

Non-Infectious Lameness

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■ Take Home Messages

- ▶ Nutrition, standing time and type of floor have a strong influence on claw-horn lesions like sole ulcers, white line disease or sole hemorrhage
- ▶ Persistent undetermined lameness not originating from the feet must be examined
- ▶ Osteochondrosis is more frequent in fast growing animals on a high energy diet
- ▶ Fracture of the metacarpus and metatarsus can be treated successfully on the farm

■ Introduction

Non-infectious lameness can be divided into 4 major categories based on its origin or location: claw-horn disease, joint diseases, traumatic injuries and neurological lameness. Claw-horn disease is often called laminitis. Non-infectious pathologies include osteochondrosis and degenerative joint disease. Traumatic injuries are rare but life threatening and immediate assistance is needed. Neurological diseases are rare. Clinical evaluation of neurologic animals is sometimes difficult and final diagnosis is often by exclusion. Non-infectious lameness is often multifactorial in origin. Nutrition, housing and genetics can be involved. Because of this, it is often difficult to control and prevent some pathology. How much time is the animal standing in a day? Is the floor slippery? Can all the cows lie down at the same time? Do the calving stalls have clean non slippery bedding? Is the cow with posted legs more susceptible to developing joint diseases? Is ruminal acidosis responsible for laminitis? Although some questions might be easy to answer, solving the problem is not so easy. We will review the most common non-infectious causes of lameness with emphasis on causes and clinical signs.

■ Lameness Examination

Observation at a Distance

A great deal of information is obtained by taking a few minutes to look at the animal quietly in its stall or in its normal environment (e.g. pasture, dry lot, group housing). Lameness is most easily assessed when the cow is observed in motion. In tie stall barns, lameness evaluation is more challenging because of limited space and movement. Displacement of cows in the alley could be difficult and hazardous.

Cattle lameness is generally assessed by observing the cow's stance. Attention should be paid to the posture of the cow, including the back, shoulders, pelvis, and major limb joints. With the animal standing, the general stance is observed first and then more specifically each limb and digit. Compare one region to the opposite side and determine if obvious swelling, wounds, shifting of weight, and foot posture such as toe touching or displacement of weight bearing on the medial or lateral claw are present.

Examination of the claws will reveal excess wear of the wall and sole of the healthy digit. In long standing diseases with severe lameness, the heels are higher and the wall longer on the affected foot compared with that of the healthy foot. A dropped fetlock (e.g. hyperextension of the fetlock joint) may be noticed on the sound limb because of excessive load on the flexor tendons and suspensory ligament. In young animals, angular limb deformities secondary to uneven weight bearing occurs rapidly with chronic lameness.

Differential diagnoses for non-weight bearing lameness should always include sole abscess, fracture, major joint luxation (e.g. hip), critical weight bearing ligament or tendon injury (e.g. gastrocnemius muscle), critical nerve injury (e.g. radial nerve, femoral nerve, sciatic nerve), septic arthritis, and septic tenosynovitis. An abnormal deviation of the limb is usually related to a fracture or joint luxation. The stance and position of the limb are abnormal with nerve damage, tendon rupture, or a severe ligament injury. Cattle affected with a radial nerve paralysis will have a dropped elbow, but this must be differentiated from a humeral fracture, radial/ulnar fracture, or septic arthritis of the elbow joint. A rupture of the tendinous portion of the gastrocnemius muscles is shown by a hyperflexion of the hock and a dropped calcaneus, but this must be differentiated from a fractured calcaneus or sciatic nerve paralysis. Careful attention should be paid to muscle atrophy because this could be caused by nerve injury or disuse. Neurogenic (paralysis) muscle atrophy occurs very rapidly (7 to 10 days) and is generally more severe. Muscle atrophy caused by disuse occurs over a longer period of time (weeks). Chronic lameness of the front limb will usually bring atrophy of the triceps, biceps and scapular muscles. The consequence of this atrophy is a more

apparent shoulder with joint instability. The animal may be falsely diagnosed with shoulder joint diseases. Similarly, atrophy of the muscles of the rear limb will make the greater trochanter of the femur prominent which may be misdiagnosed as a hip luxation.

Examination during Movement

In certain cases, lameness is subtle and other procedures are necessary to localize the lesion. Lameness' characteristics are easier to assess with the animal walking. The observer looks at the severity of the lameness and assesses the individual components of the gait including the arc of flight, position of the digit when it touches or leaves the ground, and the relative time spent in each phase of the stride. When diseases of the proximal limb, such as the hip, stifle, or shoulder are suspected, the individual structures should be palpated as the animal walks.

Examination of the Affected Limb

At this point, we should have an idea of which leg is affected. Unless an obvious lesion is apparent, we start with palpation of the limb from the digit working up the leg. The clinician should watch for pain reaction and determine if swelling, deformation, crepitation, warmth, and wounds are present. A hoof tester is used to evaluate pain of the claw. A hoof tester is often used to rule out any pain originating from the foot. Sedatives and tranquilizers should be avoided whenever pain responses are desired. After localization of the lesion, local anesthesia or sedation may be required to complete the examination.

Examination of long bones is performed by applying firm pressure in regions of minimal soft tissue covering. If the animal has an adverse response, evidenced by withdrawal, avoidance, attempts to kick the evaluator, or muscular flinching, then the opposite leg should be palpated for comparison. Most fractures are obvious, but incomplete non-displaced fractures can be suspected if there is a painful reaction after deep palpation of the limb. Each joint should be palpated separately and complete flexion, extension, abduction and adduction of the limb done. Specific physical exams are used when injury to the hip or cruciate ligament rupture are suspected.

■ Claw-Horn Disruption

Claw-Horn Disruption or Laminitis?

Claw-horn disruption is by definition any event or pathology that will influence claw-horn growth. Subclinical laminitis is often associated with horn growth abnormalities. The clinical manifestations are poor horn quality, sole hemorrhage, sole ulcers and white line disruption. Laminitis is one aspect of

claw-horn disruption. Laminitis by definition is the inflammation of the lamella of the foot. The term laminitis was borrowed from the equine condition because of similar clinical signs. The events leading to the ultimate sinking of the pedal bone seems to be different in cattle (Lischer Ch et al., 2002, Bicalho et al., 2009, Danscher et al., 2010, Machado et al., 2010). In horses and cattle, the horn is attached to the corium (sensitive part of the claw) by lamellae which are very small leaflets providing nutritive supply to the non-sensitive horn.

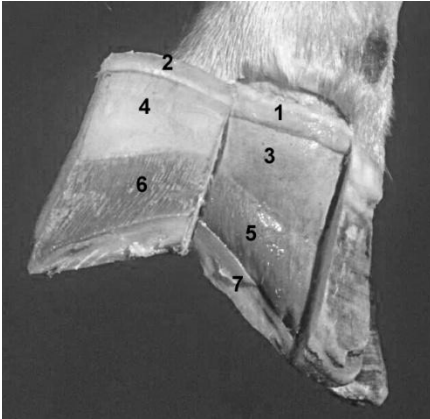


Figure 1. Cadaver limb of an adult cow. The horn of the wall has been partially detached from its corium (sensitive part).

1 = papillae of the perioplic corium; 2 = perioplic horn; 3 = papillae of the coronary corium; 4 = coronary horn; 5 = lamellar corium; 6 = epidermal corium; 7 = solar horn

The corium is attached to the pedal bone. In horses, the inflammation of the lamellae will create a separation of the sensitive and non-sensitive lamellae provoking pain and moreover sinking or a rotation of the pedal bone in severe cases. This separation is permanent. The void between the lamella will be filled up with poor quality connective tissue and secondary horn (white line horn production). The white line will then be wider and weaker. The corium will be damaged to various degrees depending on the pedal bone displacement. The origin of the detachment is different in cattle but unfortunately the results are the same. It varies from focal hemorrhages to apical protrusion of the pedal bone (Gantke et al., 1998, Bergsten, 2003).

Subclinical rumen acidosis has been frequently associated as a causative agent of laminitis in cattle (Greenough, 2007). Toxin, endotoxin and inflammation by-products (Plaizier et al., 2008) produced by the rumen flora during ruminal acidosis might have a detrimental effect on the vascularisation of the foot or directly on the suspensory tissues of the pedal bone. Livesey and Fleming (1984) studied the effect of a low fibre, high starch diet on the

incidence of clinical laminitis and sole ulcers. Cows on the high starch ration had a 68% incidence of clinical laminitis and 64% incidence of sole ulcers while cows on high fiber diet had an 8% incidence of clinical laminitis and sole ulcers. Using the oligofructose overload model, Dansher et al. (2010) showed that in fact, there are clinical (weight shifting, solar hemorrhages) and histological changes at the lamellar level consistent with laminitis. However, it does not decrease the strength of suspensory tissue of the bovine claw.

The Suspensory Apparatus of the Pedal Bone

The suspensory apparatus basically keeps the pedal bone attached and suspended into the horny shell by the lamellae, connective tissue and the digital cushion underneath. The connective tissue is on each side of the pedal bone and acts like a sling keeping the pedal bone suspended.

The digital cushion is composed of three fat cylinders with connective tissue. Lischer et al. (2002) dissected normal claws and those with sole ulcers. They found that the fat pads were thinner or gone in animals suffering from sole ulcers whereas the connective tissue was elongated. These two factors explain the sinking of the pedal bone in the bovine sole ulcer model. The downward movement of the bone will crush the sensitive tissue (corium) creating hemorrhage at different degrees changing the quality of the produced horn as well. As a result, the animal will show sole hemorrhage and sole ulcers.

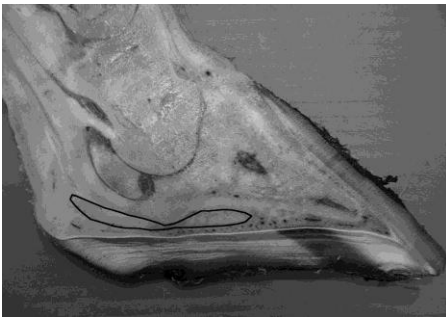


Figure 2a. Cadaver limb from a dairy cow. The digital cushion with fat cylinders is highlighted



Figure 2b. Cadaver from a dairy cow with claw-horn disruption. 1- the pedal bone is rotating and the dorsal wall is concave. 2- the fat of the digital cushion is thinner and is transformed in connective tissue (darker)

If the apex (tip) of the coffin bone sinks, the result will be toe ulcers. There is a relation between the fat pad cylinders and body condition score (BCS). Bicalho et al. (2009) studied the relationship between claw horn lesions and the digital cushion. They found that the digital cushion thickness decreased steadily from the first month of lactation to the thinnest point at 120 days of lactation. The same research group found also a relationship between BCS at dry-off period, the digital cushion thickness and claw-horn lesions (Machado et al., 2011).



Figure 3a. Heifer in a free stall barn with solar hemorrhages (1) and white line disease (2)



Figure 3b. Holstein cow with toe ulcers on both digits.

Cow Comfort Effect

There are three major causes of claw-horn diseases: nutrition, claw conformation and cow comfort. Nutrition and subclinical acidosis have been incriminated in subclinical laminitis. Poor claw conformation (long toe) is a frequent cause of sole ulcers. The biomechanics of the digits is changed with excessive pressure at a particular point of the sensitive tissue at the junction of the heel and the sole axially. It is getting clearer that cows standing on concrete for long periods of time are more susceptible to claw-horn disruption. Floor type has been reported to have an influence on claw health; however it's variable according to specific diseases. A slatted floor decreases the incidence of digital infection whereas there are fewer sole hemorrhages and other claw horn defects on a rubber floor (Fjeldaas et al., 2011, Haufe et al., 2012). Heifers can be affected as well especially if there is no gradual transition between groups. A study on heifer claw health in Austria showed a prevalence of 9.4% for lameness and 98.5% for claw lesions with white line lesions and heel horn erosions being the most common (Kofler et al., 2011). Competition between animals, standing or waiting time for feeding or milking will make heifers quite susceptible to claw-horn disruption. Rubber mats in waiting or feeding areas, short waiting time in holding pens, and an adequate

number of comfortable cubicles must be considered to decrease the incidence of claw-horn disruption.

■ Osteochondrosis

Osteochondrosis is a common disorder in horses and pigs but infrequently reported in cattle. This disease affects the growth cartilage of young animals, specifically the joint cartilage and the long bone growth plate cartilage. The joint cartilage provides smooth gliding of articular surfaces and allows some shock absorption. It is also involved in bone growth in young animals. The cartilage of the growth plate is responsible for the longitudinal growth of long bones. Some growth plate will be active for 6 months and most will be inactive by the age of 24 months. This growth plate is an area of weakness where fracture will occur in young animals. Osteochondrosis occurs when there is an abnormal ossification of the cartilage. Consequently, the cartilage cannot sustain excessive shear and compression (as it is supposed to). The surface of the cartilage will become flaky and sometimes large fragments of cartilage will detach and become loose in the joint. These loose fragments and the exposed sub-chondral bone (bone underneath the cartilage) will create inflammation in the joints resulting in severe distension.

The long bone growing cartilage can be affected as well but there is no loose cartilage. Instead, enlarged growth plates and angular limb deformities will be observed. Poor focal vascular supply of this zone could be at the origin of the defective cartilage. The exact origin of the vascular problem is unknown but repeated microtrauma is suspected.

Causes of Osteochondrosis

Predisposing factors of osteochondrosis are not well established. The most common causes are heredity, rapid growth, anatomic conformation, trauma, and dietary imbalances (Ytrehus et al., 2007). High energy and protein diets have been investigated and suspected as a cause of osteochondrosis. Animals on nutrient dense diets were more affected and the lesions more severe (Reiland et al., 1978). Low concentration of calcium was also suspected. Other nutrients like phosphorus and vitamins A and D may play a role in osteochondrosis as well (Davies and Munro, 1999). Genetics has been suspected in grazing beef cattle where affected animals, the males, shared a common ancestral sire (Hill et al., 1998). However, the caloric intake rather than the concentration of any minerals is the most important predisposing factor among all the studies. Hard flooring was shown to exacerbate methaphyseal osteochondrosis lesions in calves (White et al., 1984).

Clinical Presentation

In cattle, joints commonly affected are the stifle and the hock. Osteochondrosis lesions have also been diagnosed in the carpus, shoulder, and the pedal joint. In a study where 28,235 atlanto-occipital joints were examined, 3.8% had lesions compatible with osteochondrosis (Jensen et al., 1981). In the same study, 8.5% of the 106 lame cattle had lesions of osteochondrosis mostly in the stifle. Bilateral lesions of the same joint were present in 88% of the animals (Trostle et al., 1997). Recently, osteochondrosis-like lesions were reported in nine Holstein heifers (Mulon et al., 2009). All of the animals were of high genetic values but they were not related to the same ancestor. Bilateral lesions were present in four animals. Typically the front limbs are affected; the animals have a stiff gait and they walk on the tip of their claws. Some will stand or walk on their carpi ('front knees') (Figure 4b)



Figure 4a. Distension of the hock on a Holstein heifer caused by osteochondrosis



Figure 4b. Young Holstein heifer suffering from bilateral osteochondrosis of the pedal joint

Affected animals are young with an average age ranging between 10 and 24 months. Onset of the lameness is gradual and early stages can be missed, especially in large herds. Affected animals become stiffer and more reluctant to walk. Joint distension is variable but certainly the most significant clinical sign. Lesions are often bilateral but only one joint may be clinical. At palpation, the distended joint is soft and painless. Final diagnosis is often based on radiographic findings.

Conservative treatment for osteochondrosis should be considered first. It is stall rest, ration adjustment, and anti-inflammatory drugs if needed. Analysis of feed intake and mineral supplementation should be verified when outbreaks of lameness occur in young cattle receiving a high energy ration. If a cartilage fragment is loose and large, arthroscopic debridement is indicated. Prognosis is generally good depending on the lesion's size and the severity of the clinical signs. If left untreated, some animals may develop arthrosis.

■ Degenerative Joint Diseases

Degenerative joint disease (DJD) is also called osteoarthritis. It is characterized by a degradation of the articular cartilage leading to morphologic changes of the joint. The consequences of the cartilage degradation are inflammation of the synovial membrane and abnormal bone proliferation inside the joint. Pain is a hallmark of DJD. It is variable and inconsistent with the pathologic changes of the joint.

Etiology of DJD in cattle is either traumatic or secondary to poor conformation (straight hind limbs). In all other species, aging is certainly a common cause of osteoarthritis. The productive life of commercial cows being short, we don't see this manifestation of DJD frequently in cattle. Any ligament injuries will make the joint unstable. This instability will create excessive shear or compression stress at the cartilage level with fibrillation and degradation of the cartilage. It starts the vicious circle of inflammation and joint degradation. In fact, many by-products of inflammation are detrimental to the joint and more specifically to the cartilage. They will either block the synthesis of cartilage or activate its degradation.

Abnormal conformation like post legged cows has a slow chronic effect on joint physiology. Those joints are not meant to be straight. The joint angle is biomechanically important to propulsion while walking or shock absorption. Excessive cartilage compression and micro trauma will result from this anomaly. At the opposite of DJD from trauma, more than one joint might be involved with bad conformation. These animals have a short life expectancy especially on hard flooring.

Clinical signs and evolution are variable depending of the cause, age, activity, and housing. It generally starts with a mild joint swelling and lameness. As the disease progresses, the joint can become harder in some area (bone spurs and fibrosis) and the gait will change (stiff gait and short stride). The animal will spend more time laying down and will be more reluctant to stand and walk.

Unfortunately, when the process of DJD is initiated, there is no turning back. We can alleviate pain with anti-inflammatory and chondro protector but it will not reverse the process of degeneration.

■ Traumatic Injuries

Farm hazards make animals susceptible to trauma. Slippery floors, stall bars, and fights between animals are dangerous but unavoidable. They cause lacerations, fractures and ligament injuries. Of those, fractures are certainly the most catastrophic and impressive injuries. All the long bones can sustain

fractures but the metacarpus and metatarsus are more commonly affected. The fractures are usually accidental. In rare cases, nutritional deficiencies, congenital abnormalities or infection can make the bone brittle and more prone to fractures. As a rule of thumb, the higher is the fracture, the lower is the prognosis and the more expensive it is to treat the animal.

Veterinary surgeons have the same orthopedic tools as any orthopedists in human surgery: plates, screws, wires, pins, and interlocking nails. Some of those implants are adapted to large animals but they are very expensive. Depending on the fracture configuration, its location and the value of the animal, the surgeon will choose the appropriate technique to immobilize the fracture. These procedures are not performed on the farm because the sterility is extremely important. Bones and joints are susceptible to infection and very difficult to treat.

A more common and cheaper way to treat a fracture is with a synthetic cast made of polyurethane resin and fibreglass. The majority of metacarpal and metatarsal fractures can be treated with cast immobilization. The prognosis is generally excellent if the fracture involves the growth plate with an immobilization period of four weeks. Fortunately, this is the most common fracture in cattle. The prognosis for recovery decreases in older heavy animals and fracture complexity. If the fracture becomes open (bone through the skin or bone can be seen from a wound) the prognosis decreases significantly because the healing is delayed and infection is difficult to control.

During dystocia, the calf can sustain excessive traction. Unfortunately, there are sometime complications if traction was excessive. Many different bones can be fractured: metacarpus, femur, mandible, vertebra and ribs. Inadequate positioning of obstetrical chains around the metacarpus or mandible (abnormal position of the head) is responsible for such fractures. Vertebral fractures usually occur when the calf is locked in the pelvis and excessive traction is pursued. The calf will be paralyzed because of the damage to the spinal cord. A femoral fracture is most likely secondary to hip lock or if the stifle is flexed against the pelvis instead of being extended. Neonates with obstetrical fractures must be considered as poly-traumatized and carefully evaluated.

Fracture of the more proximal bones like the radius, tibia, femur and humerus are more difficult to treat. The heavier the animal is the poorer the prognosis for recovery and euthanasia is often recommended.

■ Lameness of Neurological Origin

This is a very complex area of medicine because we cannot easily directly access the problematic area. Simplistically the nervous system is divided as central and peripheral. The central portion includes the brain and the spinal

cord. The peripheral nerves originate directly from the brain (cranial nerves) or the spinal cord (spinal nerves). They are sensitive and motor. Spinal nerves are the bridge between the central nervous system (CNS) and organs (muscles, intestines, etc). In general, more than one leg will be affected if the lameness originates from the CNS. If the brain is affected, other clinical signs are observed as well (fever, weakness, tremor, depression, blindness). Fortunately, this is not a common cause of lameness in cattle. However, peripheral nerves are more often affected especially around calving. Fetopelvic disproportion is at the origin of some of those nerve injuries. The sciatic and the obturator nerves can be damaged. If the nerve is compressed and inflamed, the clinical signs will improve in two weeks. However, if it is disrupted or cut, then healing time is 1mm per day and it's not guaranteed. Medically speaking, there is nothing that can be done to speed up nerve healing. Downer cows laying on the same side for a long time are also susceptible to nerve injury. The peroneal nerve is typically affected in those cases. Clinically the fetlock will knuckle over and the hock will drop. If it is bilateral, spinal cord involvement should be suspected like lymphoma (bovine leukosis).

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