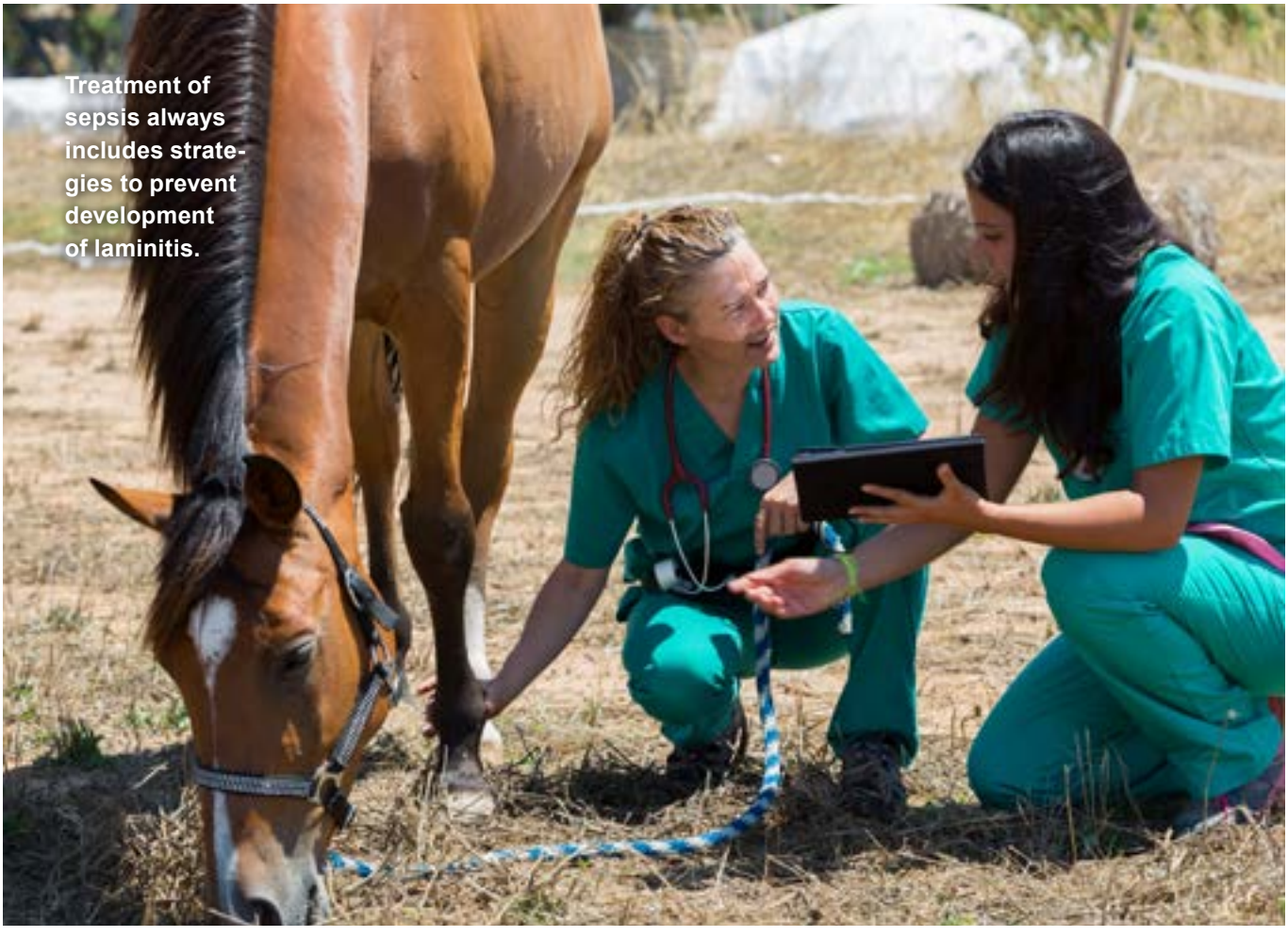

Treatment of sepsis always includes strategies to prevent development of laminitis.

Laminitis and Lungs

Information on laminitis and lung disease from the 2017 AAEP Convention.

By Nancy S. Loving, DVM

Horse owners and veterinarians are concerned about lameness and laminitis from a variety of causes. There also is a heightened awareness of equine respiratory problems across the country. The following articles are summarized from presentations given at the 2017 AAEP Convention.

(Editor's note: This article is one of four from the 2017 AAEP Convention brought to you by Boehringer Ingelheim Animal Health*.)*

Sepsis-Associated Laminitis

In the horse, systemic inflammatory illnesses—such as colitis, enteritis, metritis, carbohydrate overload and pneumonia—are often associated with bacterial infection and/or bacterial byproducts, such as endotoxin. These conditions

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lead to multiple organ dysfunction syndrome (MODS) that includes adverse effects on equine hooves. Treatment of sepsis always includes strategies to prevent development of laminitis.

There remains a limited understanding of the mechanisms that cause laminitis in a septic individual. In general, there is a loss of integrity of the suspensory apparatus in the foot, separation of the basement membrane, and crushing of solar tissue. Progression depends on the initial damage. In less severe cases, tissue reorganization can occur, but lamellar changes are irreversible. An epidermal scar called a lamellar wedge is often visible on radiographic examination.

Prevention relies on identification of a horse that is at risk for sepsis-related laminitis. Andrew van Eps, BVSc, PhD, MACVSC, DACVIM, of the University of Pennsylvania, discussed strategies to prevent sepsis-associated laminitis. He described the use of intravenous fluids, non-steroidal anti-inflammatory drugs (flunixin meglumine or pentoxyfylline), and endotoxin-binding substances such as polymixin B and hyper-immune plasma.

One extremely effective strategy to protect hooves against laminitis is the use of digital hypothermia. If done correctly, this can reduce the likelihood of laminitis by 10-fold. Cryotherapy inhibits inflammatory mediators such as cytokines, cyclooxygenase (COX-1 and COX-2), and migration of white blood cells into the tissues. Hypothermia also inhibits expression of matrix metalloproteinase (MMP) that could otherwise contribute to laminar separation. A further protective effect of cooling results from reducing metabolic demands of the tissue and mitochondrial function.

To achieve an appropriate preventive and therapeutic effect, Van Eps noted that it is necessary to apply continuous distal limb hypothermia, preferably to all four feet, until clinical signs and laboratory findings (white blood cell counts, serum amyloid A testing) suggest that endotoxemia and sepsis are resolved. The objective is to cool the hoof wall surface temperature below 10 degrees Celsius (50 degrees Fahrenheit). Application of cryotherapy from mid-cannon to include the bottom of the foot cools incoming arterial blood and keeps the hoof from being impacted by environmental temperature. An ice and water bath mixture improves the cooling process. When ice is applied without a water interface, there is a risk of frostbite.

In addition to cryotherapy, it is important to restrict activity. He recommended keeping the horse confined for one week for every day of lameness. Sedatives or tranquilizers might help to keep a horse calm and quiet. Solar support can be accomplished with the use of sand bedding spread deeply in the stall and/or application of solar support materials to the foot. The stall should also be bedded deeply with shavings to encourage a horse to lie down and relieve pressure on the feet.

Frequent radiographic monitoring enables on-going assessment and evaluation of progress and healing.

Supporting Limb Laminitis

Continuing on at the 2017 AAEP Convention with the in-depth discussion of laminitis, Van Eps discussed supporting limb laminitis. He stated that these are severe cases, usually resulting in at least 50% mortality. Some horses might be subclinical; some are affected in more than one limb. Increased risk factors for developing supporting limb laminitis include high body weight and duration of musculoskeletal repair that necessitates bearing more than normal weight on the opposite limb.

While it might seem like this form of laminitis is mechanically induced, Van Eps noted that it might have more to do with perfusion issues and energy failure. He said, "Lamellar tissue requires glucose to maintain the integrity of cellular attachments, yet there is no means for local glycogen storage." Reduction in blood supply (oxygen and glucose availability) then can lead to energy deficits in the tissue. Additionally, signaling of bone injury and inflammatory mediators to epidermal tissues might play a role in development of supporting limb laminitis.

Radiographic evaluation of horses with supporting limb laminitis demonstrated an interruption of contrast in arteries with weight bearing, indicative of poor perfusion of oxygen and glucose. The only thing that can increase lamellar blood flow is walking and/or increasing changes in weight cycling between limbs. Van Eps noted that if weight is borne consistently on one leg, that within 48 hours there are indications of ischemia. Oxygen reductions are evidenced by an increased ratio of lactate to pyruvate. Oxygen is decreased in the lamellar zone only, not globally in the foot. Ischemia and decreased limb load cycles play major roles in development of supporting limb laminitis.

In a study where nerve blocks were used to keep a horse more comfortable, heart rates were similar to control horses. The horses that received nerve blocks distributed about 10% less weight on the supporting limb than the controls.

Preventive strategies rely on enabling a horse to cycle every minute from one limb to the other through pain management and walking exercise, where possible. It is important to fully unload the foot rather than the standing horse just changing limb position. Pedometers or fitness tracking devices equipped with accelerometers could track limb cycling efforts by a horse.

Other treatments include use of non-steroidal anti-inflammatory drugs, physiotherapy, and/or solar support,

such as provided by Soft Ride boots. Partial or dynamic sling support can reduce 10% of body weight load on the feet.

Cryotherapy decreases energy demands of the hoof tissues—skin temperature must drop to <7 degrees Celsius (44.6 degrees Fahrenheit) to achieve a change in a horse's pain threshold. Sedatives might be counterproductive to limb load cycling, so Van Eps suggested it was better to use perineural catheter management or a high-volume epidural with diluted opioids. In extreme cases, forced recumbency might be necessary.


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Cryotherapy decreases energy demands of the hoof tissues and can change the horse's pain threshold.

Distinguishing Equine Asthma from Infectious Lung Disease

(Editor's note: This research report was presented during the Kester News Hour during the 2017 AAEP Convention.)

With increasing emphasis on restricting use of antimicrobial therapy to only those patients in need, a stall-side test that can help differentiate non-infectious from infectious causes is useful.

Serum amyloid A (SAA) testing seems to be able to do just that (Viner, M.; Mazan, M.; Bedenice, D.; et al. Comparison of Serum Amyloid A in Horses With Infectious and Noninfectious Respiratory Diseases. *J Equine Vet Sci* 2017;49:11-13).

The study of 207 horses identified that inflammatory airway disease cases had an SAA of <50 ug/ml, cases infected with virus or bacteria had SAA >50 ug/ml, while healthy horses had SAA of zero ug/ml. In general, there is a 10% chance of the testing yielding wrong results.

The investigators concluded that there is a threshold for SAA of 50 ug/ml, above which a practitioner should consider an etiology of infectious disease. The test does not distinguish viral from bacterial, but it does differentiate between infectious versus non-infectious pathology. EM