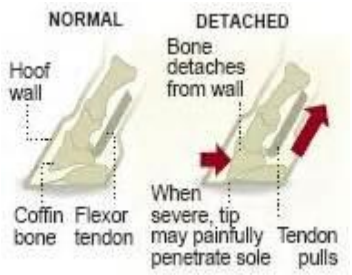
H BAR H VETERINARY CLINIC

NEWSLETTER

SUMMER 2013

Laminitis / Founder

INSIDE THIS ISSUE:



I have frequent conversations with clients and students regarding this devastating disease. It is the number two cause of death in horses, being beat out only by colic. Fifteen percent (15%) of horses will have a "bout" of laminitis in their lifetime. So we thought a review of current information would be in order.

In order to understand this disease process it is necessary to have an awareness of normal hoof anatomy and physiology. Inside the hard, normally protective hoof wall there is a bone called P3, the coffin bone or the pedal bone. This 3rd phalynx is attached to the inside of the hoof capsule by hundreds of thousands papilla called laminae. Laminae from the bone surface interdigitate with laminae from the inside of the hoof capsule much like "velcro." These little laminae have a huge job in carrying the entire weight of the horse on their "shoulders"…so they require large nutrition and oxygen delivery - through vast numbers of blood vessels. The Deep Digital Flexor Tendon (DDFT) attaches on the bottom of P3 and runs behind the navicular bone, and when muscles above the knee contract - this flexes the foot. This tendon pull becomes important with all these parts in the laminitis story we will discuss.

The back of the foot starts from the ground side with the frog which is a rubbery material that is the first part of the shock absorption apparatus of the foot. It is connected to the deeper digital cushion (fibrocartilage and fat) which continues to absorb shock and dissipate it as the foot is loaded. The final part of this shock system involves the collateral cartilages on both the inside and outside (medial and lateral) sides of the digital cushions. As load transmits to the back of the foot the frog pushes up, the digital cushion bulges out sideways (med and lat) and the collateral cartilages bow out and the large blood plexus (network of blood vessels in these cartilages) squirt blood up and out – further absorbing shock and concussion (like a built in "hydraulic shock").

The sole protects the bottom of the P3 and also prevents compression of the main feeder blood vessels that course under the foot from behind as they deliver blood up and around the coffin bone to the very important laminae.

All of the aforementioned structures work together to keep the horse sound as he or she does their job. If they are built really well and if we take care of them really well we can keep that horse sound for a lifetime.

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They also hold the coffin bone and the upper "tower of the body" on this amazing base of scaffolding. They also have a unique ability to release attachment normally in very small localized zones to ratchet down new wall growth and immediately reattach. All these laminae are attached to their opposites by simple chemical bonds (so the entire weight of the horse is held up by chemical bonds). When these laminae become inflamed, together with the weight of the horse and the pull of the DDFT, and the movement of the horse…these structures can fail and pull apart from one another. The bone can then rotate or sink inside the hoof capsule (pull away from the normal attachment – just like pulling velcro apart). When enough of the laminae fail to cause rotating or sinking of P3 we call this "founder." Blood vessels and nerves are traumatized; the blood supply running under the foot is compressed; the sole can be damaged and so on. It is very, very painful. Usually the front feet are more affected simply because the horse bears 65% of its weight on the front feet, but hind feet can also be affected.

The horse will be reluctant to walk, may lean back and try to load only the heel portion of the hoof. They get real painful when forced to turn. They might shift weight from one foot to the other. The hind legs often will be "camped under" to pick up some of the load off the front end of the horse (as well as load the back part of the hind feet). They will have a higher digital pulse intensity at the fetlock or pastern.

Normal Vital Signs of Adult Horse:

- Temp <101.5
- Pulse 36-40 bpm
- Resp 12-16 breaths/min
- Capillary Refill Time -- less than 2 seconds
- Mucous Membranes -- pale pink

Laminitis / Founder cont..

They can have many more symptoms of pain such as a higher heart rate, sweating, a stiff, pained look in face and eyes. Sometimes their lower legs might swell from inactivity and immobility. The degree of laminae damage will correlate, to some degree, with the severity of pain signs, the amount of tearing, rotating and/or sinking as well as the final outcome or prognosis.

The problem is that lots of the laminae damage and tearing occurs often times days before the actual pain symptoms occur. This is why it makes it so hard to treat…we don't have a chance to start medicating and preventing early enough!

Now let's talk briefly about the causes of laminitis, as we know them today:

As I mentioned earlier in this newsletter - endocrine problems such as EMS or PPID are probably the most common causes.

Spring or even late fall pasture associated laminitis may have an endocrine basis as above

SIRS - Systemic Inflammatory Response Syndrome. Simply means response to toxemic insult as in grain overload, or retained placenta infection in foaling mares, or severe illnesses such as colic or enteritis/colitis etc.

Black walnut extract ingestion or contact

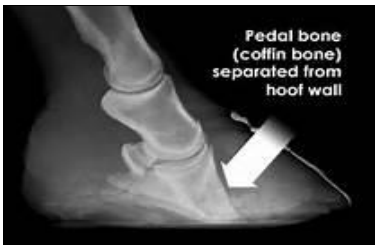
Supporting limb laminitis – as in "Barbaro." A horse cannot stand on 3 legs for a prolonged period of time. If they are forced to, the greatest risk is usually in the "good leg"

Road founder – excessive concussion to foot or hard ground (rare in my experience).

Supplements such as Biotin/Methionine can be helpful — but addressing primary cause is critical!

Treating laminitis/founder is a big commitment that can go on for a year or for the rest of the horse's life. Much more prudent to try to avoid this horrid disease…prevention is indeed worth tons of a cure.

The more you know -- the better at prevention you can be for your beloved friend…your horse!



Rabies: To Vaccinate or Not

At a recent local vet meeting, we discussed vaccination for rabies. It is on the AAEPôs (American Association of Equine Practitioners) ñcoreò vaccination list. It is in Idaho (at least at this point) at a very low incidence level (last horse case was in 1999); but it has a 100% mortality. So vaccination protection is the only way to prevent if exposed. Also, if a horse is infected it can expose you (and other people) to this terrible disease.

It is moving this way. Increased cases in Texas and now Colorado. Vaccination one time per year provides good immunity. Pregnant mares can be vaccinated prior to breeding or 4 to 6 months prior to foaling. Foals can be vaccinated at 6 months and a booster at 7 months of age.

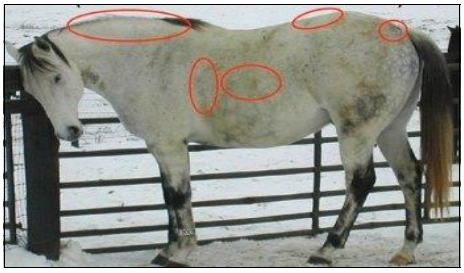
It is a good idea to vaccinate for rabies if traveling out of state to Colorado or Texas. The vaccine seems to have a low incidence of reactions.

With this update you can make a better, informed decision as to whether you will choose to have rabies included in your spring shots.

EMS– Equine Metabolic Disease

- Higher incidence to get PPID (Cushing's Disease)
- 51% of horses in the United States are obese
- Not all fat horses have EMS
- Some thin horses can have EMS
- Measuring insulin is good baseline screen
- 90% of laminitis cases have an endocrinopathy (metabolic problem) and over 1/3 of these horses will have new "bouts" with out being addressed

Makes it a good idea to check for underlying endocrine disease like EMS or PPID (Cushing's).



PSYLLIUM 20# Bucket $105

40# Bucket $185

STRONGID C 25# Bucket $65

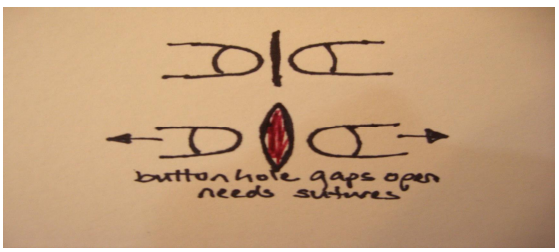
ATTENTION!!!!!

We have the best local prices on Strongid C Daily Wormer and "Psyllium" Sand Treatment!

BUTTON HOLE TEST

This is a good and useful test to determine if the wound your horse just got will require sutures or not.

- First, clean wound — just use soap and water
- It helps to clip hair around wound (for cleanliness and to evaluate better)
- Just like a button hole on your shirt — if you place a finger on each side of the wound and pull away from each other — a wound completely through the skin will gap open. These will need sutures.
- If it does not gap open — will not need sutures… just proper wound care.



SCRATCHES

A nasty, fairly common, dermatologic condition of the skin on the pastern, fetlock and even cannon region. More commonly affects white haired legs (pink skin) but can also affect other coat colors. This term, along with other pseudonyms such as pastern dermatitis, grease heel, or mud fever really doesn't point to a single causative agent or disease but rather to clinical symptoms that can be caused by a number of different etologies (or causes). So lets start with what it looks like.

The skin is flaky, reddened, sensitive, and often swollen. Many times it is first noticed when plaques (or areas) of raised, crusty, scabby lesions are present. Removing these scabs is often painful (sometimes requires sedation) and the underlying tissue appears raw and ulcerated, is oozing, red and swollen.

As stated above, there are numerous causes for this clinical picture. Perhaps one of the most common underlying causes is a severe chapping of the skin. This chapping can be brought about by several common environmental situations. Wet, as in wet dewey grasses, muddy mucky paddocks, manure and urine soaked shavings in stalls all seem to set some horses up for getting this chapping. Photosensitivity, as in UV rays from sunlight can also damage the skin and set it up to become inflamed. There are, in fact, chemical substances in many grasses and even in alfalfa that can increase a horses' susceptibility to UV radiation. Not all cases of scratches start out this way, but a vast number that we see, do. There are other, more rare causes such as pemphigus (auto immune), habronemiosis (parasitic), sporotrichosis (fungal), chorioptic mange (ectoparasitic), dermatophytosis (fungal ringworm), dermatophydosis (rain rot bacteria infection), photosensitivity vasculitis, bacterial folliculitis or even contact dermatitis (reaction to direct contact with substance that is allergic or irritant). Because there are so many "other" causes, it sometimes becomes necessary to search for the diagnosis with cultures, even biopsies of skin. But, typically most cases can be treated as if they were a chapping of the skin that may have gotten a little out of control.

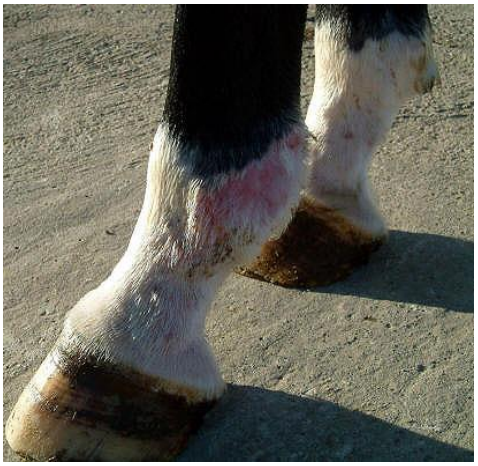
So to start with, when we have a case of scratches we may try treatment and see if we get a favorable response prior to spending money on diagnostic tests that may not be needed, and may not reveal a true etiology. Our first priority would be to remove the causative condition . . i.e. if we feel "wet" is a problem – remove it. Sometimes that means not turning the horse out on wet dewy pasture in the AM, you may need to wait until the afternoon when the dew is gone. You may have to reduce or even eliminate certain horses from being out on the pasture entirely. If it is a paddock mucky scene – clean manure daily, provide good drainage, put in raised dry areas that are matted. Clean stalls regularly and see if horse can get out of damp stall settings (even well cleaned stalls tend to get "damp" and oftentimes you can smell urine – urine will irritate pastern skin as well).

Once you have gotten rid of the "cause," you can begin to treat the actual damaged skin. We like to clip the hair from the affected area so that the oozing serum (coming from the damaged skin barrier) doesn't cling or cement to the hair which is painful and harder to clean. We then like to gently clean and debride this raw skin with an antibacterial soap (actually anti viral and anti fungal as well; in case there is any secondary infection from any of these agents.) Gentle persistent cleaning and removal of scabs with Chlorhexidine is the easiest on the skin (we stay away from betadyne or povidone iodine based shampoos, as it is a great antibacterial agent but a little hard on skin on a repeated basis). Once the skin is clipped, cleaned, debrided, and washed with Chlorhexadine, we rinse, rinse, rinse until all the soap residue is gone and then dry thoroughly. This whole process is aimed at decreasing the number of potential secondary infectious agents. Also, it will allow the skin to "breathe" briefly. Many times if you are left with raw, sensitive and swollen tissue -- it is best to bandage these areas until you get ahead of the pain and the skin begins to repair. (This can actually take repeated bandage changes until it can go into the "open air" again .)

We use a couple of different ointments on non-adherent telfa pads as follows. (Please note that there are many different "medicines" or treatments that have and can be used as well.) We like Dermalone because of its antibacterial/antifungal and anti-inflammatory properties mixed with a zinc compound to encourage healing. Bandages also serve to keep U/V light from bothering this tissue for a while. Once you see progress you can sometimes move to a couple of shampoos a week, keep dry and +/- with topical medications.

If this type of protocol is not working well, you can go to further diagnostics – such as biopsy. You must be flexible when treating scratches because it can be simple to very complex on each individual horse. Some cases may require systemic antibiotics and/or steroids.

Once again, like most things in life, prevention is real important (once you have gone through treating a nasty case in both time, effort and expense). Avoid the wet, keep legs clean and dry; keep bandages, shipping boots and grooming instruments clean. Inspect legs frequently for early signs of flaky, crusty lesions that might tip you off to an early case. Heavily "feathered" legs where the skin can't breathe because of long thick hair covering them need special attention. (If you can't clip feathers off – inspect, clean and dry regularly). Some people even go so far as to avoid all that "chrome" (white lower legs).



HAPPY SUMMER FROM H BAR H VET CLINIC!