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Hoof Mass, Motion & the Mthos of P3 Rotation

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Article Summary: Counters current thinking and hysteria over P3 rotation and (coffin bone) penetration of sole amid a new weight bearing theory.

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INTRODUCTION

Fear of P3 rotation — the painful tearing of the coffin bone from its laminar attachments to the hoof wall due to laminitis, and the consequent spectre of P3 pressing down under the horse's weight and penetrating the sole — looms greater than ever today in the minds of horse owners, vets, and hoof care practitioners contending with this dreaded pathology. Conventional therapy includes the "heart bar" horseshoe, "support" pads, drugs, lasers, resection of the hoof wall to remove or mitigate laminar "shear forces", and even tenotomy to preclude downward retro-forces on the bone caused by the flexor tendon.

What these all aim to do is stabilize or force P3's position in the capsule, prevent the bone from plunging through the sole, and, hopefully, mitigate or eliminate pain and the animal's suffering. All are doomed to failure, however, in my opinion, as P3 rotation is neither a the cause nor the source of his pain and anguish.

In this article, I will present a new weight bearing theory at the hoof, one that also provides us with a different perspective concerning P3 rotation/penetration, and why the horse would be better served if we did not obsess with it, and simply got to the actual business of facilitating relief and healing through natural hoof and horse care strategies.

PATHOPHYSIOLOGY OF P3 ROTATION

As I have written in earlier works,[1] P3 rotation follows from a corruption of the stratum internum's (inner hoof wall) lamellar "attachment-release" mechanism during laminitis. Pollit has implicated (during the onset of laminitis) the radical proliferation of metalloproteinese enzymes at the basal membrane interface with the epidermal leaves.[2] Enzymes are normally present to facilitate breakdown of the desomosome bonds during keritinization, so that the hoof wall can move past P3. But during laminitis, this breakdown occurs much faster than cell-mending keritinocytes can be deployed by the Supercorium to repair the broken attachments. When this occurs, P3, purportedly under the full bore of the horse's body weight, is said to rip ("rotate") painfully away from the hoof wall, moreover, if the laminitis trigger is severe enough, "founder" or

[1] e.g, *Founder* (1997), p.62-66.

[2] Dr. Chris Pollit's lamintis research at Queensland University (Australia) is legend in the hoof care world, and can be found just about anywhere in that circuit including the internet.

perforation of the epidermal solar shield may ensue. The latter is called "P3 penetration" — and, notwithstanding full capsule slough, constitutes the most extreme manifestation of P3 rotation.

"At all cost," goes the veterinary mantra, "P3 rotation/penetration must be stopped!"; hence, the orthopedic shoes, pads, lasers and surgeries — aided by the ever ubiquitous NSAIDS.

My problem with the above scenario is that I am dubious that P3 rotation is all that we are making it out to be. I question the explanation of the force field driving P3 downward and even P3's role in the entire affair. In short, I am suggesting that P3 rotation may not be at all what we think it is. That it may be more of a "red herring" drawing us away from the natural healing pathway that we really need to get the horse on, than a genuine pathology deserving our full, undivided attention.

What I would like to bring into the light of discussion concerning P3-rotation, is the coffin bone's relationship to: 1) the "whole horse", particularly the skeletal superstructure; 2) the stratum internum, that is, how P3 is actually "fixed" or supported within the capsule, not only in relation to the interdigitated dermal and epidermal leaves, but to surrounding soft tissue mass and circulating vascular fluids; and 3) weight-bearing forces — specifically, how downward compressional forces impact the bone during support.

In this endeavor, my goal is to question whether P3 rotation is the actual debilitating threat to the horse we think it is, and, if it isn't, then what is the cause of his agony and alternative therapeutic pathways should we be pursuing instead during laminitis.

P3 and the Whole Horse

P3 lies at the very bottom of the skeletal horse (Figure 1). It is manipulated principally by the deep digital flexor and extensor tendons. Much has been written, and I think accurately so, about the biomechanics of P3 movement specifically in response to these tendons, and also the muscles and nerves that activate and propel the whole system. So I won't go into that discussion here, except to emphasize that muscles, tendons, and nerves are, in fact, the driving force — not P3.

Instead, let's look at the weight of the 210 bones comprising the horse's skeleton. Taken together, they surprisingly constitute only 10 percent or so of the horse's total body weight — water and soft tissue mass (e.g., muscles, fat, tendons, etc. — and body wastes awaiting excretion) comprising the bulk of it. And thinking about that, I've often asked myself, being that the skeleton is such a "light weight", how much of the horse's body weight does it actually support? And what about P3, since his entire body weight is pressing down upon it from above? Before trying to answer that, let's first take a closer look at how P3 is supported inside the hoof.

P3 and the Hoof

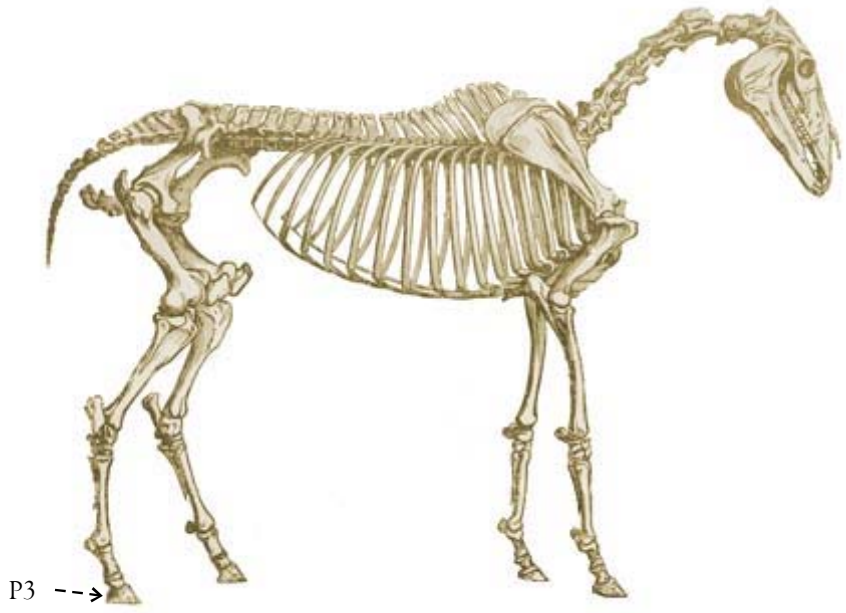

Figure 1 — P3 lies at the bottom of the horse.

Figure 2 — the epidermal leaves.

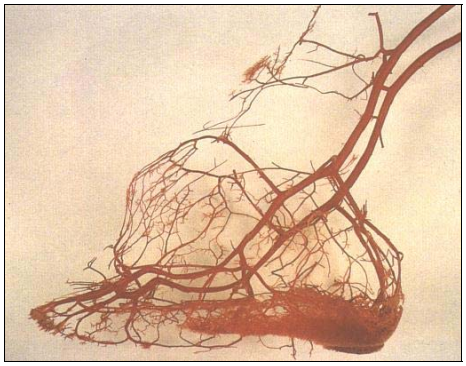

Figure 3a — corrosion of major arterial channels of the Supercorium (Pollit)

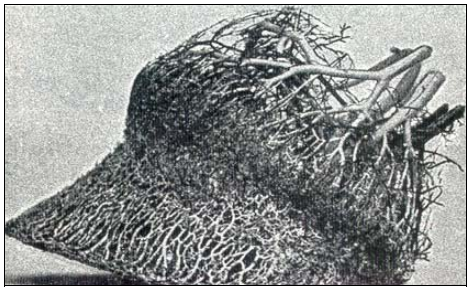

Figure 3b — corrosion of major arterial and venous channels (Dollar)

I think there is a tendency for practitioners to think of P3 as being simply slung (i.e., suspended) within the capsule via the interdigitated leaves (Figure 2) and that the horse's body weight is concentrated there. So that when the laminar attachment mechanism fails, as in laminitis, there is no more internal support structures to contrapose P3 rotation. In other words, P3 is then destined to descend under the full weight of the horse and wreak havoc on the lower solar dermis and epidermal dome causing the animal and the rest of us even more grief.

As logical as this explanation sounds, I think it's inaccurate. Between the hoof and the capsule are not only the fibrous connective tissues ("leaves") between P3 and the stratum medium, there's also a pressurized hydraulic system operative within the vascular system — namely the Supercorium (Figure 3a, b, c).[1] An extensive network of arterovenous anastemoses (AVAs), or valve-like shunts, connect the arterial and venous channels, enabling pressurized blood flow

[1]See my article and discussion of hoof hydraulics therein, "The Supercorium", Bulletin #110, Star Ridge Natural Hoof Care Series.

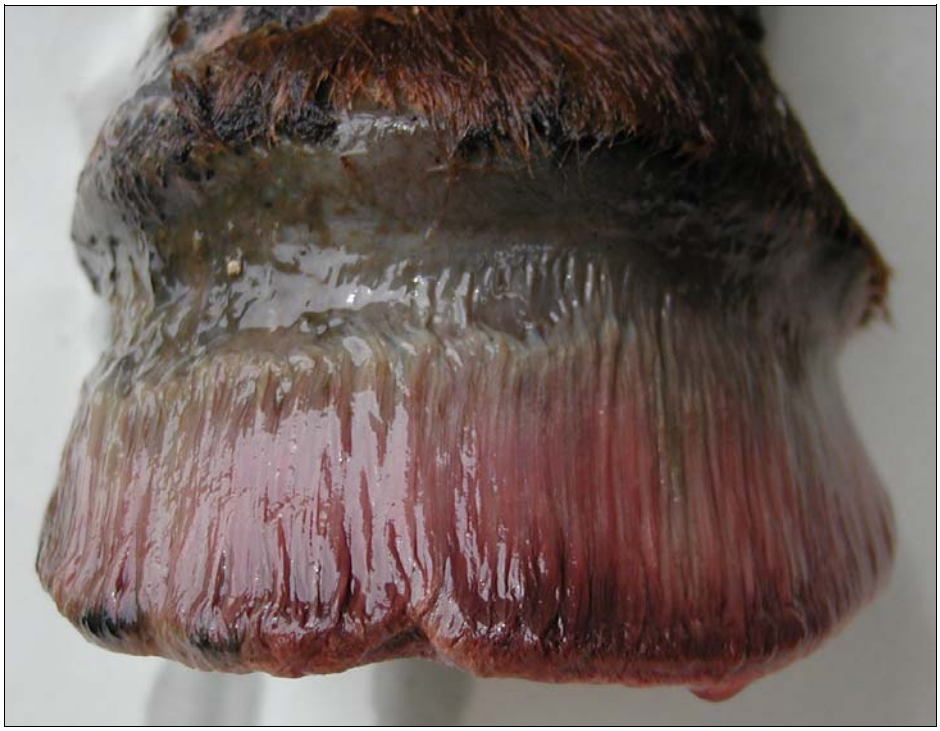

Figure 3c — Hoof capsule sloughed. P3, hidden from view, is separated from the capsule by the Supercorium (above) comprising an awesome vascular network with AVA shunts facilitating weight-bearing hydraulic counter forces within the entire hoof.

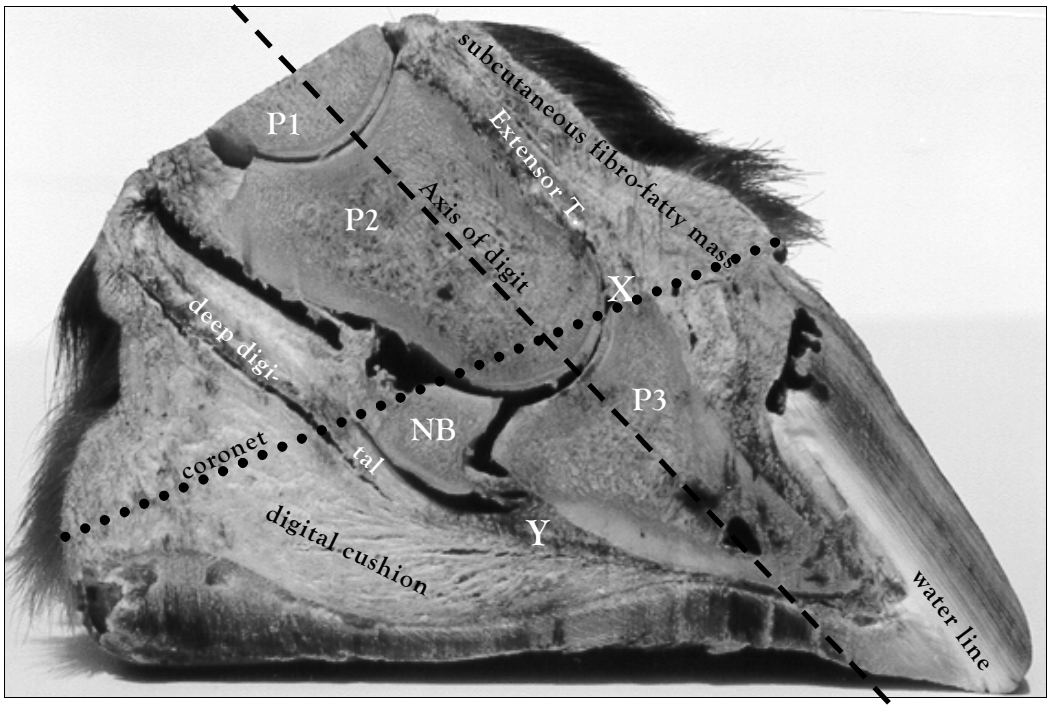

Figure 4 — digital cushion in relation to digit

within the Supercorium to bypass the capillary beds, insuring — as Pollit concludes — continuous circulation, even during acute laminitis. I have no reason to believe, therefore, that hydraulic counterforces are weakened significantly during P3 rotation.

Continuing, yet another "brace" resisting P3 rotation is in position to help: the fibro-fatty digital cushion, which lies directly beneath and behind the digit (P1, P2, P3, and NB). Figure 4 shows the relative positions of the digital bones and digital cushion. But — and this is significant — note also that a thick layer of moist, subcutaneous tissue envelops the entire digit above and below the coronary band ("coronet"). The bottom of the digital cushion is also reinforced by the highly elastic frog corium (Figure 5). Arguably, the entire environment within the capsule is a very cushioned affair!

Figure 4 also marks the extensor ("X") and major flexor ("Y") tendons and their points of insertion in P3, which further brace the entire digit. Not evident in the photo, but just as significant, is that both tendons insert at their opposite ends into major muscle groups further up the leg. These muscle groups, in turn, reciprocate with the major muscle groups (neck, shoulder, hip, thigh, etc.) we associate with thrust and propulsion .

Now, let's return to the discussion of the laminar connection of P3 to the capsule itself. If we dissect a hoof, we will find that the junction of the epidermal and dermal leaves is a relatively moist and flexible one. There's a lot of blood in there and if one grabs hold of the digit, P3 can actually be twisted clockwise and counterclockwise a bit. It's not meant to be a locked-down, "rigid" connection either. Mechanistic deformations demand a certain measure of give and take or the vascular dermal leaves would tear from their epidermal counterparts every time the hoof bore weight. My own theory, expressed in other bulletins, is that the principle function of the laminar attachments is to orient, or position, P3 within the capsule. Moreover, my feeling, based largely on common sense, is that the laminar bonds, individually or taken as a whole, aren't really that strong either. Certainly one is invited to ponder, how can 500 to 600 pairs of moist leaves bear the load of a one to two thousand pound animal pressing down upon them? This isn't to suggest that the leaves don't provide any support, only that it isn't all that we make it out to be.

What all this amounts to is that P3 is really the bottom end of a major propulsion system that simultaneously rests atop a very credible hydraulic and soft tissue braking system. Having said that, let's now consider the impact of these contiguous systems on P3.

P3 and the Hoof In Motion

The action of the muscle and tendon groups is, of course, to manipulate the bones and joints of the horse, including the digit, so that the hoof can provide support and then disengage for flight. I think it's im-

portant to emphasize that the reverse is not the case: the digit is *not* moving the muscle-tendon groups. It merely provides a "neutral" platform or framework for them to act upon. In fact, I question whether the skeletal framework even bears weight at all, including its own, during support or flight. It would appear that as long as there is muscular tension, the muscle and tendon groups are doing all the work, including weight-bearing. If this weren't the case, the skeleton would simply collapse onto the ground. Indeed, it almost seems foolish to me to suggest that the skeleton, which lacks the capacity either to propel or support itself, can somehow do for the muscle-tendon groups that which it cannot do for itself. [Of course, the muscle-tendon groups are unable to "organize" their efforts to propel and support without the skeletal framework; but this is beside the point.]

As strange as it seems — the bones neither carry weight nor propel mass! In short, they are simply "along for the ride". That nature configure them with such lightness (as well as durability and flexibility to get the job done) makes perfect sense to me now.

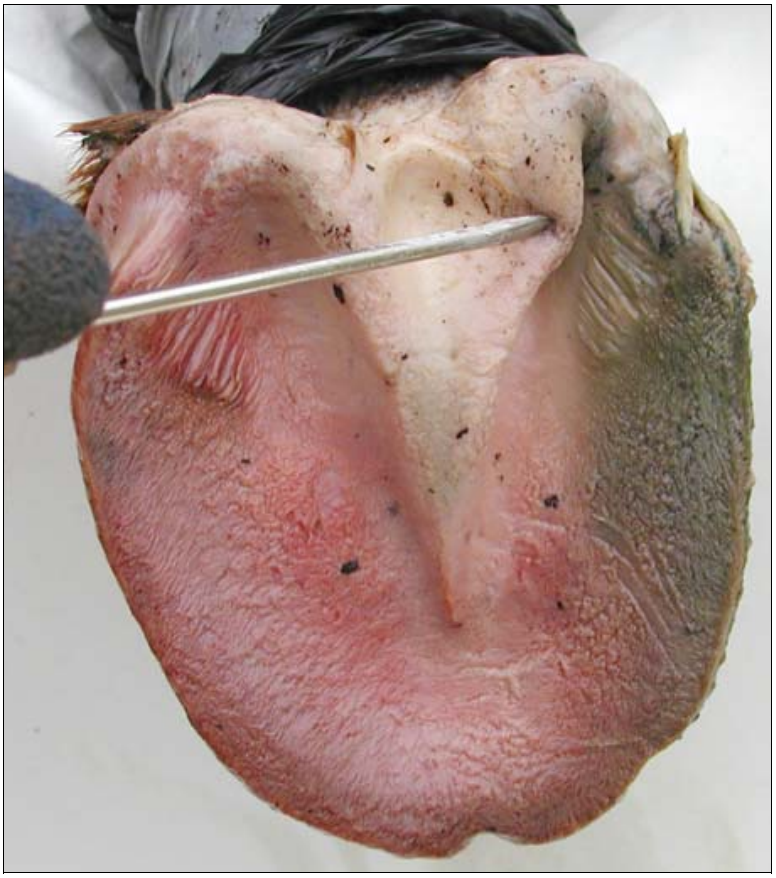

Figure 5 (top/bottom) — the digital cushion is cushioned from below by the elastic frog corium

I devised a simple way for you to test this hypothesis on yourself and others. Stand up with your legs slightly apart and your arms stretched out to your side, like the stick man in Figure 6. Now, begin to relax your arms (which are probably starting to get tired, anyway), and keep on relaxing them. If you relax them enough (or get sufficiently tired of holding them up), your arms will begin to lower, or outright collapse, to your sides. Now, go one step further, and also relax your legs. Just like your arms, they will eventually begin to give and then buckle under, until you fall on the ground.

Okay, what does this demonstration mean exactly? To me, it illustrates that the muscle-tendon groups are, in fact, doing all the work. The only thing that prevents the skeleton from collapsing and becoming totally immobile, are the muscle-tendon groups attached to it. In

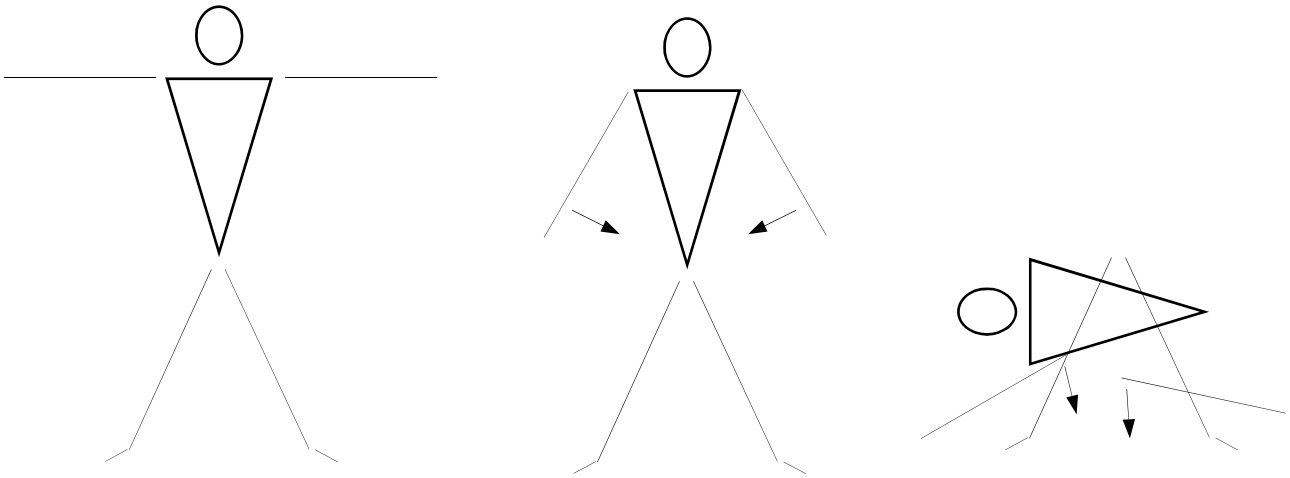

Figure 6 — the stick figure falleth!

other words, the bones are along for the ride and do nothing but provide a framework for movement and support. [This is not to "downsize" the importance of the skeleton — it also provides protection for the spinal cord, organs, etc.] The collective soft tissue mass (muscles, tendons, nerves, etc.) is the driving force that causes and sustains movement. And "locked" into place upon the skeleton, it also carries the load.

As outrageous as this may sound, I can only conclude that P3 is also along for the ride. In other words, it basically sits down there in a state of relative "weightlessness" and "motionlessness", until acted upon by the muscle-tendon groups. The interdigitated dermal-epidermal leaves — with their relatively weak bonds — are too weak to absorb the thousands of pounds of pressure during support. And that's not their job. They clearly serve to keep P3 aligned within the capsule, and facilitate downward growth of the hoof wall past the "stationary" Supercorium.

P3, not the weight-bearing structure we thought it was, is entirely buoyed and propelled by all the soft tissue mass surrounding it: the hydraulic forces of the Supercorium, the fibro-fatty digital cushion, the subcutaneous tissue surrounding the digit, the extensor and flexor actions of the major tendons and ligaments, and so forth.

So, then what is happening with P3 during laminitis, and, in particular, during P3 rotation and penetration? Somehow the bone has found its way through the sole!

The Mythos of P3 Rotation

First of all, I am dubious that the horse actually feels any pain from the "tearing" of the laminar bonds. Because I don't believe the lamina are under that kind of sheer stress. I think the pain is derived from the inflammation (heat and swelling) of the dermal lamina due to toxicity caused by unnatural diets and dangerous pharmaceuticals. During laminitis, the Supercorium is too sick to create normal attachments. So, instead of a "tight" white line formed between the hoof wall and the sole, we see a poorly formed one. This is where the familiar term, "stretched white line" comes from, because the leaves "elongate" as the hoof wall begins to separate from the horse.

If the causality of the laminitic episode is arrested, the pain will then go away. And many horses become sound even if their white lines are still "stretched". Most hoof care practitioners know this because they see it all the time. But, if the trigger is not brought under control, the pain and "stretching" will continue to manifest. Along with the characteristic "lamellar wedge" (Figure 7), "dropped sole", and exaggerated

Figure 7 — After de-shoeing, we find a severe dropped sole, deformed P3, massive lamellar wedge, and thinned out solar dome

"laminar rings" around the outer wall. Horses are typically put down at this stage of the pathology. Too, this is when P3 penetration is most likely to occur.

Putting P3 rotation/penetration in the new non-weight bearing perspective, it is logical to ask how the coffin bone, if it doesn't weigh anything, does manage to pass down through the sole? I don't think it does, or at least the way we are inclined to imagine it happening! Let me explain.

In the same way that the laminar attachments fail to connect the hoof wall to P3 (more accurately, to the Supercorium), the same is true of the sole-to-P3 attachment mechanism. Indeed, we invariably see a flat, sometimes convex, "dropped sole" with the P3-penetrated sole. The sole, like the hoof wall, is both sloughing and growing away from the horse. Its arched, concaved conformation, which once gave it strength and flexibility (— the sole epidermis is germinated in "sheets" of horn, not unlike automobile leaf-springs which provide support and cushioning to the vehicle) is now gone.

Given no choice, the weight-bearing, soft tissue mass surrounding P3, has descended too, and with P3 firmly in its grip (which is still non-weight bearing and passively along for the ride). P3 may also now be tilted anteroposteriorly due to normal interphalangeal (joint) rotation, following the loss of its indirect (via the Supercorium) connectivity to the hoof wall. It is in this position relative to the flat, weakened sole, that the latter may "split" along its sharp, distal edge.

But more often than not, this does not happen. The Supercorium will attempt to "build" a "mound" of protective horn over this edge (located on the sole just forward of the point-of-frog) to spare itself the consequences (e.g., infection) of being opened up to the environment. As another defense, it may attempt to build an "upright" club foot that transfers weight bearing more to the digital cushion and frog at the back of the hoof — thereby by relieving the inflamed dermal lamina up front of at least some of their weight bearing responsibility. I also believe that the soft tissue mass remains, for the most part, still biodynamically "operational" (i.e., capable of weight bearing and propelling) and this normally precludes P3 from piercing the sole. Otherwise, we would see a lot more P3 penetration then we do as professionals.

Many horses, however, "give up" and perish at this point from shock, or are put down by the vet — usually the latter. This is evidenced by the many laminitic cadaver hooves I see with flat soles, but no P3 penetration, when training students in our AANHCP trim workshops.

Conclusion

My feeling is that we should not obsess with P3 rotation or penetration. But, for the moment, ignore both. And then get right down to the business of naturalizing the horse's diet, living conditions, and hoof care. This is all easy enough to do. Nature will then take care of the rotation.

If he is shod, we must remove his horseshoes and allow him to go barefoot — as nature intended. Barefoot movement unleashes latent healing forces in the Supercorium. The fixed metallic shoe suppresses them by weakening the structural integrity of the hoof, deforming its growth patterns, and obstructing the natural gaits. They prevent natural wear, and so the hoof becomes longer and therefore biomechanically less efficient — a presciption for movement-based lameness. Nails, especially those driven into the water line, or worse, the white line, alert the Supercorium to environmental intrusion, causing it to contract to protect itself. No doubt, circulation is also compromised — imagine if your foot couldn't flex, as in cast if you ever worn one.

In short, P3 rotation is not the real threat. P3 simply needs time to grow a new hoof to protect its sensitive structures from environmental intrusions, to enhance its hydraulic and fibro-fatty cushioning systems, and to facilitate natural movement. And it will do this entirely on its own, if we just let it.

QUESTIONS & ANSWERS ABOUT P3 ROTATION

My horse's hooves have severely stretched white lines. Is there anything my hoof care practitioner can do to making them tighter? He moves great and doesn't have any pain. Not really, and I wouldn't worry about it either. A "tight" white line isn't necessary for soundness. Many, post laminitic horses, wear distended white lines and return to soundness. The distension may or may not disappear with time, depending on how severe the laminitic attack was, and if invasive procedures were used that may have scarred or damaged the dermis. In 1984, I observed a wild horse hoof at the BLM's Litchfield facility that had "stretched" white lines — the only time I ever saw this. He was perfectly sound and full of himself.

My horse's coffin bone (P3) experienced sole penetration, and the tip of the bone was eroded. What can my vet do to restore the bone? Nothing, and a perfectly intact coffin bone isn't necessary for a sound hoof anyway. All P3 does is provide a framework for the "work horses" of the hoof (the muscle-tendon groups I talk about in this article) to do their job of supporting and propelling the horse. In fact, in the back of the hoof, the hoof wall grows out where there is no bone. The Supercorium (dermis) has very powerful adaptive mechanisms to compensate for P3 issues. Just give your horse a reasonably natural diet, living conditions, and natural trims and he will be fine. By the way, in 1995, I was shown a radiograph at the Laminitis Bluegrass Symposium of a hoof where P3 had somehow "back fused" with the upper digital bones — then grew a normal hoof around it! And the horse moved soundly too!

My horse has severe WLD (White Line Disease), and his sole has separated from the hoof wall. My vet says he has 15 degrees of rotation. What can I do? Simple! Naturalize his diet, living conditions, and hoof care. And nothing more.

Do you recommend taping pads to the bottom of my horse's laminitic hooves to give them added support and ease his pain? I recommend that you use professionally fitted hoof boots for exercising during rehab. Use bute to reduce inflammation if he is very sore. Mainly, you need to address the causality of his laminitis. Once you get that under control, the pain and gait difficulties will begin to abate. Remove the boots when you are not exercising the horse. Let the horse move at liberty according to his own comfort level — don't force anything.

My vet has told me that my horse needs a toe wall resection and tenotomy to heal his laminitis and prevent further P3 rotation. What do you recommend? Get a second opinion. My advice would be to do neither. Resection opens the hoof to infection, and cutting the flexor tendon will compromise natural movement. The latter is necessary for short and long term healing. P3 will return to its natural position in the capsule as healthy new growth cycles in. But this will not happen until you naturalize his diet, living conditons, and hoof care.

My horse's front hooves have developed 1 inch thick lamellar wedges. The hoof wall has also grown forward such that he is walking on his soles at the toe. What can my farrier do to correct this? This condition is called a "slipper toe" and results from chronic laminitis and unnatural hoof care. First, you must naturalize your horse's diet and living conditions, or the hoof will fail to heal and will continue to look abnormal and not move naturally. AANHCP natural hoof care practitioners actually trim the slipper toe exactly the same as a "normal" foot. But this requires understanding of how such hooves transform during natural healing, plus technical know-how they gain from specialized training. Practitioners without this background are likely to cause harm.

My hoof care provider has cut the toe wall back to the sole on my laminitic horse. Does this help or hurt my horse? Commonly called "trimming to the white line", it is a violation of the wild horse model and, therefore, is not recommended. The practice stems from the believe that removing hoof wall that connects poorly to the sole due to a stretched white line, removes sheer forces further up in the laminar corium

where new growth and laminar attachments germinate. Many natural hoof care practitioners have experimented with this method, only to reject it as it causes soreness in many horses. Deformity of the capsule has also been reported. My feeling is this, why do such a thing if it isn't necessary? Further, there is no clinical evidence to suggest that not trimming to the white line, such that one turns the mustang roll outside the water line, does not work. As long as this is the case, and horses can be healed (that is, brought to soundness without causing harm, such as soreness) by less invasive methods, we should respect nature's model.

[1]*The Natural Horse,* p. 73

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