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

The Vascular Cushion of the Frog: Avoiding Consequences of Laminitis through an Understanding of Fascia, Microvessels, and Dissipation of Energy

Robert M. Bowker, VMD, PhD

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

INTRODUCTION

Perhaps the most under-appreciated area of the equine foot is the frog. Our understanding of frog function affects how we view the importance of its function and our resulting husbandry practices.

As previously discussed in, **"Understanding Laminitis: How We View 'Normal' Function"**, we have found long-term effects of peripheral loading of the foot to include the following:

1. The foot becomes *asymmetrical.* Steep versus flared sides are accepted as normal in greater than 90% of equine feet. Asymmetry affects the hoof wall, P3, and P2 .
2. *Bone is lost* from distal parts of the coffin bone, creating the potential for pedal osteitis, crushed toes in laminitis, and an increase in biomechanical issues.
3. The *long toe* alters the vasculature within the solar dermis, decreases energy dissipation and support, creating biomechanical issues involving the deep digital flexor tendon (DDFT), decline in frog, tendon and ligament health, as well as issues further up the leg.
4. *Vascular perfusion* is altered, creating extreme detriment to foot function, i.e., energy dissipation and support.

ENERGY DISSIPATION

When the foot hits the ground it is often as forceful as a strike with a hammer. When the hoof wall is loaded, in a shod foot or in a barefoot horse on a hard surface, energy dissipation becomes compromised as the vasculature and connective tissue are damaged.

Gradual deterioration of foot tissues is due mainly to our husbandry practices. It used to be that a shod horse was only shod for a portion of the year and then allowed to "rest" when not in training; but now they are often shod year-round due to the schedules of the various industries. An unshod horse only means that the horse does not have shoes on! The owner must pay attention and examine the foot on a daily basis if the foot is to remain healthy, and relay any concerns to the trimmer. Having horses on hard surfaces for housing and work or training results in high-impact energies passing through primarily the hoof wall, instead of passing through the solar surfaces of the foot. All tissues within the foot will be affected. Energies are not restricted to just the foot, however, and with time, their effects radiate up leg.

Research from Europe has shown that when shoes are applied to the foot, vibration frequencies are created that can exceed a frequency of 2,000 to 3,000 Hz.[1] When barefooted horses move on these same hard surfaces the frequencies are less but still they are high. These vibrations cause the vessels to constrict over long periods of time, which can have additional, deleterious effects on tissues within the foot. The connective tissues begin to become deficient in the supply of oxygen and nutrients needed to sustain and support surrounding connective tissues.[2]

Consistent with this idea is in human research in the study of carpal tunnel syndrome and in construction workers using jack hammers.[3,4] In the rat model of this research, when a rat tail with its main artery is subjected to 15 seconds of vibration at 250 - 400 Hz, the arterial vessels are directly affected by the vibrational energy:

they constrict and they remain constricted for three days via the vibrational actions on the receptors on the vessels.[5]

When the vibrational energy due to impact is not reduced, it starts to affect blood flow and to produce degeneration and pathology within the foot tissues. In Sweden, construction workers are now permitted by law to use a jack hammer for only 15 minutes per day, which is down from 30 minutes per day a few years ago.

VIBRATION, NAVICULAR SYNDROME AND UNDERSTANDING LAMINITIS

For nearly 200 years, observed pathology in navicular syndrome horses at necropsy has been associated with the navicular bone (NB) and deep digital flexor tendon (DDFT).[6]

While several different hypotheses have been proposed for navicular syndrome, the main one is: Biomechanical concussive loading forces via the pressure of the deep digital flexor tendon (DDFT) against the navicular bone (NB) causing cartilage degeneration and eventual tendon and bone damage of the navicular apparatus. Amazingly, after all this time, while the diagnosis of navicular has improved as a result of technology, the treatment for navicular has not changed significantly in 200 years. The most recommended treatments continue to be a padded shoe with raised heels. When this does not work, heroic procedures are introduced such as Navicular Suspensory Desmotomy, Stem Cell and Platelet-Rich Plasma therapy (PRP), core decompression[7] to stimulate bone growth, and drug therapies such as Tildren and OsPhos, injectable biophosphonate solutions.

We believe that by appreciating the functions of the interior of the foot on both gross anatomical and histological levels, significant improvement can be made for the horse without traditional navicular treatment or other heroics. By recognizing early changes, such as actual pathology in other areas within the foot, we can positively affect the long-term outcome for these horses and avoid the too-common consequences of not only navicular syndrome, but also laminitis.

With peripheral loading supporting the hoof wall and heel bulbs, vibration radiates through the foot and the forelimb of the horse. With the load primarily on the hoof wall, the inside of the foot changes in a negative way. Gradual deterioration of foot tissues are unrecognized before clinical signs are evident. In cadavers of young horses, we have observed that the lateral cartilages, the impar ligament, the distal annular ligament (DAL), the DDFT, the frog, and coffin bone density are affected negatively. Our research continues to find that negative changes are not just confined to the foot but, with time, affect tissues further up the leg.

These same tissues are similarly affected in horses with peripheral loading of the foot, i.e., deterioration of the foot's support structures. These deteriorating changes begin to set the horse up for potentially serious consequences — even subclinical laminitis — as the coffin bone has become osteoporotic, especially in the distal third of the bone. Thus when laminitis begins to develop, the coffin bone can no longer support the weight of the horse, often resulting in the toe of the coffin bone being crushed: these changes can be seen, before the actual acute phase when clinical signs appear and diagnosis is usually made.

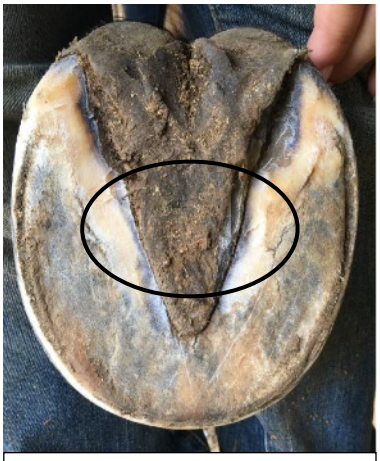

Figure 1. The cuneate frog and open central sulcus.

SIGNIFICANCE OF THE CUNEATE FROG AND THE CENTRAL SULCUS

Cuneate in anatomy jargon means wedge-shaped. Here we are describing the *cuneate frog* as the wedge-shaped portion that should be evident in the forward one-half of the frog in front of the central sulcus. (Figure 1) To function properly it should be large and obvious. Frogs today are regularly trimmed which

discourages the cuneate frog from developing the ligaments and vasculature necessary to support the horse and dissipate energy the way the horse's foot was meant to do.

Some literature speaks about fat in the frog; however, a healthy frog should have very little fat or at least not a lot of fat in it. What is thought of as "fat" should really be myxoid tissue, which encourages the formation of connective tissue of ligaments and fascia as well as promoting angiogenesis, the formation of blood vessels. Allowing the frog to develop a diamond-shaped central sulcus and full cuneate frog helps the frog do its job.

The central sulcus should be open and diamond-shaped in most horses. The exception to this idea are those horses moving up and down a mountainous terrain, when they utilize their heels more. When landing or standing it should just kiss the ground and not be loaded heavily. An open, diamond-shaped, correctly placed sulcus is derived from the toe and hoof wall trimming approach, bringing the toe back, unloading the walls, and bringing the heels back to correct position under the boney column. In frogs that are not working correctly, the central sulcus is closed and deepened and oriented differently from that of the healthy cuneate frog.

In general, the frog does not like to have a lot of weight or constant pressure on it. When too much frog pressure is applied to the frog, we see it become bruised and it usually will shrink in size as it disappears. With a long toe, underrun heels, and loaded hoof wall, the central sulcus closes and migrates to the rear of the foot. The frog stay is then closer to the pastern bones and it will not function as it should in supporting the foot.

HOW THE FROG SHOULD FUNCTION

Erich Blechschmidt (1904 – 1992) was a German anatomist who described the human heel pad in 1933, with chambers present between the calcaneus and skin for dissipation of energy.[8] Between the heel and the skin, these chambers have walls formed by the connective tissue ligaments that contain fluid inside. Interestingly, the walls are broken down in plantar fasciitis.[9]

In the 1930s, Schummer, et al.,[10] described chondropulvinale ligaments (CPLs) between the lateral cartilage and the tissues under the coffin bone. They believed that these ligaments may function as a hammock underneath the navicular bone and the DDFT, to support the bony column during movements. These ligaments were just described as ligaments with no findings of the massive microvasculature present there.

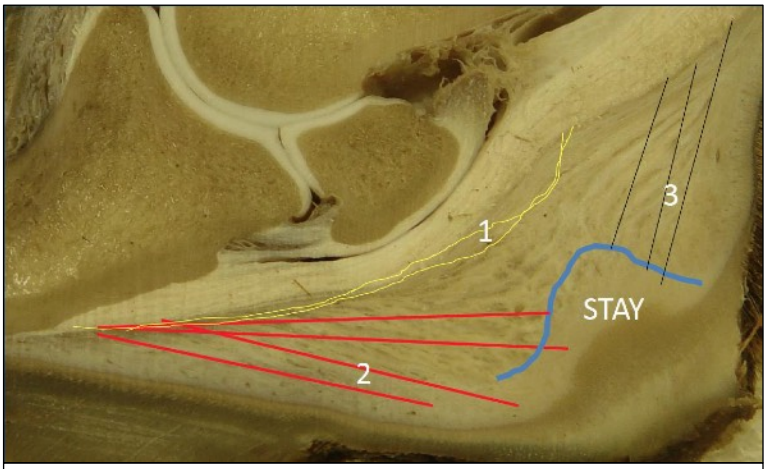

Figure 2. Three compartments in the caudal foot, each with different functions, creating support and dissipating energy.

STRUCTURALLY DIFFERENT FROG COMPARTMENTS SUGGEST DIFFERENT FUNCTIONS

Our laboratory has further identified in the horse the enormous microvasculature contained between the cuneate frog ligaments in the cuneate frog; these ligaments become organized into forming three compartments in the caudal foot. Each compartment has different functions in creating support and negative pressures for energy dissipation. (Figure 2)

Our descriptions of these and other ligaments create a unique structure or compartment, which indicates that they have a highly critical and functional organization within the horse's foot. For example, the distal annular

ligament (DAL) represents a major ligament within compartment 1, but is not part of the CPLs. We have found that DAL connections are different from the earlier descriptions referenced above.[11]

In early descriptions, compartment 2 was not recognized by the Germans. The ligaments were likely destroyed in many horses seen with foot problems when examined in necropsy. Furthermore, they never examined the frog tissues for any vessels. We have seen that when viewed anatomically, as well as through MRI technology, most of the microvessels are present here. This makes compartment 2 the most critical.

Compartment 3 in the caudal aspect of the foot, includes the frog and portions of the digital cushion in the rear of the foot. We have further identified the frog stay as a critical component in good foot function when it works in association with this caudal compartment. Small ligaments, including some condrocompedoungulare (CPU), originate from the frog stay and pass up the pastern and attach at various and multiple levels to the fetlock. When the central sulcus is on the ground, i.e., just kissing the ground upon impact, these ligaments all become tight/taut, which creates the negative pressure needed to dissipate energy from the foot fall.[12] These small and short ligaments are very numerous but also variable between animals. Depending on husbandry practices and the movements made by the horse, they will form but can also can be damaged and destroyed. As a result, the anatomical structure will be variable and different in each horse.

THE MICROVASCULAR NETWORK

Several studies have focused upon the circumflex artery as one of the main arterial blood supplies to the hoof wall.[13] This artery courses around the perimeter of the coffin bone with numerous smaller branches supplying the dermis between the epidermal laminae. However, there are other branches from the circumflex artery coursing under the coffin bone towards the center of the foot, which in turn contribute to the cuneate frog and dermis underneath the coffin bone. Thus this vessel represents only one of many arteries that provide the major avenue of blood flow to the tissues of the foot.

There are several major arterial branches that supply other areas. These branches all come together to provide a vascular network through the palmar foot. It should be noted that this extensive blood flow provides very little nutritional benefit to the frog and digital cushion, as they are composed of myxoid tissues which require minimal need of the nutritional function of blood flow. We have found, in fact, that most of the blood flow to the foot serves other functions besides nutrition and oxygenation to the tissues. Two of these other functions include support of the boney column and dissipation of vibrational energy.

This extensive and unique microvascular network of the equine foot is comprised of small vessels that are 5 to 25 microns in diameter, with some vessels being as large as 100 microns, with a layer of involuntary smooth muscle supporting the vessels.[14] The latter vessels are still very small in diameter. While most of the vessels in frog compartments are 5 - 25 microns in diameter and red blood cells are often larger — 7 - 8 microns in diameter — a healthy red blood cell has little difficulty passing through these small microvessels, as they can fold up to fit into these very small vessels. Red blood cells of humans with Sickle Cell Anemia are very rigid and are unable to bend and fold as they pass through small diameter vessels, hence the formation of serious vascular disorders in these affected people.

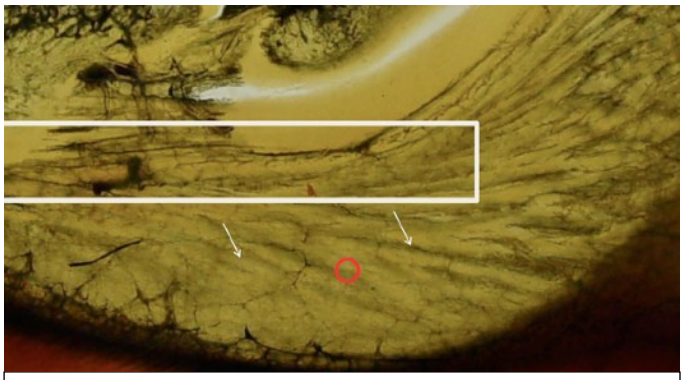

Figure 3. Microvessels filled with india ink to be seen; the white area in between the black vessels is ligament.

The microvessels are too small to normally visualize with most clinical and some anatomical techniques. To be seen with radiographic techniques, vessels need to be greater than 700 - 900 microns. Vessels must be 800 - 900 microns to be seen on a cat scan. A micron is 1,000th of a millimeter.[15]

Vessels throughout the compartments support the boney column and dissipate energy. (Figure 3) In horses with navicular syndrome, laminitis, or other lameness, these vessels are constricted or closed. This is visible under the microscope via antibody staining. In cadaver feet, micro vessels are still constricted in some individuals, meaning that that when they were alive, there was hardly any blood going through the back part of the foot.

We also found no vessels under the coffin bone in navicular affected horses. These feet have gone through the constricted phase and the vessels in the entire ventral part of the foot are just dying.

Figure 4. Fascia functions as a parallel nervous system transmitting information from the foot, as it hits the ground, to the spinal cord.

Very little blood goes through the vessels because a protein that constricts the endothelial lining may be high due to metabolic issues. Endothelin-1 has been found to be high in endocrinopathic horses. Nitric oxide is a natural antagonist of endothelin-1. In a normal cell there is a balance between nitric oxide production and endothelin-1, but in the EMS/IR horse, the endothelin-1 is prevalent.[16]

THE IMPORTANCE OF FASCIA

The hoof is a very smart structure that knows it is going to hit the ground before it actually does.

We have found fascia in the equine leg and foot to be a parallel nervous system. (Figure 4) When the foot hits the ground, information is transmitted up to the spinal cord. This information is also informing fascial slips along the leg that support/aid the function of the structures in the foot via Ruffini Corpuscle Receptors (light pressure) located in the fascia over the distal limb.

Navicular horses we have observed do not have facial slips. The fascia band going down to the digit is much thinner and underdeveloped. Vibration has come up the leg and inhibited the development of fascia.[17]

TENSEGRITY: TENSION & INTEGRITY. HOW IT WORKS FOR THE HORSE TO DISSIPATE ENERGY

The internal frog stay is, as the name suggests, the "stay" or stabilizer on all the ligaments within the foot. Many clinicians and researchers believe the frog stay pushes the blood out of the foot. We have found, however, that with the appropriate development of the frog apparatus and fascia, the stay works to open blood vessels in the foot to dissipate energy through negative pressure.

The frog stay is like the mast stay in a ship which holds the mast steady. Ligaments from compartment 2 under the NB and DIL attach to the stay. Additionally, ligaments in compartment 3 go up the foot from the stay and attach to the pastern. The frog stay keeps the ligaments — primarily located in chambers 2 and 3 — in extension when on the ground.[18]

The cuneate frog of the foot increases in volume via the chamber microvessels and increases the negative pressure necessary to absorb the energy created by the foot fall. This negative pressure allows blood to be sucked up through the back part of the foot. (See Poiseuille's Law[19])

If we have disturbed, limited, or rearranged this complex-but-elegant anatomical structure, areas that are not designed to absorb this energy are now forced to, and suffer as a result.[20]

By getting the frog stay closer to the boney column instead of centered and open in the caudal foot, the ligaments in compartment 3 become less taut. There is less negative pressure within the frog, less energy dissipation, and the frog becomes smaller and smaller. It atrophies.

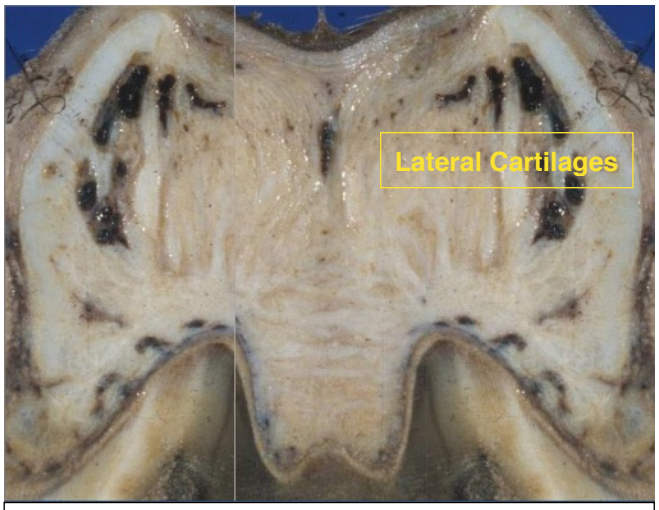

Figure 5. A good foot with thick lateral cartilages within the frog.

When the central sulcus goes rearward and up toward the fetlock, this creates the skinny, tight cleft so often treated for thrush.

Constant pressure on the frog via boot frog insert, or compression material, collapses the ligaments within the frog and it will not function properly.

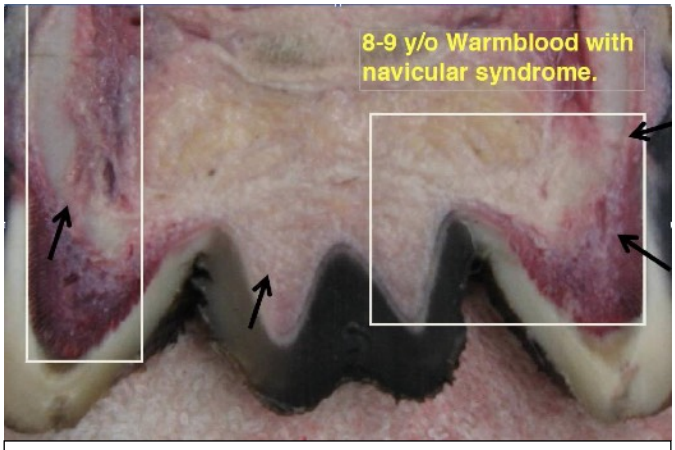

Figure 6. Compared to the foot in Figure 5, this young horse shows destruction of lateral cartilages and a cuneate frog absent of fascial sheets across the width of the frog.

POOR FROG FUNCTION AFFECT ON TISSUES

We have observed hundreds of cadavers. In two- and three-year-old horses, the lateral cartilages (LCs) extend ventrally and fill space between bars and wall. The cartilage is very thick.

Horses without foot problems have very dense, **thick LCs** with thickness greater than 25 - 36% of the caudal foot, greater vasculature and superstructure, and much greater fibrocartilage in the cuneate frog ligaments. (Figure 5) These horses have something to stand on. Good LCs contain proteoglycans that ***provide hydration and swelling pressure to the tissue, enabling it to withstand compressional forces.***[21,22]

Conversely, in individuals as young as 8- and 9-years-old, we found LCs deteriorating, primary epidermal lamina damaged, and digital cushion destroyed. With no robust tissue within the foot, they are standing on the bone and hoof wall. When the LCs are thin and mushy, the vibration has to go elsewhere to be dissipated.

Damage to foot tissues and fascia via vibration destroys the way in which the frog functions. After about 3 years of age, individual cuneate frogs examined began to diverge structurally with deterioration of the entire foot versus healthy augmentation of cuneate frog tissues, depending upon how vibrational energy was dissipated.

In the foot in Figure 6, the LCs are destroyed, and the walls of the internal cuneate frog are disintegrating. There is an absence of facial sheets and ligaments in the cuneate frog. They are chopped up in small pieces

from the vibration. With loss of support in the cuneate frog, the navicular bone is no longer supported by compartment 2 during loading.

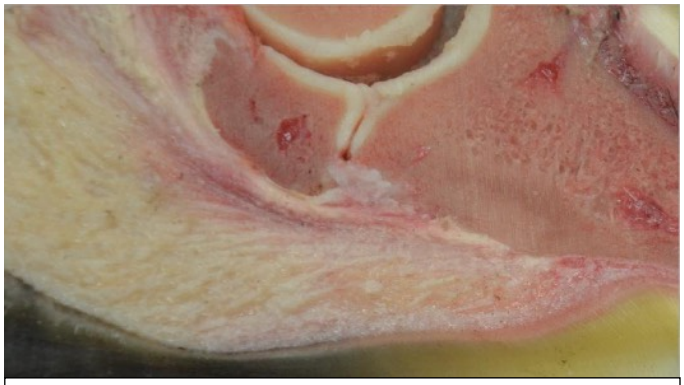

Figure 7. Ligaments in compartment 2 are damaged.

The gel pad which lines the underside of the coffin bone and LC, is also comprised of proteoglycans and microvessels. When viewing radiographs, the depth of sole measurement should be mostly gel pad which protects the bone. The sole itself doesn't do much for protection. As an example, consider hitting the medial surface of your tibia. It hurts because you have only keratin on the bone and no gel pad for protection.

The gel pad should be 4 - 6 mm thick in good feet; however, we have found less than 1 mm in laminitic and navicular-syndrome-affected horses. This further reduces the ability of the foot to absorb impact and protect the coffin bone.

UNDERSTANDING SOFT TISSUE PATHOLOGIES EARLIER

When the central sulcus is not open in a diamond formation, the frog does not function properly. Trimming the frog discourages formation of the compartments within. The horse loads the toe and front half of the foot, further compounding the problem.

Histologically, one can see many changes in the ligaments in the cuneate frog and DC before any changes in the DDFT or navicular bone are seen because it hasn't happened yet. These changes, however, are progressing up the leg. In the warmblood foot (Figure 6) above, the LC is deteriorating ventrally in the foot and cuneate frog. Laminae and bone are further stressed by the resulting toe loading. This damage can be seen in fuzzy dorsal and ventral bone on radiographs.[23,24]

In compartment 2 you will start to see larger holes that are not microvessels between the ligaments. In laminitic horses, holes in the structure of compartment 2 are huge. These holes can also extend to the LCs allowing less and less blood going to the back part of the foot.

Damage in compartment 2 is present 3 - 8 years **before** clinical signs appear. (Figure 7) Seeing these early pathologies allows us to make changes before acute or chronic event occur.

When compartment 2 is destroyed, there is no longer anything to cushion the weight of the horse coming down through P2, and through the NB, DIL, and soft tissue. The boney column comes down and beats the internal tissues into hamburger with every step. Recognizing this early damage allows us to treat earlier.

Early changes in compartment 2 can be viewed via ultra sound. Pathologies can be seen in shod horses and in barefoot horses that have had long toes and/or worked on a hard surface. Reduction will vary, but we often see 30 - 40% of vessels remaining in a damaged foot, versus 100% found in a normal or young horse.[25]

In chronically laminitic horses the frog often gets bigger; however, it is not functioning correctly. The vessels in the caudal frog are dilated because the blood cannot get to the chambers. Chamber vessels that are dead or dying prevent blood from getting through the foot, which makes the frog artificially larger.

SUMMARY

The frog is not vestigial like equine extra toes. It is not *degenerate, rudimentary, or atrophied, having become functionless in the course of evolution*,[26] except as we have made it so through husbandry. It has a specific function with energy dissipation being only one of these functions. Its purpose is underappreciated. Recognizing how the frog functions and how we affect frog function in our trimming practices will help more horses not fail.

When we do a better job recognizing and questioning what is considered *normal*, we help the horse to avoid the consequences of laminitis, or to better survive the rehabilitation from laminitis. We have within our grasp the ability to rehab most horses with gate abnormalities, including laminitis and caudal heel pain (navicular syndrome). We have the ability to affect their long-term health.

REFERENCES

1. Dyhre-Poulsen, P, Smedegaard, HH, Roed, J, and Korsgaard, E. 1994. Equine hoof function investigated by pressure transducers inside the hoof and accelerometers mounted on the first phalanx. Equine Vet. J. 26: 362-366.

2. Lanovaz JL, Clayton HM, Watson LG. In vitro attenuation of impact shock in equine digits. Equine Vet Journal Suppl, (1998).

3. Grant KA1, Congleton JJ, Koppa RJ, Lessard CS, Huchingson RD. Use of motor nerve conduction testing and vibration sensitivity testing as screening tools for carpal tunnel syndrome in industry. J Hand Surg Am. 1992 Jan;17(1):71-6.

4. Occupational Safety and Health Administration (OSHA), *Preventing Muskuloskeletal Disorders in Construction Workers.* http://elcosh.org/document/1648/d000560/preventing-muskuloskeletal-disorders-in-construction-workers.html

5. Krajnak K, Miller GR, Waugh S, Johnson C, Li S, Kashon ML. *Characterization of frequency-dependent responses of the vascular system to repetitive vibration.* J Occup Environ Med. 2010 Jun.

6. Dyson S and Ross M. *Diagnosis and Management of Lameness in the Horse*. Copyright © 2003 Elsevier Inc.

7. Jenner F and Kirker-Head C. *Core Decompressioin of the Equine Navicualr Bone: An In Vitro Biomechanical Study* 2011. https://doi.org/10.1111/j.1532-950X.2010.00766.x

8. Blechschmidt E. *The Structure of the Calcaneal Padding, Foot and Ankle*, March,1982 (translated from the original1933 German article). First Published: http://journals.sagepub.com/doi/abs/10.1177/107110078200200503

9. Narváez JA, Narváez J, MD , Ortega R, Aguilera C, Sánchez A, MD, Andía E. *Painful Heel: MR Imaging Findings, 2000.* http://pubs.rsna.org/doi/full/10.1148/radiographics.20.2.g00mc09333

10. Nickel R, Schummer A, Seiferle E, Frewein J, Wilkens H; Wille K-H, Siller W. G (Translator). Stokoe W M (Translator), *The anatomy of the domestic animals. Volume 1. The locomotor system of the domestic mammals,* 1986.

11. Bowker RM. *Unpublished.*

12. Ibid.

13. Mishra PC and Leach DH. *Extrinsic and intrinsic veins of the equine hoof wall.* J Anat. 1983 May; 136(Pt 3): 543–560.

14. Bowker RM,. *The Vascular Cushion of the Frog: What Does It Do? (2015) ECIR Group Inc., 2015 NO Laminitis! Conference, Austin, TX, USA.*

15. Ibid.

16. Kellon EM. *The Role of Endothelin-1 in Laminitis,* ECIR Group Inc., 2013 NO Laminitis! Conference, Jacksonville, OR, USA.

17. Bowker RM. Unpublished.

18. Ibid.

19. Ibid.

20. Ibid.

21. Bowker RM. *Contrasting Structural Morphologies of 'Good' and 'Bad' Footed Horses,* 49th Annual Convention of the American Association of Equine Practitioners, 2003, New Orleans, LA, USA.

22. Yanagishita M. *Function of proteoglycans in the extracellular matrix*. Acta Pathol Jpn. 1993 Jun;43(6):283-93. https://www.ncbi.nlm.nih.gov/pubmed/8346704

23. Bowker RM. *Understanding Laminitis: How We View "Normal" Function*, ECIR Group Inc., 2017 NO Laminitis! Conference, Tucson, AZ, USA.

24. Bowker RM. *The coffin bone: How does adaptation impact the foot during laminitis?* ECIR Group Inc., 2017 NO Laminitis! Conference, Tucson, AZ, USA.

25. Bowker RM. Unpublished.

26. https://www.merriam-webster.com/dictionary/dictionary

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.