LAMINITIS (founder)*

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Definition. Laminitis, literally, “inflammation of the laminae,” is a disease that causes degeneration, necrosis, and inflammation of the dermal and epidermal laminae in the hoof wall of horses and ruminants.

Etiopathogenesis. Because the epidermal laminae suspend the distal phalanx and therefore the body weight of a horse, laminar degeneration destroys the suspension mechanism and permits weight-bearing forces to push the distal phalanx ventrally. Failure of the laminar suspending mechanism causes a painful and potentially crippling lameness.

Laminitis is commonly a sequela of digestive disturbances and other disorders that cause endotoxemia and elaboration of inflammatory mediators. Unless preventive measures are taken, laminitis often occurs after colonic torsion, enteritis, grain overload, pleuropneumonia, and septic metritis (i.e., postparturient retention of the placenta). In horses it is sometimes seen following changes in feed, excess intake of cold water after hard exercise, grazing on lush spring grasses containing highly available carbohydrates, or persistent feeding of a high concentrate ration. Laminitis also may be precipitated in horses by administration of high levels of corticosteroids, which decrease protein synthesis and potentiate digital vasconstriction and microthrombosis. Excessive weight bearing in the support limb during severe lameness of the contralateral limb can produce laminitis, as can hard work on hard ground or extreme exhaustion and dehydration. A water-soluble toxin in black walnut shavings also has been shown to induce laminis in horses. In cattle it is most commonly seen right after calving in fat heifers that have been fed excess concentrates and kept on concrete surfaces.

Pathophysiology. The pathophysiology of laminitis has not been totally elucidated; however, laminitis is often considered a local manifestation of a variety of disorders that cause a generalized metabolic disturbance. Several factors may produce laminar degeneration.

The integrity of the laminar suspending mechanism depends on maintenance of proteins in the cytoskeletal networks and intercellular junctions of the epidermal laminar cells. This process is energy-dependent, and disorders that decrease laminar perfusion or decrease protein synthesis have the potential to initiate laminar degeneration. In addition, laminar degeneration may be initiated by disorders that cause the elaboration of factors that are cytotoxic to the epidermal layer by disorders that increase the tension on the laminae and their sustaining vessels confined within the rigid hoof wall, factors that increase tissue swelling, such as inflammation and edema. Because the laminae and their sustaining vessels are confined within the rigid hoof wall, factors that increase tissue swelling, such as inflammation and edema, also increase the interstitial tissue pressure beyond critical capillary closing pressure, predisposing the animal to develop compartment syndrome and functional ischemia of the coronary. Opening of arteriovenous shunts within the coronary occurs during carbohydrate-overload, but such shunting has not conclusively shown to be a major factor producing laminar degeneration.

Laminitis is often a sequela of diseases, such as gram-negative sepsis and endotoxemia, but exact administration of endotoxin has failed to reproduce the condition. Overingestion of grain or other feed containing large amounts of highly available carbohydrates, however, is thought to produce endotoxemia as the most common cause of acute laminitis. Carbohydrate overload results in bacterial overgrowth in the stomach, lactic acidosis, decreased colonic pH, colonization, slough, and death of colonic bacteria with consecutive liberation of endotoxin. Degeneration of the digital laminae is thought to allow endotoxin to gain access to the portal circulation. The mechanistic links between endotoxemia and laminar degeneration are not well understood; however, hyperimmune serum to negative core antigens has a strong protective effect in horses at high risk of developing laminitis as a consequence of metabolic crises or carbohydrate overload.

Sequential biopsies of the epidermal laminae of horses during the development of laminitis indicated that initial laminar degeneration was most compatible with ischemic or cytotoxic injury, that inflammation, edema, and microthrombosis were not necessary for laminar degeneration, and that increases in catecholamines that potentiate peripheral vasconstriction and further diminish laminar perfusion.

Clinical Signs. The signs of acute laminitis are lameness, depression, anorexia, and reluctance to move. Early in the disease affected animals often show subtle changes from walking on the hoof wall of one foot to the other. Insensitivities of the superficial anal gland and increased sensitivity over the sole at the toe, and tapping the hoof wall at the toe may elicit pain. Severe lameness may lead to
may be unwilling to pick up a forefoot or
foot because of a reluctance to bear full weight on
contralateral foot (Oibel grade III lameness.176). The
lesions are usually affected more often and more seri-
ously than the hind feet in horses, and the most dorsal
regions are more severely involved than laminae in the
hindlimbs. Therefore horses with laminitis commonly
bear the hindlimbs under the body and place the
hoof forward to shift weight to the hindquarters
and the heels more than the toes. In ruminants the
hindlimbs are most commonly involved, and affected
foot characteristically become recumbent. In severe
cases laminitic degeneration circumferentially in-
the foot, a noticeable depression can be palpated
the coronary band. In such cases exudation is
caused by the distal phalanx has shifted distally with respect
to the hoof wall (i.e., severe rotation or sinking of the
distal phalanx) and suggest a grave prognosis. With
removal of the distal phalanx, the sole loses its
hollowed appearance and is flat or bulges between
the frog and apex of the frog. Pulse and respiration rates
usually increase, and other clinical signs reflect
the disease processes.

Chronic laminitis are lameness and abnormal
formation of the foot. The sole is flat or dropped,
white line is widened, and the hoof wall shows signs
of abscessation. Irregular rings of horn, closely
parallel to the uniocular and more widely spaced near the heels,
the hoof wall. In ruminants the same effects and
light yellow discoloration. Hemorrhages can
be identified in the abaxial white line region, and
are parallel to the coronary band may be
in the hoof wall. The signs of subclinical abscessation
times mimic those of laminitis; however, abscesses
commonly involve only one foot and rarely cause
pain, depression, or elevation of the pulse and
respiration rates.

Clinical Pathology and Radiology. Clinical path-
findings during the development of acute laminitis
commonly represent alterations associated with
dying disease processes, such as enteritis or metritis
and are not pathognomonic for laminitis. During
set alimentary laminitis, packed cell volume,
plasma protein, heart rate, respiratory rate, rectal
body temperature, and blood glucose level are commonly
raised. Arterial blood pressure is usually elevated in
but depressed in ruminants.171 Neutrophilia and
anemia also are often seen. These changes are
consistent with release of adrenal glucocorticoids
and catecholamines. Horses with chronic severe
in which euthanasia was deemed necessary,
total white blood cell (WBC) counts that were
significantly elevated (total WBC 15,000 to 18,000/μl)
over control horses and horses that recovered from less
severe bouts of laminitis.172 The persistent neutrophilia
was presumably a response to infection and was thought
to signify an unfavorable prognosis.

Radiographic examinations should be performed for
the affected digits of horses suspected to be developing
laminitis. The initial examinations should include latero-
medial and 65-degree dorsoproximal-palmarodistal
projections. These views should be taken to assess the
appearance of the distal phalanx, the soft tissues of the
dorsal hoof wall and corium, and their relationship. Latero-
medial examinations are periodically repeated to check the
progression of the disease. Radiographic signs of
laminitis include ventral displacement of the extensor
process with respect to the coronary grove of the hoof
wall, increased distance between the dorsal cortex of
the distal phalanx and the surface of the hoof wall, and
ventral rotation of the tip of the distal phalanx. Linear
radioluencies are noted inferior to the hoof wall in
cases where the corium has separated from the epider-
mal laminae. Increasing degrees of rotation of the distal
phalanx and increases in the distance between the
dorsal surface of the distal phalanx and the hoof wall
indicate progression of the disease (Fig. 36-22).

Because variations in technique affect subsequent
radiographic distance and angle measurements, it is
essential to standardize the radiographic procedure to
detect small changes between examinations. For the
lateromedial radiograph, the foot is cleaned and placed
on a wooden block approximately 3 inches thick. A
radiopaque marker can be embedded in the dorsal
surface of the block and along the dorsal surface of the
hoof wall to aid in determining the amount of rotation
of the distal phalanx. If the same marker or a marker of
known length is placed along the hoof wall for each
examination, it can be used for calculating the amount
of radiographic magnification to permit an accurate
comparison of measurements among radiographs with
different magnification factors. A tack, screw, or groove
can be placed in the proximal dorsal hoof wall as a
reference for measuring vertical displacement of the
distal phalanx in repeated radiographs.

The radiographic beam should be perpendicular to a
sagittal plane through the digit and should be centered
midway between the toe and heels, 2 to 3 cm above the
bearing surface of the wall. The radiographic cassette
should be parallel to the sagittal plane through the digit
and should be placed as close to the foot as possible.
Using consistent focus-object and object-film distances
or correcting for radiographic magnification and per-
forming the examination in this standardized manner
permit straight lateral radiographs to be produced and
allow accurate quantification of radiographic param-
eters so that subtle changes may be identified.
FIG. 36-22 A. Lateromedial radiograph of a normal digit. Two radiopaque markers can be seen. One has been placed on the block below the foot to mark the bearing surface of the wall, and the other marker identifies the location of the dorsal surface of the hoof wall. Notice that the dorsal surface of the hoof wall and the dorsal cortex of the distal phalanx are parallel and that the distance between them, the soft tissue thickness (T), is approximately 25% of the distance from the tip of the distal phalanx to the articulation of the distal phalanx and the navicular bone, i.e., the length of the distal phalanx (L). B, Lateromedial radiograph of a digit from a horse with severe laminitis. The distal phalanx has dropped ventrally without rotating. This phenomenon is seen in some horses with laminitis. The most consistent radiographic manifestation in such cases is an increased distance between the dorsal cortex of the distal phalanx and the dorsal surface of the hoof wall. The soft-tissue thickness as measured between the dorsal cortex and the dorsal surface of the hoof wall in this case is 45% the length of the distal phalanx. The soft-tissue thickness is normally less than 28% of the distal phalanx length for thoroughbred racehorses.
FIG. 36-22, cont'd C, Lateromedial radiograph of a digit from a horse with severe laminitis. Note the linear radiolucency dorsal to the distal phalanx (open arrow). The presence of this lucency indicates that there has been a separation between the corium and primary epidermal laminae, and the lucency marks the inner aspect of the hoof wall (arrows). The dorsal cortex of the distal phalanx is rotated approximately 14 degrees with respect to the inner surface of the hoof wall. Note that the dorsal and inner surfaces of the hoof wall are not parallel. This is the result of rasping along the distal portion of the dorsal surface of the hoof wall. The soft-tissue thickness in this case is markedly increased to nearly 42% of the distal phalanx length.

Epidemiology. A survey of the risk factors associated with laminitis indicated that intact mares and stallions are at greater risk of developing laminitis than geldings. Horses also accounted for a significantly greater number of laminitis cases than was expected on the basis of their proportion of the caseload. The peak incidence of new cases also corresponded with growth in spring grasses, suggesting that ingestion of large quantities of fresh grass is also a significant risk factor for laminitis in mature horses. Other risk factors include diseases that cause reduced blood flow or trauma to the digit, and diseases that produce endotoxemia. Persistent feeding of a high grain ration, stabling on concrete surfaces, long trips, and exposure to, or ingestion of, black walnut wood products are also thought to be associated with an increased risk of laminitis. In addition, horses that have previously had laminitis are at greater risk of developing it again than are other horses.

Necropsy Findings. Peracute cases may have total separation of the secondary epidermal laminae, which causes a separation between the primary epidermal laminae of the hoof wall and the collagen fibers of the corium. Abscessation may occur in the necrotic laminae or subsolar tissues. The distal phalanx may sink or be rotated ventrally with respect to the hoof capsule, and the tip may penetrate the sole (Fig. 36.23). Severe cases are accompanied by fractures of the solar margin, osteomyelitis, or severe resorption of the distal phalanx. The necropsy findings generally demonstrate a variable degree of elongation of the epidermal laminae, which depends on the severity and duration of the problem (Fig. 36.24).

Treatment. Treatment of animals developing acute laminitis should be considered an emergency. Laminar degeneration is underway by the time clinical signs of lameness appear, and even a few hours of delay in treatment can mean the difference between success and failure. Therapy should be initiated before the development of clinical signs in circumstances in which the untreated animal is at high risk of developing laminitis (e.g., animals that have recently ingested a large quantity of grain, mares with retention of the placenta, and horses with strangulating colon torsions).

General principles of therapy are aimed at eliminating the cause, promoting digital circulation, reducing tension on the laminae, and administering nonsteroidal anti-inflammatory agents to minimize digital inflammation and necrosis and to relieve pain.
FIG. 36-23 A. Midsagittal section from the foot of a horse with a normal digit. Note the distance between the dorsal surface of the dorsal cortex of the distal phalanx (open arrows) and the inner surface of the hoof wall (arrows). The dorsal surface of the hoof wall and dorsal cortex of the distal phalanx are parallel. Compare with Fig. 36-22, A. B. Midsagittal section from the foot of a horse with severe laminitis, a “sinker.” Note the increased distance between the dorsal surface of the dorsal cortex of the distal phalanx (open arrows) and the inner surface of the hoof wall (arrows). Also note that the distal phalanx has not rotated with respect to the hoof wall. Compare with Fig. 36-22, B.
**FIG. 36-23, cont’d** C. Midsagittal section from the foot of a horse with severe laminitis. There is approximately an 18-degree rotation of the distal phalanx, and its tip has penetrated the sole (*curved arrow*). Note the increased distance between the dorsal surface of the dorsal cortex of the distal phalanx (*open arrows*) and the inner surface of the hoof wall (*arrows*). Compare with Fig. 36-22, C.

**Mechanism of cause.** A laxative or purgative should be administered to animals that have ingested a large amount of grain. In such cases 3 to 4 L of mineral oil may be given through a nasogastric tube. Intravenous administration of balanced electrolyte solution is usually indicated for horses with laminitis resulting from exposure to heat, dehydration, and hypovolemia. Retained placenta should be expelled within 3 hours after parturition in all horses. Antidotoxin hyperimmune serum may be indicated for horses at risk of developing endotoxaemia as a result of colon torsion, toxic diarrhea, toxic proximal enteritis, septic metritis, grain overload, or other disorders.

**Steroidal antiinflammatory agents.** Phenylbutazone is the recommended antiinflammatory agent, and it is usually given orally once a day. The dose is 4.4 mg/kg (orally, twice a day) for 4 days. The dose should be tapered to 2.2 mg/kg (twice a day) as soon as possible. Flunixin meglumine* is sometimes given concurrently for the first 4 days in severe cases (1.1 mg/kg intravenously, once a day). Dimethylsulfoxide (DMSO) also may be given daily (0.2 to 1 g/kg) for 2 to 3 days. To administer it intravenously in a 450-kg horse, 250 ml of the 90% solution is mixed in 3 L of balanced electrolyte solution of 5% dextrose and given slowly. DMSO should be diluted to less than 20% concentration to avoid hemolysis when it is given intravenously. The use of aspirin (10 mg/kg, orally, once a day) is sometimes advocated for its antiinflammatory and antiplatelet activities. Corticosteroids and adrenocorticotropic hormone (ACTH) are contraindicated because they decrease protein synthesis, and they may potentiate peripheral vasoconstriction and microthrombosis.

**Reduction of tension on the laminæ.** The force related to suspending the weight of the horse by the attachment between the hoof wall and the distal phalanx is likely to be a major factor producing laminar deformity in horses with laminitis. Reduction of laminar tension may be achieved by focusing the forces of weight bearing more on the frog and sole and reducing the amount of weight taken by the hoof wall. This can be accomplished by using frog support bandages or shoes, sole casts, or sand stalls. Recently, elevation of the heel with an 18-degree wedge has been advocated to reduce the pull of the deep flexor tendon and decrease the tension on the laminæ. This elevation

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*Note: The asterisk (*) indicates a non-verified compound or technique.*
FIG. 36-24  A, Section of a healthy foot. The section was cut parallel to the coronary band, midway between the coronary band and the bearing surface of the hoof wall (inset, S). The length of the epidermal laminae (L) is approximately 33% of the thickness of the hoof wall (W) in normal horses. The distance between the dorsal cortex of the distal phalanx (open arrows) and the inner surface of the hoof wall (arrows) is normally less than 75% of the thickness of the hoof wall. B, Foot section, cut in a manner similar to that of A, from a foot of a horse with moderate laminitis. Note the increased length of the epidermal laminae (L). The increase in epidermal laminar length has allowed the distance between the dorsal cortex of the distal phalanx (open arrows) and the inner surface of the hoof wall (arrows) to become nearly as large as the thickness of the hoof wall (W).
achieved with a plastic-cuff shoe* that is taped, screwed to the hoof wall and used with a frog pad† and an 18-degree heel wedge.

Horses with severe acute laminitis, do not lower in the acute stage and avoid shoes that require to bear full weight on one foot for a pro- period while the shoe is being nailed on the foot. Using shoes that increase laminar tension may benefit by weight bearing forces to the hoof. A plastic-cuff heel-wedge shoe can be taped temporarily to the hoof with minimal trauma and effort. If the shoe is more comfortable, the shoe can be in place. Frog support shoes continue to put on the frog when the horse is recumbent and predispose to subsolar necrosis. Frog support ban- ner Lilly Pads provide satisfactory support and el the complications associated with shoeing. The cal should be dubbed off to decrease the lever arm that a long toe has on plying the wall away from phalangeal during break over.

14. Horses should be encouraged to lie down to laminar tension. This goal usually can be accom- plished with sedation. The stall should be heavily bed- straw and pine chip shavings to a depth of 30 to 60 cm for comfort and to reduce the risk of pressure sores.

**Promotion of digital circulation.** Walking with frog supports in soft ground for 5 to 10 minutes every 3 to 4 hours is beneficial for nonlame horses during the developmental stages of laminitis. It may increase the amount of laminar deformity when used in horses with lameness, or in nonlame horses that have a depression at the coronary band, indicating that laminar degeneration has already begun. Perineural anesthesia of the palmar digital nerves, at the level of the apex of the proximal sesamoid bones, may decrease pain-related peripheral vasoconstriction and thereby promote digital circulation. A 3- to 4-ml dose of 0.75% bupivacaine hydrochloride (Marcaine HCl)* may be used at each site at approximately 12-hour intervals for 1 to 2 days. Severely lame horses should not undergo nerve blocking because it may prompt them to put more stress on tenuous laminar attachments. α-Adrenergic blocking drugs such as acepromazine, phenoxybenzamine, and prazosin also have been advocated to decrease peripheral vasoconstriction and promote digital circulation. Acepromazine (0.02 to 0.04 mg/kg intramuscularly, four times a day) may be given for its theoretic effect on

*Winthrop-Breon Laboratories, Division of Sterling Drug Inc., New York, NY, 10016.
digital circulation and for the sedative effect that encourages the horse to lie down and reduce laminar tension. Before lameness develops, heparin may be administered (40 to 100 U/kg, SC, two to three times a day) to provide laminitis prophylaxis by attenuating potential microthrombosis. This therapy significantly reduced the number of horses developing laminitis after proximal enteritis when given before the onset of laminitis.175

**Other treatments.** Antibiotics may be indicated in severe cases to reduce the risk of secondary sepsis in the foot. Methionine (20 to 60 mg/kg, PO sid) and biotin (0.03 to 0.2 mg/kg PO sid) have been used for their effect on keratinization.

**Prognosis.** Owners should be advised that it is often hard to arrive at an accurate prognosis for up to 6 weeks after the original insult. Redden176 has suggested the following general guidelines regarding prognosis:

- Horses that become sound within 24 to 48 hours of the onset of treatment remain sound, demonstrate no radiographic changes, and have no palpable increased pulsation of the digital arteries after they have been off of all medication for 5 days have a good prognosis. They should be given 10 additional days of stall rest, after which they can be vanned or put back to regular work.

- Horses that develop 2 to 5 degrees of rotation within the first 30 days of onset but then become sound, remain sound, and show no further radiographic progression after an additional 45 days without treatment have a good prognosis. They may resume light exercise, but they should not be shipped long distances for several months, and they should be considered to have an increased risk of recurrence.

- Horses that develop 5 to 10 degrees of rotation in the first 6 weeks but then have no further radiographic progression should receive an additional 90 days of stall rest. If they remain sound without medication, they may resume light exercise with caution after they have been turned to pasture for an additional 12 months. Such horses will not return to their previous level of performance and are not suited for racing or endurance, but may function as pleasure horses.

- Horses that develop 10 to 15 degrees of rotation within the first 4 to 6 weeks have a poor prognosis. The tip of the distal phalanx often penetrates the sole. Necrosis of the dermal and epidermal laminae and subsolear tissues usually occurs. Drainage commonly is noted at the coronary band or heels and is an indication of subcapsular abscessation. Gas or fluid pockets may develop between the hoof wall and the dorsal surface of the distal phalanx. Such cases require drainage and debridement of the necrotic tissue, which may be accom through an anterior hoof wall resection. A keratinized sole is underrun, and it should be elevated off the underrun area with antiseptic solution. Daily bandage changes and antisepsic flushing or soaking are required to prevent infection. A thin layer of keratinized sole may be left in place, provided it does not cause the horse discomfort. A thin keratinized tissue reduces the potential for ant granulation and seems to increase the rate of reepithelialization across granulating wounds on the sole.

Horses with this degree of laminitis require months stall rest and are chronically crippled, at best. Prevention of laminitis requires several thousand dollars of care and barn space just to stabilize the foot. The foot usually is chronically painful, and, if so, euthanasia is justifiable on humane grounds. Tenotomy of the deep digital flexor tendon is beneficial in these cases. It seems to protect severely affected horses to become more comfortable, enhances reepithelialization of defects in the hoof, and permits the anterior hoof wall to grow better.

- Horses that have circumferential laminitis where the distal phalanx drops 2 cm or more to 20 degrees with respect to the hoof wall within the first 4 to 6 weeks of onset carry a poor prognosis.

**Prevention and Control.** Prevention should be aimed at controlling as many risk factors as possible. Restricted grazing on lush spring grasses should be avoided, especially in areas where horses have developed laminitis in preceding years and especially if a horse has a history of laminitis. Horses should be fed a ration that primarily consists of concentrates. Factors that cause gastrointestinal upset should be avoided; for example, changes in ration should be made slowly, and overfeeding should not be allowed to promote condition. The mare should be treated with oxytocin or other hormones 24 hours after parturition.

Preventive measures should be instituted before the development of clinical signs for horses that are at risk of developing laminitis from conditions such as metritis, torsion of the colon, pleuropneumonia, and severe diarrhea. Preventive therapy should involve frequent walking, frog support bandages or stabilizing soft surfaces, and administration of nonsteroidal anti-inflammatory agents and antiendotoxin hyperimmune rum.
Diseases of the Bones, Joints, and Connective Tissues

Signs of acute fluoride toxicity include restlessness, stiffness, anorexia, agalactia, salivation, vomiting or regurgitation, urinary incontinence, diarrhea, clonic convulsions, hyperemia, weakness, severe depression, and cardiac failure. Necrosis of the gastrointestinal mucosa and high concentrations of fluoride in plasma and urine are present in acute fluoride toxicity. Chronic fluoride toxicity is most commonly referred to as fluorosis, a general term that includes osteofluorosis and dental fluorosis. The most common sources of excess fluorides in the diet are (1) water with a naturally high fluoride content, (2) forages contaminated with fluorides from nearby (upwind) industrial plants (e.g., phosphate processing plants, aluminum plants, smelters), (3) mineral (nondefluorinated rock phosphorus) and feed supplements with excessive fluoride content, (4) forages contaminated by soil or water (particularly sprinkler irrigation water) with a high fluoride content, or (5) volcanic activity, which can deposit fluoride-containing ash on soil, plants, or in water used for agriculture.

Clinical Signs, Differential Diagnosis, and Pathophysiology. Clinical signs of fluorosis are usually first recognized as either dental fluorosis or osteofluorosis. Developing teeth are very sensitive to the ingestion of excess fluorides. The deciduous teeth rarely show signs of dental fluorosis, as a partial placental barrier to the accumulation of fluorides appears to be present in the fetus. During tooth development excess fluorides cause ameloblasts to prematurely reduce in size and the enamel epithelium to form an irregular matrix. This matrix does not calcify normally, producing defects in the mature teeth. Cattle are susceptible to dental fluorosis during enamel matrix formation occurring from approximately 6 months to 3 years of age. Excess fluoride intake after 3 years of age does not result in the typical fluoride-induced dental lesions. Changes in incisor teeth are observed most commonly and include chalkiness, motting (striaions or patches in the enamel), hypoplasia (defective enamel), and hypocalcification. Clinical lesions can be graded from normal to excessive. Factors that influence dental fluorosis include the amount of fluoride ingested, the animal's age, the duration and consistency (intermittent vs. continuous) of exposure to fluoride, and the source and chemical form of fluoride ingested. Although diagnostically useful, dental lesions alone should not be used as the sole criterion to determine the degree of fluorosis.

Fluoride accumulation in bone occurs over a prolonged time; osteofluorosis can eventually develop if excessive fluoride is ingested. In cattle the first palpable lesions occur on the medial surface of the proximal third of the metatarsal bones. Later, lesions can be palpated on the mandible, metacarpal bones, and ribs. Radiographically, osteofluorotic bones are thickened.