Laminitis Update

3-20-05 Revised 12-05 Pete Ramey

Important new research added 8-7-07: Finally, this is what we've been waiting for! Katie Asplin, et al., at University of Queensland have released the "study of the century (in my opinion)". They've proven that laminitis can be induced by prolonged high insulin levels! This will be a critical turning point in laminitis research, and should immediately cause the veterinary world to place primary focus on diet and exercise for both prevention and treatment of laminitis.

[Asplin K.E., et al., Induction of Laminitis by Prolonged Hyperinsulinaemia… , The Veterinary Journal (2007) doi: 10.1016/ j.tvjl.2007.07.003]

Laminitis research is a fast-moving field, with new discoveries being reported almost every day. I am not a scientist; just a farrier who specializes in founder rehabilitation. I spend my days working on foundered horses and my nights reading veterinary research papers, so I'm writing this only as an interpreter; providing reference to some of the latest understandings of laminitis to help farriers keep themselves current. Also of great importance are references I will make to human medicine. The more I study contemporary laminitis research, the more parallels I find to human diabetes research, and there are many answers waiting there for those seeking a deeper understanding of what is happening to our horses. It seems few farriers have time or inclination to wade through the complex jungle of veterinary research papers, so for the sake of the horse, I'll provide this simplified shortcut; a brief overview and a guide through the actual papers referenced at the end for those interested in deeper study.

The word "laminitis" means simply the inflammation of the laminae. In the past, we thought of it as a circulatory problem with inflammation, swelling and a lack of circulation within the dermal (the "living half" of the) laminae and the bypass of the laminae by arterial blood through AVA's being the primary problems. (AVA's are "side tracks" that can open and shut at will, diverting blood from artery to vein; bypassing the intended destination of the blood.) Resulting reduced blood flow was thought to starve the laminae and cause their destruction. Certainly all of this **has** been proven to happen, along with excruciating pain caused by inflammation and swelling of soft tissue trapped between the coffin bone and the hoof wall, (and equally painful inflammation of the frog and sole coriums) but in light of more current research, it appears that all of this happens in **response** to the damage of the laminae, rather than being the actual cause.

In fact, it has been proven that "normal" circulation is necessary to deliver laminitis triggers to the hoof. Experimental restriction of blood flow to one hoof will keep it from developing laminitis while the other three hooves are afflicted (after carbohydrate overload) (Pollitt). The closer we can get to a true of understanding the primary problems, the more effective our prevention and treatment can be. New research has given us powerful ammunition in the war against laminitis; knowledge we can deliver to the horse immediately.

The epidermal laminae are produced at the coronet and are literally a part of the hoof wall itself. They are intricately intermeshed with the dermal laminae which are firmly attached to the coffin bone. Between the two "halves", is a tough "skin" called the basement membrane. The basement membrane is the point of attachment of hoof to horse. At the distal border of the coffin bone, the dermal laminae end, and a second epidermal "half" is produced that bonds the wall to the sole. This "non-living" extension of the lamellar bond is called (for some unknown reason) the white line (I've never once seen a white one).

The hoof wall and the epidermal laminae grow past the stationary dermal laminae while being constantly, firmly attached to each other. There is around ten square feet of total attachment per hoof that must be constantly modified as this movement occurs. This amazing process is done with the aid of remodeling enzymes (MMP's) that constantly remove the anchoring filaments that tie the epidermal laminae to the basement membrane. In a normal situation, this "destruction" is timed perfectly with the rate of reconstruction and growth of the wall can occur while the healthy laminae remain almost impossible to tear apart.

Dr. Chris Pollitt (University of Queensland) has shown that during the developmental stage of laminitis, the MMP's go through a population explosion and destroy all of the anchoring filaments and sometimes even the basement membrane itself. This means that the hoof is literally no longer firmly attached to the horse. As a result of this damage the horse **then** goes through excruciating pain, inflammation, a disruption of normal circulation and often the weight of the horse drops the entire skeleton to the ground (through the hoof capsule), leaving the coffin bone far removed from its natural position within the hoof capsule. This is very important to understand, because in spite of this research, many professionals still treat laminitis primarily as a circulatory problem (by attacking **symptoms**). Pollitt has shown that **before** any of the clinical signs of laminitis are detectable, the damage is already done. The very term "laminitis" may be a complete misnomer.

This important "which came first?" revelation has quickly led to even deeper understanding. Pollitt has searched for other factors that can separate the hoof from the horse. He has studied the effects on the laminae by most of the "classically proposed"

trigger factors and to date has found only one that actually causes separation of the laminae. This is the hindgut bacteria streptococcus bovis.

The horse's natural diet of mixed grasses provides structural carbohydrates like cellulose, hemicellulose and lignin, but fructans usually dominate the engineered grasses and hays we feed our domestic horses. The horse (or any other mammal) has no enzymes to digest the non structural carbohydrate; fructan. They are present in great quantities in our sweet feed, rich, cultivated pastures and our beautiful "horse quality" hay. Fructan, sugars and starches (All three of which are combined in the total measure of non structural carbohydrates, or NSC) pass undigested into the hindgut where they undergo rapid fermentation to lactic acid (which can then be utilized by the horse, but not exactly naturally). S. bovis is the bacteria primarily responsible for this and can undergo a 1680 fold increase in numbers during a carbohydrate overload that leads to laminitis. While no one has scientifically proven that these bacteria leak into the blood stream and cause lamellar separation in naturally occurring laminitis, Pollitt has proven that if they reach the laminae they trigger MMP-2 production and cause the failure of the attachment of the epidermal laminae to the basement membrane.

More recently, Pollit has found that damaged intestinal walls can cause one of their components; Laminin-5 to access the bloodstream. When they reach the laminae they also cause MMP release and destruction of the anchoring filaments (also made of Laminin-5).

Another important direction to be aware of is that Pollitt has proven that the laminae readily separate when they are starved of glucose. Glucose starvation causes the destruction of important "spot welds" (hemidesmosomes) between the epidermis and the basement membrane that the anchoring filaments actually grow from. When this happens, the laminae are easily torn apart under load. This opens up a vast area of study and more new ways to understand laminitis. The dermal laminae each have over a hundred secondary laminae growing from them. These secondary laminae are made of keratinocytes, or simply keratin producing cells. They are the same producers of hoof wall and the horse's skin, as well as our own skin, hair and fingernails and those of other animals.

While Pollitt found that the endotoxins often previously blamed for laminar separation (in the wake of a dietary upset and a "die-off of digestive bacteria) do not directly cause laminar separation, the resulting metabolic stress caused by endotoxemia is still thought to cause it indirectly. A major feature of any metabolic stress is a reduction of glucose supply to the extremities. This is done to preserve the more vital organs. When the digestive bacteria in the hindgut go through the massive die-off often associated sudden change in diet and with the development of laminitis, the resulting lack of digestive bacteria creates an immediate "starvation" within the horse. Each hoof of a **standing** horse normally uses more glucose than the horse's whole head. Moving? It stands to reason that the same shutdown of glucose supply to the keratinocytes of the laminae is happening to the horse's skin, soles, frogs and etc., but the forces required to "finish the job" and cause physical separation are much greater in the hoof walls (when they are in a solitary support role without aid from a well callused sole).

This is one of several reasons we see so more much resistance to founder in hooves that are maintained in a natural form. These sheer forces are greatly reduced. Aside from the front hooves of horses under competent natural hoof care, we also commonly see increased resistance in the hind feet, regardless of the type of care provided. Although the fronts and hinds are identical at birth, the hinds are used much more aggressively by the horse throughout life, so domestic hind hooves tend to develop more naturally and completely than the fronts (Read "Digging for the Truth About Navicular Syndrome" at www.hoofrehab.com for more on this). The proper development of the back of the foot allows much better energy dissipation, which greatly decreases stress on the laminae. In addition, in a naturally shaped hoof, the laminae may be equally weakened, but the coffin bone has "nowhere to go" because the sole and thus the solar surface of the coffin bone is aiding in direct support of the horse. (Dr. Robert M. Bowker)

Also, naturally shaped and developed hooves have much more thorough blood circulation in place. Read Dr. Bowker's "Theory of Hemodynamics". The blood pumping action of the foot is much more advanced and difficult to obtain than most farriers realize. It goes far beyond simple frog pressure and requires proper development and flexion of the foot from the moment of birth. It is not only important for energy dissipation and for easing the burden on the heart, proper hoof function and circulation also gives the foot profound advantages and resistance when laminitis does make it to the acute stage and circulation loss becomes a more important factor. The foot must be allowed to flex and function at this stage to aid in circulation.

While visiting my diabetic grandfather, I noticed his fingernails were rippled, discolored and partially separated from his fingers. From underneath the deformed nails, soft, new fingernails were protruding. Sound familiar? If you've worked with foundered horses it certainly does! This led me down the road of studying human medicine and the role of blood sugars and insulin on keratinocytes. Being a person who thrives on new information, I felt like a kid who just walked in a candy store! Imagine my surprise when I found medical recommendations from the American Diabetes Association that, "All individuals with diabetes should receive an annual foot examination to identify high-risk foot conditions. This examination should include assessment of protective sensation, foot structure and biomechanics, vascular status, and skin integrity. Diabetic patients are urged to be evaluated for:

i) Peripheral neuropathy with loss of protective sensation
ii) Altered biomechanics (in the presence of neuropathy)
iii) Evidence of increased pressure (erythema, hemorrhage under a callus)
iv) Bony deformity
v) Peripheral vascular disease (decreased or absent pedal pulses)
vi) A history of ulcers or amputation
vii) Severe nail pathology

The skin should be assessed for integrity, especially between the toes and under the metatarsal heads. The presence of erythema, warmth, or callus formation may indicate areas of tissue damage with impending breakdown. Bony deformities, limitation in joint mobility, and problems with gait and balance should be assessed." **(Remember, they're talking about people, here!)**

I believe much of the information we need to know about how an unnatural diet high in sugars can effect hoof wall attachment has already been done for humans. It is the veterinary researchers who will have to assess the relevance of this, though, and determine its application to the horse. Until then, I will tell you about some of the other things I was able to dig up:

The secondary laminae are made up of keratinocytes. French researchers studying human skin show that increased insulin levels cause migration of keratinocytes! This is a potentially important tidbit that no one has studied in the horse (to my knowledge) If an increase in blood sugar causes an increase in insulin levels, this alone could start the whole process of laminar destruction by displacing the secondary laminae. **(I was right!)** While it is relatively common knowledge that insulin levels are often above normal in laminitic horses, I haven't found a scientific study that actually monitors insulin levels in horses after a carbohydrate overload that induces laminitis. Does this always occur? Knowing that this migration of the keratinocytes (the secondary dermal laminae) could possibly begin the separation of the laminae, I would be really interested in such a study to determine how consistent the feature of increased insulin is, among horses in the early developmental stage of laminitis. I have to wonder if the increased population of MMP's are trying to keep up with the repair of the resulting damage, rather than being a key destructive force (or both, which would create a vicious cycle). Research for human medicine often shows them coming along to aid in the repair of damage in the wake of injury.

From the American Diabetes Association, a research project done on mice (Gale group), found that high glucose levels inhibit the proliferation of keratinocytes and that increased insulin levels increase their proliferation. Either way, a disruption or unnatural situation is directly created by alteration of blood sugars. New intertubular horn tubules constantly grow from the dermal laminae and aid in the movement of the ever-growing hoof wall, past the coffin bone (Dr. Robert M. Bowker). They normally grow from the dermal laminae tips, through the epidermal laminae and merge with the rest of the hoof wall growing from the coronet. This is why the hoof walls are not stretched to the thickness of a coin by the time they reach the larger diameter at ground level. If an increase of blood glucose decreases this proliferation, it stands to reason that the hoof growth from the coronet could quickly "grow past" the laminae, causing the very beginnings of lamellar separation.

On that same note, if high insulin levels increase this new hoof horn growth, it could push the dermal and epidermal laminae apart as the excess new hoof horn piled in between them. In other words the lamellar wedge (I'll get to that later) could possibly be a cause, rather than an effect.

This could explain the two types of separation we see in the field. Sometimes we see the white line perfectly intact and the hoof wall pulled away from it. Other times, we see the dermal and epidermal halves separated from each other. The former would cause the hoof wall to separate from the "still-intact" laminae. The latter would cause the dermal laminae and the epidermal laminae to part ways from each other. I'm speculating, here, but I feel it deserves veterinary research.

From the same research team: Too much glucose actually starves the cells by decreasing its own utilization by skin keratinocytes. In this study, they actually found that the cells were receiving **more** glucose at a low 2 mmol/l level, than at an experimentally induced 20 mmol/l level in the blood. Hyperglycemia leads to increased insulin resistance as well as a reduction in general glucose utilization. In other words an excess of sugars, whether from grass, hay, grain or molasses can, in effect, starve the laminae to death on its own. They also found that keratinocytes flatten and loose their correct orientation at this higher glucose level. This research was for humans, but it also sounds very familiar, doesn't it?

In fact it is well known that horses with decreased insulin sensitivity have a very high risk factor for laminitis; there is simply not enough understanding within the horse-owning public as to why? Dr. Philip Johnson writes: "There is a very strong association between the development of obesity, metabolic syndrome and the risk of developing laminitis. Commonly, at initial veterinary examination, there already exists both physical and radiographic evidence of long-standing laminitis in these horses although reputable and credible owners and managers report that there have been no prior signs of laminitis or any obvious explanation. In these horses, visible changes that are commonly attributable to laminitis (including prominent growth lines, palmar divergence of growth lines, and a convex sole) may be evident in the absence of laminitic pain or any history of laminitis or lameness."

It is my firm opinion that these "credible owners and managers" simply must learn to read this "physical evidence". Over and

over, I am called to "new" founder cases only to find that the "writing on the wall" has been present, but unrecognized for years. Often the obvious warning signs are more evident in the "healthy" hooves at the same facility that haven't foundered yet. The eventual breakdown could be both easily predicted and easily prevented.

Throughout my career, working with countless laminitic horses, I have observed that often there is nothing really wrong with most horses that are considered insulin resistant or diagnosed with equine metabolic syndrome. Most horse owners call them "easy keepers" and it can seem impossible to keep them from being overweight or to grow healthy hooves. The real problem is that we are taking animals that evolved to thrive on sparse desert rangelands, and are forcing them to try to survive in a "donut factory". This is why the "desert breeds" carry a higher risk factor for problems processing sugars and thus for laminitis. I know this because I have watched many "hopeless" cases thrive, when their diet was changed to a simpler and more natural mixed-grass hay diet, and the bulk of the sugars and fructan-rich, lush grasses and grains taken away. The resulting increase in health has a very positive effect on their attitude, their bodies, and of course their completely sound hooves. The problem is not so much that they had trouble with sugars and starches in general; just that they had trouble with the unnaturally high amounts they were previously receiving. This is not to say that a change in diet should replace all medical intervention; but that it should be the first place we start.

In a nutshell, the same thing that causes fat causes laminitis; excess or improper feeding! Not that a horse has to be fat to suffer from laminitis, but if all horse owners recognized that increasing fat on a horse was reason for sheer panic, laminitis would quickly become almost a distant memory of the "way things used to be". I have also seen countless horses that were underweight, who were foundered by caring owners spending $60 per day feeding the horse, while the horse refused to gain weight. Over and over, these horses will finally gain weight and also grow healthy hooves when the diet is switched to a simple free-choice, mixed grass hay diet (plus salt and minerals). The reason is that the horse has no means to utilize the rich (high NSC) feed. The owners are just keeping the horse in constant metabolic distress and foundering the feet, while creating very rich, valuable urine. The nutrition in the rich feed is wasted, at best. At worst it can do great harm.

In true cases of insulin resistance or in perfectly healthy horses simply receiving excess, the cells eventually become so impervious to glucose that the horse goes into starvation mode, burning fat and then muscle because the mechanism to burn glucose has totally collapsed. Improving insulin sensitivity by feeding low sugar hay (and increasing exercise) may actually allow more glucose to be utilized once the vicious cycle has been broken. (From human medicine and anecdotally in horses, magnesium supplement can increase insulin sensitivity as well.)

It would be nice if we could violate the evolution of the horse and make its digestive tract just like ours. It is much more convenient to provide a couple of rich meals per day, rather than a constant uptake of lower nutrition feed. It just doesn't work very well for most horses. Don't blame a horse for being a horse. It is our responsibility to provide for their needs. Type-2 diabetes is a "human condition" in which the cells become increasingly resistant to stimulus from insulin to absorb glucose. If both insulin and blood sugar are high, it's called type II diabetes. If only insulin is abnormally high but still getting the job done of keeping glucose normal it is generally considered insulin resistance.

There is increasing suggestion that it usually does not begin as a disease at all, but that the effected individual is simply "built" for a much leaner, more natural diet than he/she is receiving. If an individual receives an excess of blood glucose (excess NSC) the body responds by increasing insulin levels to handle the glucose. Over time, the beta cells that produce insulin get "exhausted" and begin to loose their ability to produce insulin, snowballing the problem.

Why is the study of human medicine important? We currently know a great deal more about maintaining health and blood sugar levels in humans than in horses and the understanding of the similarities between the two can give help to foundered horses now, while laminitis research continues to advance. We still have hoof professionals treating laminitis as a circulatory problem while completely ignoring the diet and exercise of the horse. The circulatory problems and inflammation have been proven to be only symptoms of laminar destruction caused by an unnatural diet too high in non structural carbohydrates (read grain, molasses, rich pastures and high NSC hay).

Most horse owners trust the farrier to take charge of the health of the hooves and only include the vet when things go horribly wrong. Should routine hoof care be a veterinary procedure? Maybe so, if farriers ignore this knowledge: Currently, only one part of the body of one animal on the planet can be legally immobilized or have its bone alignment altered by someone who is not a doctor. While it is illegal for a lay farrier to diagnose laminitis in a horse, we must deal with its consequences to some degree on **almost every domestic hoof we see**. The more you learn about the dietary destruction of the laminae, the more you will start to see it happening everywhere. If you understand that a "too-rich" diet weakens the attachment of hoof to horse, it spills over daily into the traditionally "non-medical" job of simply maintaining a healthy hoof. Farriers must either get educated in the ways the diet effects the hooves' attachment to the horse and advise clients accordingly or ignore the facts and pretend hoof management is just the art of shaping of some non-living, non-moving "block of wood", which doesn't bode well for the horse. Educated, alert farriers can prevent laminitis. I have personally chosen to buck tradition and try to learn about, and then educate clients about the effects of diet on their hooves, because I feel it

is the only competent way I can do my job.

In light of modern understanding of diet and laminar integrity farriers need to shift more of their focus toward helping horse owners with dietary management. If lay farriers are to be responsible for maintaining the health of the hoof, they must become more aware of the body it is attached to. Every day, horse owners face a barrage of commercial advertisements and dietary advice from other horsemen that recommend the routine feeding of the very things that have been scientifically proven to destroy the hooves' attachment to the bone! This loss of attachment is what makes most horses "need" hoof protection to begin with and it is usually readily visible long before total breakdown and chronic founder. As professionals we must use the latest available science to combat this problem. It is our responsibility to study and interpret this new information and deliver it to our client's horses.

Understand that this discussion is not just for the horse diagnosed with laminitis or chronic founder. By the time things advance to this stage the real problems have generally been at work for quite some time. Although researchers are still figuring out all the reasons why; we know for a fact that carbohydrate overload weakens the hoof to horse attachment. What happens next depends on the mechanics already in place. If the walls are excessively long and the coffin bone is not receiving natural support through the thick, callused sole of the horse, the weight of the horse simply drops toward the ground, creating coffin bone rotation or distal displacement (sinking). The lower coffin bone position (relative to the hoof capsule) causes a "flat foot" or a bulging sole, with the apex of the frog lacking depth within a solar dome. Most professionals consider this to be a very sudden thing, but it usually happens over time, long before anyone notices a problem. **Routine rasping of this growing bulge (read undermining the coffin bone or thinning the sole and overexposing the corium) is usually what actually brings the problem to a horribly painful head for the horse; not the laminitis that has been constantly affecting the horse for years.**

The dermal and epidermal halves of the healthy laminae are impossible to tear apart with stress on the hoof walls. Instead, when mechanical stress alone causes failure, the laminae stay bonded together and both are separated from the wall itself. According to Pollitt, normal locomotion exposes the hooves to 1/10th of the stress required for failure of the hoof wall, yet the inner wall itself is the "weaker link", when compared to normal, healthy laminae! Therefore, if the two halves have become separated from each other, you can be assured one or more of the factors we have discussed were present. When the dermal and epidermal halves of the laminae are correctly bonded together, the keratinocytes of the secondary dermal laminae constantly add new hoof horn tubules to the mass of the hoof wall; from the inside out, not just from the top. (Dr. Robert M. Bowker) When the laminae separate from each other, even very slightly, the dermal laminae continue to grow the new horn tubules, but they now pile loosely between the dermal and epidermal laminae, pushing the two farther and farther apart and creating the lamellar wedge. This presence of hoof horn between the dermal and epidermal laminae is proof in a given situation that this has been going on for quite a while, usually while the owner and farrier perceive the horse to be "sound".

Another key feature of the laminitic hoof is that when the laminae separate, the new wall growth at the toe is no longer forced to grow down the bone to meet the ground. As long as the diet continually destroys the attachment, the new wall growth is free to follow the path of least resistance. At the toe, it will thus grow straight out, perpendicular to the laminae. Since the growth at the toe is not moving downward like the heel growth, it gives the **illusion** that the toe walls are growing slower than the heels. In fact, if you cut a cadaver hoof down the middle that has the classic "fan-shaped" growth lines of founder, you can measure the curved horn tubules and see that the toe and the heel actually grew at the same rate, just not in the same direction. **Occasionally you can find toe growth actually outrunning heel growth (visible in cross section) in hooves that appear from the outside to have no toe growth at all.** The fan shaped growth lines, along with the "filled in" area between the coffin bone and a flared or rotated hoof capsule are definite signs that the original separation of the laminae occurred many months ago. It is frightening how few hoof professionals recognize this fact. In the rare case that a coffin bone rotation does happen suddenly, there will be nothing but air between the two halves of the separated laminae; you can see or probe all the way to the coronet from the bottom of the foot (between the wall and the bone), and again, this is not the way it usually happens in the real world.

Most commonly, after a dietary insult to the lamellar bond, if the horse is provided with natural support of the coffin bone through the sole, the result is much less dramatic. Constant flaring, wall separation, "white line disease" develops instead, and dealing with these things falls on the shoulders of the farrier. If you try to treat these things with only mechanics, you will never see complete results. The diet must be addressed as well. Otherwise, you can soak hooves in anti-fungal solutions indefinitely, shoe, trim, or epoxy however you want and you will make only minimal progress with a case of "white line disease" or wall flaring. Keep in mind that that destruction of the lamellar bond can happen without clinical signs of laminitis. It happens from "top to bottom", and takes a full growth cycle to grow out in the best of circumstances. If a horse has a dietary insult every spring, when the grass fructans heavily increase and every fall because of acorns or crab apples, the hooves may appear to be constantly suffering from fungal invasion or "white line disease". I do regret the unsuccessful anti-fungal soaking programs I once put horse owners through, only to later watch as a grazing muzzle completely fixed the feet.

In most cases of "seedy toe" or white line disease, this process is well under way and only the epidermal half of the laminae are visible at ground level. The half produced from the dermis is back with the coffin bone where it belongs and a true white line (involving both halves) doesn't exist. Worrying about the fungal part of "white line disease" when the horse doesn't even have a true white line to begin with, is obviously a prescription for "spinning your wheels". The fungal complications of a white line separation do sometimes need to be treated, but understand that fungi do not really bother healthy hooves. The true lesion will be present first to create a home for the opportunistic pathogens that then attack the hooves.

So dealing with one horse with a 15 degree coffin bone rotation and another with a case of constant wall flaring is exactly the same. Generally speaking, the horse is growing well attached laminae from the top every second of every day. From a trimming standpoint, we need to load the coffin bone naturally through a dense callused sole, and provide some relief to the stresses on the disconnected walls. This in turn relieves the mechanical tearing apart of the laminae, allowing them to grow in correctly.

These mechanics do nothing, however, to stop the process of constant dietary destruction of the lamellar bond. This is often happening as quickly as the horse can grow well connected laminae. We must, at the same time naturalize the diet. This means providing free choice mixed grass hay or pasture that is as low as possible in non structural carbohydrates (particularly sugar and fructan) and the elimination of all or most of the starches, grains, fruits, acorns, molasses, and high NSC pasture from the horse's diet. (Often a grazing muzzle is all it takes) Horse owners are usually shocked when they see how well their horses thrive with such a diet. They just don't realize that the horse has little or no means to utilize most of the things they were previously feeding. The study of the nutrition of grasses and hay is an incomplete, but fast moving field in its own right. Please study www.safergrass.org for more details. It is the best source I know of to help you figure out the safest diet available in your area. **No one** can tell how much NSC is in a pasture or hay just by looking. This website will teach you how and why to get testing done. It's surprisingly cheap and easy to do.

As with humans, exercise will greatly enhance the effects of a healthier diet and also help make up for many shortcomings. The extra movement also increases hoof wall and sole growth; speeding up the healing process. The advancements in the hoof boot industry in the last year have been amazing. Hoof boots with thick foam insoles added, are very useful for providing enough comfort and protection for exercising the foundered horse while allowing excellent trim mechanics, and tape-on pads are a better choice when protection is needed during turnout. Stall rest is undesirable if a rehabilitative trim is well applied. If you set up proper mechanics with the coffin bone in a natural position (relative to the ground **during impact**; not just while standing) and relieve the damaged laminae from stress, every step will cause a bit of well connected new growth. That said, do not "force-walk" a lame horse. Offer the **choice** to move until you can get true comfort in the boot/pad system, or on firm, but yielding terrain. Then, and only then, begin a true exercise program; increasing work as the horse becomes more able.

Any means of support that is rigidly attached to the hoof wall itself is contraindicated. One reason is that the walls themselves are not properly attached, so can't provide support stable enough to allow well connected growth to move in from the top. It will be constantly damaged as it grows. Mechanically, the laminae are very strong when they are all working together, but tear with the slightest pressure when they are partially separated. Think of a piece of cloth that you cannot tear, but a little "starter cut" is all you need to easily rip it apart. This is where the "old" laminitis ideas come into play. We still do have to worry about them. Even if you provide a satisfactory diet, if pressure on the walls and/or lamellar wedge overstresses or irritates the new growth, inflammation, pain and swelling will occur, along with the mechanical separation. This swelling and capillary damage makes normal blood flow through the laminae impossible. Blood is then redirected through AVA's, bypassing the restrictions in the dermal laminae and starving the cells, thus adding to the damage and making healing impossible. "Hanging" a horse from weakened, separated laminae simply doesn't work and repeated unsuccessful attempts have left most farriers wrongly feeling that the reversal of a 10+ degree rotation is impossible.

Secondly, the walls are constantly growing. If you attach perfect coffin bone support to the walls today, by tomorrow your support has crept away with the ever-growing hoof capsule, along with the bone you are trying to stabilize. If you are willing to support the bone column with the ground, through the well callused sole and unload the laminae, you will be able to set up the mechanics to grow out the worst of "rotations". If the diet is right, the rest is just a matter of waiting for the growth cycle to complete itself; a new, well connected wall is simply regrown around the bone.

Every day, more is understood about how the horse's diet effects hoof wall attachment, but this just leaves many farriers and horse owners falling farther and farther behind. We need to teach everyone associated with horses to recognize the warning signs **before** they come to a head and cause severe lameness and pathology. Ripples or red stripes on the hoof walls, abscessing, fan-shaped growth rings, obesity, abnormal weight loss, dull, shaggy coats or an unwillingness to shed winter coats, flat soles, wall flares or white line separation, negative "attitude", "laziness", and hoof sensitivity should be cause for immediate concern for anyone associated with horses. It should be an immediately recognized fact that there is likely to be very serious and potentially dangerous dietary problems, and it should be considered irresponsible to simply wait and see if things get worse. This new science should have already brought about sweeping changes across the horse world, but the "tradition driven" horse world makes any changes very slowly and only

then while "kicking and screaming". Yes we have learned to heal most chronic founder cases, but the truly exciting part is that **all** founders could have been easily prevented, if only the horse owners and farriers were more educated.

A part of what is slowing down the horse-using public's awareness of obvious danger signs is the simple tradition of misunderstanding what a truly healthy hoof is capable of. Many professionals and owners still consider a truly laminitic hoof that is "sound" **only** while a half inch of steel keeps it completely out of contact with the ground to be "normal" and healthy. This thinking keeps blinders on us and masks obvious signs of troubles until they go too far. It sets the bar for gauging hoof health way too low. It allows professionals to accept true pathology as a perfectly normal condition and stop far short of achieving a truly healthy hoof. Who would try to evaluate the soundness and proper function of a splinted leg?! If that splint was required for the horse to have any use of its leg, who would accept that the horse is sound and refuse to dig a little deeper for answers? This is not such a wild comparison when you really think about it. The only differences lie in our traditionally accepted viewpoints, which should not have any influence on the veterinarian or the educated farrier.

A bare, unprotected hoof that cannot function comfortably and properly in the terrain the horse normally lives and works in is no less "sick" than any other part of the body that is not capable of doing its intended job. When any other part of the body is not functioning correctly, we immediately try to fix it. When the hooves aren't functioning correctly, tradition demands that we just try to cover them up. The problem is that it only works for a little while and actually brings the hooves farther out of normal function. This simple understanding is the heart and soul of founder cure and prevention. Given the recent major advancements in the hoof boot industry (Please read the how-to article "Boots and Pads" on www.hoofrehab.com) and ever-increasing knowledge of proper barefoot trimming, fixed metal horse shoeing is no longer necessary for bridging the gap between living and riding terrain anyway. It only serves as an unnecessary barrier between us and a true understanding of the current health and status of our horse's hooves.

For case studies, clinic schedule, more of Pete's articles and links to these research papers, please visit www.hoofrehab.com .

References and further study:

www.safergrass.org and personal consultation with its author, Kathryn Watts

INSULIN STIMULATES HAPTOTACTICMIGRATION OF HUMAN EPIDERMAL KERATINOCYTES (Benoliel AM, Kahn-Perles B, Imbert J, Verrando P.)

HOOF WALL WOUND REPAIR (Pollitt, Daradka)

FUNCTIONAL ATANATOMY OF THE CARTILAGE OF P3 AND THE DIGITAL CUSHION AND A HEMODYNAMIC FLOW HYPOTHESIS OF ENERGY DISSIPATION (Bowker, Van Wulfen, Springer, Linder

EQUINE LAMINITIS: INCREASED TRANSCRIPTION OF MMP-2 (Kyaw-Tanner, Pollitt)

BIOCHEMICAL INDICES OF VASCULAR FUNCTION, GLUCOSE METABOLISM AND OXIDATIVE STRESS IN HORSES WITH EQUINE CUSHINGS DISEASE (Keen, McLaren, Chandler, McGorum)

EQUINE LAMINITIS: LOSS OF HEMIDESMOSOMES (French, Pollitt)

SENSORY RECEPTORS IN THE EQUINE FOOT (Bowker, Brewer, Guida, Linder, Sonea, Stinson)

EQUINE LAMINITIS: CLEAVAGE OF LAMININ 5 ASSOCIATED WITH BASEMENT MEMBRANE DYADHESION (French, Pollitt)

THE GROWTH AND ADAPTIVE CAPABILITIES OF THE HOOF WALL AND SOLE: FUNCTIONAL CHANGES IN RESPONSE TO STRESS (Bowker)

Also read all of Bowker's papers from the link provided on my site.

GLUCOSE EFFECTS ON SKIN KERATINOCYTES: IMPLICATIONS FOR DIABETES SKIN COMPLICATIONS (Gale Group: Spravchikov, Sizyakov, Gartsbein, Accili, Tennenbaum, Wertheimer)

EQUINE LAMINITIS: GLUCOSE DEPRAVATION AND MMP ACTIVATION (French, Pollitt)

ENDOTOXIN INDUCED DIGITAL VASOCONSTRICTION IN HORSES (Bailey, Menzies-Gow, Marr, Elliott)

EQUINE LAMINITIS: A REVISED PATHOPHYSOLOGY (Pollitt)

METABOLIC SYNDROME IN HORSES (Johnson)