyard Equine Medical Institute, 4250 Ironworks Pike, Lexington, KY 40511; e-mail: rhunt@hagyard.com.

1. Introduction

Over the past 20 yr, extensive research efforts as well as clinical experience has greatly increased our knowledge base of equine laminitis. Our present understanding of the disease is that there is failure of the lamellar attachment between the distal phalanx and hoof wall.[1–7] This, in turn, initiates a sequence of events ranging from no permanent structural damage to loss of the hoof capsule. Affected individuals are often plagued with chronic pain, and they may suffer chronic hoof abscessation or abnormal hoof-wall development with recurring bouts of laminitis.

The preponderance of laminitis research has been aimed at defining the events that occur systemically and locally within the digit that result in clinical laminitis. Although we have identified many early events that occur, we have yet to determine a specific triggering mechanism that initiates the pathological sequence. Importantly, there is a disconnect between research and clinical medicine in that much of our acquired research information is not used clinically. This is partly because of the narrow time frame of the prodromal and acute stage of laminitis compared with the relatively late clinical presentation and relative long-standing nature of the disease. Also, because of the variation in clinical case presentation and response, our treatments are anecdotal, based on clinical signs, and in the long run, often ineffective.[8–10] Numerous treatments have, therefore, phased in and out of vogue over the years with only a few, such as the non-steroidal anti-inflammatory drugs, withstanding the test of time. The objective of this discussion is to review the salient features of laminitis and provide a realistic view point for the practitioner to consider when managing the patient and the client.

2. Overview

The foot is a highly resilient and durable structure able to tolerate extremes in load and concussion on a variety of surfaces under a variety of environmental conditions, such as excessive moisture or dryness, freezing temperatures, or extreme heat. It is well designed and able to withstand notable insult externally and internally. Considering the normal rigors that the equine foot endures, it is almost incomprehensible to think a variation in normal physiology can result in such devastation as that seen with laminitis.

Anatomy and physiology of the equine foot have received considerable attention.[11–18] The most relevant structures of the foot pertaining to laminitis include the vascular architecture and the dermal epidermal interdigitation of the primary and secondary lamella supporting the coffin bone to the hoof wall.[11,12] The vascular supply to the digit and lamellar bed is extensive and functions to provide nutrition, structural support, and hydraulic damp-

NOTES

ening against concussive shearing. The circumflex artery provides a large portion of the corium vascular supply through the laminar arteries coursing in a distal to proximal direction. Venous drainage accompanies the arterial supply in a proximal to distal direction. The arteries of the digit are highly muscular, which likely function in the dampening mechanism as well as allow for little compliance. There are an abundance of arteriovenous shunts throughout the vascular bed that allow bypass of the capillary bed as well as rapid alterations in hoof temperature and capillary nutrition.

Ultrastructurally, the hemidesmosome maintains the attachment between the secondary epidermal lamella basal cell and its basement membrane. The sequence of events that occur during experimental laminitis results in a disassociation of this junction beginning at the basement membrane level of the secondary lamella from cleavage of the hemidesmosome.[3] Irrespective of the initiating cause, there seems to be some overlap in the pathogenesis of the syndrome, because the end result is loss of mechanical support caused by laminar detachment. Imposed biomechanical load on this compromised junction causes further lamellar disruption. Additionally, it results in additional physical separation and displacement of the coffin bone from the hoof wall.

Several hypotheses have arisen from a sound body of research to explain the pathophysiology of laminitis. Presently, it is not possible to apply a single theory to every case, and the pathogenic pathway likely varies depending on the type and severity of the inciting cause. The various hypotheses may be classified as vascular, metabolic/toxic, inflammatory, or traumatic (mechanical) in origin.[7] There is likely some degree of interplay and overlap in the various mechanisms, but the connection has not been fully proven. It is beyond the scope of this presentation to provide a detailed literature review on these theories. From a pragmatic clinicians' perspective, it seems that a destructive endogenous agent (metalloprotease or others), vascular ischemia, or mechanical overload of tissue results in separation at the basement membrane, loss of structural integrity, and separation of the coffin bone caused by mechanical load on the digit. We need to be cognizant that data are lacking to support these theories on large groups of horses in a clinical setting, and because of the lack of reproducibility of this syndrome, it remains difficult to prove.

3. Clinical Presentation and Diagnosis

Understanding the pathophysiology of laminitis is important to the understanding of the disease; however, it provides only a small piece of the puzzle when managing a clinical case. Accurate assessment of the whole patient with consideration for signalment, occupation, and owner expectations should be considered in every case when attempting to provide appropriate treatment as well as prognosis. One must ascertain if the horse does actually have laminitis and attempt to determine the underlying cause. It cannot be overemphasized that this is a dynamic disease that progresses at variable rates. Clinical laminitis is appropriately referred to as a syndrome because of the differing presentations and stages. Managing an unstable acute sinker is vastly different than working with a horse with a chronic stable laminitis that is able to perform athletically.

Diagnostics remain basic for laminitis, but thoroughness must be emphasized. The physical evaluation and in particular, detailed evaluation of the feet is mandatory. Assessment of the quality and integrity of the feet, intensity of digital pulse, and temperature should be made. The coronary band should be assessed for the presence of edema (swelling), depressed areas (sinking), or palpably tender areas (possible abscess or separation of hoof wall). The shape and position of the sole is observed for degree of concavity or protrusion, soft spots, or excessive thinning. The size, shape, and integrity of the feet are especially important when designing a podiatry program for the horse and for monitoring change associated with the progression of the disease. The single evaluation provides limited information relative to the long-term management of the patient with laminitis, and the clinician must look for trends in disease progression over time to guide appropriate therapy.

In most instances, observation of the stance and gait are strong indicators of the presence of laminitis. The characteristic stilted camped out front legs are believed to redistribute load to the hindlimbs.[5] Variations on this stance likely occur because of the presence of pain in the rear feet or variations in the location of pain in the front feet. It is not often necessary to perform diagnostic nerve blocks to diagnose laminitis; instances of low-grade bilateral pain associated with chronic laminitis can be an exception.

There are three vital pieces of information to obtain when evaluating a patient and formulating a diagnostic, therapeutic, and prognostic plan. The clinician must determine the reason and source of pain, the location of pain, and the degree of instability within the foot. It may take several visits and serial evaluations spread over days to weeks to accurately make this assessment. Ultimately, one hopes to determine if the horse can continue to generate a viable and functional hoof capsule, and this is presumably correlated with the integrity of the germinal layers.

The source or cause of pain within the foot should be determined, and differentiation should be made between septic processes, non-septic processes, bruising, laminar shearing, ischemia, and bone pathology. Digital sepsis or abscessation may occur without loss of structural integrity to the lamella. Consideration of the etiology and stage of laminitis should be given when assessing the source of pain.

A horse with chronic laminitis with keratin hyperplasia resulting in an overgrown foot with seedy toe and prolapsed sole is more likely to have a subsolar abscess contributing to the lameness than a horse with acute laminitis with radiographic evidence of sinking. With acute distal displacement, pain is probably associated with laminar shearing, digital collapse, and pressure on inflamed sensitive tissue. In general, it is counterproductive to open the sole in this situation.

The location of pain is likewise important to determine from a therapeutic standpoint. Hoof-tester evaluation is useful when positive, but a negative response does not rule out foot pain or laminitis. It is common to have a negative hoof-test response in a horse with thick soles and hoof capsule. Horses with metabolic syndrome also often have a negative response. Bilateral diffuse solar pain across the toe and dorsal wall is considered characteristic for laminitis; however, bilateral foot bruising may yield similar findings on a single evaluation. Focal pain anywhere in the foot is generally associated with abscess, but the horse may display a "laminitic" gait to relieve load on the foot. Pain predominantly located in the toe and dorsal wall and sole is managed differently than pain in the heel region or along the medial wall quarter. Variations of the stance and gait are recognized when pain originates in areas other than the toe and dorsal wall. Laminitis involving the heels will present with a toe-first gait or flat-footed landing, or the feet may be camped under the body; the heels may collapse or detach at the coronary band. Hoof-wall collapse along the medial wall is another recognized entity associated with distal displacement of the coffin bone. It is not fully understood at this time if this is attributable to a greater degree of lamellar damage in this region or simply to regional mechanical overload along the medial wall. Interestingly, this is seen in some horses as they age and become more pigeon toed in conformation.

The most important determinant of long-term outcome in the acute active laminitic patient, and one of the most difficult to determine, is the degree of instability between the coffin bone and hoof wall. At present, we have few, if any, means to make this assessment beyond serial radiographs, clinical evaluation, and in some situations, the venographic study. Differences in coffin bone position between loading and non-weight-bearing stance may provide some indication, but at present, this information is theoretical. Impending instability is difficult to determine given our evaluation techniques. Therefore, by the time active displacement is recognized, instability is present.

Radiographic evaluation is the only imaging modality as valuable as the physical evaluation. When using conventional and digital radiography, techniques and views are well standardized.[19] Standard views including the lateral to medial, dorsal palmar, and 45° dorsal palmar projections should be performed routinely. Findings should be assessed in light of the rest of the clinical picture, which must consider the stage of the disease and especially, the rate of change of the position of the coffin bone within the hoof capsule. It is important to note that the radiographic study represents a static image of a dynamic model and disease. It is most useful when combined with the clinical evaluation during serial sessions.

Radiographic assessment has changed from only measuring rotation of the coffin bone; however, this is still popular because of the relative standardization and familiarity of the technique. Other objective parameters currently assessed include the palmar angle of the third phalanx, horn-lamellar (H-L) zone width proximal and distal, extensor process-coronary band distance, sole depth at the tip and wing of the distal phalanx, and soft tissue.[19] Integrity of the coffin bone is likewise evaluated for proliferative or erosive changes, relative density, and porous or pathologic fracture. Again, the progression of radiographic changes over time is important to consider.

The use of digital venography varies among clinicians but there is definite merit to the procedure. Obviously, it is not indicated in every horse with laminitis or even the majority. The primary indication is to gain an appreciation of the vascular perfusion of the digit for prognostic and therapeutic purposes. The technique has been described in the literature.[20] A venogram that shows normal fill of the vascular space generally carries a much better prognosis for the horse than a venogram with no perfusion. Relative poor perfusion is accounted for by vascular compression, vascular damage from soft tissue disruption, possible influence of arterio-venous shunts, or technical errors associated with the procedure. Venography allows evaluation of the digital vasculature for deficits in the coronary circulation, coronal circulation, terminal arch, dorsal papillae, bulbar circulation, and circumflex vessels.[20] As with other diagnostic aids, the whole clinical picture must be considered in interpretation.

Differentiation of the etiology of laminitis is important given that different forms of laminitis seem to manifest themselves in a unique and often predictable clinical scenario. For example, laminitis as a result of retained placenta or septic metritis typically follows a course of extreme pain (Obel grade 3 or 4) for 3–5 days followed by a moderate improvement and a more clinically stable appearance (Obel grade 2) for the following 4–8 wk. After this period, there is commonly a recrudescence of pain accompanied by further displacement of the coffin bone. This is different than a horse with laminitis associated with administration of corticosteroids, which often undergoes no displacement of the coffin bone; however, the patient may remain persistently foot sore and unusable athletically. When steroid-induced laminitis does result in displacement of the coffin bone, it is generally severe and may result in sloughing of the hoof capsule in a

matter of days to weeks. This is often the case with horses suffering from severe diarrhea or proximal enteritis in which the severity of clinical laminitis and displacement of the coffin bone seem to have a direct correlation. Severely toxic horses may undergo displacement of the coffin bone to the point of detachment of the hoof capsule in days to weeks. Horses with unilateral laminitis as a result of overload generally progress very slowly up to a certain point at which extreme displacement and instability occur over a relatively short period. It is unknown if these clinical manifestations have different events involved in the developmental stages leading up to clinical laminitis.

4. Prevention and Treatment

Therapy for laminitis is based on the underlying cause and the stage of the disease; however, all forms of treatment are controversial including medical management as well as shoeing and surgical management. Given the fact that a specific mediator has not yet been identified, there is not a specific preventative agent or treatment. It is an academic argument to debate the efficacy of many of these drugs against laminitis, because they are generally administered for the primary disease.

Recognized high-risk individuals are those suffering from a toxic event such as acute metritis, proximal enteritis, enterocolitis, grain overload, intestinal strangulation, or pleuropneumonia.[1,7,21–24] Obese individuals are at an increased risk as well, especially if obesity is accompanied by metabolic syndrome. There is usually a time frame of 24–72 h from onset of clinical signs of toxemia until signs of laminitis are seen. Clinically, the most commonly used drugs for prophylaxis and treatment are non-steroidal anti-inflammatory drugs (NSAIDs). Flunixin meglumine[a] (1.1 mg/kg) has been shown to be effective at dampening clinical signs of endotoxemia. Phenylbutazone[b] (2–4 mg/kg) clinically seems more effective at providing pain relief; however, this has not been proven in a laminitis model nor has the combination of flunixin and phenylbutazone been proven more effective than either alone. It is still common to use these drugs in combination in a clinical setting. IV lidocaine[c] infusion (0.05 mg/kg/h as a constant-rate infusion) is often used to treat ileus associated with enteritis or to manage the patient after abdominal surgery. This has been shown to decrease leukocyte activation and may have benefit in the developmental stages of laminitis. Polymixin B[d] is commonly used to ameliorate clinical signs of endotoxemia; however, it was not effective at preventing laminitis in a grain-overload model. Dimethylsulfoxide[e] is commonly used as a free-radical scavenger, but data proving its efficacy experimentally and clinically is lacking. Work showing normally low laminar superoxide dismutase activity suggests that drugs used to combat reactive oxygen species may be beneficial.

Although they have not been shown to have benefit at preventing or treating laminitis, medications altering hemodynamics and blood flow are still in common usage. Acetylpromazine[f] is commonly given for its vasodilator properties and has been shown to be effective at increasing digital blood flow at a high dose. Unfortunately, the effects in the clinical patient are unclear other than that the drug depresses the patient. Pentoxifylline[g] has been used as a rheologic agent, but its efficacy in experimental laminitis or the clinical patient has never been substantiated.

Fig. 1. The Falkner Walker[h] with support sling. This allows mobility and manipulation of the patient without fully loading the limbs.

Regional hypothermia to prevent laminitis in an experimental model has been documented.[25] Clinically, the treatment has not been implemented enough to obtain sufficient numbers of cases to document its efficacy. It is also frequently being used after the onset of clinical signs, and this is probably too late in the disease progression to have merit.

Physical measures to alter laminar load are often taken too late. It is not known if frog pressure devices, elevators, or other foot devices have any benefit at preventing laminitis associated with a toxic event, but regardless, we continue to routinely use them.

Measures to prevent contralateral limb laminitis are mostly common sense and include reestablishing normal weight bearing as soon as possible and attempting to reduce excessive load on one foot. Horses wearing a cast after limb injury should have the opposite foot elevated so that they bear weight evenly if comfortable. Any foot apparatus should provide a uniform cushioned layer for weight bearing. Facilitating load reduction with a sling is labor intensive and requires a patient that is tolerant. Portable mobile sling devices[h] (Fig. 1) are available; these slings are helpful with rehabilitation, because they reduce load bearing as well as support the patient during bandage changes, trimming, and shoeing.

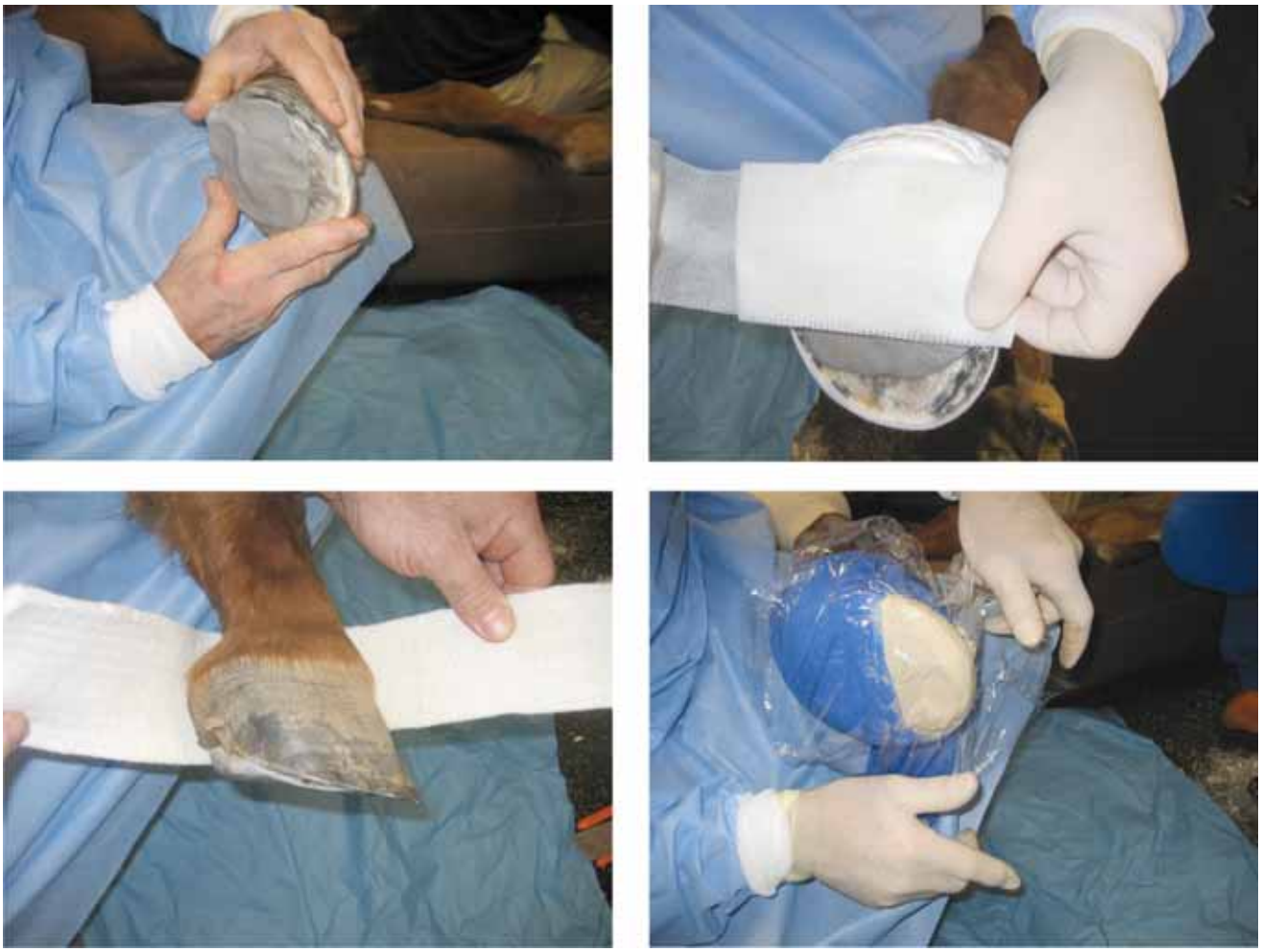

Fig. 2. Application of a foot cast.

Specifics regarding podiatry will be discussed elsewhere, but the limitations and goals of foot management must be mentioned. One must keep in mind that there are no controlled studies documenting the efficacy of any podiatry procedures. Techniques seem to change almost yearly, and for the most part, they are based on an idea. Thus, as with systemic medications, podiatry remains anecdotal and is based on the stage of the disease, clinical experience of the veterinarian, and response of the patient.

The most obvious limitations of podiatry include the facts that we have such a relatively small area to work with in an attempt to offset profound vertical forces imposed on the digit and that we are applying forces through compromised tissue and therefore, may cause further tissue damage. The type of damage within the foot may change rapidly, because the disease progresses or may change from our intervention. In general, goals of foot therapy include relieving pain within the foot and providing support to structurally stable areas of the foot to prevent further tissue damage. The basic principle of using the "good area" of the foot is safe so long as it is not overused. There are no quick fix shoes that consistently work on horses with laminitis, and in most instances of acute laminitis, shoes provide little to no benefit and may cause more damage. One can achieve adequate uniform support with suitable bedding that will conform to the soles of the feet. Application of a heavily padded foot pack, Styrofoam,[i] impression material,[j] or commercially available boots[k] with a soft weight-bearing surface will accomplish this purpose as well. After a chronic state is reached and the foot is stable, shoes will provide protection and allow a broader range of corrective changes to the foot. Changes made to the foot are somewhat limited and include the length, angle, breakover, balance, and focally loading or unloading regions. These procedures may help stabilize the foot, reduce loading on painful areas, and facilitate revascularization and foot growth.

One useful technique that the veterinarian or farrier may use is application of a fiberglass foot cast with compliant impression material against the sole (Fig. 2). This is particularly beneficial in the patient with chronic laminitis that has poor quality

and poor integrity hoof wall that is continually suffering from bruising of the feet. Applied properly, these may be worn >1–2 mo before changing is necessary. After proper trimming, impression material is applied to the palmar one-half to two-thirds of the foot. Cast padding[l] or other cushion is placed over the heel bulbs, and one roll of 3- or 4-in cast material[m] is applied to the foot. The material may be manipulated to alter the angle of the foot, point of contact, and point of breakover. The toe region should be protected with equilox[n] or other composite material to prevent premature wear. I prefer the cast over composites applied to the foot, because the circumferential support to the wall prevents the spreading of the laminae at the ground surface. Also, the cast material allows the foot to expel moisture better than most composites, and thus, it remain a healthier environment.

Surgical management for laminitis may be placed in 1 of 3 categories: hoof debridement and resection, deep digital flexor tenotomy, and digital unloading by transfixation pinning or foot amputation. The most common procedure involves debridement of necrotic material in chronic laminitis. The intent is to allow drainage of purulent material and allow regrowth of healthy horn. Hoof-wall resection is indicated when there is compression of vasculature or germinal tissue from displacement within the hoof capsule. Wall resection may be performed as total removal of the dorsal wall from the solar surface of the toe to the coronary band or as removal of a 1- or 2-cm horizontal strip at the coronary band. A more aggressive resection will require more external support of the hoof in the form of shoeing, casting, or bandaging.

In general, tenotomy is most beneficial in the patient with chronic laminitis that has chronic recurrent abscessation. Clinically, there may be improvement in the acute patient, but long-term outcome does not seem to be appreciably altered. Techniques have been described in the literature.[26–28]

Transfixation pinning is usually reserved for severe laminitis with extreme compromise of the vasculature and soft tissues, and it is, unfortunately, often an end-stage case. The major limitation of this procedure is the duration of time required to allow for clinical stability and regrowth of the foot. The most common complication encountered is pin loosening or fracture of the cannon bone through pin holes. These complications may be lessened through appropriate placement technique.[29]

5. Client Management

Client communication is one of the most important but least discussed aspects of case management. Salient points have previously been discussed that pertain to client education.[30] Accurate conveyance of information on a regular basis is critical to avoid client dissatisfaction, unpaid debt, and possible litigation. A policy of open, honest communication that tempers false expectations must be used. Horses rarely die from laminitis; euthanasia is generally performed electively for humane purposes. Clients read and talk. Given the seriousness of a case of severe laminitis, they will most likely look into other sources regarding the management of their horse. It is imperative that the treating veterinarian and farrier are well versed in the common inquiries and that these are addressed prospectively. They should present a unified approach to case management with neither party questioning the procedures of the other to the client.

I preface many conversations with clients that have horses with laminitis with a basic premise. With the exception of resolution of a foot abscess, if it happens fast, the laminitis is usually bad; it is pretty easy to tell when things are going bad. The client needs to understand that this is not a disease that will suddenly resolve itself, and with severely affected patients, it is not uncommon to expend 1 yr or more of effort only to end up with a permanently lame or dead horse.

Accurately predicting the outcome of horses with laminitis is controversial. This is understandable given the number of variables associated with management of the clinical patient, which includes not only the feet but the rest of the patient in addition to client variables. Several studies have attempted to provide clinical parameters that are useful in prognosticating. These include radiographic findings correlating the degree of coffin bone rotation to soundness and clinical outcome or the presence of distal displacement of the coffin bone, and the severity and duration of pain having an influence on the outcome.[24,31,32] However, as with most clinical entities, there are exceptions to every rule. We largely rely on clinical experience and temper this with our recognized guidelines. These general guidelines are influenced by other clinical findings such as the accompanying disease, stage of laminitis, and degree of instability within the hoof capsule. For example, a horse with chronic laminitis that has 10–12° of rotation but is stable has a much better prognosis than an acute laminitis case with 3–4° rotation that has occurred over a 1-wk period and displays severe pain.

The impact of the disease in the equine community and the importance of determining the underlying cause and having the ability to offer prevention has come to the forefront in the equine community. We must also remain vigilant in our pursuit of treatments to halt the progression and provide humane treatment for those with advanced stages of the disease. As veterinarians, it is our duty to eliminate pain and suffering. When there is no possibility in sight for an acceptable quality of life, we should remain professional, be honest with ourselves and the owners, and keep the best interest of the patient in mind.

References and Footnotes

1. Colles DM, Jeffcott LB. Laminitis in the horse. *Vet Rec* 1977;100:262–264.

2. Chapman B, Platt G. Laminitis in the horse, in *Proceedings*. 37th Annual American Association of Equine Practitioners Convention 1984;94.
3. Pollitt CC. Equine laminitis: a revised pathophysiology, in *Proceedings*. 45th Annual American Association of Equine Practitioners Convention 1999;188–192.
4. Pollitt CC. Basement membrane pathology: a feature of acute laminitis. *Equine Vet J* 1996;28:38–46.
5. Hood DM. The mechanisms and consequences of structural failure of the foot. *Vet Clin North Am [Equine Pract]* 1999; 15:437.
6. Weiss DJ. Equine laminitis: a review of recent research. *Equine Pract* 1997;19:16–19.
7. Hood DM. Laminitis in the horse. *Vet Clin North Am* [*Equine Pract*] 1999;15:287–284.
8. Belknap JK, Black SJ. Review of the pathophysiology of the developmental stages of equine laminitis, in *Proceedings*. 51st Annual American Association of Equine Practitioners Convention 2005;383–388.
9. Pollitt CC. Medical therapy of laminitis. In: Ross MW, Dyson SJ, eds. *Diagnosis and management of lameness in the horse*. St. Louis: W.B. Saunders Co., 2003;329–332.
10. Parks AH. Treatment of acute laminitis. *Equine Vet Edu* 2003;15:273–280.
11. Kainer RA. Clinical anatomy of the equine foot. *Vet Clin North Am [Equine Pract]* 1989;5:1–27.
12. Bowker RM, Van Wulfen KK, Springer SE, et al. Functional anatomy of the cartilage of the distal phalanx and digital cushion in the equine foot and a hemodynamic flow hypothesis of energy dissipation. *Am J Vet Res* 1998;59:961–968.
13. Stump JE. Anatomy of the normal equine foot, including microscopic features of the laminar region. *J Am Vet Med Assoc* 1967;151:1588–1598.
14. Ratzlaff MH, Shindell RM, DeBowes RM. Changes in digital venous pressures of horses moving at the walk and trot. *Am J Vet Res* 1985;46:1545–1549.
15. Adair HS III, Goble DO, Schmidhammer JL, et al. Laminar microvascular flow, measured by means of laser Doppler flowmetry, during the prodromal stages of black walnut-induced laminitis in horses. *Am J Vet Res* 2000;61:862–868.
16. Allen D Jr, Korthuis RJ, Clark ES. Capillary permeability to endogenous macromolecules in the equine digit. *Am J Vet Res* 1988;49:1609–1612.
17. Allen D, Clark ES, Moore JN, et al. Evaluation of equine digital Starling forces and hemodynamics during early laminitis. *Am J Vet Res* 1990;51:1930–1934.
18. Allen D Jr, Korthuis RJ, Clark S. Evaluation of Starling forces in the equine digit. *J Appl Physiol* 1988;64:1580–1583.
19. Floyd AE. Grading the laminitic horse. In: *Equine podiatry*. St. Louis: Saunders Elsevier, 2007;320–327.
20. Rucker A, Redden RF, Arthur EG, et al. How to perform the digital venogram, in *Proceedings*. 52nd Annual American Association of Equine Practitioners Convention 2006;526–530.
21. Goetz TE. The treatment of laminitis in horses. *Vet Clin North Am [Equine Pract]* 1989;5:73.
22. Eades SC, Holm AMS, Moore RM. A review of the pathophysiology and treatment of acute laminitis: pathophysiologic and therapeutic implications of endothelin-1, in *Proceedings*. 48th Annual American Association of Equine Practitioners Convention 2002;353–361.
23. Parsons CS, Orsini JA, Krafty R, et al. Risk factors for development of acute laminitis in horses during hospitalization: 73 cases (1997–2004). *J Am Vet Med Assoc* 2007;230:885–889.
24. Hunt RJ. A retrospective evaluation of laminitis in horses. *Equine Vet J* 1993;25:61–64.
25. Van Eps AW, Pollitt CC. Equine laminitis: cryotherapy reduces the severity of the acute lesion. *Equine Vet J* 2004; 36:255–260.
26. Allen D Jr, White NA II, Foerner JF, et al. Surgical management of chronic laminitis in horses: 13 cases (1983–1985). *J Am Vet Med Assoc* 1986;189:1604–1606.
27. Eastman TG, Honnas CM, Hague BA, et al. Deep digital flexor tenotomy as a treatment for chronic laminitis in horses: 35 cases (1988–1997). *J Am Vet Med Assoc* 199;214:517–519.
28. Hunt RJ, Allen D, Baxter GM, et al. Mid-metacarpal deep digital flexor tenotomy in the management of refractory laminitis in horses. *Vet Surg* 1991;20:15–20.
29. McClure S. How to construct and use a transfixation cast, in *Proceedings*. 44th Annual American Association of Equine Practitioners Convention 2006;205.
30. Moyer W, Schumacher J, Schumacher J. Chronic laminitis: considerations for the owner and prevention of misunderstandings, in *Proceedings*. 46th Annual American Association of Equine Practitioners Convention 2000;59–61.
31. Stick JA, Jann HW, Scott EA, et al. Pedal bone rotation as a prognostic sign in laminitis of horses. *J Am Vet Med Assoc* 1982;180:251.
32. Baxter GM. Equine laminitis caused by distal displacement of the distal phalanx: 12 cases (1976–1985). *J Am Vet Med Assoc* 1986;189:326–329.

[a]Phenylbutazone Equi-Phar, Vetco Inc., St. Louis, MO 64507.
[b]Banamine, Schering Plough Animal Health, Union, NJ 07083.
[c]Lidocaine HCl 2%, Butler Animal Health Supply, Dublin, OH 43017.
[d]Polymixin B, Abraxis Pharmaceutical Products, Schaumburg, IL 60173.
[e]Dimethylsulfoxide, Butler Animal Health Supply, Dublin, OH 43017.
[f]Aceproject, Butler Animal Health Supply, Dublin, OH 43017.
[g]Trental Oral Gel, Hagyard Pharmacy, Lexington, KY 40511.
[h]Falkner Equine Walker, Paris, KY 40361.
[i]Styrofoam Pads, Equine Digital Support System, Penrose, CO 81240.
[j]Equine Digital Support System Sole Support Impression Material, Penrose, CO 81240.
[k]Soft Ride Equine Comfort Boots, Soft-Ride, Inc., Vermilion, OH 44089.
[l]Orthopedic Felt, Victor Medical Co., Irvine, CA 92630.
[m]Vetcast Plus, 3M Animal Care, Minneapolis, MN 55415.
[n]Equilox Adhesive System, Equilox International, Pine Island, MN 55963.