Equine Laminitis: Part 1: What is it?

Laminitis (a/k/a “founder”) is a disease of the feet common in equines – horses, donkeys, and mules. This disease is one of the most heartbreaking and costly to the equine industry. It can cause severe and debilitating lameness and may necessitate euthanasia. It can seemingly come out of nowhere, or can follow as a sequel to other serious diseases that have been successfully treated and resolved.

The mechanisms of laminitis have been studied intensively for many years but there is still much that is not understood. Recent findings have contributed more to our understanding of this disease process. The purpose of this first article is to define laminitis and discus current ideas on how it happens. The second part discusses some mainstream approaches and new ideas with respect to treatment and prevention.

SIGNS OF LAMINITIS

Laminitis typically results in very severe lameness that is usually worse in the forelimbs than hind limbs. Anyone who has witnessed a horse with laminitis is unlikely to forget it. In an effort to avoid bearing weight on the very painful front feet, horses show a characteristic stance with the hind limbs pulled far under the body and the front feet far out in front. They may even lie down to get off the feet entirely. Less painful laminitis often results in a horse that looks like he or she is “walking on eggshells.”

EQUINE FOOT STRUCTURE & LAMINITIS

While other grazing animals may get laminitis, none show the dramatic signs that equines do. In order to understand the mechanism of laminitis, it is important to understand the basics of equine foot structure. Equines are the only family of mammals that have evolved to walk on the tip of one finger

(digit). The equine foot is a miracle of engineering. The entire weight of the horse is borne by the coffin bone, which is suspended above the sole of the foot by its attachments to the outer hoof wall. This unique attaching layer is called the “laminar layer.”

The key to understanding laminitis is to understand that a mechanism exists in the foot that allows the coffin bone to be securely suspended by the hoof wall, but also allows the hoof wall to slide over the bone as it grows. The coffin bone (pedal bone, or P3) is attached to the outer hoof wall by the laminar layer. You can see the laminar layer on the sole of a newly trimmed foot as the “white line,” just inside the outer hoof wall. Microscopically, the white line is made up of two layers of laminae, one that is attached to the outer hoof wall and one attached to tissues firmly glued to the underlying coffin bone. The attachment between these two laminar layers contains an active process involving thousands of interconnected and branching microscopic fingers of live tissue between the coffin bone and outer hoof wall. The millions of live cells making up these fingers of attachment are under precise control by signals from the body and local signals from the foot tissues.

I like to use Velcro as a model to illustrate the laminar layers. Imagine the coffin bone glued to the soft Velcro layer. Now imagine the outer hoof wall glued to the rough Velcro layer. The Velcro surfaces come together at the white line. Now imagine that there is precise control of this attachment which allows limited sliding of the hoof wall over the coffin bone. Tiny releases and reattachments constantly occur to allow the hoof wall to grow down from the coronet band (the hairline of the hoof).

Protruding into and between these laminar layers is a fine web of tiny blood vessels that bring oxygen and other essential nutrients to the cells. At any one time, the cells making up these fingers are mainly adhered to one another. Precise

control of the laminar cells allows movement of the hoof wall along the coffin bone while never completely releasing it and thus never allowing it to alter its position within the foot.

THE MECHANISM OF LAMINITIS – "LAMINITIS TRIGGERS"

The word laminitis means inflammation of the laminae. It is a disease that involves dysfunction of this unique system of attachment discussed above. It can progress to allow complete breakdown of the structure of the foot.

The old mechanism for laminitis that I learned as a veterinary student involves blockage of the circulation to the laminae, leading to cell injury and stretching of the laminar attachments. New research has shown that certain biochemical "laminitis triggers" can cause over-activation of this release mechanism, allowing the coffin bone freedom to move within the hoof. Horses undergoing this "over-release" show the signs of laminitis.

To what degree the coffin bone moves is dependent on many factors but, in general, the more movement of the coffin bone, the more severe the damage, and the less likely the horse is to make a complete recovery. Understanding the new concept of laminitis triggers is important. Any substance that can function as a trigger can cause the mechanism of laminitis. The blockage of blood supply to the laminae is likely a later contributor, but the main event in initiating laminitis is this over-release of the laminae caused by the laminitis trigger.

Where do these laminitis triggers come from? Classically, founder has been thought of as a disease resulting from grain overload. Unlike cattle, which ferment feed in a rumen (or foregut), horses are known as hindgut fermenters. This is because the large specialized hindgut, or large colon is the place where the tough structural components of grass are broken down by the action of bacteria. Ordinarily, simple sugars and starch are absorbed upstream of this by the small intestine before many reach the colon. An overload of this

system with sugar or starch from a sudden load of grain or other high starch feed allows increased starch or simple sugar to reach the colon. This can result in a die-off of the normal bacteria here and a shift to new types and numbers of bacteria. Some of these new bacterial types may produce substances that act as triggers, and result in the signs of laminitis.

However, there are many other laminitis triggers. Bacterial toxins coming from retained placenta, abdominal infection, or from intestine damaged by a colic episode, also can be triggers. There are specific toxins found in nature that are known to be direct triggers, such as Black Walnut wood shavings.

PREDISPOSING CAUSES

Anything that makes the laminar cells more susceptible to a trigger will increase the chances of a horse developing laminitis. Genetics plays a role in the sensitivity of a given horse to laminitis triggers. Some breeds are more sensitive than others. Endocrine disorders, hormonal problems such as Equine Cushing’s disease (also known as PPID) or Equine Metabolic Syndrome (EMS), can predispose horses to laminitis by increasing sensitivity to triggers. A horse that has had prior laminitis episodes is more sensitive to recurrence as well.

For a variety of reasons, horses that are abnormally fat are more predisposed to developing laminitis than a horse in normal body condition. Obesity may cause specific problems with the circulation in the foot, interfering with delivery of oxygen and nutrients to the live cells of the laminae. Because of this, these laminar cells may be more sensitive to the action of a trigger.

Reaction to any trigger causes the same end result: Either over-activation of the releasing mechanism, or damage to the live cells themselves. Either alone or together, these two factors allow movement of the coffin bone within the hoof.

Once the coffin bone rotates or moves within the hoof, the live layers of the sole may be crushed and the blood supply to the laminae damaged. This further contributes to a cycle of destructive events in the hoof, which ultimately can break down the entire structure.

In Part 2 of this article, I discuss ways to prevent and treat laminitis in its acute and chronic forms. I pay special attention to some new research regarding feeding and grazing management to avoid this serious disease.

By Douglas O. Thal DVM Dipl. ABVPBoard Certified in Equine PracticeThal Equine LLCLast Updated August 2011