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Laminitis Update

Supercoriaitis — Laminitis Re-defined

Jaime Jackson

Article Summary:
The author, arguing that the term *laminitis* is essentially incorrect, incomplete, and misleading, provides an alternative conceptualization for the age old hoof disease — and a new definition.

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INTRODUCTION

The conventional veterinary definition of laminitis is *the failure of the attachment between the coffin bone and the inner hoof wall.*[1]

In this interpretation, the coffin bone, or P3, is said to be suspended within the capsule by a "bridge" of leaf-like structures called the sensitive and insensitive lamellae or "lamina". The bridge spans the outer, periosteal surface of the bone to the inner surface of the stratum medium ("middle wall") of the hoof wall. When this lamellar bridge fails, according to laminitis researcher C. Pollitt, P3 plunges "into the hoof capsule, shearing and damaging arteries and veins, crushing the corium of the sole and coronet, causing unrelenting pain and a characteristic lameness."[2] The alleged devastating descent of P3 referred to by Pollitt is commonly referred to as *P3-rotation* in the veterinary and farriery communities. It is a term, unfortunately, that is now all too common among horse owners, as well.

With some notable misgivings, this is pretty much the definition I gave in my book, *Founder: Prevention and Cure the Natural Way*,[3] I now believe, however, that this definition is essentially incorrect, incomplete, and misleading.

The purpose of this article, then, is to re-cast the laminitic hoof in a new light, explain what is happening from this new perspective, and then re-define the word accordingly. In fact, I propose to throw out the word laminitis altogether from the vernacular of the natural hoof care movement and replace it with one that truly makes sense for what is happening in this most dreaded of hoof-related pathologies.

LAMINITIS: "A FOOL'S PARADISE"

The statement that laminitis implies an attachment failure has occurred between P3 and the coffin bone is not incorrect. The problem is that it ignores the fact that the attachment failure is happening elsewhere in the hoof as well. And this is my principal, although not the only, grievance I have with the word laminitis: it steers us to one part of the hoof—the coffin bone and its laminar attachments—rather than the "whole" hoof, where a more complete picture of the problem can be found.

For example, the cartilages fused to the palmer processes of P3 are also connected to the capsule via the lamina.

[1,2]Chris Pollit, "Equine Laminitis: A Revised Pathophysiology," (8/2000) European Farriers Journal, p. 10

[3]Jaime Jackson, *Founder: Prevention and Cure the Natural Way* (2000: Star Ridge Publishing).

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Are we to ignore this? Indeed, when these posterior attachments fail, the back half of the hoof, well behind the coffin bone, is also subject to disengagement from the horse. This is no better evidenced than by a sloughed hoof.

But there is more. The *sole* is also subject to attachment failure during laminitis, witness again the sloughed hoof — when "everything" goes (below, "Hoof Slough"). And even though there is no lamellar bridge! Ditto the *frog*, *heel-bulbs*, and *periople*. To make a long story short, everything below the hairline is subject to attachment failure during "laminitis", even though the lamina may not be involved.

Which is to say that the term laminitis holds us hostage to the false perception that during a laminitic attack, the only attachment failure we ought to be thinking about is the lamellar bridge to P3. This misperception is so ingrained, for example, in both the farriery and veterinary communities (not to mention horse owners), that I have on more than one occasion personally witnessed heart-bar shoes—designed to counter P3-rotation—applied to hooves that were on the verge of sloughing. Common sense tells us that there is no point in nailing that or anything else to the hoof, if the whole hoof is getting ready to go. I'll take up the futility of such measures shortly, but they are, in my opinion, misguided efforts spawned by the perceptual illusions inherent in the conventional definition of laminitis.

Indeed, if we move beyond this laminitis-induced myopia, we are drawn outward into the "whole" hoof. And from there upwards to the "whole" horse, where the only effective treatment for laminitis can be initiated. Laminitis is, holistically speaking, a "whole horse" disease, with the ailing hoof but a symptomatic smokescreen shrouding a digestive nightmare. Nothing can be done at the hoof to stop or prevent laminitis. It is a fool's paradise, filled internationally with the carcasses of untold numbers of dead equines.

C. Pollitt

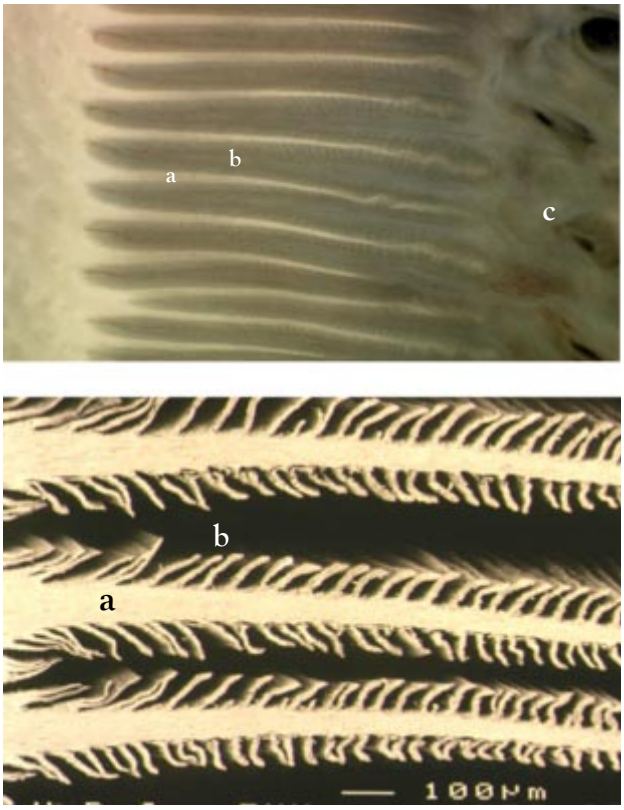

Figure 1 Intermeshing of (a) insensitive and (b) sensitive lamina. (c) marks the Supercorium. P3 is to the right of the Supercorium in the images above.

The Laminitis "P3-to-Inner Hoof Wall Suspensory Failure": "A Pathophysiological Red Herring"

Hand-and-glove with the hoof-to-bone laminitis illusion is the widely held belief that P3 is "suspended" in the capsule by the lamellar attachment mechanism (Figure 1). And that this "slung" orientation is what prevents, in the words of Pollitt, "the weight of the horse and the forces of locomotion [from driving] the bone down into the hoof capsule," wreaking the havoc he describes above in this article's introduction. While

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Hoof Slough: In the most extreme case of laminitis, the Supercorium "kicks off" the capsule completely . . . a death sentence for most horses receiving conventional veterinary and farriery care.

C. Pollitt

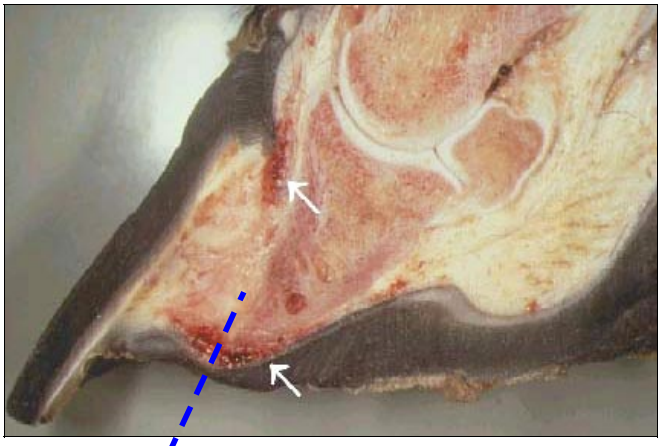


Figure 2 P3 Rotation A pathophysiological Red Herring and ultimate consummation of laminitis mythos

it is true that P3 is attached to the capsule via the lamellar bridge, I don't believe the "leaf like" lamina perform a true suspensory function, although the basic architecture of this bridge suggests that they do.

Structurally, in fact, the "leaves" are actually weak and incapable of supporting or resisting the tremendous weight-bearing forces descending upon them during support. One can demonstrate this impotence on a fresh cadaver specimen by removing the sole from the capsule, severing the integument at the coronary band, and pushing down on the bone column. It requires very little effort to press P3 out the bottom. Imagine what the weight of a horse could—and does—do! It would take leaves of steel to counter such overwhelming compressional forces.

Yet, the conventional definition of laminitis mandates this support-based suspensory function, and *P3-rotation* is offered as proof of the inevitable consequences of the lamellar suspensory apparatus gone awry—but this is a pathophysiological red herring, in my opinion (Figure 2).

Once more, if we back up from the pathology, which nails our perceptions to the immediate P3-inner hoof wall interface, we may look beyond to the "whole hoof" for a truer picture of how P3 resides and functions in the capsule. From that vantage point, we will discover that the actual P3 suspensory mechanism is far different than the laminitic reasoned one.

Briefly, P3 is attached rather "loosely" to the hoof wall via the intermeshing lamellar leaves. I say loosely because the insensitive (epidermal) leaves are adjoined by the highly vascular sensitive leaves, the latter being extensions of the Supercorium (discussed in greater detail later in this paper). During the hoof's support phase, this environment is flooded with blood as the hoof capsule expands under the horse's weight. Blood is alternately squeezed out of the Supercorium as the hoof contracts and enters its flight phase. This flexing of the hoof capsule is called the "hoof mechanism".[1] Collectively, the lamina in a healthy hoof must "give and take" with the mechanism.

If, as I've argued, the lamina lack the strength and rigidity to suspend P3 in the capsule, then what do they do? Several things. One, they position or orient P3 inside the capsule, like "guy lines" holding a ship at dockside. I believe this role supercedes any notion of suspensory function. Two, they connect P3 to the hoof wall, but, again, they do not support the horse's weight because they lack the structural integrity to do so. Three, by means of specialized "rivet cells" (i.e., desmosomes), the leaves cooperate to allow the stratum medium (main hoof wall) to grow past the Supercorium and P3. Last, I believe they form vertical "shunts" which the Supercorium utilizes to expel bacterial debris out the coronary band interface during abscessing.

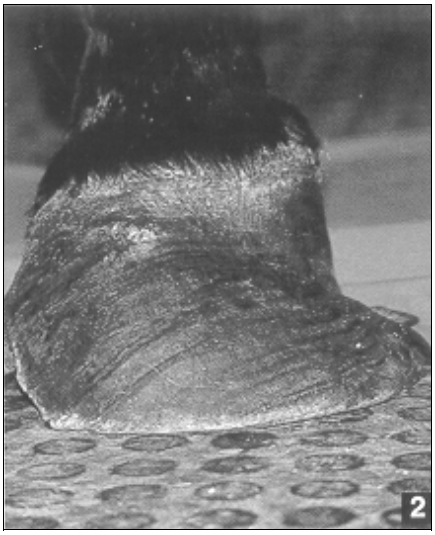
Finally, if not by means of the lamellar attachments, how is P3 "supported" in the capsule? *By the hydraulic action of the hoof mechanism*. As described above, the Supercorium "loads" with blood, and P3, fully aligned by the lamina, is buoyantly secured within a highly compressed capsule. A fascinating description of P3's hydraulic orientation in the capsule, taken from a century old British War Department Manual for hoof care, is worth remembering:

> The blood supply is so bountiful that in addition to giving nourishment to the foot, it acts like a water bed, helping to equalize the tremendous pressures to which the whole structure is constantly subjected, and keeping the bones and sensitive parts contained in the hoof buoyantly supported, like a big ship in a little dock.[2]

[1]Jaime Jackson, *Horse Owners Guide To Natural Hoof Care* (2000: Star Ridge Publishing) pp. 70-71. This is the European model for the hoof mechanism. For my expanded model, see "A *New Mechanistic Theory*: Time and Mass In A 4th-Dimensional Hoof Mechanism", Bulletin #108, SRP Natural Hoof Care Series

[2]*Animal Management, Prepared in Veterinary Department of the War Office.* London: His Majesty's Stationery Office, 1908. p. 221.

European Farriers Journal



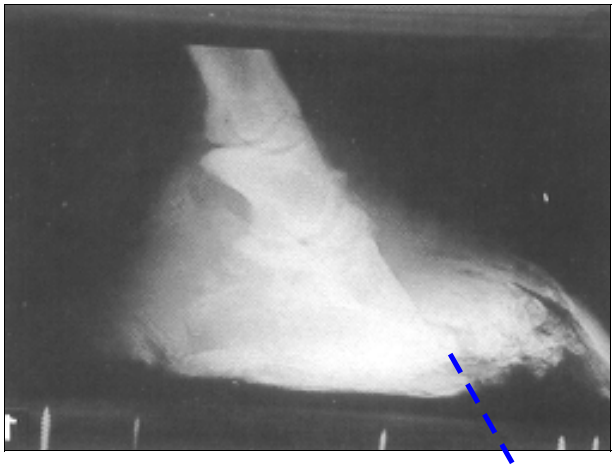


Figure 3 P3 Mythos "Right where it is supposed to be"

P3-Rotation: "A Bloody Red Line"

Whoever that ancient mythographer of laminitis was, his grand delusion has infected the better judgment and common sense of veterinary and hoof care practitioners ever since. P3-rotation is the ultimate consummation of the laminitis mythos. *There is no such thing as P3-rotation, then, now, or ever*: it lives solely in the imaginations of the mislead, including myself at one time. The sharpest radiograph, the most convincing photo, the eeriest dissection, and the worst alleged case of sinker or P3-penetration imaginable, each and every one only prove one thing: P3, with nothing more than a reasonable natural trim, if even that, *is right where it is supposed to be*—directly under the horse (Figures 2 and 3). It is the hoof wall, not P3, that is in the wrong place . . . and probably in the wrong shape too.

One can draw a bloody red line straight to the heart bar shoe (below) from the illusion. It is the last futile effort of the mythographer's descendents to prevent the bone from going exactly where it wants, needs, and *has* to go if the horse is to find relief and a healing. Well, almost the last measure, because if the orthopedic procedure fails, the veterinary surgeon has discovered he can pull the plug out completely by cutting the deep digital flexor tendon so P3 may never find its way home (Figure 4).

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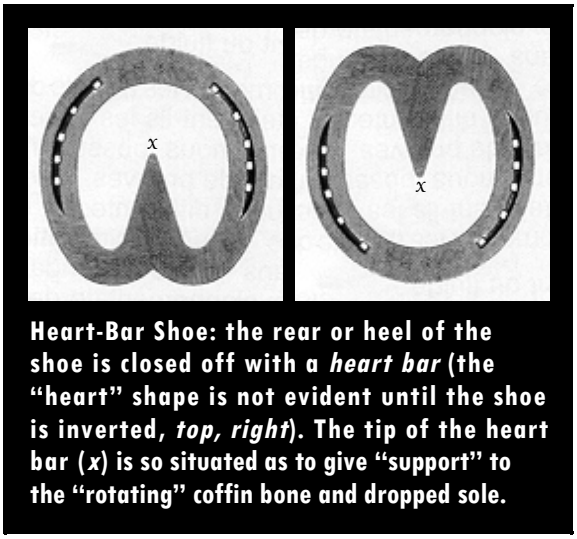

Heart-Bar Shoe: the rear or heel of the shoe is closed off with a *heart bar* (the "heart" shape is not evident until the shoe is inverted, *top, right*). The tip of the heart bar (*x*) is so situated as to give "support" to the "rotating" coffin bone and dropped sole.

Laminitis: What Is This Word?

Here, at the cutting edge of the laminitis anti-mythos "counter culture", I cannot even accept the ancient lexicographer's choice of words, "laminitis", as being valid! What does the word *laminitis* mean, exactly?

Literarily: "Inflammation of the lamina." Forgetting, for the moment, that the rest of the coria are also inflamed, and the entire capsule is ready to go, what is a "lamina(e)"? It is a Latin term, meaning "a thin plate". And what does a "thin plate" have to do with a horse's foot that is coming apart? Absolutely nothing!

The idea the Ancient Greeks were getting at, of course, was a system of stacked or overlapped plates, like plates of armor or the "laminated" veneers comprising plywood. But not *laminitis* in equines. That, the Greeks, ever astute observers, called "barley foot", recognizing the dietary implications. So, whoever later coined the term laminitis was really astray, in my opinion.

But back to this business of inflamed lamina. While the inner hoof wall attachment mechanism (the "leaf-like" lamina, Figure 1) is, indeed, inflamed, "angry" and dysfunctional, so potentially is the rest of the hoof-to-horse attachment apparatus. Since I refuse to ignore the

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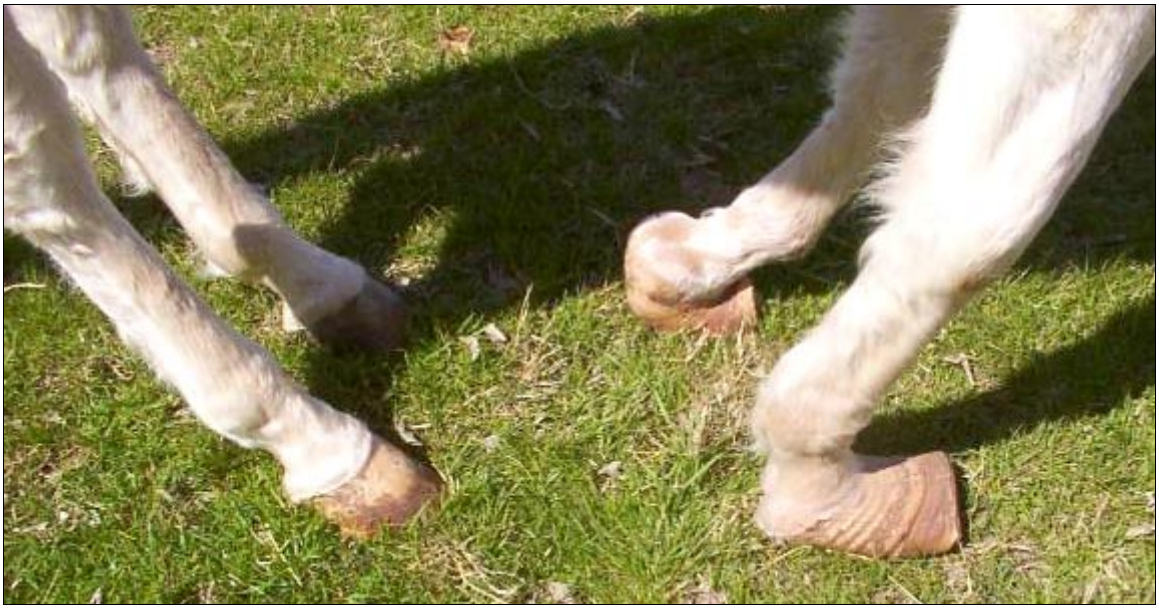


Figure 4 Tenatomy "Pulling the plug" so that P3 can never find its way home.

rest of the capsule attachments, on the basis of a bad word, then, once more, I am compelled to look beyond to the "whole hoof"—and, ultimately, to the whole horse—and make my case there for a better word with a truer definition.

In conclusion, I would, once and for all, like to throw out the term "laminitis" altogether, along with all its myths, bar shoes, pads, and unnecessary pharmaceuticals,[1] and start over from scratch. So, why don't we?

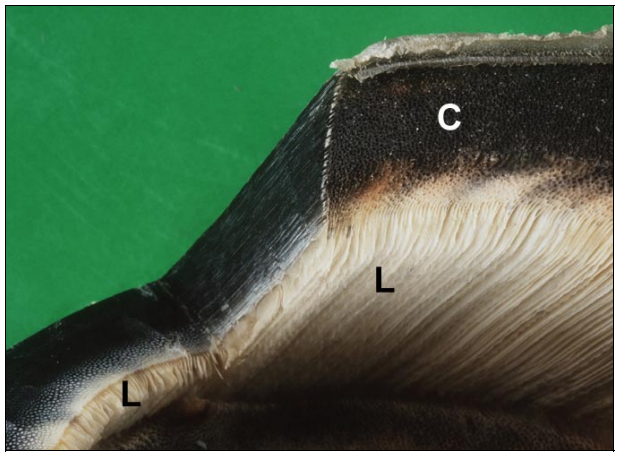
Supercoriaitis: Laminitis Re-Defined

First, I propose to re-define laminitis as *the dietary induced inflammation of the Supercorium resulting in the failure of the horse-to-hoof attachment mechanism*. And if we dispense with the word laminitis itself and seek a replacement term, then the new word, logically, is *Supercoriaitis*, (pronounced *Super-ko-ree-i-tis*, after *coria*, Latin plural of *corium*), since we are talking about the inflammation of a composite of interconnected coria that are collectively at the foundation of the horse-to-hoof connection. Let's look closer at this term Supercorium, the structures for which it is named, and how it serves in the "whole" hoof-to-horse connection mechanism.[2]

The Supercorium

The Supercorium is the inner dermal matter of the hoof that produces the epidermis, or hoof capsule.[3] Except through dissection, the Supercorium cannot be seen with the naked eye as it lies behind the capsule out of view —and harm's way, hopefully (Figure 5). It is actually an extension of the dermis (skin) above the hairline, thus, the Supercorium is an integral part of the "whole horse."

C. Pollit



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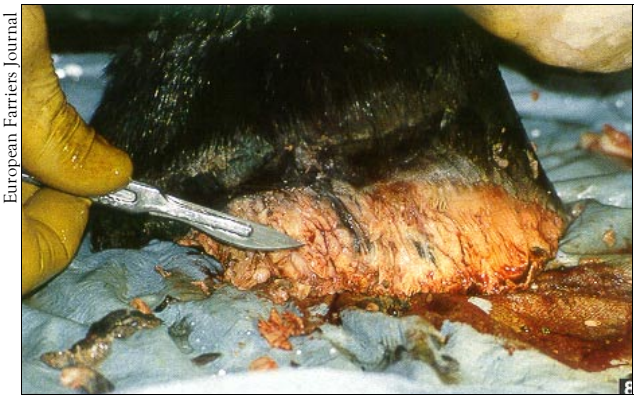

Figure 5 (*top*) Healthly insensitive lamina ("L") teased from sensitive lamina (i.e., the Supercorium, not seen in photo); (*bottom*) diseased lamina exposed under resected capsule.

[1]Less I be misunderstood, I want to be clear to readers that I am not against pharmaceutical intervention. For example, if a drug can bring pain relief to the horse while he heals, I'm for it.

[2]"The Whole Horse Trim," Bulletin #109, SRP Natural Hoof Care Series

[3]"The Supercorium," Bulletin #110, SRP Natural Hoof Care Series

The *hoof capsule*, I should point out from the outset, comprises all the epidermal structures below the hair-covered integument of the lower leg, the division commonly being referred to as the "hairline". The Supercorium, thus, comprises all the sub-coria that produce the various epidermal parts of the capsule visible to the naked eye. These include the *coronary band*, *periople*, *hoof wall* (including the bars), *sole*, *frog*, and *heel-bulbs*.

In precisely the same way that all these epidermal structures are joined contiguously together, so too are the sub-coria (sole corium, coronary corium, etc.) comprising the Supercorium which produce them. Collectively, they form a living "sock", or "super sock" of sorts, hence, the name, "Supercorium". In fact, I think of the Supercorium in terms of a sock, like a sock you would put on your own foot and subsequently cover with a shoe (tantamount to the hoof capsule). And like a sock removed from your foot, the Supercorium, if surgically removed from the capsule, and the horse too, would become an amorphic, rumpled mass.

Unlike your sock, however, the Supercorium is nutrified by blood and is *sensitive*, that is, it is vascular, contains nerves, and is able to receive and transmit information. Its architecture is complex. On its inner side, it forms a tough, fibrous connection, not unlike Velcro, to the horse via the coffin bone, cartilages, and digital cushion. As stated, on its "outside", the Supercorium produces, or manufactures, the hoof capsule. As the capsule is formed, the Supercorium simultaneously creates an extraordinary attachment apparatus to make sure the hoof stays connected to the horse. The actual attachment mechanism is complex and lies beyond the scope of this paper. But to the main point, each sub-coria is involved and each undergoes disengagement during the Supercoriatic attack.[1]

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Founder Stance

exotoxins (cell body wastes) to be absorbed into the bloodstream. Soon these reach the Supercorium. Once there, they trigger a massive proliferation of specialized enzymes normally involved in the natural breakdown and repair of the horse-to-hoof attachment mechanism. The enzymes then destroy the attachments quicker than keritinizing germinal cells in the Supercorium can re-build them.

But long before the naked eye can perceive deformities developing in the capsule associated with the attachment failure—for example, a "stretched white line" at the toe wall—the Supercorium begins to lose its grip on the capsule which, as stated, it is simultaneously charged with creating. Eventually, it lets it go, causing the shear force and excruciating pain described by Pollitt. Typically, the horse then assumes the characteristic "founder stance" (*above*).

P3, nevertheless, does not rotate during all of this, but stays put where it belongs: underneath the horse, and fully connected to the digit bones of the lower leg, while the hoof separates from the horse. The sheer

SUPERCORIAITIS

Supercoriatis is set off by "indigestion": dietary distress in the horse's hindgut. The Supercorium becomes sick and incapacitated—unable to produce normal hoof-to-horse attachments.

Briefly, the horse's digestive system, due to unnatural feeds (including pharmaceuticals) and feeding behavior, is taken over by harmful bacteria. These bacteria produce toxins which kill off normal digestive bacteria. Eventually, ulcers develop in the horse's gut, which allow bacterial

C. Pollit

Figure 5 Degradation of "hoof hydraulics". Right hoof is hydraulically normal; left hoof is hydraulically compromised.

[1]"The Supercoriatic (Laminitis) Pathway" Bulletin #103, SRP Natural Hoof Care Series

force and pain ensue because P3 is no longer sustained by the capsule's hydraulic system described earlier. Figure 5 demonstrates the degradation of the Supercorium's vascular system behind the hoof wall. As the wall moves away from P3, a gap forms which fills with blood, serous fluids, bacteria, toxic bacterial waste, phagocytes, and other debris. The lamina also become deformed, giving rise to the stretched white line so characteristic of this disease. If the attack is sufficiently severe, the capsule falls off and a temporary state of desiccation envelops the region as the Supercorium retreats to P3 (and other areas of attachment to the horse, as the case may be).[1] Significantly, the Supercorium is capable of regeneration as new capsule is laid from above by the sub-coria germinal cell matrices.

Supercoriaitis and the Natural Trim

Another laminitis myth, ironically fostered by elements of the "barefoot" hoof care movement, including horse owners themselves who simply pass around bad information given to them, is that a barefoot trim—particularly the "natural trim"—can "cure" the horse of Supercoriaitis ("laminitis").

Nothing could be further from the truth. As stated in this document many times, Supercoriaitis is a "whole horse" disease caused by unnatural diet and unnatural feeding behavior. Unless those issues are aggressively confronted, and resolved, the horse's condition will not improve, but remain chronic. Natural trimming is not a "silver bullet": it cannot right a harmful diet.

The function of the genuine natural trim (and I'm not talking about just any "barefoot" trim), is to aid the Supercorium in its efforts to restore a stable, naturally shaped hoof capsule for the horse to move and heal upon. *But the dietary causality must first be brought under control*. The natural hoof care practitioner simply shapes the healthy, new growth as it emerges from the germinal cells of the Supercorium (Figure 6). Just as is done with sound, healthy hooves.

Unnatural trimming practices, and shoeing, I do want to emphasize, can and do impair the natural healing processes. An unnaturally trimmed hoof, like the horseshoe (which causes hoof contraction[2]), obstructs the

Richard Drewry/Jaime Jackson

Figure 6 This Supercoriatic hoof is well on the road to recovery following a strict regimen of natural boarding and trimming, the latter conducted at 4 week intervals. (*top*) Before first trim; (*center*) 10 months later before 10th trim; (*bottom*) After 10th trim. The pony, previously completely lame, had been earmarked for euthanasia; now trots and gallops soundly. [Richard Drewry, AANHCP Practitioner]

[1]Pollit has shown that another extracellular structure, the *basement membrane,* lies at the outer surface of the Supercorium (which he calls the "dermis") like a layer of skin. The BM lies everywhere between the capsule and the Supercorium, and is intrinsically involved in the hoof-to-horse attachment mechanism. Significantly, BM retreats with the Supercorium when the attachment mechanism fails. For more on this, see my article, "The Supercorium," Bulletin #110, SRP Natural Hoof Care Series

[2]See "Does Horseshoeing Cause Hoof Contraction," Bulletin #111, SRP Natural Hoof Care Series

hoof mechanism and impedes the Supercorium's efforts to produce a naturally shaped, balanced hoof.

Summary

"Laminitis" continues to plague horses and their owners because the very definition of the word not only suggests an incomplete picture of the problem, it seduces horse owners into treatment strategies that contraindicate healing. Only a genuine "whole hoof" and "whole horse" approach to treatment will bring . lasting relief and soundness to the horse.

This paper has attempted to redefine laminitis on all fronts: the word itself, the meaning behind the word, and how we should perceive the malady from the new perspective as front-line natural hoof care practitioners.

QUESTIONS & ANSWERS ABOUT SUPERCORIAITIS

My farrier and vet both say that P3-Rotation will permanently maim and possibly kill my horse if I don't use heart-bar shoes or other "support" shoeing systems. How can you say that P3-Rotation is normal and natural and puts the horse's foot where it belongs? P3-rotation isn't going to kill your horse. It's the way you are feeding your horse that is going to kill him.[1] It's important to understand that your horse's hoof will continue to move away from the horse ("P3-Rotation") until you "naturalize" his feeding program. Use natural hoof care methods to nurture and expedite healing.

My horse's front feet both have "sinker" (dropped soles). The sole has pushed down, forming a reddish mound of horn forward of the frog. My farrier wants to know if you think he should elevate the soles off the ground with pads/shoes to relieve the sole of weight-bearing pressure and damage from the ground? Tell him that it isn't necessary, or desirable. The "mound" you are describing is the Supercorium's effort to build-up protection and support below the coffin bone: a "natural pad", if you will (see Figure 6 in this article). If you look closely too, you will see that the Supercorium will also attempt to render the mound passive by growing extra hoof wall. Let this happen. This temporarily simulates the hoof's natural relative concavity seen. Your farrier can stimulate this by following my "Rules of Rasping" for natural trimming.[1] The reddish sole will eventually shed, leaving P3 and the solar dome up where they belong. Move the horse temporarily to soft ground during the initial healing phase to ease his comfort. Have the vet administer bute, making sure it is not formulated with fructose-based (e.g., apple powder) fillers.

My farrier wants to know how far back the "run away" toes of my foundered horse can be backed up? He or she should refer to my updated trimming guidelines for supercoriatic hooves as well as the "Rules of Rasping" bulletin.[2] Specifically, the toe is reduced as a consequence of precision rasping and harmonizing of the capsule's Divergent Toe Angles.[3] Your farrier should avoid penetrating the cornification zone during back-up.[4] This approach to toe reduction yields excellent results, efficiently and safely.

I'm somewhat troubled by your new definition for laminitis, "Supercoriaitis". From what I can see, the only separation occurring between my horse's hoof and him is at the toe. The rest of the white line, quite frankly, looks perfectly normal, as does the sole, etc. Even the stretched white line at the toe "looked normal" when your horse first became "laminitic". You can't "see" Supercoriaitis until the damage is done. Hence, it is a false sense of security to think that just because you don't see it yet, that it isn't underway. I doubt too, that as a horse owner, you possess the critical evaluation skills to discern whether or not the white line behind the toe wall is, in fact, inflamed. As

[1]"The Supercoriatic (Laminitis) Pathway," Bulletin #103, SRP Natural Hoof Care Series

[1]"*Rules of Rasping*: Eliminating Wall Flare, Splits, and Run-Under Heels," Bulletin #106, SRP Natural Hoof Care Series

[2]"Trimming The Supercoriatic (Laminitidc) Hoof," Bulletin #104, SRP Natural Hoof Care Series

[3]"Trimming For Natural Toe Angle, Toe Length, and Heel Length," Bulletin #101, SRP Natural Hoof Care Series

[4]"The *Correct* Mustang Roll," Bulletin #107, SRP Natural Hoof Care Series

for the sole, when it begins to "drop" on you, you'll wish you never made that statement.

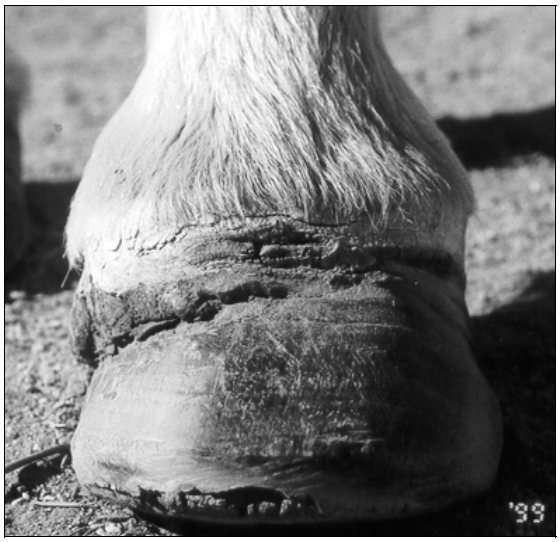
I just can't accept your argument that P3-Rotation puts my horse's foot "right where it belongs", even when the coffin bone is penetrating the sole. Aren't you being a bit irresponsible? I understand your frustration with this concept. But let me repeat what it means: P3 has *not* rotated, it is simply "right where it belongs". It is the hoof wall that has gone awry. And it will continue to go awry until you institute natural boarding (i.e., feeding) practices, augmented by genuine natural hoof care. Myself, and many other natural hoof care practitioners, have *responsibly* tested this theory on countless horses. It is no longer theory, in our opinion, *it is fact*. It is now the responsibility of horse owners to desist in *irresponsible* and unnatural horse care practices that are leading too many horses to early and painful deaths.

Last spring, both of my foundered gelding's front feet sloughed off. At my vet's recommendation, I had him put down. Do I understand you correctly that euthanizing my horse was uncalled for? Euthanasia is normally the last step that vets recommend when all else fails and the horse's obvious suffering necessitates humane intervention. Given your situation, with the knowledge you and your vet had in hand at the time, euthanasia was probably the humane thing to do. But is euthanasia *the* answer so far as hoof slough is concerned? Not from the standpoint of whether or not the Supercorium can restore a sound, functioning hoof capsule, because it *can*. The real question is whether horse owners are willing to negotiate the soundness and lives of their horses, in order to continue feeding "laminitis" diets?

My farrier says that my mare's foundered hooves continue to grow excessive heel, but little or no toe? I know that you have written that a natural hoof requires short heels. But every time he tries to shorten the heels to a natural length, she ends up walking painfully on her quarter and toe soles. What should we do? This problem is quite common. Fortunately, it is very easy to remedy. First, try to understand that naturally short heels go only with naturally worn hooves as we seen among wild horses and sound, fully transitioned domestic horses that are worked hard on abrasive services. We must not expect this type of heel configuration in Supercoriatic horses until *after* they are healed, and, as stated, only *if* they are subjected to rigorous use, natural boarding practices, and genuine natural hoof care.

The Supercorium will begin to produce substantial toe growth with normal attachment once the horse's digestive system is sufficiently de-toxed. The "excessive" heel you are seeing is the Supercorium's attempt to create a "club foot", that is, a natural "crutch" which aids in the healing process. This is discussed in a separate bulletin, which includes specialized trimming instructions.[1]

My vet says I should have his farrier remove the hoof wall over the toe, to relieve pressure on the inner hoof wall, and to make room for healthy new growth? He also mentioned "coronary grooving" to relieve pressure. What do you advise?
I advise that you don't do either. First, you "want" pressure on the inner hoof wall. This gets back to "hoof hydraulics," which is discussed in some detail in a separate bulletin.[2] However, as I pointed out earlier in this article, that pressure is meant to be there in order to support P3 hydraulically and optimize blood circulation. You also open the capsule to infection if you remove the toe wall. "Grooving" the hoof wall below the coronary band may prolapse or herniate the Supercorium (*below*), and you don't want that! By keeping the wall in tact, the supercorium will be able to do more to



Coronary Groove

[1]"Trimming The Club Foot," Bulletin #112, SRP Natural Hoof Care Series
[2]"The Supercorium," Bulletin #110, SRP Natural Hoof Care Series

promote healing "behind the scenes". Follow the "Rules of Rasping" for normal wall "back up". Then let Nature run her course.

My vet says that, due to laminitis, adequate blood cannot enter the inner wall (i.e., the lamina) of my foundered pony to facilitate healing. He recommends giving blood thinner medications to achieve this? I don't know what to do. New research confirms what natural hoof care practitioners have known all along: there is more than adequate blood reaching the inner hoof wall to facilitate healing.[1] Once more, the problem is diet. Until the horse's natural digestive system is restored to accommodate healthy digestive bacteria, abnormal proliferation of the MMP enzymes (described earlier) will continue to wreak havoc on the horse-to-hoof attachment mechanism.

My vet has recommended that a tenotomy be performed on my laminitic horse. This operation, he explains, will remove posterior tension from the coffin bone, so that it will be less likely to rotate downward and away from the hoof wall? What do you say? The deep flexor tendon has been engineered by Nature to reciprocate with the major extensor tendon (inserted at the front/top of P3) in the natural movement of the hoof and horse. Severing or diminishing the flexor tendon's connection to P3 undermines the function of the tendon, and, thus, the capacity of the hoof to support natural movement. Thus, from a biomechanical standpoint alone, there are grave risks in performing tenotomy. But the issue here is the logic of performing this surgical procedure to prevent P3 rotation. There isn't any, because P3 has rotated nowhere. Once more, it is the hoof that has left P3 and the horse. P3 must be allowed to remain where it is, and you must now naturalize your horse's diet and other horsekeeping practices. The hoof will "return" to P3 once you have done these things.

[1]Ibid., Pollit, p. 20

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