Laminitis/Founder/Rotation:

A common hoof deformity defined by conventional veterinary medicine as "Founder", involves the displacement of the coffin bone. Rotation is defined as a deviation from the normal alignment of the bones around a joint.[1] When we apply this definition to the bones of the foot (Founder), it is understandable how the function of the lamellae have been misinterpreted. As previously stated, lamellae attachment has long been thought of as crucial in the conventional hoof model. Once more, this because conventional veterinary medicine practices under the premise that this attachment in responsible for the position of the coffin bone within the hoof capsule, and that the conformation of the capsule is thus responsible for the position of the coffin bone about the coffin joint.

Further, we need to be clear on our definition of "rotation." Rotation in the conventional sense does not always accompany a hoof deformity. The conventional definition; any deviation from the normal alignment of the bones around a joint constitutes a rotation. That definition does not work within the parameters of AEP. AEP recognizes that the load surfaces of a joint must be in balance. In other words, all load surface of a joint must receive uniform load. That is not to say that all surfaces of a joint are receiving load simultaneously, it is to state that the surface under load is receiving even distribution of that load.

The common practice of defining rotation by the extent of lamellae separation is inadequate. Separation can be evidenced on x-ray; however the joint surfaces can be in balance, receiving uniform load throughout the motion range of the joint. It needs to be understood that with stress resulting in the forces that induce rotation, that those same forces are utilized by nature to place the affected joint's load surfaces back into balance. Change is the result of modeling, and/or remodeling of bone and cartilage. These changes in bone and/or cartilage are often viewed as deformations. AEP views these changes as appropriate changes to the normal conformation of the foundations of the equine foot under the given set of circumstance, and appropriate for the forces being applied. *Appropriate change* does not define the change as healthy; it simply means that we understand the cause for the change. Note: These changes do not always result in soundness for the intended use of the horse.

The types of hoof deformity that we are currently discussing (rotation) often exhibits high heel wall, uneven growth patterns, slight dishing of the toe, a separation or stretching of the golden line, laminar horn, and corium within the toe region. Remember, where conventional veterinary medicine targets laminar to hoof wall integrity as being the predisposing factor in this type of hoof deformity, AEP recognizes the subtle changes taking place in the *suspensory and supportive foundations of the caudal foot*.

[1] H.Strasser, S. Kells, Hoof Orthopedics and Holistic Lameness Rehabilitation, Qualicum Beach, BC Canada, S. Kells, 2001

Internal Arch Rotation

How the Internal Arch Apparatus is suspended within the hoof capsule is one aspect that is not addressed by conventional veterinary medicine. When you have a clear understanding that the suspension of the Internal Arch Apparatus is achieved by the relationship the coronary band holds to the ungular cartilages, and that this relationship is influenced by the conformation of the hoof capsule, your focus and perspective on hoof deformity changes. How rotation occurs: *The focus for the cause of rotation is no longer lamellae integrity, but instead falls upon the deformation of the ungular cartilages, the foundation responsible for the support and suspension of coronary band, and ultimately the Internal Arch Apparatus within the capsule. A change in the conformation of the foundation of the caudal aspect of the foot (ungular cartilage) results in stresses about the coffin joint that can lead to rotation. The loss of integrity of the laminar to horn union is in all probability a secondary condition, though the pathophysiological cause of tissue damage, both in the ungular cartilage (Elastosis) and lamellae dermis (basement membrane degradation) is likely the same. Histological studies indicate that a breakdown of the lamellae tissue is a result of enzymatic remodeling gone wrong (Pollitt, 1990). It is my contention that mechanical stresses created by the same enzymatic action affecting the support and suspensory structures of the caudal foot, result in the actual mechanical separation. If we can stem the change in conformation of the Ungular Cartilages long enough to see stabilization to normal enzymatic action, separation and rotation should be avoidable. See Illustration.*

Having described how rotation occurs, it is important that we recognize at what stage of pathophysiological change the horse is experiencing at this moment. Over time modeling of the bones and cartilages within the foot, will likely place the joint's load surfaces into balance and what was once viewed as *rotation must at some point be viewed as a conformational change* of the foundations of the foot. Remember that the process of change will follow this given sequence; first to change is soft, then dynamic, then static tissue. In treating the deformed hoof, we must identify the current state of conformation, and how far along is conformation from what is conventionally considered normal. This thought process has come to be known as *"Physiological Sequencing"*.

Applying the Knowledge

Developing a treatment protocol for most hoof deformities do not have to be complicated, we simply have to accept these facts:

1. Hoof capsule shape is responsible for the stresses conveyed to its *foundations* (cartilage & bone).

2. Conformational changes of the foundations are the result of how the forces of stride are delivered via the hoof capsule; this is nothing more than a cause and effect statement.

3. It is further understood that the foundations of the Internal Arch Apparatus play host to the dermis that produce our hoof capsule (epidermis), therefore hoof capsule conformation is directly proportionate to foundation development.

The science of AEP recognizes that the internal structures of the foot (foundation structures) are a mirror of the structures the dermis produce (the hoof capsule).

Conventional veterinary medicine utilizes static mechanics placing great emphasis on hoof angle. Unfortunately proper hoof angle, as defined in conventional veterinary medicine, or by many natural hoof models has far less to do with correct foot function than proper orientation of the coronary band. The orientation of the coronary band is established by the conformation of the ungular cartilages and not simply by trimming the dorsal wall to match the dorsal surface of P3.

The conformation of the ungular cartilages is maintained by the dynamic distribution of forces to the coronary band, by way of the coronary groove of the hoof capsule. With this in mind it can be argued that hoof wall angle does play a role in delivery of these forces, and it does. Unfortunately, *the root cause for the change of conformation of connective tissue and cartilage begins with a loss of elastic potential. Cartilage has the ability to resist deformation (maintain conformation), this is what is described as having elastic potential, with a loss of elastic potential deformation results in conformational change.* With a loss of elastic potiential pain often results; pain changes how a horse loads the hoof. Changes in load can result in the incorrect distribution of forces to the coronary band, resulting in conformational changes within the ungular cartilages. This type of hoof deformity is more often progressive in nature, but whether gradual or acute a loss of elasticity will result in hoof capsule deformity to some degree.

Dynamic Balance

When the Internal Arch Apparatus is correctly suspended by the coronary band and accompanying structures, the Internal Arch Apparatus will be placed in the proper position to receive the stresses induced by the stride, with the distal border of the coffin bone at approximately a five degree inclination to the Live Sole Plane. This orientation is said to place the *healthy* Internal Arch Apparatus into dynamic balance within the hoof capsule. The position of P3 is ultimately a function of the whole (IAA), and should never be used in and of itself as a denominator in the equation for achieving dynamic balance. You should always remember that balancing the solar aspect of the hoof capsule to the Live Sole Plane simply places P3 into the desired position within the capsule. It does not return health to the IAA. Dynamic equilibrium is defined as all structures functioning optimally at any given moment of time.

Deviation of the balance of the solar aspect of the hoof capsule, from that of the LSP results in incorrect stresses being imparted on the coronary band. This results in abnormal stresses being placed on the ungular cartilages, ligaments, and the dermis of the Internal Arch Apparatus, a loss of dynamic balance. It is not the conformation of the coronary band or

cartilages directly, but rather *what caused the change in conformation*. Stresses result in conformational change of cartilage, when elastic potential is compromised. Horn, because of its relatively low elastic potential readily conforms to the stresses imparted on it, i.e. flaring. Once more we need to be remembered of the process involved here; first we identify soft tissue changes, then dynamic tissue (horn) change, and finally static tissue (cartilage and bone) changes. It is sometimes necessary to reverse the process, working from x-ray, and this is fine provided you apply your knowledge of physiology and don't fall into the trap presented by static mechanics. Scrutinizing the palmar processes of the distal phalanx (P3) can help in our assessment of the ungular cartilages. Observing the health of its (P3) solar margin, and the solar canal will help us in physiological sequencing for the condition presented. Has there been long standing inflammation, evidenced by abnormal development of vascular channels? Has modeling of the tip of P3 occurred? All of this information can be used in developing a treatment protocol and ultimately formulating a prognosis.

Deformity Case Study (referred to our office for comment)

Perhaps the following correspondence will serve well as a review for the above information, presenting the information in a real world scenario. It is intended to illustrate how a shift in perspective can spur a shift in the hoof care provider's paradigm.

Horse Owner Comments:

This year for the first time, one of our Peruvian Paso horses came down with laminitis--too much fresh, spring grass and she was a little overweight to begin with. We called the veterinarian. He treated her, and she is much more comfortable, now healing I hope. At least her back isn't arched like it was; she isn't depressed anymore, etc.

Here's the problem. Our natural foot farrier came to trim the other horses last night, and when she saw the way the vet treated the mare she became very upset. Our veterinarian had trimmed our mare according to the contour of her foot, and then trimmed back the toe somewhat. He made a rubbery compound that he fitted into the sole of her hoof underneath, and set the hooves on a hard plastic shoe that was wedged, lifting the heels. He then covered the whole thing with a short rubber boot and wrapped it all up (vet wrap tape). Two days later he returned; seeing that she was doing pretty well, he took off the wrap and glued the rubber cuffs onto the hooves. The overall effect was that she was wearing modest high heels, maybe an inch and a half of lift in the heel. The plan was to remove one of these wedges in four weeks and the rest in another four weeks. During this time she is supposed to stay in her stall, or in an outdoor stall which we built just outside the barn on level ground.

Our natural farrier described this treatment as "the old way" that no one does anymore, because it never works. This surprised us, because our vet is not an old guy. He and two others work almost exclusively with horses and are well liked around here. The natural farrier has ranted about vets before, so this is nothing new. We want to do what is right for the mare. Our

natural farrier said she would have stopped with a trim, and that it sounded like a trim quite similar to what the vet had done, but with no wedges, and definitely no stall rest. Get her out and walking, lead her along a roadway for a mile or more twice a day. She had done that with others with good results, according to her. She cited the Institute in support of her recommendation.

What do we do?

My response:

Your natural trimmer's response is understandable, but was it appropriate? She is correct in stating that the technique used in the treatment of your horse is somewhat antiquated in *our* minds, though it is taught as the latest method for the treatment of acute laminitis in many of the conventional veterinary medicine circles. Problems do arise however when we view things from different perspectives, not knowing whether one is more appropriate than the other.

1. Your natural trimmer is viewing acute laminitis from a, (for lack of a better term) *natural* perspective. I myself find this perspective a bit naïve, and I mean that in a respectful way. The natural perspective is based in part on circulation, or lack of circulation, what they perceive as the "why or cause." Therefore they develop a "how to protocol" based on this "why" perspective. How do we aid the horse in returning correct circulation? The natural perspective does not concern itself with the root causes of the problem, "the true why," but rather become more concerned with the *how*, this often to the exclusion of all else. How can we fix this lack of circulation, rotation, or contraction? How do we de-contract or de-rotate? I ask you, can we really fix anything without a clear understanding of the process that led to the condition? No, we are simply shooting in the dark in developing a how to protocol, leaving it up to God (read nature if you prefer) to fix it.
2. The veterinarian (conventional veterinary medicine) too believes that they understand the why, though this is not always as clear cut as a lack of circulation and must be reviewed on a case by case basis. In other words, they too are coming from a perspective of "why," but viewing the condition from a slightly different perceptive. The result is that they initiate techniques that are preventative, and reactive, based on their belief of *what the progression of the pathology is*. How can we prevent further damage, until which time we can arrest the pathophysiological process? How can we return circulation, or cease inflammation? Both parties do ask, what caused the inflammation, or lack of circulation.

I do believe that only God (read nature if you prefer) can heal, but I also believe that God provides us with the knowledge to establish an environment that is conducive to his healing. It is this given knowledge that empowers us with the ability to develop a protocol to aid in the healing process. Our knowledge must be based on our understanding of the *root causes*, and the *overall effects of the pathophysiological process* and not on a simplistic view of the symptoms associated with the condition.

You may be saying to yourself; in both examples they are asking "why" and you are correct. The true nature of the problem is that *they are asking the wrong why question*.

Maybe this will help. Both your natural trimmer and veterinarian are going on the premise that the integrity of the lamellae is responsible for maintaining the position of the coffin bone within the hoof capsule.

Your veterinarian has been taught that with inflammation and the loss of circulation to the lamellae (laminitis), the integrity of the attachment to the coffin bone is also lost. The pull of the deep flexor tendon is thought to be a cause of rotation when this occurs. Rotation increases damage to the lamellae, and to the solar dermis from pressure necrosis as a result of this downward rotation of the coffin bone. This is why they elect to utilize the *wedges, to relieve the pull* of the deep digital flexor tendon, DDFT. The packing is to protect the sole, and to *support the internal structures*, in hope that they can figure out how to eliminate the inflammation and return normal circulation to slow the degradation of the lamellae attachment before any further rotation occurs. This is also why your vet suggested stall rest, this in hope of *reducing the induced stresses created by movement*. Let's recap the conventional veterinarian perspective. Current belief is that lamellae integrity is responsible for the position of the coffin bone within the hoof capsule. Laminitis (inflammation of the lamellae and / or loss of circulation) is the result of a metabolic insult of some sort.

There are two popular theories that support the conventional veterinary medicine perspective; the edema theory, and the ischemia theory. These are conventional veterinary medicine's answer to their "why" question.

1. The edema theory states that inflammation caused by a metabolic insult results in the reduction of circulation to the lamella, leading to loss of integrity of lamellae attachment due to death of cells within the lamellae.
2. The ischemia (deficiency of blood supply) theory states that there is a functional constriction, or diversion of blood to the lamellae, resulting from a pathophysiological process. This process is often referred to as AVA shunting, (arteriovenous anastomosis), where blood is shunted from the artery directly to a vein, bypassing the capillaries. It is believed that this occurs at the coronary band, in the toe region of the equine foot with acute laminitis. The end result death of the cells within the lamellae.

A third theory was introduced by Chris Pollitt, BVSc, PhD of Australia.

3. The Haematogenous Laminitis Trigger Theory states that high concentrations of blood born factors are cause for the triggering of laminitis. He states that his studies opened up the possibility that laminitis could be caused by the arrival in the foot of blood borne laminitis trigger factors (LTF's) that activated constituent enzyme systems that in turn *attacked individual connective tissue elements*.[2]

[2] Pollitt C., Laminitis & Diseases of the Foot, 3rd Int. Equine Conference, Palm Beach, Florida, 2005; 52

While the Natural practitioner may subscribe to one, both or all three of these theories in an attempt to validate their own "how" to approach, there are additional theories on cause that some advocates of the natural approach to laminitis and founder (rotation) subscribe to.

1. Mechanical forces acting on the coffin, often present for a long time before laminitis was triggered causes separation of the coffin bone from the hoof wall. These forces are said to result from imbalance of the hoof capsule; high heels, wedge pads, and excessively short toes are said to create such forces.
2. A *lack of nutrition resulting from impaired circulation.* Which is said to have several causes including; lack of movement, high angle of hoof causing a pinching of the digital arteries about the navicular bone, constant pathogenic pressure on the corium do to hoof capsule contraction, and increased vibration (from shoes) causing capillary damage (Raynaud's syndrome) It is said that often many of these causes are present at one time.[3]

Regardless of the combinations of the causes listed above, the primary cause is said to be a lack of circulation and as a result the "How to" protocol is developed based on this premise.

Your natural trimmer based her "How to" protocol on the above listed beliefs. She suggested removal of the remedial shoes, because the wedge pads place the coffin bone at to high an angle. She suggests walking the horse each day to increase circulation through movement. She is aware that with lack of healthy structure of the sole, the functions of the foot are compromised. She feels the boot is needed for protection.

Both conventional veterinary medicine and natural hoof care practice start with a model of the equine foot. The two are decisively different, yet both are rooted in static mechanics. Static mechanics is the branch of mechanics that describes two bodies which are acted upon by balanced forces and torques so that they remain in uniform motion. Utilizing static mechanics as a foundation has forced the use of fixed levers (bone) and semi rigid (horn) structures to facilitate the development of a workable model. That is what we mean when we say that the conventional farrier practice views the skeletal, and tendon structures as levers and pulleys. There is a place for static mechanics in the study of the equine foot, but we must delve far beyond static mechanics to the study of Dynamics (The branch of mechanics that is concerned with the effects of force on the motion of a body) and to Energetics (Study of the flow and transformation of energy). In the practice of Applied Equine Podiatry, we have utilized each of the above named branches of science to develop a model for the treatment of the equine foot.

Let's continue by defining how AEP views Laminitis and the possible causes of the above described pathology.

Laminitis (Equine Digital Elastosis)

AEP views laminitis (inflammation of the corium of the lamellae) as a symptom, not as a defined disease process. Our research has taken us down a different road, one that leads to

[3] H.Strasser, S. Kells, Hoof Orthopedics and Holistic Lameness Rehabilitation, Qualicum Beach, BC Canada, S. Kells, 2001

Equine Digital Elastosis (degradation of connectivetissue of the equine foot) or EDE. EDE is viewed as the primary cause for the pathophysiological changes we observe in the equine foot that conventional veterinary medicine diagnosis as acute laminitis. The *Equine Digital Elastosis Theory* is supported by the Haematogenous Laminitis Trigger Factor Theory, in that blood borne triggers activate constituent enzyme systems that in turn attack individual connective tissue elements. The root cause in each theory is likely to be the same, however our investigations are governed by *physiological sequencing* and *how the resulting loss of elastic potential effects dynamic equilibrium of function within the foot.* Unlike conventional veterinary medicine, I do not feel that the degradation of the lamellae and their dermal to epidermal union to be responsible for rotation, this is because of my understanding that the lamellae are not the primary structure responsible for the suspension of the coffin bone within the hoof capsule.

Stress induced Equine Digital Elastosis

As stated earlier, many of our domestic horses have a predisposition to hoof deformities. Most domestic horses suffer from some form of metabolic stress; it is simply the nature of the beast. Environmental influences weigh heavy on our horses. Metabolic disturbances can and often do trigger Elastosis (degradation of connective tissue).[4] Severe bouts of Elastosis can easily be construed as a component of Laminitis by conventional veterinary medicine. Some of the triggers of laminitis are believed to be; slight increase in normal blood protein levels, lack of essential amino acids, forcing a horse to metabolize muscle tissue to obtain the necessary amino acids, releasing excess protein into the blood stream; hormonal changes, such as those that occur during the birthing process; toxin from poisonous plants, or chemicals (vaccines, steroids, and wormers); long term fasting (neglect) or abrupt changes in diet[5]. Changes in Glucose metabolism could play a significant role in the development of Elastosis[6]. The major feature of acute metabolic stress that attends acute fulminating diseases such as colitis, metritis, and carbohydrate alimentary overload is the reduction in glucose consumption in many peripheral tissues. The purpose of this change is to maintain glucose, and therefore energy supplies to the vital organs, this at the expense of other tissues.[7] Metabolic stress is regulated by the hormones insulin, glucagon, cortisol, and adrenaline. Insulin promotes glucose utilization; glucagon promotes glucose production, especially by the *liver*. Cortisol and adrenaline promote glucose production from other substrates and *reduce glucose consumption in peripheral tissues* such as skin, and Horn. Conventional veterinary medicine cites decreasing local concentrations of glucose as a cause for lamellae separation, going as far as to acknowledge that *other epithelia may be similarly weakened*. The *EDE theory* sites the *weakening support of the ungular cartilage* through (loss of elastic potential) to be the primary reason for displacement of the Internal Arch

[4] Blood D.C., Studdert V.P., Gay C.C., Comprehensive Veterinary Dictionary, 3rd edition, London, Saunders, 2007;594
[5] H.Strasser, S. Kells, Hoof Orthopedics and Holistic Lameness Rehabilitation, Qualicum Beach, BC Canada, S. Kells, 2001
[6] Pollitt C., Laminitis & Diseases of the Foot, 3rd Int. Equine Conference, Palm Beach, Florida, 2005; 53
[7] M.W. Ross, S.J. Dyson, Diagnosis and Management of Lameness in the Horse, St Louis, Mo., 2003; 34:328

Apparatus; this displacement can range from a chronic stressing of the supportive and suspensory structures of the foot, leading to deformity, to an assessable rotation of the Internal Arch Apparatus. Metabolic imbalances could result in a chronic condition characterized by mild Elastosis, with the horse exhibiting intermittent sub-clinical lameness. A manifestation of hoof deformity is the first sign of such an imbalance, largely because of the mechanical forces generated on the coronary band / ungular cartilages by weight bearing. A slow progressive change in the conformation of the ungular cartilages due to mild chronic Elastosis would result in having the metabolically challenged horse prone to acute bouts of laminitis (stress induced inflammation of the lamellae). In such cases, any further metabolic disturbances could easily shatter the fragile balance and trigger EDE, resulting in marked conformational changes to the ungular cartilages. Severe metabolic disturbances resulting in the loss of elasticity to the foundation of the coronary band (ungular cartilages) in the caudal region of the foot could also result in rapid rotation of the Internal Arch Apparatus, this occurring in only a matter of hours. Those horses displaying deformity of the coronary band and ungular cartilages (DHS) have more often been sufferers of mild Elastosis, taking place over months or even years.

Diagnosis

Stages of Elastosis (Conventionally viewed as Laminitis)

1. Developmental Phase: Starts when the horse is exposed to the causative agent and ends with the first signs of lameness. This is the phase when load forces upon those structures exhibiting loss of elasticity may begin to show signs of conformation change. The induced stress results in the strain of lamellar integrity, though no signs of foot pain exist. • This phase lasts between 24 and 60 hours.

2. Acute Phase: Begins when clinical changes occur in the horse's feet. Bounding digital pulse, heat, sensitivity to hoof testers, and lameness, the "Camped in Front" stance may be seen. This phase starts with the first signs of lameness and continues until 72 hours have passed without physical or radiographic evidence of mechanical collapse of the foot or conformational changes to the caudle foot. There is an abrupt ending of the acute phase with foot collapse (IAA displacement or sinking of the bone column.)

3. Sub acute Phase: This is a mild period with less severe clinical signs, and without mechanical failure of the foot. Often ends without observable conformational changes of the ungular cartilages, or permanent connective tissue damage. This is the ideal resolution of the acute phase.

4. Refractory Phase: Does not respond, or is minimally responsive to treatment within 7-10 days.

5. Chronic Phase: Begins with visibly noticeable conformational changes of the proximal aspect of the palmar ungular cartilages, accompanied by the separation of the pedal bone from the dorsal hoof wall. The chronic phase can last indefinitely with clinical signs ranging from:

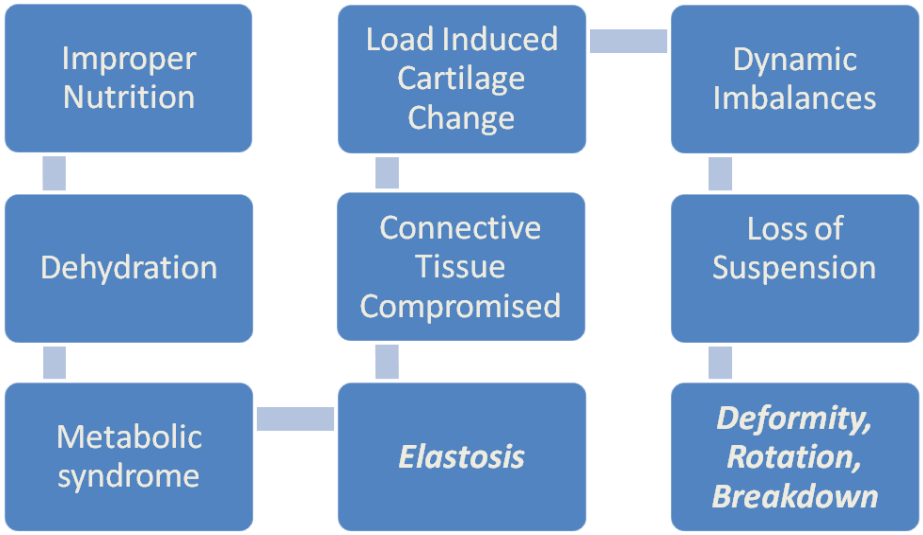
continuous mild lameness, severe foot pain, continued degeneration of the connective tissues accompanied by conformational change of the caudal aspect of the foot (ungular cartilage) and Hoof wall deformation. In severe cases, sloughing of the hoof capsule can occur.

Sub phases of the Chronic Phase

a. Early Chronic: Resolves or continues for months.

b. Chronic Active: Recurrence of pain after a period of improvement. Chronic reoccurring abscesses are common.

c. Chronic Stable: IAA is stable. Hoof and Sole grow is healthy, with steady clinical improvement. The Applied Equine Podiatrist proceeds with the understanding that the foot has undergone a change to the conformation of its foundations.



Chronic Equine Digital Elastosis

Displacement of the Internal Arch Apparatus can be evidenced in any number of hoof deformities. How the IAA is displaced is dependent upon the forces applied to the supporting structures over the period of time in which a loss of elasticity exists. This type of deformity should not be confused with tubule migration of the sole and wall, or flares. True IAA displacement is evidenced by a displaced coronary band, and a change in the normal conformation of the ungular cartilages. In long standing cases involving mild to moderate elastosis IAA displacement can result in separation of the laminar dermis/epidermis union. This *separation can be symmetrical, occurring in the frontal area, or asymmetrically involving lateral structures* i.e. quarters. With a symmetrical displacement, we often see that the bones remain in

alignment (often viewed by conventional veterinary medicine as hoof capsule rotation). Separations of the dermal/epidermal junction with displacement of P3 within the IAA, seen on x-ray as misalignment of the coffin joint, is viewed by conventional veterinary medicine as Founder. In such cases close observation of joint spacing, and the conformation of P3 is crucial.

The foot stricken with asymmetrical displacement of the IAA exhibit greater hoof capsule distortion, often accompanied by lateral imbalances within the coffin joint. Uneven compression rings evidenced in the hoof wall provide detailed information as to how the coronary band has been loaded, and to what effect this has had on the conformation of the ungular cartilages.

Growth rings in the toe of the foundered horse indicate that the health of the Stratum Internum is poor. Incorrect growth of the stratum internum results in a poor matrix of coronary band and lamellae derived epidermal cells in the area just distal to the coronary band. When sub-clinical Equine Digital Elastosis and/or laminitis are present, the growth of horn is altered. Intercellular spaces in the epidermis are enlarged and the process of keratinization is altered. Recent studies show that horn cells in the horse experiencing laminitis either acute or chronic contain less keratin, and deposition and organization of intercellular lipids is lost. This results in increased intercellular spaces in the horn. The barrier to the entry of micro-organisms is broken and white line infections will often result.[8]

In those horses stricken with laminitis, it is a common belief that heel growth is faster than toe growth; this because there is greater pressure exerted on the angle of the bar. This is likely the case, but the fact that the keratinization process is altered in the toe region also plays a role in the development of DHS. The mechanism for the downward progression of hoof wall is likely not as significantly affected in the heel region; this due to there being less mechanical stresses imparted on the lamellae dermis. In the heel, the forces are directed proximal to the coronary band and ungular cartilages, as opposed to the rotational stresses imposed on the lamella of the toe.

Heel growth is of major concern in those horses experiencing EDE. With a loss of elasticity the foot undergoes a loss of suspension. With a loss of suspension the downward forces exerted by the bone column can induce inflammation of the foot's solar dermis (dorsal solar). In an attempt to reduce pressure on the dorsal solar dermis, the horse will shift weight bearing to the heels and caudal aspect of the foot. The increased force on the hoof wall of the heels translates to excessive forces on the coronary band and its foundation the ungular cartilages. With increased pressure, we see increased growth, placing the ungular cartilages under increased force resulting in changes to their conformation. This is a vicious cycle that must be broken if we are to have any hope of treating the foot afflicted with EDE, Laminitis, Founder, and DHS.

Treatment

[8] Kempson S., Robb R. , Corrective Farrier, Newmarket Farrier Consultancy, Newmarket, UK., 2006; 436

Despite all of the misinformation, and misunderstanding surrounding laminitis, it is generally accepted that the resulting deformities of the foot are a secondary problem. AEP views the primary cause as loss of elasticity, and not circulation. The loss of circulation is a result of stresses induced by incorrect load and distortion. If you have not grasped the concept behind the displacement of the IAA due to a change in cartilage conformation please review the previous paragraphs concerning EDE.

Treatment of the horse stricken with EDE, Laminitis, Founder, and the subsequent DHS depends on your ability to determine the level of functionality present in the caudal foundations of the foot. Conventional veterinary medicine has made attempts to support the bone column through various means, including heart bar shoes, the Equine Digital Support System, Lilly pads, and too many variations of support pads to list. Many of these devices have merit, but only if the correct "why" question is being asked. To what end is this device being used?

Why does a heart bar shoe sometimes work? Why does the equine digital support system have a better success rate than the heart bar shoe? Why do some horses do better when bedded in deep shaving than others? Why do some horses do really well in foam pads alone? Answers to all of these questions lay in your understanding of the underlying cause of the changes in caudal foot conformation.

If you subscribe to heel load as being the dominant factor in conformational change to the caudal foot, it is quite simple to explain why each device may work some of the time. If for instance, a heart bar shoe is applied to the horse suffering elastosis, and the *frog support bar* transfers load away from the angle of the bar and places that load on the frog, then the forces responsible for stress induced changes to conformation are reduced. Increased stresses on the dorsal sensitive structures are also reduced, which may offer temporary relief from pain. I said temporary, this because hoof wall growth will result in the load being transferred back the angle of the bar.

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