

Acute laminitis

Shifting weight from one foot to the other (paddling) is the first clinical sign that lamellar degeneration is occurring (Obel grade I laminitis) and usually occurs in the forefeet (**Figure 4.1**). Occasionally, the shifting weight sign appears in just the hind feet. Careful observation may reveal that one foot, already with more severe lamellar pathology, is being lifted more frequently, and for longer, than the contralateral foot. It is worth spending some time quietly observing sick horses for the first appearance of paddling as early institution of treatment can do much to halt the progression of further lamellar damage.

The hooves of horses developing laminitis after carbohydrate overload (and possibly laminitis associated with other endotoxic/ septic shock syndromes) are palpably warm. If the ambient temperature is cool, hoof temperature can be measured using a thermographic camera or a hand-held infrared temperature scanner. Hoof temperatures of 30°C for more than 24 hours during the developmental phase usually indicates impending laminitis. Unfortunately, if the ambient temperature is above 30°C measuring hoof temperature becomes difficult to interpret. Bounding, exaggerated pulses in the digital arteries over the fetlock (**Figure 7.1**) are invariably present, but may be difficult to palpate if there is much subcutaneous oedema of the distal limbs. Sometimes pulsation in the digital arteries is so pronounced it is visible to the naked eye. A bounding digital pulse is not specific (pathognomonic) for laminitis, however, and occurs after exercise and in association with a number of other foot conditions, such as sole abscessation and fractures of the distal phalanx. Thumb or hoof tester pressure over the sole at the toe usually elicits a pain response, but not always, so a negative response should not be used to rule out a diagnosis of laminitis. Tapping the dorsal hoof wall with a hammer or dropping a flexed limb so that the toe strikes the ground may also elicit a pain response. Lameness during a trot-out may be obvious only when the horse turns sharply. An abaxial sesamoid nerve block abolishes the foot pain of laminitis.



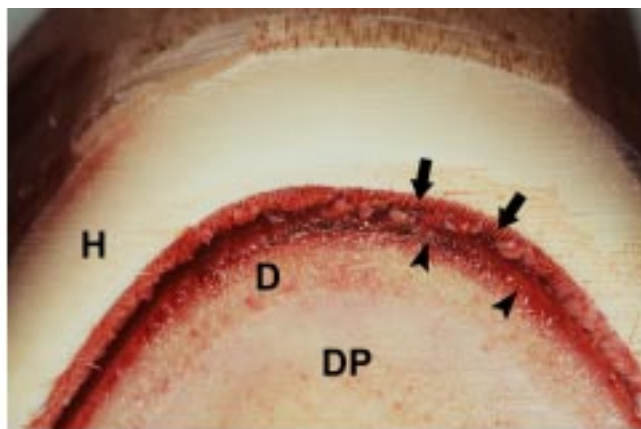
◀FIGURE 7.1 Palpating the digital pulse. The medial and lateral palmar digital arteries should be palpated with the foot in the weight bearing position. In the normal horse the pulse of the arteries is slight and often barely detectable. The digital pulse will be dramatically increased in amplitude (bounding) in cases of laminitis, distal phalangeal bone fracture, foot sepsis and immediately after exercise.

Although laminitis pathology appears to weaken the lamellar attachments of all the feet, more severe lesions usually develop initially in the forefeet. This is because the forequarters support a greater proportion (around 65%) of the horse's weight. Similar logic dictates that if the horse is forced to walk, locomotory forces and the rotatory force of the deep flexor tendon cause the lesions to worsen in the lamellae of the dorsal hoof wall of the forefeet.

Horses that develop more extensive lamellar pathology will exhibit more obvious clinical signs (Obel grade 2 laminitis). They may tremble and sweat and appear distressed. The heart and respiratory rates may be increased. They can be mistakenly diagnosed as suffering from "tying-up" (rhabdomyolysis), pneumonia or even a broken pelvis. Severely affected horses may refuse to pick up a forefoot or a hindfoot because full weight bearing on the contralateral foot causes extreme pain (Obel grade 3). When standing still, the forefeet will be placed forward of the normal position so that the heels are loaded more than the toes (**Figure 7.2**). If forced to walk the horse will arch its back and place the hindlimbs forward, under the abdomen, to shift as much weight as possible to the hindquarters. The horse half rears before stepping forward in front. The posterior phase of the forelimb stride is kept short to minimize the painful downward rotating force of the deep flexor tendon on the distal phalanx.



▲ **FIGURE 7.2** The laminitis stance. The forefeet are placed forward of the normal position so that the heels are loaded more than the toes. When walking is attempted, the horse will arch its back and place the hindlimbs forward, under the abdomen, to shift as much weight as possible to the hindquarters.



▲ **FIGURE 7.3** Acute severe laminitis: *sinker*. Sometimes, laminitis occurs with such rapid and devastating severity that there is total separation of the epidermal (arrows) from the dermal lamellae (arrowheads) and the distal limb sinks into the hoof capsule (hence the term "sinker"). When this foot was sectioned, in the transverse plane, the whole of the distal phalanx (DP) and the attached dermis (D) was floating unattached within the hoof capsule (H). The photograph shows the toe region. All four feet were affected, the hind worse than the fore, and the horse was so lame it could not move. The stance of the horse was not typical of laminitis; weight was evenly distributed to each limb and there was muscle trembling, sweating and signs of severe distress.

When the hindfeet are more severely affected than the fore, the order of the laminitis stance is reversed. The forelimbs are placed back under the abdomen and the horse leans over its forequarters, lowering its head and neck as a cantilever, to relieve the hindfeet of weight bearing. Weight will be shifted from one hind foot to the other.

With severe lamellar failure in all four feet, horses are immobile and extremely distressed ([Figure 7.3](#) and [Figure 8.4](#)). They frequently lie in lateral recumbency with all limbs extended.

Chronic laminitis

The anatomical disintegration of the hoof, which accompanies the acute episode, is for the most part, invisible to the naked eye. With the passage of time however the hoof begins to display the effects wrought upon on it by the pathology of the acute phase. Lameness persists and its severity correlates with the extent of displacement of the distal phalanx within the hoof capsule.

Coronary band changes

When the majority of the lamellar attachments fail, as they do in severe cases, the distal phalanx descends deeply into the hoof capsule, taking with it the attached coronary subcutis. This creates a deficit in the coronary band and the sharp edge of the proximal hoof wall becomes palpable with a finger ([Figure 7.4](#)). Initially the deficit may be palpable only dorsally, over the extensor process of the distal phalanx. If it extends around the coronet to the quarters and heels, the prognosis is grave as this is an indication that the lamellar suspensory apparatus no longer exists; that it is totally destroyed. Sometimes the skin may separate at the hair-line of the coronet and exude serum.

Sole changes

Within a few days of the acute episode, a convex bulge in the sole (the so-called dropped sole) may appear beneath the descending distal phalanx ([Figure 7.5](#)). Initially, fine cracks appear over the bulge in the sole and in severe, deteriorating cases the resultant pressure necrosis causes the distal phalanx to prolapse completely through the horny sole ([Fig 7.6](#)). In less severe cases, where descent or rotation of the distal phalanx has been slight, the sole may lose its normal concave appearance and be flat. Careful shaving of the



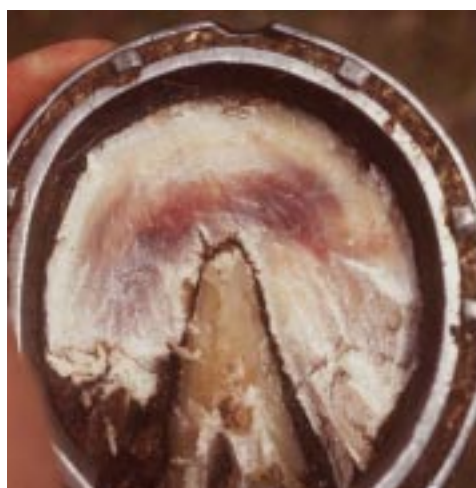
◀ **FIGURE 7.4 SEVERE EARLY CHRONIC LAMINITIS.** As the hoof/distal phalanx attachments progressively fail, the bone and the attached subcutis descend into the hoof capsule. A distinct deficit at the coronet develops that can be palpated with the finger. The top of the hoof palpates as a sharp edge, instead of full and rounded. This is a grave clinical sign, especially if the deficit extends to the quarters and heels, because it means that lamellar destruction is severe and extensive.



▲ **FIGURE 7.5 The dropped sole of severe chronic laminitis.** A characteristic of chronically foundered feet is a downward bulging of the sole (dropped sole). As the bond between the hoof and distal phalanx fails, the bone descends into the hoof capsule forcing the sole downwards. The sole appears convex or "dropped" in appearance, instead of the normal concave shape. The solar corium, trapped between the tip of the descending distal phalanx and the rigid horny sole, is crushed. It is important to shoe foundered horses so that this painful, compromised region is kept clear of the ground.



▲ **FIGURE 7.6 Chronic laminitis: prolapse of the solar corium.** This 3-year-old Arabian mare accidentally gorged on grain and developed moderately severe laminitis. The distal phalanx has descended into the hoof capsule and destroyed a crescent shaped band of solar corium. The dead sole has been resected and the photograph shows the exposed solar corium (arrows) covering the distal phalanx.



◀ **FIGURE 7.7 Pony foot with early chronic laminitis.** This obese pony mare developed laminitis on Spring pasture six weeks previously. At the time the photograph was taken, the pony would stand for long periods painfully shifting its weight from one front foot to the other and walked with the typical laminitis gait. When the outer layers of the sole were pared away an ominous, crescent-shaped, dark-red bruise was visible. The shape of the bruise and its position are evidence that the distal phalanx has detached from the lamellae of the inner hoof wall and has descended into the hoof capsule, crushing the solar corium against the horny sole. The bruise stains the horny layers of the sole and after a few weeks, grows out. It is clearly visible when the sole lacks pigment.

sole, with a sharp hoof knife, may reveal a red, crescent-shaped bruise (**Fig 7.7**). This is evidence of trauma from within, inflicted on the sole by the descending margin of the distal phalanx which is also crescent shaped. Haemorrhage from blood vessels in the solar corium, crushed under the distal margin of the bone, occurs and haemoglobin and red blood cells become incorporated into the horny sole. They appear at the ground surface of the sole after a period of sole growth.

Bilateral, crescent-shaped bruising of the sole must not be dismissed as “stone bruising”. A penetrating wound of the sole and consequent abscessation could mimic laminitis, but would usually involve only one foot and would not cause the array of clinical signs associated with the diseases that induce laminitis.

Hoof wall changes

After the acute episode of laminitis and the consequent shift in the position of the distal phalanx within the hoof capsule, the horse is left with a legacy of deformed hoof growth. Dorsal hoof wall growth is retarded, while growth at the heels proceeds at the normal rate. In the normal hoof, minor fluctuations in growth rate produce in the wall a series of concentric rings, parallel to the coronet, clearly visible on the surface of the hoof. In a hoof affected by chronic laminitis the growth rings are no longer parallel; they converge at the toe where the growth rate is least (**Figure 7.8**). In severe cases, the divergency of growth between toe and heel produces a dramatic upturning of the toe and the hooves take on the appearance described as “Aladdin’s slippers” (**Figure 7.9**). If neglected, toes affected by chronic laminitis can become unnaturally long and make locomotion for the foundered horse or pony extremely difficult.

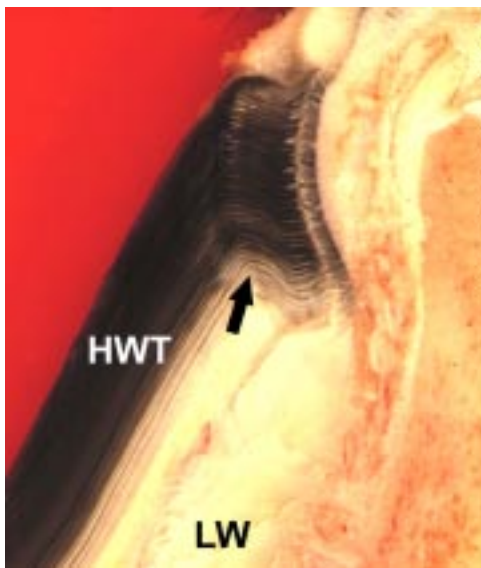
During the acute phase, when the lamellar attachment apparatus fails, the descending distal phalanx takes with it the dermal papillae of the coronary groove. The normally straight papillae become kinked, but the basal cells of the coronary epidermis usually continue to elaborate hoof wall, albeit more slowly and in a distorted direction. The kink imposed on the papillae is reflected in the hoof wall tubules: they are kinked too. After the acute episode the coronet usually recovers to some extent and produces hoof tubules with a more correct orientation. However, the kinked tubules; remain in the hoof wall until they grow out at the ground surface. Kinked hoof wall tubules can be seen in sections of chronically foundered feet post mortem (**Figure 7.10**). A groove in the hoof wall, corresponding to the zone of kinked tubules, is visible on the surface of the hoof wall. The depth of the groove gives some



▲FIGURE 7.8 The asymmetrical growth of chronic laminitis. Nine months of clinical laminitis is recorded on the surface of the horse's hoof. Five months before the photograph was taken this overweight pony mare was allowed unrestricted access to lush spring pasture. She developed laminitis and the impact of the pathological changes that occurred to the growth regions of the hoof are visible. The onset of chronic laminitis is marked by the groove half way down the side of the hoof-wall (arrowed). Distal to this groove the hoof-wall appears normal; the remaining in the wall, basic changes in hoof structure (widened and evenly spaced laminae) are visible (laminitis). Proximal to the arrowed groove the rings are non-parallel because the internal, pathological changes of chronic laminitis are widespread growth in the dorsal aspect. The growth rings converge in the dorsal hoof wall above the arrowed groove, but are virtually normal in the heel. The hooves of horses and ponies being examined prior to purchase should be closely scrutinised.



▲FIGURE 7.9 Severe chronic laminitis untreated for 18 months. Gorging on wheat grain is a common cause of laminitis in Australia. This pony gained access to a ripe wheat field and, after surviving the severe digestive disturbance and endotoxaemia that this generated, developed severe laminitis in both fore and hind feet. Unless effectively treated in its early stages the disease causes a chronic growth disturbance to the hooves. The heel grows faster than the toe, and the hoof, if left untreated and given enough time, (in this case 18 months), will assume the classic shape of Aladdin's slipper.



◀FIGURE 7.10 Chronic laminitis causes deformation of tubular hoof wall growth. The distal phalanx and the dorsal hoof wall have lost their attachment and have become widely separated. The tissue now between the inner hoof wall and the bone is abnormal and consists of epidermal tissue proliferating to form a weak, disorganised mass called the lamellar wedge (LW). The descent of the unattached distal phalanx into the hoof capsule has distorted the growth of the proximal hoof wall tubules which are now kinked (arrowed). Distal to the laminitis episode the hoof wall tubules (HWT) are normal.

indication of the severity of the acute episode. The distance of the groove from the coronet can be used to estimate (based on a normal hoof wall growth rate of 7 mm/month) the retrospective timing of the acute episode.

Diagnosis

It is not difficult to make a presumptive diagnosis of laminitis, based on history, the characteristic clinical signs and a physical examination of the feet. However, the additional data generated by good quality radiographs will allow a final decision regarding severity, chronicity, type of treatment and prognosis to be made with more confidence. A radiographic examination of the feet is an essential step in the diagnostic work-up of the laminitis case.

Whenever possible, radiographs should be made as soon as the clinical signs of laminitis appear. An early appreciation of the subtle, radiographic changes of acute laminitis may enable further deterioration to be halted if treatment procedures are effected promptly. It is also important to take serial radiographs so that the effectiveness of treatment, or conversely, deterioration, can be monitored over a period of time. The owners of horses in the process of foundering usually appreciate being kept up to date with the clinical status of their animal. In the critical, early stages of laminitis where the final outcome depends so much on the extent of the acute lamellar pathology, every effort must be made to support, mechanically, as well as medically, the damaged lamellar attachment apparatus. Radiographs enable the

status of the relationship between the distal phalanx and dorsal hoof wall to be assessed with precision.

Key Points

- Early clinical signs of laminitis include shifting weight from one foot to the other, high hoof temperatures for a prolonged time and bounding pulses in the digital arteries. A pain response elicited by hoof tester pressure may not always be present, nor marked lameness during a trot-out.
- After the development of more extensive lamellar pathology, foot pain increases and its severity is proportional to the extent of displacement of the distal phalanx within the hoof capsule. A characteristic stance and gait is adopted by the horse to minimize the pain in its feet.
- Chronic laminitis is marked by persistent lameness and anatomical disintegration of the hoof that includes changes to the coronary band, the development of a dropped sole and deformed hoof growth.
- Radiographic examination of the feet should be performed as soon as clinical signs of laminitis appear and during the course of treatment.