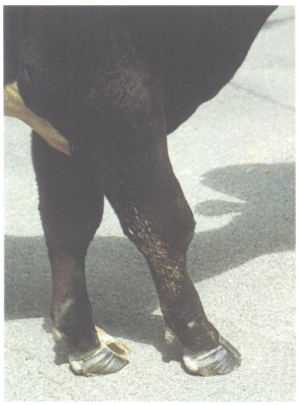

Case of acute laminitis. The cow has adopted a cross-legged stance is an effort to take the weight off the inner claws. Despite the pain, there are no visible lesions in the hooves at this stage

Bovine laminitis: the lesions and their pathogenesis

PETE OSSENT AND CHRISTOPH LISCHER

CAREFUL postmortem examination and interpretation of findings, assisted by good clinical records, does much to throw light on the pathogenesis and nature of hoof disorders in farm animals and horses. Although there is much speculation surrounding the pathogenesis of laminitis in cattle, the principal mechanisms have largely been determined. These mechanisms and their associated lesions are described in this article.

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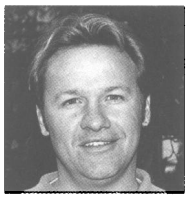

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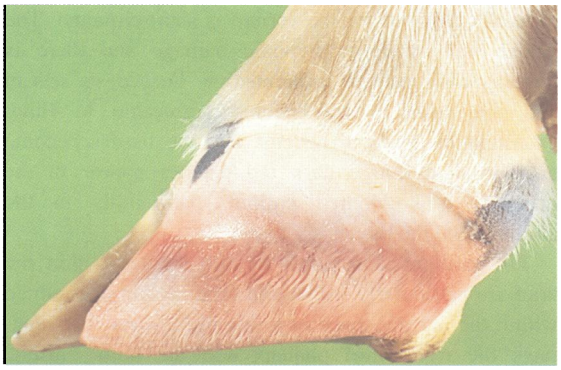
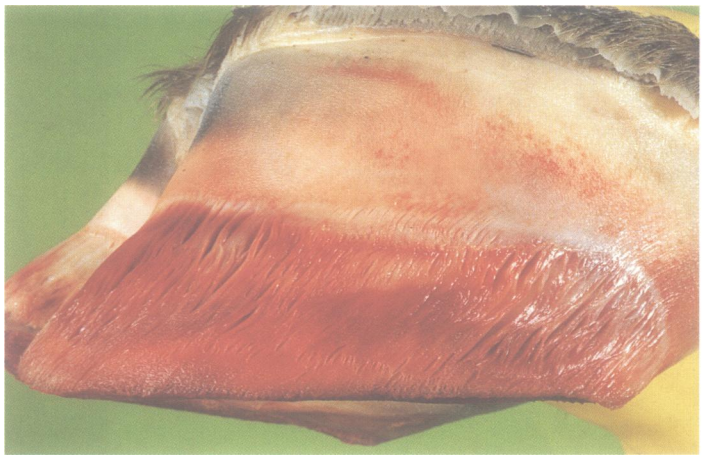
PHASES IN THE PATHOGENESIS OF BOVINE LAMINITIS

An earlier article, which described the techniques for postmortem examination of hooves (Ossent and Lischer 1997), explained how removal of the horn capsule can reveal a wide spectrum of changes in the wall and sole of the corium, as well as inside the horn capsule and within the horn itself, long before they become clinically apparent. There are strong indications that the crucial mechanism in the pathogenesis of laminitis in cattle centres on changes at the dermal-epidermal junction in the hoof. The resulting lesions range from impaired horn production, with diffuse softening, discolouration and haemorrhages in the horn of the sole and heel and the white line; through the gradual development of double soles, heels and walls, with separation of the white line; to (eventually) the formation of ulcers in the sole and heel. In even more chronic cases there is a deformation of the whole claw. These changes, which until quite recently were regarded as separate hoof conditions, are often sequels of laminitis. One lesion may develop into another and what is encountered depends on which stage in the pathological process the examination takes place.

In order to gain an insight into the underlying mechanisms, it is useful to divide the pathogenesis of laminitis into three phases, as described on the following pages.



Normal hooves of a cow. The horn capsule has been removed and from the side view (above) the pink parietal and lamellar corium of the wall can be seen
Reproduced, with permission, from Lischer and others (1994)
(below) Ventral view. The corium of the sole and heel is pink or white. The inner surface of the horn capsule should be the same colour, with an intact firm surface. The walls have been removed to allow a better view

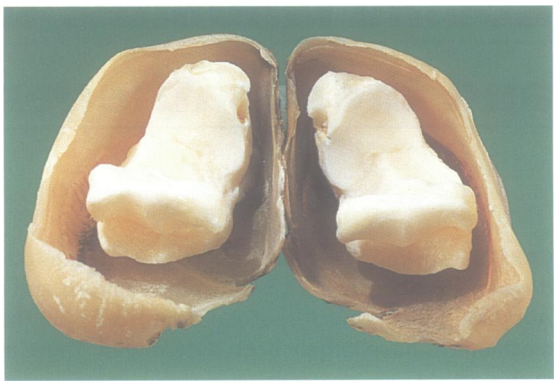


Phase 1: Congestion, oedema and haemorrhage. (above left) The lamellar region of the wall corium, in particular, is severely congested and oedematous and there are several haemorrhagic patches. Such discolourations are sometimes difficult to differentiate from the red patches caused by irregular congestion that may be encountered in perfectly normal hooves. In such cases it is important to correlate the postmortem findings with the clinical history. (above right) Large areas of the corium are intensely reddened and haemorrhagic. This case was so acute that not enough time elapsed for blood to discolour and to be incorporated into the corresponding inner surface of the horn capsule. (In this photograph the horn capsules were mistakenly arranged next to the wrong claw)

PHASE 1: VASCULAR DISTURBANCES IN THE CORIUM OF THE HOOF THAT LEAD TO DEGENERATION AT THE DERMAL-EPIDERMAL JUNCTION

The precise aetiological factors that induce laminitis in cattle remain controversial. It is widely accepted, however, that it is the release of systemic mediators (probably vasoactive substances) that triggers an intricate pathological response in the blood vessels in the corium of the hoof. The blood stagnates due to vessel wall paralysis and vasodilation. Haemostasis and congestion ensue, resulting in erythema of the corium which lasts the duration of the acute phase. The claw's arteriovenous shunts open and the main flow of blood normally destined for the corium is short-circuited. The vessel walls undergo hypoxic damage and there is possibly also reactive inflammation. Diapedesis sets in and the vessel walls become permeable to fluid. Transudation leads to oedema and the tissues become haemorrhagic. Large dark red patches appear in the normally pink-coloured corium, particularly in the laminae, and the tissues are moist.

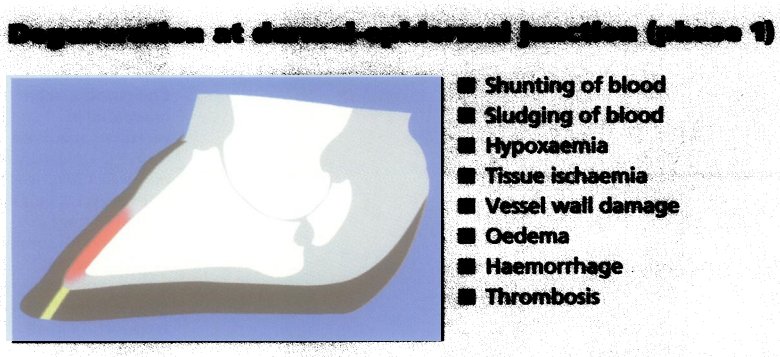
For anatomical reasons the soft tissues within the hoof are particularly vulnerable. The corium is confined within the narrow space of a few millimetres that separates the rigid third phalanx and the horn capsule. Oedema amplifies the tissue pressure within the horn capsule, causing intense pain in acutely affected animals since the corium is abundantly innervated. A vicious circle develops whereby the increased pressure within the horn capsule reduces the blood flow even further. Thrombi commonly occur.



The very narrow space that the corium occupies between the rigid bone of the distal phalanx and the horn capsule can be seen in these specimens of macerated hooves. It is easy to appreciate how oedema or displacement of the bone would lead to compression, pain and, eventually, lesions in the tissues
Reproduced, with permission, from Ossent and others (1997)

The horn-producing and proliferating basal epidermal cells are supplied with nutrients and oxygen from the blood vessels in the underlying dermis. The demands of these cells are higher than those of any other tissues in the vicinity. Impeded metabolic exchange and a reduced nutrient supply lead to the synthesis of inferior keratin and, thereby, an ultimate deterioration in the horn and loss of structural integrity at the dermal-epidermal junction. The junction in the interdigitating lamellar region of the wall is of particular anatomical importance. A significant proportion of the animal's weight is suspended from the hoof wall and the normal carrying function of the foot depends on the integrity of the these cell layers; a deterioration in the dermal-epidermal (lamellar-laminar) link would lead to a failure in this function. If the link begins to separate, the whole foot sinks within its horn capsule (as illustrated at the top of page 418).

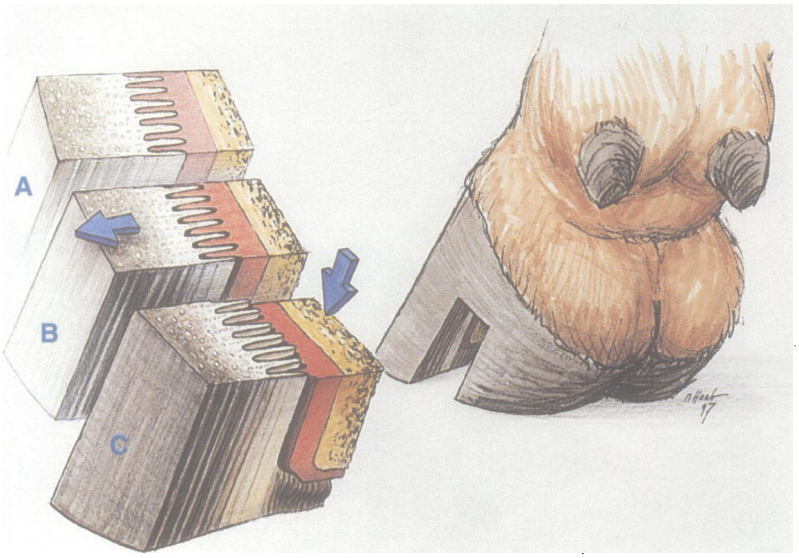
In cattle it is not clear how, and at what stage, inflammatory components contribute to these degenerative events or even whether they are necessarily involved at all.



Key: red = haemorrhage, yellow = white line

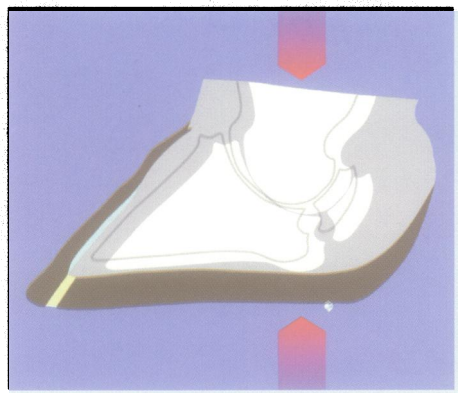
SUBCLINICAL LAMINITIS

The laminitic process may persist in phase 1 without sinkage to produce a very widespread chronic state, not associated with lameness, defined as 'subclinical laminitis'. In these cases, the horn is of inferior quality, diffusely softened and either yellow from transudate or discoloured by escaped blood that gradually forms a dark red white line or patches in the sole and heel.



(A) Normal hoof wall. A substantial part of the animal's weight is suspended from the hoof wall. Clearly the forces can only be transmitted to the wall, and thence to the ground, if the interdigitating lamellar structures of the horn and corium are intact. Grey = horn capsule, red = corium, yellow = distal phalanx. (B) Phase 1: Degeneration at the dermal-epidermal junction. Circulatory disturbances in the corium lead to structural changes in the carrying layers. Eventually the lamellar junction disrupts and the wall separates from the corium. (C) Phase 2: Sinkage of the distal phalanx. Once the degree of separation is too severe to suspend the weight of the animal, the distal phalanx sinks within the horn capsule and compresses the corium beneath it

Sinkage of the distal phalanx and compression of the corium in the sole and heel (phase 2)



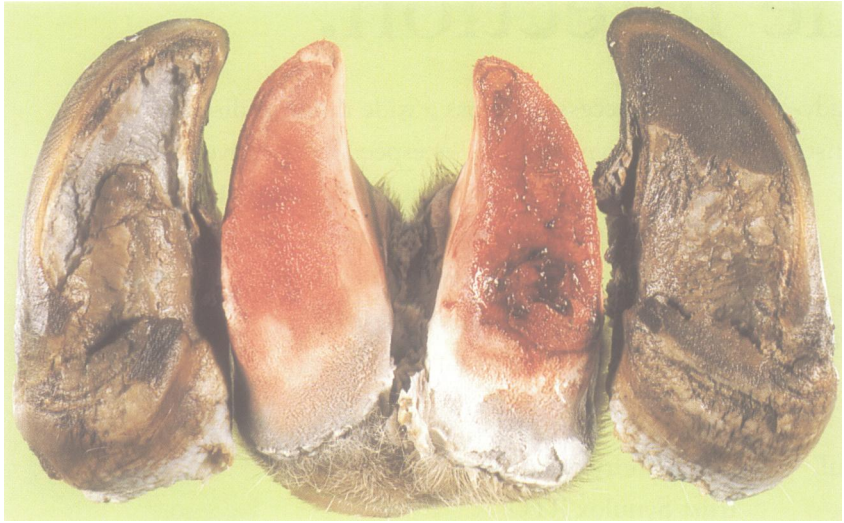
- Haemorrhage
- Thrombosis
- Oedema
- Ischaemic necrosis
- Inflammatory infiltration

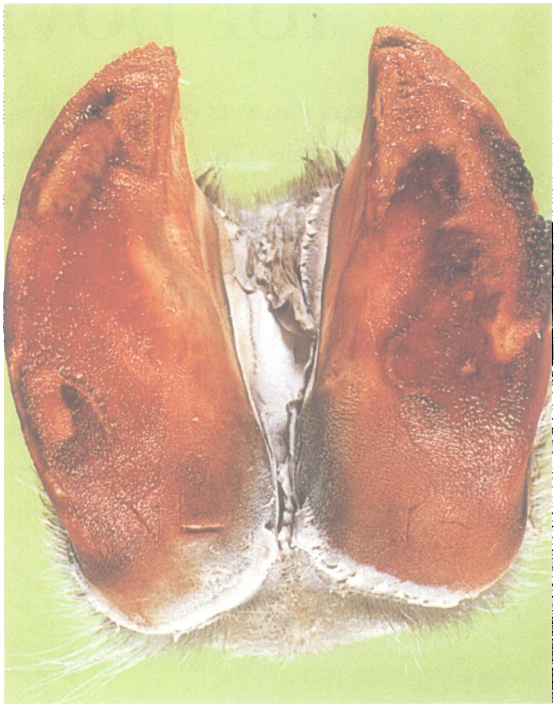
Key: blue = lamellae and laminae, yellow = white line

PHASE 2: SINKAGE OF THE DISTAL PHALANX AND COMPRESSION OF THE CORIUM IN THE SOLE AND HEEL

The thin layer of tissue immediately beneath the distal phalanx is at risk of compression from abnormal weightbearing, oedema in combination with the pressure exerted by normal weightbearing, and sinkage of the phalanx. Prolonged compression of the corium of the sole and heel leads to further episodes of capillary damage, haemorrhage, thrombosis, cellular inflammatory reaction and, eventually, ischaemic necrosis. The areas of necrosis are generally focal. These lesions are ultimately life-threatening as they may induce extensive damage with severe pain and lameness.

In the earlier stages of phase 2, the corium of the wall regions is reddened and oedematous but, with time, it reverts to a normal colour unless exudate has accumulated in the separated layers. It is important to realise that



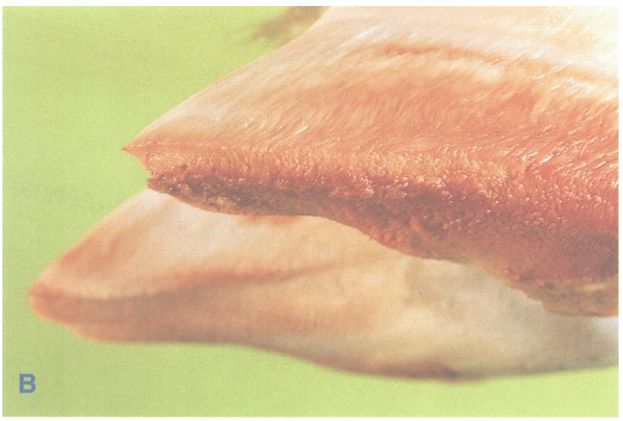


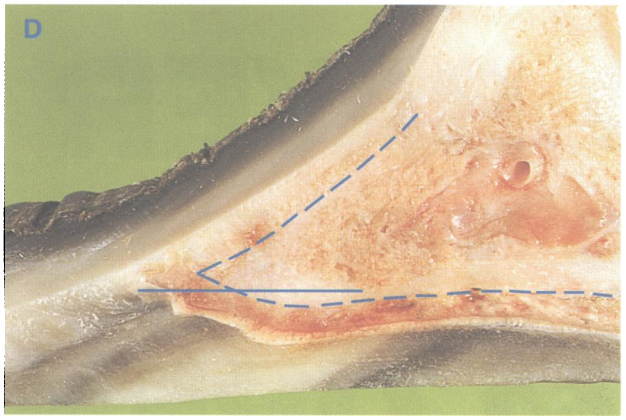
Early phase 2: Haemorrhages and necrosis in the corium. (above) There is widespread necrosis and haemorrhage in the corium of the outer hindclaw (on the right of the picture) which bears more weight. The centre of the lesion is located immediately under the flexor process of the distal phalanx. The inner claw is merely hyperaemic. Still nothing is visible clinically at this stage. (right) Both claws of this foot are severely affected. The sharply demarcated yellow areas in the toe correspond exactly to the contours of the underlying bone that compressed the corium, causing ischaemic necrosis and haemorrhages along the edge of the claw on the right

there are no overt clinical signs at this stage although several weeks may have elapsed since the onset of laminitis.

Further specific signs of chronic sinkage are a groove in the corium along the edge of the sole and a corresponding ridge in the inner horn along the junction between the sole and wall of the horn capsule. The groove results from the sole corium directly under the sinking distal phalanx (and the horn sole with it) being displaced several millimetres distally to a new level (akin to a footprint in soft ground). A ridge, the width of which corresponds to the thickness of the wall corium, is formed in the junction between the wall and sole due to the fact that the wall structures remain in place (ie, are not compressed). The ridge in the horn capsule fills the groove.

Early phase 2: Focal necrosis in the corium and horn. The sharply demarcated necrotic tissues are situated directly beneath the flexor process at the caudal edge of the distal phalanx. The production of horn has ceased and there is a 1 to 2 mm deep mirror-image 'impression' of the corium lesion on the inner surface of the horn capsule





Late phase 2: Signs of chronic sinkage of the distal phalanx within the hoof.
(A) The tip of the horn capsule of this claw has been removed to show several features resulting from sinkage. The corium of the sole is displaced ventrally (between the arrows) immediately under the sunken distal phalanx. There is a resulting groove in the corium along the junction between the sole and wall surfaces (arrowheads) and a corresponding ridge on the inner surface of the horn capsule. In this hoof the ridge consists of haemorrhagic, necrotic cell debris and horn of inferior quality. With time, these lesions grow out to cause a defective white line.
(B) Lateral view of the corium showing the groove along the junction between the sole and wall surfaces. This is an unmistakable sign of ventral displacement of the distal phalanx.
(C) Floor of the horn capsule of the claw pictured in A and B, showing the imprint of the third phalanx in the horn sole and the ridge along the junction between the sole and wall surfaces. There is also haemorrhage and slight necrosis in the toe area.
(D) Chronically laminitic claw in sagittal section with a very obviously concave dorsal horn wall. The distal phalanx (broken line) has sunk to the original level of the inner surface of the horn capsule (solid line) and, in so doing, has displaced the horn sole ventrally to cause a horn ridge in the tip of the toe

PHASE 3: DEVELOPMENT OF LESIONS IN THE HORN CAPSULE

The fact that there is a time-lag of at least six weeks before lesions in the corium manifest clinically in the horn capsule explains why the connection between laminitis and many hoof lesions was not recognised for so long. Some changes seen in the chronically deformed hoof are the result of repeated bouts of laminitis.

SOFT YELLOW HORN

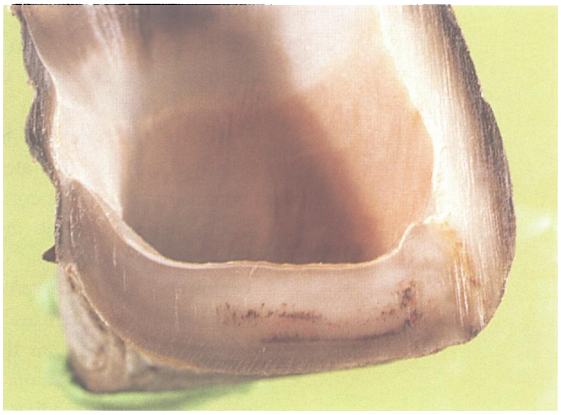
Horn that is soft, yellow and in some cases friable, occurs very commonly in diffuse areas of the sole and heel. These changes are at least partially due to serous fluid being incorporated into the horn and are the principal signs of chronic subclinical laminitis.

RED DISCOLOURATION (PATCHES)

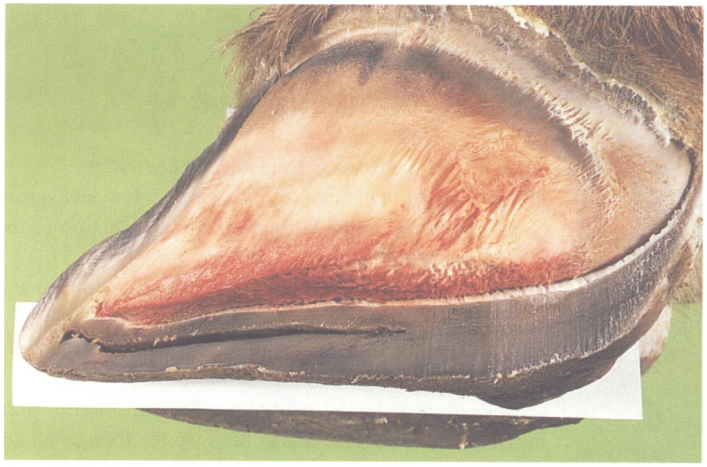
Blood pigments deposited in the horn tubules or lamellar layers following mechanical or other injury to the vasculature in the corium result in an area of red discolouration within the sole and heel horn. After about six weeks growth, this emerges at the surface as a dark red patch opposite the site at which injury occurred. Lesions in the white line take longer to appear if they occurred further up the wall. Repeated incidents of injury cause successive layers of blood-stained horn.

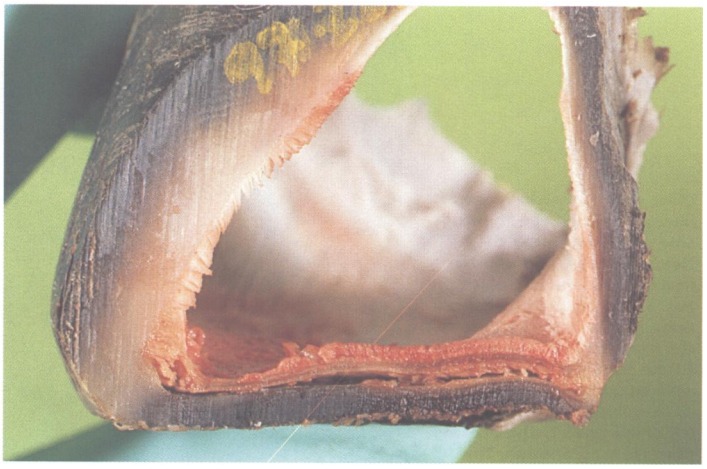
DOUBLE SOLE OR HEEL

In more severe cases, mechanisms similar to those causing red patches lead to accumulations of necrotic cell debris on the surface of the sole or heel corium and a temporary discontinuation in horn production due to separation at the dermal-epidermal junction. With time, the epithelial basal cells recover and produce new horn that covers and 'seals in' the debris. The incorporated deposits dry out to form a hollow or cleft that appears at the outer surface weeks later as a double sole or heel. The horn layers in the heel may separate fully to produce a flap or 'underrun heel'. Such lesions provide potential sites of access for infection.

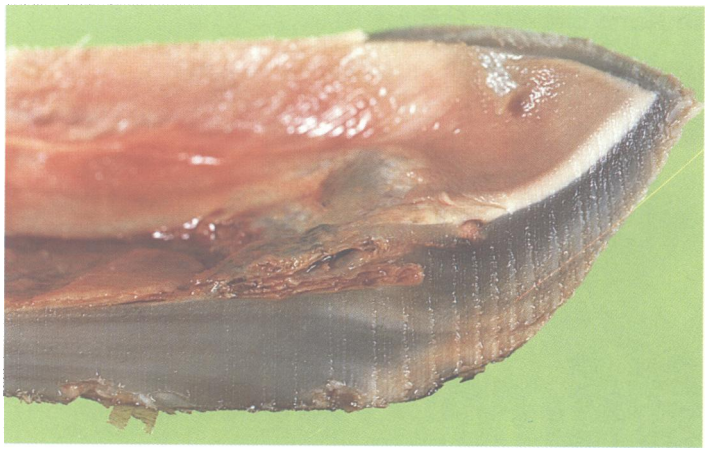


Phase 3: Haemorrhages in the sole. The horn capsule has been sawn vertically to reveal a section of the sole and walls. This cow clearly suffered several consecutive bouts of laminitis with haemorrhage since the blood pigments have discoloured the sole horn in several layers. Such discolourations are encountered commonly when trimming hooves

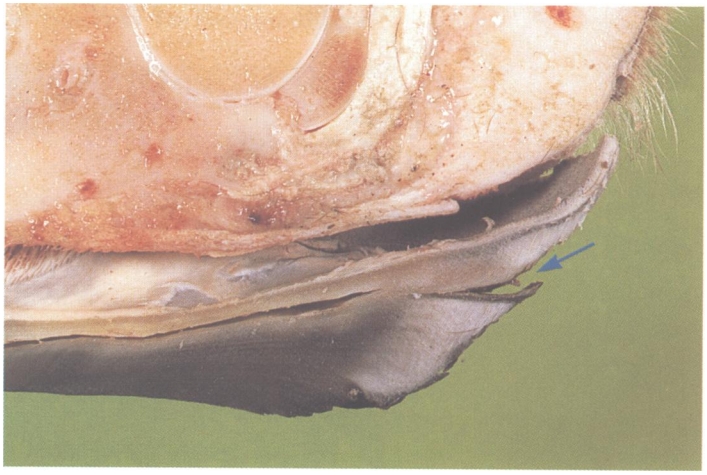




Phase 3: Double sole. (above left) Half of the horn capsule has been removed to show a section of horn containing a cleft. This is the result of cell debris and blood drying out after having been incorporated into the horn. The layer of new horn between the cleft and the corium is healthy so at this stage the double sole would hardly have caused the animal any discomfort. (above right) Empty horn capsule in cross section with the tip removed to reveal a double sole. In such cases nothing is visible on the outer or inner surfaces of the capsule



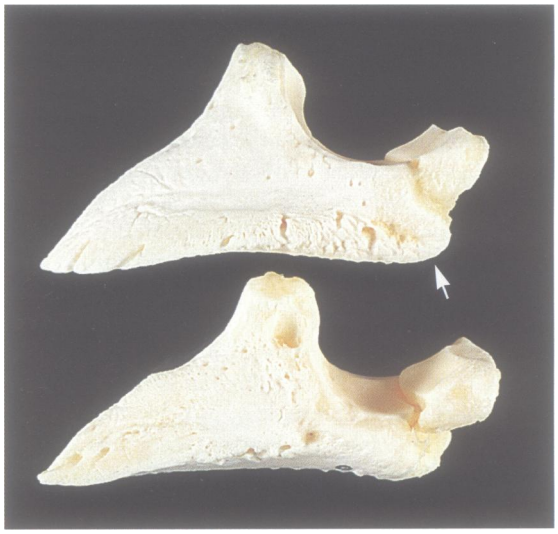
Phase 3: Double heel (acute). The heel region of the horn capsule has been sectioned in a sagittal plane and the wall removed. The white layers of new horn on the inner surface have recently covered up the damage and enclosed the blood and cell debris in a hollow



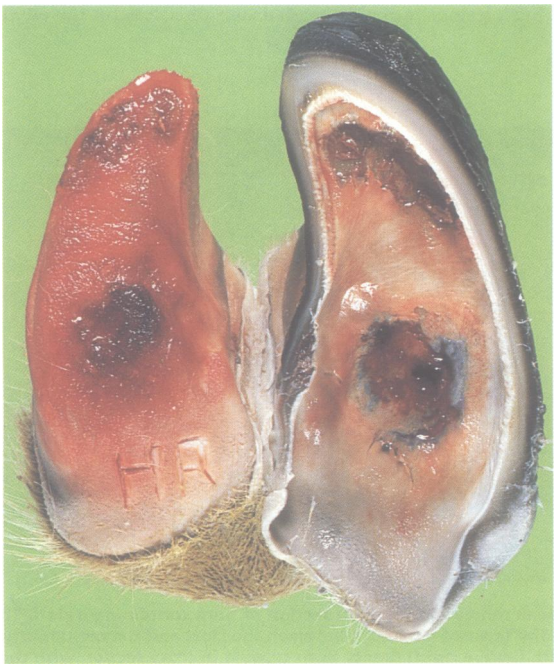
Phase 3: Double heel (chronic). In this case a double heel has lead to the development of a cleft at the caudal surface – an 'underrun heel' (arrow). (The gap between the corium and the horn capsule is artificial)

ULCERS

An ulcer – a perforation of the horn layer – develops when tissue necrosis is severe and extensive enough to hinder horn production permanently. Necrosis in the corium of the sole and heel, and therefore ulcers, are usually focal (as illustrated below). The site of an ulcer depends on where the corium is being compressed. This, in turn, depends on the angle at which the distal phalanx sinks and on the bone's surface configuration. Most commonly, the flexor process (the most distal point of the sunken bone) causes the compression and an ulcer develops beneath it, at the 'typical site'. The situation becomes critical as soon as necrosis spreads, since the typical site is immediately beneath the flexor tendon, bursa and interphalangeal joint. Toe ulcers are caused by the tip of the rotated distal phalanx, while the inner regions of the white line are affected when the bone's sharp outer edges compress the corium (the bony protuberances or edges are often palpable under the necrotic areas in the corium of claws from which the horn capsule has been removed).



Distal phalanx viewed from both sides showing the protrusions and sharp edges that correspond to the primary sites of injury in the corium of the sole and heel when compression occurs. The 'typical' site for sole ulcers is immediately under the flexor process (arrow). Rotation of the bone results in a lesion at the toe or even a toe ulcer. The sharp outer edges may compress the corium along the white line



Claw with a haemorrhagic necrotic area at the 'typical' sole ulcer site under the flexor process, and another less haemorrhagic area at the toe. The bony protuberances were palpable at the base of the necrotic foci. The damage to the sole is deep in this case because the production of horn has been interrupted for some time. If the process persists long enough, the whole sole will perforate, resulting in a sole ulcer. This takes two to three months to become clinically visible

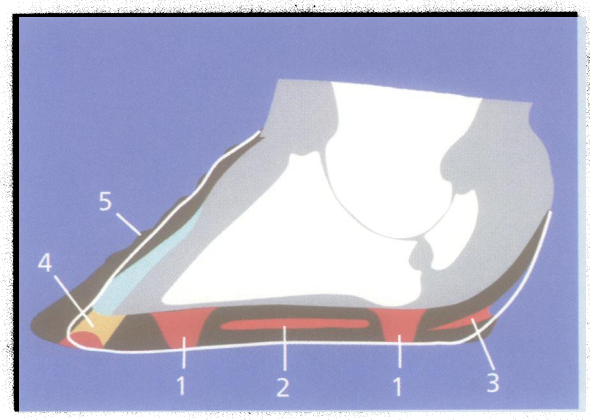
WIDENING OF THE LAMINAR ZONE (WHITE LINE)

Widening of the laminar zone may be caused by:

- Accumulation of fluid, blood or cell debris and subsequent separation at the dermal-epidermal junction of the wall;
- Separation of the same cell layers due to sinkage and hyperplasia of the laminae in the chronic phase.

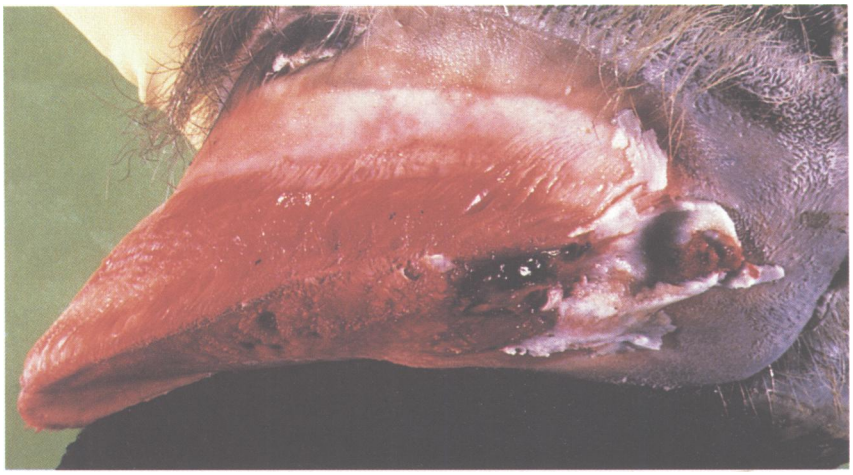
Once the involved regions in the wall have grown down to the weightbearing surface, they appear as a broadened, discoloured white line. The soft inferior horn may erode and provide a port of entry for foreign matter and infection. A double or hollow wall ('white line disease') is seen in severe cases of separation and erosion.

Development of lesions in the horn capsule (phase 3)



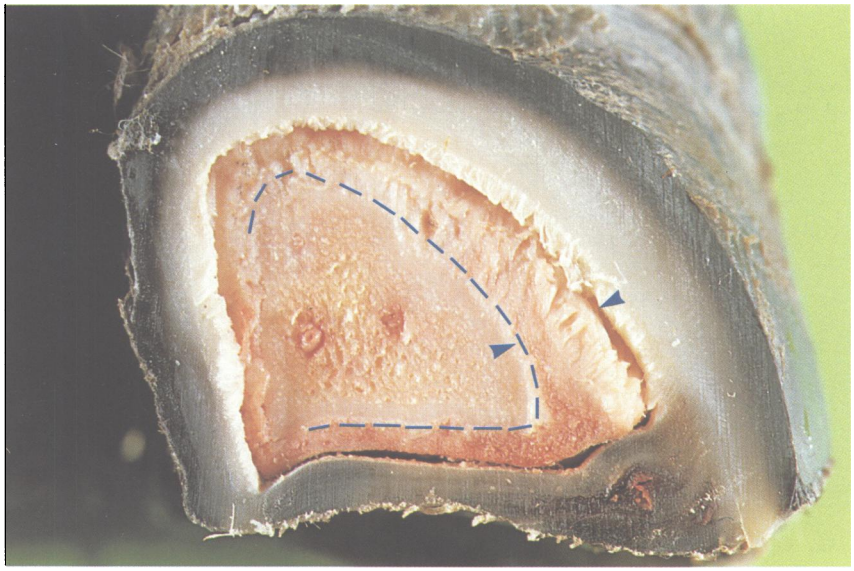
- Haemorrhagic patch or ulcer (1)
- Double sole (2)
- Heel horn separation (3)
- White line separation (4)
- Deformation of horn capsule (5)

Key: blue = widened lamellae and laminae



Phase 3: White line lesions (acute). Acute haemorrhages, as seen here at the base of the laminae at the outer edge of the claw, will eventually appear as lesions in the white line

Phase 3: White line lesions (chronic). This more chronic and severe case shows how the inner wall of the horn capsule is affected at the site of necrotic areas in the corium. (There are additional subacute to chronic lesions in the heel)



Phase 3: White line lesions (chronic). The tip of the toe has been removed to show an abnormally wide wall corium (arrowheads) that has resulted from partial separation at the dermal-epidermal junction after sinkage and reactive hyperplasia of the laminae. This widening of the wall corium means that the wall horn grows away from the distal phalanx – one reason why chronically laminitic claws are broader. In addition, the sole and heel corium immediately under the sunken distal phalanx is very thin and compressed. The broken line shows the outline of the distal phalanx. (The cleft between the corium and horn capsule is an artefact)

DEFORMATION OF THE WHOLE HORN CAPSULE ('SLIPPER FOOT')

The wall of a typical chronically laminitic claw is grooved and concave, while the entire hoof is broad and flat. The heels of some claws develop V-shaped furrows and the whole sole may bulge.

Horizontal ('hardship') grooves

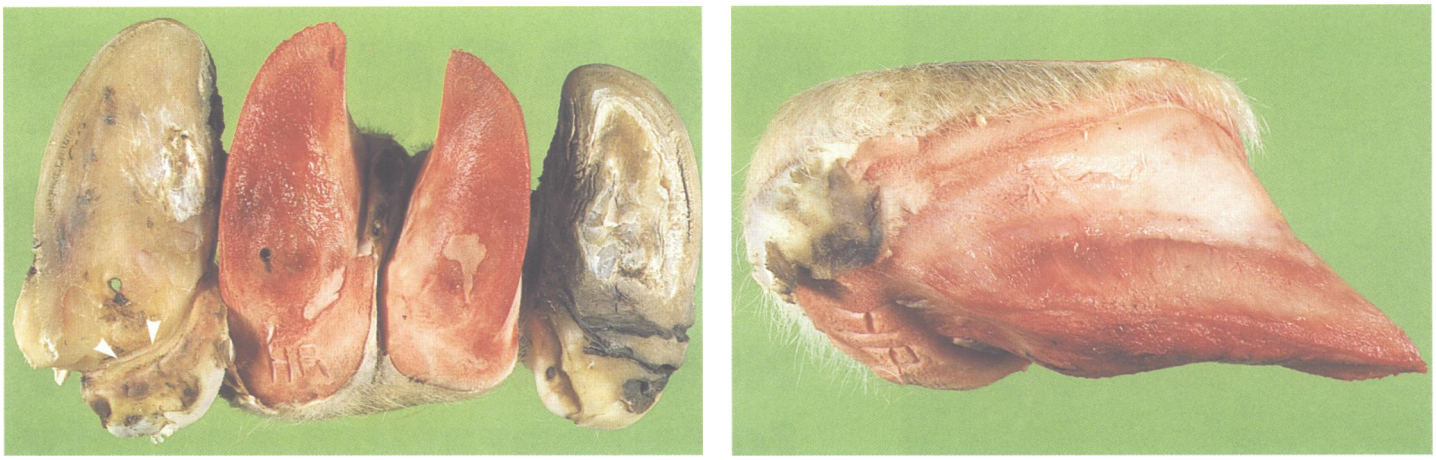
Horizontal grooves develop after a bout of laminitis (or any other severe illness). The epidermal stratum germinativum, which generates the structures of the wall, temporarily slows down its horn production; the layers get thinner; the new (still soft) tissues buckle due to the mechanical forces exerted by the sinking distal phalanx; and, as a result, a roughly horizontal 'hardship' groove emerges at the coronary band. It is possible to calculate when an insult occurred from the rate of horn growth and the distance of a groove from the coronary band. Chronically laminitic cows typically develop a series of horizontal parallel grooves that coincide with successive episodes of laminitis (less pronounced physiological grooves also develop in the horns and claws during gestation).

If the insult is so severe that continuity of the wall is lost, a horizontal fissure may develop in the horn capsule. As the fault grows out, the apex of the hoof becomes mobile and forms a 'thimble' which is painful until the fragment breaks away. The rough, ragged claws that result are referred to as 'broken toes'.

The hardship groove, horizontal fissure, thimble, and concave and broken toe represent different stages or degrees of the same phenomenon.

Concave walls

The direction of horn growth alters by a few degrees at each hardship groove. Horn that normally grows parallel to the surface of the distal phalanx gradually curves away so that the dorsal and abaxial walls typically assume a concave shape (this is one reason why the whole hoof becomes broader). At the same time, the distance between the wall horn and the distal phalanx increases when the bone sinks, so that the corium is



Phase 3: Deformed claw. (above left) Ventral view of a right hindfoot of a chronically laminitic cow; the deformed lateral claw on the left is far wider, larger and flatter than the medial claw. The horn quality is inferior, being softer than normal, yellowish to pink, and flakey. There are very clear signs of sinkage: the sole is bulged ('dropped sole') and the heel cushion has also dropped because the soft heel horn of the bulbar cup is not rigid enough to contain it. The bulbar cushion 'overflows' and a typical V-shaped furrow results (arrowheads). (above right) Lateral view of the lateral claw showing the part of the heel cushion that was contained within the soft bulbar cup, separated by a furrow. The bulging sole is also clearly visible Pictures reproduced, with permission, from Lischer and others (1994)

wedge-shaped when sectioned in a sagittal plane. A similar wedge is seen on the rare occasions that the distal phalanx rotates; in these cases the dorsal wall remains straight and the corium is compressed at the toe.

Restitution of the hoof requires a sufficiently long laminitis-free period to allow the whole length of the wall to grow out; this rarely occurs in older cows that commonly suffer repeated episodes of laminitis. Young animals appear to recover more readily from a laminitic incident, possibly because there is less scar tissue and the corium is capable of developing an efficient compensatory circulation.

Bulged (dropped) horn sole

A bulged or dropped horn sole is an advanced sign of a sunken distal phalanx. The pressure displaces the whole sole and heel to such an extent that the capsule bulges enough to become clinically apparent.

V-shaped furrow in the horn

Running along the junction between the bulbar cushion and the hard heel in chronically laminitic hooves is typically a V-shaped furrow in the horn. The bulbar cushion is not compressed directly by the sinking distal phalanx since it does not lie immediately under the bone; however, the soft tissues are dragged downwards indirectly. The cushion is partially contained by the rigid, upwardly curving edge of the heel horn (the inner edge of the furrow). Further up, analogous to an obese person's heel that sags over the sole of a slipper, the remaining heel cushion 'overflows' the bulbar cup where the horn is softer, and depresses it (to form the outer edge of the furrow).

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