Nutritional and Animal Welfare Implications to Lameness

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Abstract

Rumen acidosis is associated with fermentive disorders occurring secondary to the consumption of feedstuffs that contain relatively large amounts of highly fermentable carbohydrates. It may be complicated by diets that offer less than adequate levels of effective fiber. Lactic acid accumulates within the rumen, leading to rumenitis and a change in the rumen flora that favors lactate production. The change in rumen flora is also associated with the release of endotoxins within the rumen. These endotoxins, along with various other vaso-active substances (including lactate), are absorbed into the blood stream where they disrupt the normal flow of blood within the tissues of the corium. Inflammation of the corium initiates the release of potent metalloproteinase enzymes that cause destruction of the suspensory apparatus of the 3rd phalanx. Sinking and rotation of the 3rd phalanx follows with compression and damage to the supporting tissues and structures of the corium that precipitates the development of sole ulcers and white line disease. In recent years, research from the United Kingdom suggests that damage to the suspensory apparatus is likely to occur by other mechanisms. Their work demonstrates that as animals' progress toward calving, rigidity and strength of the collagenous connective tissue of the suspensory apparatus is significantly reduced (Tarleton et al., 2002; Tarleton and Webster, 2002). They propose that these changes are potentially a consequence of both the activation of metalloproteinase enzymes and peripartum hormones, such as estrogen and relaxin. The implications of this are that in addition to feeding and nutrition, dairy farmers must pay particular attention to cow comfort during the transition period. The digital cushion consisting of fat and loose connective tissue is an important support structure in the claw. Recent work suggests that body condition score mirrors size (i.e. fat content) of the digital cushion and may be very important to the integrity and health of claws. Whereas, most have thought that lame cows become thin, their research suggests that thin cows become lame. Maintenance of good body condition throughout the first 120 days of lactation may prove to be a very important feeding objective. The welfare implications of lameness are huge. Lameness affects performance, causes pain, and disrupts the cow's ability to express normal behavior. The underlying causes of lameness are multi-factorial, but based upon the preponderance of information on the pathogenesis of lameness in recent years, there is little question that housing is a major contributor to the development of lameness disorders. Overcrowding, stall design and stall surfaces, flooring, and specific management practices restrict resting and force cows to stand for longer periods. Smooth and manure slurry covered or wet floors are slippery and may precipitate injuries due to falling. Abrasive flooring conditions cause excessive wear and lameness due to thin soles. Anything that increases the incidence of lameness contributes to poor animal welfare. Treatment of lameness needs to be timely and carefully administered.

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Rumen Acidosis and the Vascular Disturbances Contributing to Laminitis

Rumen disorders that result in acidosis are usually associated with the ingestion of large amounts of highly fermentable carbohydrate-rich feeds, resulting in the excessive production and accumulation of lactic acid in the rumen (Nocek, 1997). In its acute form, rumen acidosis is a life threatening disease characterized by severe toxemia, ataxia, incoordination, dehydration, ruminal stasis, bloat, weakness, recumbency, and ultimately death. The subclinical form of rumen acidosis (better known as SARA, for SubAcute Rumen Acidosis) occurs more commonly. Clinical manifestations would include: variable feed intake, depressed fat test, poor body condition despite sufficient energy intake, mild to moderate diarrhea, and occasional cases of epistaxis (nose-bleed) or hemoptysis (the expectoration of blood from the mouth).

Relatively few studies have been able to establish a direct link between rumen acidosis and laminitis, yet observation and clinical experience suggests feeding is an important underlying factor (Nordlund, 2002). Acidosis causes varying degrees of rumenitis which permits absorption of lactate, endotoxins (from the death of gram negative microorganisms in the rumen), and bioactive messengers, such as histamine and serotonin, from the rumen wall. These combined with vasoactive substances, such as epinephrine and norepinephrine, cytokines, prostaglandins and other substances, have direct effects on the vascular endothelium that initiate a cascade of events, including a decrease in blood flow caused by the simultaneous dilatation of arterioles and constriction of venules. Damage to walls of blood vessels leads to the extravasation (leaking) of blood and blood fluids into extravascular (into the tissues surrounding the vessels) tissues of the corium. This is complicated still further by stagnation of blood within the capillaries and other small blood vessels, resulting in thrombosis (clotting), ischemia (lack of blood flow), hypoxia (low oxygen), and eventually arterio-venous shunting (Vermunt and Greenough, 1994).

The end result of the above events is inflammation, accompanied by swelling, hemorrhage, and death of corium tissues. Functional disturbances follow, including the activation of matrix metalloproteinases **(MMP)** that degrade the collagen fiber bundles of the suspensory apparatus of the third phalanx (i.e. P3, the bone within the claw capsule). These changes are exacerbated by the activation of epidermal growth and necrosis factors that contribute to structural alterations involving the basement membrane and capillary walls. Although somewhat complicated, these are the major lesions associated with laminitis at the cell and tissue level. A more comprehensive discussion of these may be found in other recent publications (van Amstel and Shearer, 2006; Greenough, 2007).

Laminitis (Pododermatitis aseptica diffusa)

Laminitis, also known as founder, is an important predisposing cause of disorders affecting the digit in cattle. It is characterized by disrupted blood flow (as described above) that results in inflammation and the activation of potent enzymes (metalloproteinases) that damage tissues responsible for suspending the third phalanx **(P3)** within the claw horn capsule. To understand this phenomenal suspensory system and what happens when it fails is fundamental to understanding the pathogenesis of claw disorders (i.e. ulcers and white line disease) in cattle.

The suspensory apparatus of P3

The laminar corium (sensitive lamina) of the claw is the primary suspensory tissue for P3. Cows essentially "hang in their claws" by virtue of a series of laminar folds that are anchored on the abaxial, dorsal, and axial surface of P3 and extend outward to interdigitate with the lamelle of the wall. Beneath P3 are tissues which make up the underlying support

structure for P3. This tissue is composed of loose connective tissue from the solar and perioplic corium and caudally by the digital cushion. The digital cushion is an important support structure composed of loose connective tissue and varying amounts of adipose tissue. It has become the object of attention by several researchers in recent years as recent observation suggests that the size and type of fat within the digital cushion may have important implications in the occurrence of lameness (Lischer et al., 2002; Raber et al., 2004).

Inflammation leading to destruction of the dermal-epidermal junction results in weakening of the suspensory apparatus that predisposes to the downward displacement and rotation of the P3. The result is compression of the corium and supporting tissues that lie between P3 and the sole which predisposes to the development of sole ulcers (Ossent and Lischer, 1998). In some cases, rotation of the apex of P3 is severe enough to cause dysfunction of the corium in this region and predispose to a toe ulcer. If, on the other hand, sinking of the P3 is such that the rear portion sinks the furthest, compression at the heel-sole junction will result in the development of a sole or heel ulcer. Sole ulcers are one the most common claw lesions in dairy cattle and constitute one of the most costly of lameness conditions.

Displacement of P3 by Alternate Mechanisms

While most have considered rumen acidosis to be the primary cause of laminitis, lameness researchers in recent time suggest that it is more complicated than previously believed. In fact, the claws of cows and particularly heifers are less resistant to compressive loading forces than previously thought and especially so during the peri-partum period. The confinement of cows on hard surfaces is potentially one of the single most important predisposing causes of lameness in dairy cattle.

Activation of matrix metalloproteinases by "hoofase"

Researchers from the United Kingdom studied the supportive capacity of the suspensory apparatus of P3 in first lactation heifers and age-matched maiden heifers during the peripartum period (Tarleton et al., 2002; Tarleton and Webster, 2002). They observed increased laxity, reduced rigidity, decreased load-bearing capacity, and a clear deterioration in the structural integrity of hooves in first lactation heifers. Furthermore, these changes appeared to be progressive over the period of 2 weeks prior to calving until 12 weeks post-calving. These hoof characteristics were not observed in age-matched maiden heifers. Workers suggested that these changes would permit sinking of P3 and thus predispose affected animals to compression of the corium and sole ulcers.

Biochemical explanations for these observations were explored, and in the process, researchers identified a unique ~ 52kD gelatinolytic enzyme they termed as "hoofase". This enzyme was isolated from all specimens derived from calving heifers; however, none was found in specimens from the maiden heifers. Researchers also sampled each of the study groups to determine if there was a relationship between hoofase and the types and levels of MMP isolated from the connective tissue samples. Interestingly, researchers observed the highest levels of hoofase in pregnant heifers approximately 2 weeks prior to calving. In addition, they found highly significant increases in the activated form of MM-2, a very important metalloproteinase involved in the mediation of collagen remodeling in normal animals (Tarleton and Webster, 2002). On the other hand, levels of metalloproteinase-9 (MM-9), the enzyme most consistently associated with inflammation as related to rumen acidosis, was not found in significant quantities in either the first lactation or maiden heifers. This suggested that the classical form of rumen acidosis-induced laminitis was not a cause of the changes observed. There

were marginal increases in "proMM-2" (a metalloproteinase normally responsible for physiological and pathological remodeling of connective tissues). Researchers concluded that these results indicate that hoofase plays a very important role in the activation of MM-2 and weakening of the suspensory apparatus by a mechanism quite different than that related to rumen acidosis. Considering the observations of this study, indicating that peak hoofase activity occurs approximately 2 weeks prior to calving and that it continues well into the early postpartum period, one must conclude that hoofase may have a very important role in the cause of claw lesions.

Peripartum hormonal effects

Researchers from the United Kingdom have suggested yet another mechanism for weakening of the dermal-epidermal segment between the wall and P3. Their work suggests that weakening of the suspensory tissue may be the result of hormonal changes that normally occur around the time of calving (Tarleton and Webster, 2002; Webster, 2002; Knott et al., 2006). In particular, hormones such as estrogen and relaxin, responsible for relaxation of the pelvic musculature, tendons, and ligaments around the time of calving, are thought to have a similar effect on the suspensory tissue of P3 as well. Whereas this is likely a natural phenomenon around the time of calving, housing animals on soft surfaces during the transition period (4 weeks prior to calving through 4 to 8 weeks after calving) may be an important management procedure to reduce or alleviate the potential for permanent damage to these tissues. Researchers base this opinion on observations that fewer claw lesions occurred in heifers housed in straw yards compared with those housed in free stalls during the transition period. Researchers concluded that first lactation animals, in particular, would benefit from softer flooring surfaces during the peripartum period .

German researchers suggest that sinking and rotation of P3 may be associated with as of yet unexplained structural alterations occurring on the surface of P3 where the suspensory tissues are anchored (Mulling and Lischer, 2002). It is clear that despite the preponderance of information linking laminitis to feeding conditions and rumen acidosis, softer flooring surfaces and cow comfort cannot be overlooked as requirements for animals during the transition period.

Ulcers of the Toe, Sole and Heel (Pododermatitis circumscripta, Rusterholtz Ulcer/Sole Ulcer, Toe Ulcer and Heel Ulcer)

Displacement of P3 results in compression of the solar and perioplic corium between P3 and the sole. Contusion and bruising of the corium at the toe, sole ,and heel causes injury and dysfunction of the corium. In cases where displacement of P3 involves severe rotation of its apex, a toe ulcer may develop. If, on the other hand, sinking of P3 is such that the rear portion sinks furthest, compression of the solar and perioplic corium of the heel will lead to development of a sole ulcer at the heel-sole junction (Raven, 1989). Toussaint Raven has characterized this as the "typical site", i.e., the site most commonly associated with the development of sole ulcers.

An ulcer is defined as a full-thickness defect or break in the epidermis that exposes the corium. One of the earliest indications of a developing sole ulcer is hemorrhage in the sole, particularly when it occurs at the heel-sole junction. If the animal exhibits pain when pressure is applied to this area, it offers good evidence that the ulcer is in the clinical stage. With additional time and trauma associated with weight bearing, this lesion will likely progress to a full-thickness horn defect or ulcer. In the pre-clinical or early stages of development, despite the size of some hemorrhages, pressure as might be applied with a hoof tester causes little or no discomfort. Treatment of these cases may be accomplished by

lowering the heel on the affected claw so that it may have time with reduced weight bearing for rest and repair. If, on the other hand, one is able to illicit pain by putting gentle pressure over this area with a hoof tester, one should not only lower the heel on the affected claw but also consider application of a foot block to the healthy claw to insure complete relief from weight bearing. When identified early, these cases will usually recover quite rapidly (within 3 to 4 weeks).

Mature ulcers are usually accompanied by lameness and even gentle pressure with a hoof tester over the ulcer site will illicit a positive pain response. Removal of superficial layers of horn may uncover an area of exposed corium that is extremely sensitive. Assuming minimal damage of the corium, these may be treated by thinning horn around the base of the ulcer and lowering this area relative to the weight bearing surface of the healthy claw. It is also advisable to avoid leaving a crater or hole in the sole that will fill with organic matter. Instead, slope the sole axially toward the interdigital space. Recovery time for ulcers requires a minimum of 20 to 30 days, and based on studies by European workers, recovery may take as much as 50 to 60 days in severe cases. The objective should be to provide relief from weight bearing on the affected claw for at least 1 month by means of a foot block and an additional 20 to 30 days by corrective trimming to adjust load bearing between the two claws. It is important to remember that if there has been significant damage to the corium, recovery may be extended. In some cows, this may require that a new block be applied as soon as the first one is no longer functional due to wear (van Amstel and Shearer, 2006).

For chronic ulcers where long-standing inflammation has resulted in granulation tissue formation: 1) apply the corrective trimming procedures described above, 2) carefully remove the granulation tissue with a sharp hoof knife. Be careful not to damage adjacent normal tissues of the corium. Granulation tissue bleeds freely and recurrence rates for ulcers with exposed granulation tissue are high.

To fully understand the pathogenesis of ulcers, one must recognize their multi-factorial etiology. The metabolic factors responsible for sinking and rotation of P3 include rumen acidosis and laminitis and also the effects of enzyme activity and hormonal changes that are most common during the transition period. The mechanical factors of greatest importance are those that contribute to unbalanced load bearing within and between claws. The work of Toussaint Raven (Raven, 1989) demonstrates that weight does not distribute itself evenly but more so to the outer claw. This increased weight bearing leads to accelerated horn growth and overloading of the outer claw, the consequence of which increases weight load and pressure on the corium. The biomechanics of loading within the weight bearing surface are also affected by toe length. When the toe is long, the sole in the region of the toe is always thicker. This creates a greater distribution of weight caudally toward the heel-sole junction and is therefore believed to be a contributor to the development of sole and heel ulcers. The purpose of maintenance or preventive hoof trimming procedures is to re-establish appropriate weight bearing within and between claws by eliminating abnormal overgrowth that leads to overloading of the claw.

Finally, size and type of fat within the digital cushions has become a source of interest in understanding the pathogenesis of sole ulcers (Bicahlo et al., 2009). The digital cushion lies immediately above the loose connective tissue of the corium and beneath P3. It consists of 3 roughly parallel cylinders (axial, central, and abaxial) composed primarily of fat which serve as shock absorbers in the heel. Studies by Swiss researchers have found that the digital cushion of heifers is smaller in size and contains more saturated fat, which reduces it cushioning value as compared with mature

cows. These characteristics of the digital cushion in heifers may increase the vulnerability of young animals to claw diseases, particularly sole and heel ulcers. Furthermore, observation of the feet of animals suffering laminitis indicates that the sinking of P3 leads to damage of the digital cushions and replacement of the fat with firmer connective (even cartilaginous or cartilage-like) tissue. The combination of a harder less flexible digital cushion and compression of the corium caused by sinking of P3 results in greater damage to the corium in the heel and consequently, a greater risk of sole and heel ulcers.

Effects of Body Condition on Claw Disorders

The impression of most people is that lame cows lose weight. This is logical since lameness causes pain and reduces their interest in walking or standing and therefore the number of trips a cow might be willing to make to the feedbunk. However, a recent study suggests that rather than lame cows becoming thin, it may be that thin cows become lame (Bicalho, et al, 2008). These researchers investigated the relationship between claw lesions (ulcers and white line disease) and thickness of the digital cushion **(DC)** in 501 lactating Holstein cows. They found that the prevalence of sole ulcers and white line disease increased as thickness of the DC decreased. They also observed that digital cushion thickness decreased steadily throughout lactation, reaching nadir (i.e., its lowest point) at 120 days after calving. Body condition scores **(BCS)** of cows were positively associated with thickness of the DC, whereby an increase in BCS was associated with a corresponding increase in mean thickness of the DC.

Results of this study add further credence to the idea that claw lesions bear a close relationship to external factors, in particular, confinement housing and hard flooring surfaces. Furthermore, it is tempting, if not reasonable, to theorize that the mobilization of fat in early lactation is a significant risk factor for lameness. The results of several studies are now beginning to suggest that when the shock-absorbing properties of the DC are compromised, the corium becomes vulnerable to mechanical injury. Knowing that cows mobilize fat from multiple body locations, it is reasonable to assume that they would likely mobilize fat from the DC as well. In the study described above, researchers noted a decrease in thickness of the DC and also evidence of a change in the DC composition as BCS decreased (Bicalho et al, 2008).

The Cornell study (Bicalho et al., 2008) demonstrated that the highest prevalence of sole ulcers occurred near peak lactation (i.e., 60 to 100 days in milk); the point at which shrinking of the digital cushion was approaching nadir. This is not unlike observations from other studies and also supports an association with a thinner, less functional digital cushion. However, the rumen acidosis-laminitis complex, the effects of hoofase or activation of metalloproteinase activity, and/or the impact of peri-partum hormonal changes can all be theorized as causes of these conditions in a similar time-frame. Therefore, these observations neither preclude nor reduce the significance of other causative factors as mentioned earlier in this chapter. Rather, they highlight lameness's complicated pathogenesis and its multi-factorial causes.

Welfare Implications of Lameness

The primary concerns in animal welfare typically include 3 basic questions: 1) is the animal functioning well (in other words, is it producing well), 2) does the animal have pain or is it distressed, and 3) is the animal able to express or perform natural behaviors (Frazer, 2008; Von Keyserlingk et al., 2009)? When we consider lameness in the context of these concerns, we fail to achieve the objectives of good welfare by any of these measures. Lameness reduces milk production and reproductive performance. It causes pain and interferes with the animal's ability to move about freely or confidently

to interact with herd mates in behaviors such as estrus behavior or interactions to establish dominance.

Housing considerations to improve welfare of lame cows

Poor cow comfort is not only an important predisposing cause of lameness, but it's also what keeps cows lame. Key factors are overcrowding, poor stall design, inadequate bedding and grooming of stalls, abrasive flooring surfaces, slurry covered or wet and slippery flooring surfaces, and management practices that contribute to excessive standing and reduce time available for resting (Shearer and van Amstel, 2007). In short, failure to maximize cow comfort is likely to increase the prevalence, incidence, and duration of lameness, while simultaneously extending time required for recovery from lameness disorders.

Overcrowding

Leonard evaluated the effect of lying time on first-calf heifers in overcrowded conditions (Leonard et al., 1996). He found that heifers which spent 10 or more hours per day lying down had significantly better claw health than those that spent 5 hours/day or less lying down. Cows will normally lie down and rest for as much as 11 to 14 hours/day. Less time resting usually means less time ruminating or "cud-chewing". When cud-chewing time is reduced, the natural buffering of rumen contents by saliva is decreased.

Stall design and stall surfaces

Design features of stalls that appear to be most important are: 1) the provision of sufficient space for movement associated with lying and rising, 2) adequate resting space within the stall, and 3) a well-cushioned bed. Placement of the neck rail appears to affect perching and time spent standing in stalls. Generally speaking, stall dimensions for a large Holstein cow include: head end against a wall, minimum length should be 9.5 to 10 feet; stalls oriented head to head, 8 to 8.5 feet, and recommended stall width varies from 46 to 50 inches (Faull et al., 1996; Anderson, 2002; Cook, 2009).

Lame cows have a particularly difficult time rising or lying down in poorly designed stalls bedded with mattresses. They fare much better in sand stalls because the foot is able to gain much better traction in the loose bed of sand (Cook, 2006). This alone is believed to contribute significantly to the duration of lameness in individual cows. The ideal housing condition for a lame cow is a soft surface (as might be found on an earthen surface), without the normal restrictions that come with a stall. Where weather conditions are unfavorable, special needs barns with bedded packs provide friendly conditions for lame cows.

Flooring

Cows are land animals. Hard surfaces contribute to claw horn overgrowth, which leads to overburdening or overloading of claws and ultimately contributes to the development of disorders of the foot, such as ulcers and white line disease (Raven, 1989). Furthermore, the manner in which concrete is finished has significant consequences for foot and leg health. Rough finishes increase the rate of claw horn wear and has been associated with a higher incidence of lameness (Wells et al., 1993). New concrete is particularly abrasive because of the sharp edges and protruding aggregate that naturally develop as it is cures. These may be removed by dragging heavy concrete blocks or a steel scraper over the flooring surface. They may also be removed mechanically by grinding or polishing of the surface.

Smooth concrete reduces wear and contributes to claw horn overgrowth that may require more frequent trimming of claws. Smooth surfaces are also slippery and predispose to injury,

usually of the upper leg and hip from falling. Grooving the surface of smooth concrete floors increases traction and reduces injuries from falling. Most recommend grooving a parallel or diamond pattern in the floor to maximize traction. Grooves should be 3/8 to 1/2 inch wide and 1/2 inch deep. When grooves are wider than 1/2 inch, the floor is less comfortable because support at the weight bearing surface is less uniform. For the same reason, it is advised that the floor area between the grooves be kept flat and uniform as well. Grooves in walkways that run in a parallel pattern should be 2 to 3 inches apart, whereas grooves on a diamond pattern may be slightly wider at 4 to 6 inches on center. The diamond pattern is considered to be particularly useful in high traffic areas. As much as possible, avoid orienting grooves at right angles to the direction of the manure scraper travel (Shearer and van Amstel, 2007).

In confinement conditions, feet are continually exposed to manure slurry and moisture. Since claw horn absorbs moisture readily, feet of cows in free stall housing systems are softer. In housing systems where floors are abrasive, wetter and thus softer, claws wear more rapidly, predisposing to thin sole problems. But, in addition to effects on horn hardness, at least one study indicates that the exposure of claws to manure slurry has very detrimental effects on the intercellular matrix claw horn (Kempson et al., 1998). In other words, the health and integrity of claw horn is reduced for cows with near constant exposure to manure slurry. Possibly, the best example is heel horn erosion, a common disorder in confinement housed animals. It is believed that manure slurry not only increases the susceptibility of heel horn to erosion but also provides the ideal environment required to support the growth of organisms that actually breakdown and destroy the heel horn.

In recent years, some operations have incorporated rubber belting along feed mangers and in alleys or walkways to and from the milking parlor. Observation of cow behavior indicates that cows prefer the softer surface offered by the rubber belting. In fact, in barn conditions where the stall is poorly designed or inadequately bedded or groomed, cows often find the rubber flooring more comfortable than the adjoining stall. When this happens, cows may block access to the feed manger. Properly textured rubber surfaces generally provide improved traction because of its compressibility (Rushen and Passille, 2006). It can also be slippery when walking surfaces are slurry covered or wet. Grooving the belts (only belts without reinforcing wires) helps reduce slipping injuries.

Primary problems with rubber belting are related to manure handling and securing it to the underlying floor. For example, in flush barns where rubber may not be properly secured, manure and other debris may become entrapped beneath the rubber making for a very uneven surface. In barns that scrape manure, depending upon how the rubber is secured to the floor, scraping may result in frequent displacement of the rubber. Rubber flooring must be secured in such a way as to make it resistant to displacement by either the twisting or turning action of the wheels or the scraper itself. Despite these drawbacks, rubber belting is a flooring modification that appears to improve cow and foot comfort, but research has yet to establish its value in reducing the incidence of common claw lesions such as sole ulcers (Vanegas et al., 2006), with one exception. That exception is excessive wear from aggressive concrete. When rubber is strategically used in areas such as parlor holding areas and exit and travel lanes, it can reduce some of problems with rapid wear rates (Shearer and van Amstel, 2007). But, it is not a substitute for a poorly designed stall or management errors that contribute to increased standing time.

Treatment of Lameness Conditions

Disorders of the claw horn, such as sole ulcer or white line disease, generally require corrective trimming procedures and in most cases application of a foot block to relieve weight bearing on diseased or damaged claws. All loose and/or undermined horn should be removed without causing damage to the underlying nerve-rich corium. A sharp hoof knife is the fundamental requirement for conducting these procedures since careful dissection of claw lesions to distinguish healthy from diseased tissue is the primary objective of corrective trimming techniques. Severe lesions may require extensive work and thus corrective trimming procedures may be very uncomfortable for the animal. When pain is severe, it may be necessary to stop and apply local anesthesia via ring block or intravenous regional anesthesia techniques. Anesthesia reduces discomfort for the animal and permits the removal of necrotic tissue with less risk of inadvertent damage to surrounding normal tissues (Shearer, 2008).

Many people are under the erroneous impression that the proper treatment of claw lesions requires sufficient damage to cause hemorrhage of corium tissues. In fact, excessive hemorrhage is simply a signal that one is damaging healthy tissues. Although some hemorrhage is likely to occur in the process of corrective trimming, excessive bleeding is never desirable. Necrotic or dead tissue does not bleed, and there is no sensation because nerves and nerve supply to these tissues is disrupted or no longer exists. Trimming of claw lesions should cease when pain and hemorrhage are severe. The same comments apply to the cauterization of corium tissues with hot irons to control bleeding. The corium is the only tissue in the cow's body capable of producing claw horn. Every attempt possible should be made to avoid excessive damage of these unique and delicate tissues.

Aggressive trimming of claw lesions with little regard for peripheral damage to normal corium tissues causes excruciating pain and also delays recovery, both of which are welfare negatives. Trimmers should approach corrective trimming tasks with compassion and sensitivity to the fact that these are already painful conditions, and with careful techniques, they can provide prompt relief. Careless disregard for the pain a cow may experience during and following treatment of these conditions amounts to little more than torture.

Pain Management

Options for management of pain in cattle are limited by the lack of drugs or information on analgesics in cattle, for economic reasons, and concerns relative to food safety. Flunixin meglamine (Banamine, a product of Schering Plough, Whitehouse Station, NJ) is frequently used for pain in cattle, but its value as an analgesic is probably overestimated by most people who use it for that purpose. Its primary use is as a non-steroidal anti-inflammatory agent. Aspirin is another option, but most consider it to be of doubtful value in cattle. Pain management, beyond local anesthesia, remains a major weakness in livestock disease therapy.

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