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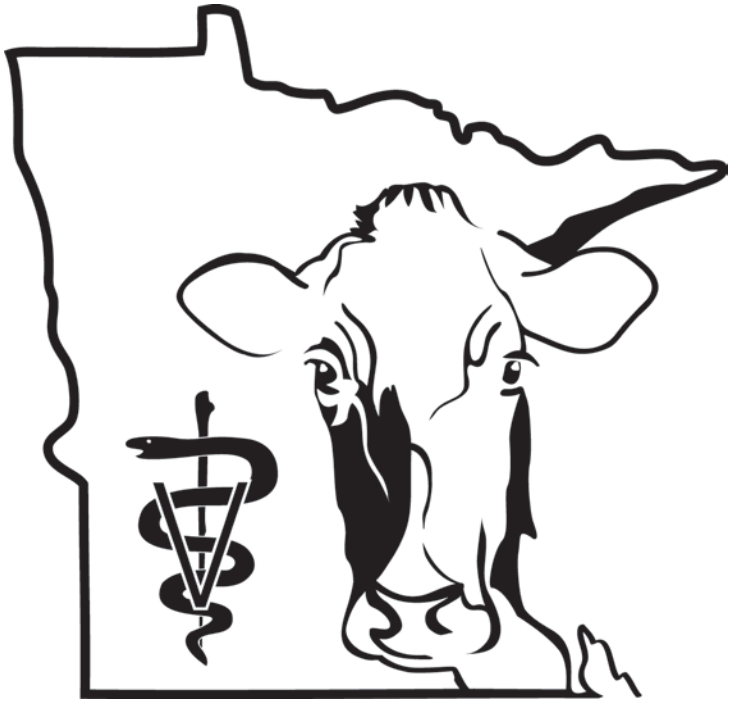

ST. PAUL, MINNESOTA
UNITED STATES OF MINNESOTA

FUNCTIONAL ANATOMY OF CATTLE DIGITS: WHAT GOES WRONG THAT RESULTS IN LAMENESS

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The structures of the bovine digit that support her body weight and work in normal locomotion can and do become diseased in predictable fashion. The bones, tendons, ligaments, corium, and hooves are all potentially involved when things go wrong. A better understanding of the biomechanical relationships in the digits of cattle can help with more rational therapeutics and appropriate preventive maintenance. The things that go wrong with the digits from a mechanical perspective are related to the environmental influences of moisture, infection pressure, and standing surfaces. The distance walked and the characteristics of the substrate walked on also result in predictable problems. This paper will describe the interplay between external forces and physiologic and pathologic events in the digits.

What we see as the current condition of the claws on any cow is a result of continuous growth, continuous wear, and intermittent trimming. The growth rate of hooves is relatively constant but subject to minor modifications. Nutrition can influence hoof growth rate. Hooves do not grow as fast during starvation as during adequate feeding. Since dairy cattle are never intentionally starved this effect is unimportant. A small variation occurs during the lactation/gestation cycle and with season. The reports on this effect are difficult to interpret since the cattle calved seasonally. In a study we conducted at Cornell on mid-lactation cows, growth rate was influenced by floor surface within the freestall pens. Cows grew hoof faster on concrete than rubber floors. The implication is that hoof growth can respond to environmental conditions by making more hoof when the standing or walking environment is more mechanically insulting. Typical growth rates are about 6 mm of hoof wall per month with variations due to environment less than 10% of control rates. Wear rates are much more variable and depend on the abrasiveness of the walking surface and distance walked. Data is not available to compare the wear rates of dry versus wet hooves on the same walking surfaces.

Why do we see more disease in rear feet than fore? Why is there more disease in lateral rear claws than medial? The fore versus rear argument in dairy cattle has 2 components. The rear limbs of dairy cattle are forced to carry weight in excess of the original design criteria. The wild-type cow which gave us our modern dairy cows never had a large udder, even at calving. As we have selected for more milk production, I do not think we have been able to simultaneously modify the musculoskeletal system to accomodate this extra weight at the rear of the cow. Secondly, rear feet are always more exposed to the bacteria and moisture of manure and urine. The skin near the hooves is more likely infected with bacteria due to maceration by this moisture and the hoof capsule is softer due to greater hydration. The medial versus lateral argument is potentially more confusing. Lateral claws grow about 10% faster than medial claws and wear about 8% more in freestall housed Holsteins. Thus lateral claws can progressively outgrow medial claws. They are larger even in fetal calves. Larger lateral claws are more heavily loaded than medial claws. Larger loads result in more potential for mechanical insults. Cows may

adopt a toed-out posture to help equilibrate the weight between the rear claws when overgrowth and some discomfort occur. This toed-out posture or being "cow hocked" can be used as an indicator of the need to trim an individual cow or by population evaluation when a group or herd needs trimming.

Body weight is supported by the column of digital bones resulting in the load being approximately evenly divided between the eight digits with normal claws and conformation. The third phalanx or P3 is the end of these columns. The load on P3 is supported by several structures of importance in our concern for lameness. There are laminae in the mural corium, which is tightly attached to lateral and cranial portions of P3, that interdigitate with laminae in the hoof wall. These have less surface area of mutual contact per unit of supported weight than in the horse. Therefore the laminar region of the bovine digit while very important is not as signifigant as in the horse for support. There is also support of P3 by ligaments that suspend the caudal portion of the bone and blend with the interdigital cruciate ligaments axially and with the laminar corium abaxially. The tension of the deep flexor tendon on P3, in addition to fixing the bony column in a nearly vertical formation, pulls the distal tip of P3 ventrally and transfers some weight forward in the claw. Between P3 and the solar corium is a complex arrangement of fat deposits that cushion and distribute weight transferred to the sole. The fat pad is thickest at the heel and plays a dynamic role in cushioning during walking.

The structure of the horny capsule of the claw is different in different regions. Everyone who has trimmed a hoof knows this. The wall horn is the most rigid and hardest. It has the hightest density of horn tubules that are arranged in parallel and develop from papillae in the coronary corium. The horn tubules are much less numerous in the sole which makes it more flexible. The horn of the heel has the lowest density of horn tubules and it is very pliable. The only non-tubular horn of the claw is called cap horn and is produced at the distal ends of the laminae. It serves to cement the sole to the wall and is visually identified as the white line. This cap horn seem particularly vulnerable to the effects of laminitis. It may fail mechanically allowing the wall and sole to separate or fall out in portions of the white line allowing entry of foreign matter. All of the horny tissue of the claw is able to absorb water. The higher the water content, the softer and more flexible the horn. The most noticeable effect of continuous hydration of the claw is in the sole. The horn of the sole flakes away leaving a concave surface and a relatively thin and constant thickness sole when the hoof is dry most of the time. This occurs by slight contraction of the cells of the sole during dessication and fracture along horizontal planes during walking. Under dry environmental conditions the moisture within the sole is derived from the corium which diffuses at a constant rate into the sole resulting in a constant thickness. In contrast, in freestall housed cows where slurry maintains the sole continuously moist the sole does not flake away and it must be worn or trimmed away.

When the events commonly known as laminitis occur, the vascular disturbance affects the corium of the laminae and the non-laminar corium as well. The edema and resulting swelling reduces the ability of normal circulatory dynamics to oxygenate the corium. Some anoxic damage may occur. The mechanical properties of the corium that have suffered the distortion due to the edema and the biochemical changes due to anoxia can be altered with the result of lower tensile strength. As a consequence, P3 may move within the horny capsule beyond the

limits occurring in healthy claws. With the exaggerated movement of P3 2 specific lesions may develop. If P3 moves closer to the sole, abnormal pressure may cause further anoxic damage to the solar corium. If mild, this may appear as hemorrhage in the sole at a later trimming. If severe, the solar corium may die and result in a sole ulcer. In housed cattle the ulcer is most common at the caudal portion of P3 in proximity to the flexor tuberosity where the subcorial fat pad is thin and in extensively managed cattle it more often occurs under the distal portion of P3. The abnormal movement of P3 relative to the laminar corium may result in ruptured blood vessels that leads to hemorrhage. If the hemorrage is very mild it is later seen as a red line in the sole at the sole-wall junction or white line. If the hemorrage is more extensive, it can result in a hematomma that later becomes either a sterile abscess or a septic abscess if the white line is separated and permits entry of environmental bacteria. Cattle that must make sharp turns on rough flooring may experience more white line lesions due to the lateral forces placed on the wall. During a turn there may be claw deformation that can pull the wall from the corium or shear the corium if the structural integrity of the tissue is already compromised by edema.

It is important to note that the lesions of the corium that we recognize as and call laminitis require weight bearing during the period of primary damage to the corium. No one knows how long episodes of altered permeability and edema last following the chemical messengers from ruminal acidosis. However, if the cow did not stand during that period, there would be no mechanical damage to the corium. In the vast majority of cases, the lesions within the claw that we call laminitis are the consequence of standing or walking on damaged corium. Standing is perhaps a worse insult to the corium than walking. With each step there is normal movement of P3 within the horny capsule of the claw. This movement results in periodic perfusion of parts of the corium. When a cow stands without shifting her weight these periodic changes in blood flow within the corium are probably interrupted. Thus standing motionless is potentially more damaging to an already insulted corium than walking. Only in the rarest cases do the lesions of laminits actually develop while cows are lying. Thus the great stress in recent years on cow comfort and maximizing opportunities through time management and providing attractive lying surfaces are actually anti-laminitis efforts.

Claws with abnormal shape, particularly of the ground contact surfaces are more prone to mechanical insult to the corium. This is most commonly seen when excess horn production occurs at the axial border of the claws near the heel. The horn is probably being produced at an accelerated rate by this portion of the sole in response to stimulation by chronic dermatitis caused by *Dichelobacter nodosus* which is recognized to cause skin hypertrophy of the heels and interdigitally. Unfortunately, this site on the sole where an excess rate of horn production is observed is also that of the common sole ulcer. During weight bearing the corium deep to the horn buildup will be compressed in a fashion similar to when P3 movement within the claw is excessive.

Complications of the simpler lesions of the white line may occur when the pressure accumulating within the space between the hoof wall and the mural corium is not released to the exterior. The pressure within the abscess may be great enough to dissect along whatever path presents the least resistance. This may be proximally to the coronary band, axially across the sole, or caudally

under the heel. Such abscesses result in greater disruption of the mechanical stability of the claw and the necessity of more horn removal.

Complications of sole ulcer are due to extension of necrosis to the nearby structures around the coffin joint. The navicular bursa, deep flexor tendon, and coffin joint are all at risk of sepsis from free entry of bacteria through devitalized tissue. It is unclear whether necrosis of these connective tissue structures must precede invasion by bacteria or whether bacterial infection of a sole ulcer can proceed to extend into these other tissues if they are healthy.

Abnormally shaped claws may develop from genetic traits such as corkscrew claw or secondary to chronic or recurrent episodes of laminitis. The laminitic changes may manifest as concavity of the dorsal hoof wall due to displacement of the wall from the corium. Weakness of the capsule-corium attachment or corium-P3 attachment in conjunction with mechanical pressure on the hoof at the toe tip can result in turning up of the toe. Care must be taken in trimming of these claws since Dutch rules will not work unless the dorsal wall is first straightened. The thickening of the white line may be more widely distributed around its entirety resulting in laterally flared claws. The claw capsule may also seem to twist on its long axis due to laminitis resulting in so-called screw claw although this is distinct from the genetically controlled condition. All of these claw shape abnormalities are likely at least uncomfortable for the cow to walk and stand on. In addition, they predispose to more severe lameness conditions due to abnormal load