PROCEEDINGS
NO LAMINITIS! 2017 CONFERENCE • TUCSON, AZ
Equine Cushing's and Insulin Resistance Group

The Coffin Bone: How Does Adaptation Impact the Foot During Laminitis?

Robert M. Bowker, VMD, PhD

Professor Emeritus of Anatomy, College of Veterinary Medicine, Michigan State University, East Lansing, Michigan, USA.

The suspensory apparatus, adaptation to load forces, and a porous coffin bone all play a role in how well an equine individual will survive acute or chronic laminitis.[1] We believe we can develop an ability to recognize early signs of bone and foot deterioration, before pathologies occur. By developing this skill, we can then intervene to minimize the devastating effects of laminitis, or even prevent it.

IS THE EQUINE FOOT UNIQUE?

The theoretical belief that the equine hoof wall is the main support structure has long been the cornerstone of the notion that the horse's biomechanical support system is unique in the animal population — the notion that the hoof wall is the primary load support results in the actual "suspension" of the coffin inside the hoof wall.

Most mammals and marsupials, however, walk on the ventral surface of their foot — the solar pad. Ruminants, including dairy cows weighing 2,000 to 2,500 pounds, walk on a solar pad rather than the hoof wall.

Experiments have never been conducted to verify the exact amount of load the equine hoof wall should take. I believe the hoof wall is primarily a "decoration" and should not function as a primary loading apparatus. We believe that this "decoration" should only support a small percentage of the load being placed upon the foot, somewhere between 5 and 20%.

In real life, the solar surface of the foot is usually filled with dirt or the contents of the ground surface of the barnyard; this dirt plug is the interface between the bony column and the weight bearing structures. With my herd I can take a thin ruler and pass it around the entire hoof wall, as little to no weight is being placed on the walls, even in my two Belgians.

Figure 1 displays a tragic example showing why the hoof wall is not designed to be the primary loading structure.

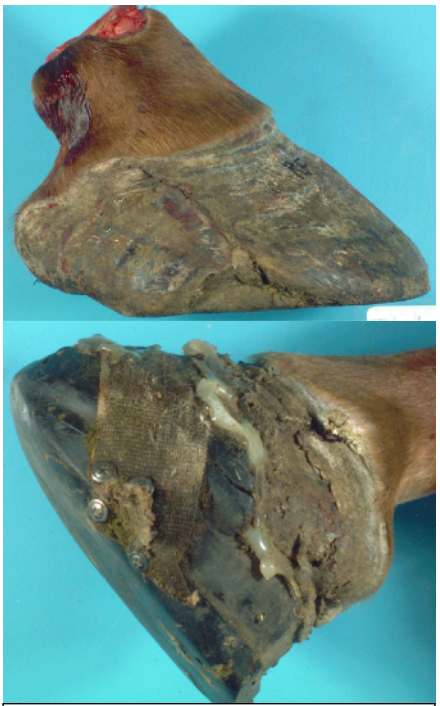

Figure 1. Double-loaded hoof wall split from ground to coronet band (top). Repair device continued to load the hoof wall (bottom).

During a routine appointment, the right shoe was removed from a 1,400 - 1,500 pound, 11-year-old quarter horse. When the opposite or left foot was elevated to remove the shoe, the right foot and hoof wall was supporting virtually all of the weight on its foot. During this "double loading of the wall" period of the right foot, the hoof wall split from the ground to the coronet band. The foot was repaired with a device that continued to put all of the load on this now severely compromised hoof wall. The compromised wall

could not support the weight. We received this foot as a specimen at the lab as a result. (Figure 1)

When the hoof wall grows beyond the sole, it becomes to a varying degree, the primary loading structure. In addition to the example above, we have all seen examples of walls on a harder surface that start to break off. This is a hint that the wall is not designed to primarily bear most of the weight of the horse.

Smaller horses and ponies can get away with such loading, as the hoof wall tissues are similar in both small and large horses, so there will be minimal weight on the extended wall of a small horse. However, if a “large rider” is placed on the back of the small horse the result might well be like that seen in the above picture. It is basically biomechanics and laws of physics: tissues cannot support the loads if the weight exceeds the tissue’s physical properties.

SUSPENSORY APPARATUS OF THE DISTAL PHALANX (SAPD)

The Suspensory Apparatus of the Distal Phalanx (SADP) has long been viewed as crucial to maintain a healthy foot. What makes a healthy suspensory apparatus?

per·i·os·te·um
ˌperēˈästēəm/
noun Anatomy
plural noun: periostea
- a dense layer of vascular connective tissue enveloping the bones except at the surfaces of the joints.
https://www.merriam-webster.com/dictionary/periosteum

In current and historical literature, drawings of the suspensory apparatus illustrate the common belief that the hoof wall “suspends” the coffin bone through attachments from the Primary Epidermal Laminae (PEL). The pathological condition of a penetrating laminitis supports this notion, as during a severe bout of laminitis, the toe rotates, sinks, or penetrates the bottom of foot.

I agree that, when the hoof wall is loaded, it does suspend the foot — but, should it? Ruminants even heavier than equines walk on their pads. What is going on? When you load the equine hoof wall you suspend the coffin bone inside the hoof capsule. When you do not load the hoof wall — when the equine foot is loaded primarily through the sole — we have seen that there is no suspensory apparatus.[2]

THE ROLE OF THE PERIOSTEUM

Many people have stated that the horse is unique because the coffin bone does not have a periosteum. This is where the unique argument of the “equine suspension” system being a crucial and healthy component in foot support, starts to break down.

When load is preferentially through the solar tissues the periosteum is present. (Figure 2) When the load is primarily through the wall, the periosteum adapts and realigns to suspend the horse via the coffin bone.

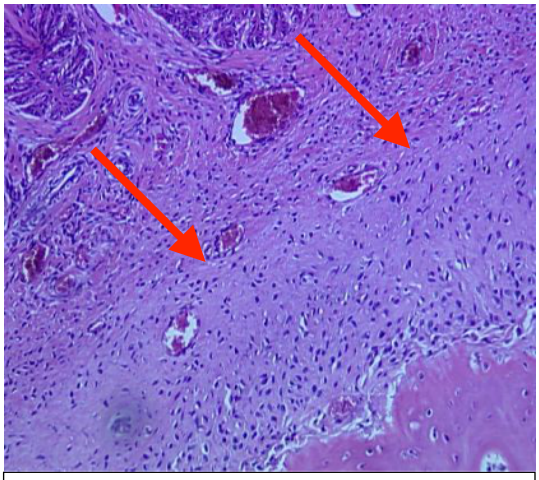

Figure 2. Periosteum connective tissue can be seen between red arrows. Normal covering for bone consists of a thin layer called periosteum, which is 4-5 proteins bound together. The solar side of the foot also has a periosteum. Nuclei — small blue dots — tend to be oriented perpendicularly to the periosteum collagen fibers.

In our work on the horse foot and how it functions, we have shown the lack of a periosteum to not be consistently true. Like all other bones, when viewed histologically, the coffin bone from young or older, primarily barefoot horses, does have a periosteum. Composed of 4 - 5 proteins, the periosteum is like a plastic wrap with collagen fibers being aligned to hug the bone.

When we start to load the foot through the hoof wall the periosteum

changes. Energy comes up the hoof wall, through the lamina, and goes into the bone itself. Instead of aligning parallel to the bone, the collagen fibers that make up the periosteum then begin to orient perpendicular to the bone.

Periosteum starts to reorient to make connections between the hoof wall and the coffin bone as well as the laminae. They start to reach out — away from the bone — to grab on to the tips of the laminae, in order to stop, or at the very least, reduce the movement between the hoof wall and the bone.

The equine foot is a “smart” structure. The periosteum is there. All coffin bones have periosteum until the interior of the foot starts to adapt to loading forces of the hoof wall and it can no longer be seen.

Figure 3. Is the SADP structure and the porous coffin bone consistent with a healthy foot? If true, then why is it potentially or unhealthy in humanoids?

THE POROUS COFFIN BONE

It is believed that, under normal conditions, the coffin bone is very porous with many small pores or holes located in the dorsal cortical bone. (Figure 3) It is depicted as normal because it is seen all the time. It is sometimes described as a special feature of the horse, to give them a better ability to run and jump. A similar finding in the legs, feet, and back of women, however, is viewed to be unhealthy. We call it osteoporosis, a pathological condition as the bones are unable to support the weight of the woman. This term — osteoporosis — in women is based upon the studies of millions of women, with the age of a 29-year-old being the baseline. The bones of all other women are based on this age group: all other bone densities are relative to this group of young women. In horses there has been little work examining this question.

Figure 4. Coffin bones in populations of presumed healthy horses have few pores on their surfaces. Are these bone less healthy than those with porous consistency?

What is the “normal” biology of the distal phalanx in the horse’s foot? Are both notions of unhealthy osteoporosis in women, yet healthy porous coffin bones in equines, possible and correct?

Is the suspensory apparatus of the distal phalanx (SADP) structure and the porous coffin bone consistent with a healthy foot? If that is true, then why is it potentially bad or unhealthy in humanoids? Why do cadaver feet from horses, primarily supported through the sole, not present this way?

In hundreds of cadavers that we have examined, many bones are “normally” dense with few connections or vascular channels between the coffin bone and the hoof wall. (Figure 4) They have few pores on their surfaces, except for the larger vascular channels they are born with. The periosteum lays against the coffin bone. The bone over all is dense and the distal edge is also dense and round. In

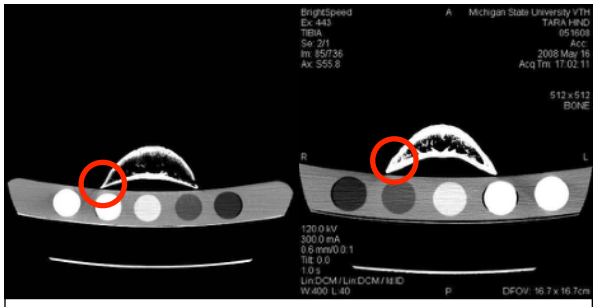

Figure 5. CT scans bones of same width. Density of bone will vary depending on how they were loaded. A peripherally loaded foot will be less dense, while a solarly loaded foot is dense and symmetrical.

these horses 100% of their weight has not been on their hoof wall, but varying amounts have been placed upon the sole.

The MRI in Figure 5 shows that in barefoot horses, solar loading promises increase in bone deposition with no, or less, loss of bone.

BLOOD VESSELS

Coffin bone holes have been described as important to supply vessels for blood supply[3] to the interior of the hoof wall — the laminae.[4] The inner wall laminae are believed to have a high metabolism — higher than the horse's brain.

In many cadaver feet, more blood vessels are seen and are considered normal. However, most tissues of the foot do not require high levels of nutrition and oxygen. Cartilage has no blood supply to it. The digital cushion has very little required in the way of energy. Tendon and bone do not function at a high metabolic rate.

Vessels for energy and blood would be more uniform around the entire surface. The pores are different between the medial and lateral side of the bone due in part to the landing and the disparity of movements between the hoof wall and the coffin bone and the two sides of the foot. The foot is "looking" for less movement between the dermis and the coffin bone.

To illustrate, when bones are broken and reset, the two bones of the fracture will set well together as good strong bone is created. If movement is significant between the two bones, then the fracture site is infiltrated by blood vessels, and fibrocartilage predominates within the callus as opposed to bone. Little to no calcium is laid down to repair the bone. This result is caused by the movement between the two pieces of bone.[5]

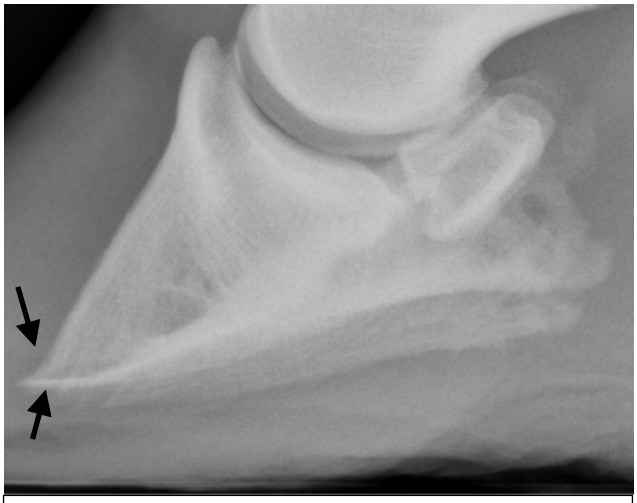

Figure 6. Bone changes seen radiographically with thin cortex dorsally and in sole.

RADIOGRAPHS

Earlier recognition of pathology can help us help the horse.

An early indication of bone loss can be seen when radiographs start to get fuzzy along the edges of the bones within the foot. Calcium in a healthy bone stops the radiographs ray from penetrating. Lack of calcium creates dark spots, making the bone look less opaque, with a fuzzy appearance at the edges. (Figure 6)

When viewing a radiograph, focus your eye and go all around the surfaces looking for fuzzy areas. These are easily seen once your eye is trained. When the radiograph starts to look like "cotton candy", there is something going on: it may be early stages, there may be no clinical signs, but it is time to make husbandry decisions.

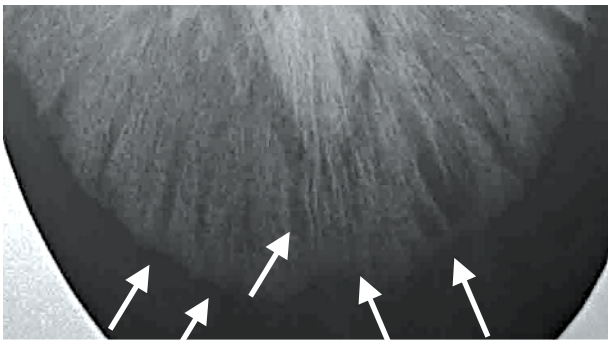

Figure 7. The coffin bone edge is beginning to get "moth-eaten", with enlargements of the vascular channel. Notice the pores are also evident.

Actual bone will look worse than the radiograph suggests. This is early pathology. Thinness at the distal edge of the coffin

bone is due to the peripheral loading; porousness is due to the foot trying to stop the movement. (Figure 7)

As fuzzy readings increase, the dorsal surface contains less and less bone and cannot support the horse if laminae connections are lost. Peripheral loading has changed the bone distribution and density. Horses with this type of bone will have a much harder time withstanding a laminitic event.

True “slipper toe” happens much earlier, beginning as in the photo above. What is often described as a slipper toe is really a fracture of the dorsal cortex and solar bone at P3. The distal end of P3 is fractured with one-quarter to one-third of the coffin bone crushed as a result. When bone breaks off, very painful pedal osteitis develops along the edge of peripheral bone.

Fracture does not happen when the coffin bone is dense and the foot is supported through the sole.

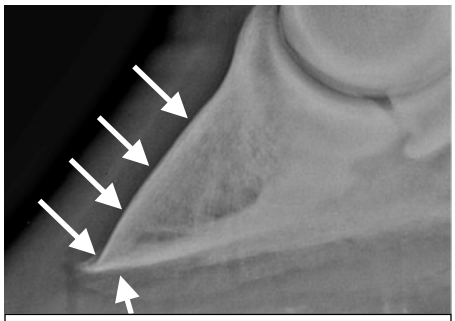

Figure 8. A bulge in the dorsal cortex, fuzzy edges of bone and remodeling on the dorsal edge are evidences of bone deterioration.

HOW RECOGNITION WILL HELP IN PREVENTION

If thinning cortex and trabecular are recognized as early pathology, steps can be taken to avoid crushed bone in laminitis. One can see the bulge in the dorsal cortex of P3. It should be smooth. The fuzzy appearance at the tip of P3 is also indicative of deteriorating bone. Addressing this now through trimming to change the load will help this horse return to a more dense P3. (Figure 8)

Calcium in human bones is replaced every five years and this physiological aspect of the horse is likely the same. Slipper toe will disappear and the toe will come back in most individuals if you load the foot primarily through the sole.

Current literature describes a “sinker” as a 15 - 18 mm difference between the coronet and extensor process. If displacement is recognized earlier, adjustments can be made to minimize the damage from a laminitis event. We believe that at 12 - 13 mm, trim issues should be addressed. If you wait for a 15 - 18 mm difference, events have already happened. The horse is lame. Waiting gives you the Quarter Horse referenced above. Waiting does nothing for the horse.

Use plastic disposable rulers to measure everything you can, every time you view radiographs. The more you measure the more you will see. Measure from coffin bone to exterior hoof wall both proximally and distally and you may see the distances are not equal. Measure the space between the coronet and extensor process; the more you measure the more you will pick up subtle differences and become able to recognize issues before they become issues.

REFERENCES

1. Orsini JA, Parsons CS, Capewell L, Smith G. *Prognostic indicators of poor outcome in horses with laminitis at a tertiary care hospital.* https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2871359/

2. Bowker RM. *The Vascular Cushion of the Frog: Avoiding Consequences of Laminitis through an Understanding of Fascia, Microvessels, and Dissipation of Energy,* ECIR Group Inc., 2017 NO Laminitis! Conference, Tucson, AZ, USA

3. Pollitt CC. *Clinical anatomy and physiology of the normal equine foot,* Equine Veterinary Education, 1992. https://doi.org/10.1111/j.2042-3292.1992.tb01623.x

4. Ibid.

5. Augat P, Burger J, Schorlemmer S, Henke T, Peraus M, Claes L. *Shear movement at the fracture site delays healing in a diaphyseal fracture model,* Journal of Orthopaedic Research 21 (2003) 101 1 1017.

This research was made possible by the generous support of the American Quarter Horse Association and the United States Equestrian Federation.

All photos Robert Bowker files.