

Question: We cull a lot of our dairy cows because of lameness and get a very low price. Is there any way to decide which cows to treat, which cows to cull, and when to cull?

Answer: About a decade ago lameness surpassed mastitis as the health issue in the dairy industry causing the greatest economic loss. The average cost of a lame dairy cow is estimated at approximately \$350! Lame cows that are not successfully treated and managed, lose weight, drop in milk production, do not breed back, and are predisposed to secondary diseases. If held too long, these cows have little value at slaughter because of low yield, poor quality, damaged hides, and excessive trim due to bruising and infection.

The first step toward successful intervention is early recognition of the lame cow. Mild lameness should be monitored closely. If she is getting worse, she should be examined. Moderately or severely lame cows should be examined promptly. A good examination requires clean and well lit restraint facilities with adequate hardware to elevate any foot to a comfortable position for the operator. The goal of the exam is to locate the source of pain and/or locomotion dysfunction followed by a specific diagnosis if possible. If a diagnosis cannot be made and the lameness has not improved, I recommend a re-examination in 5-7 days. If the cow is not doing well in the tie stall or the free stall she should be moved to a well bedded box stall or pack where she can rest, lunge and rise safely and comfortably, and has minimal competition at the feed bunk.

Ninety percent of lameness originates in the foot, the vast majority of which are in the outside claw of the hind foot. Most front foot lameness is in the inside claw. Unless proven otherwise, assume the problem is in the foot. Diagnosis and treatment always begins with proper trimming of both claws. Improper or overly aggressive trimming may do more harm than good so know your limits. Some hoof trimmers are skilled at treatment of lameness; seek the assistance of your veterinarian when in doubt.

Lameness that does not originate in the foot (below the dewclaws) is referred to as upper limb lameness or may be caused by a spinal lesion. The latter often results in downer cows and is very difficult to diagnose. Spinal lesions include trauma resulting in vertebral fracture, herniated disc, or hemorrhage, tumors, or abscess. Upper limb lameness that I see commonly include fractured or dislocated hip, ruptured ligaments or tendons of the stifle, infections around the hock from repeated concussion and trauma in poorly bedded or designed stalls, infection inside a joint capsule or tendon sheath, and long bone fractures. Generally there is no treatment for these conditions in the field other than nursing care and prognosis is usually guarded or poor. These cows are often in chronic pain. If their condition is declining or they show no improvement in a matter of days or weeks, culling should be considered. Clean, well bedded hospital facilities often make the difference between success and failure.

Lameness in the foot can be categorized as soft tissue or claw lesions and superficial or deep. A little anatomy is in order here. Below the ankle is a pair of digits consisting of three bones each, two joints, and a multitude of associated tendons and ligaments. These are analogous to your two middle fingers with a

hoof rather than a fingernail wrapped around the last bone like a shell. Directly under the hoof is the corium, a protective tissue layer, and between that and the bone is the lamina, a network of small blood vessels that nourish and support the bone structurally. Superficial lesions include skin, sole, and hoof wall and the immediate tissue underlying them. These are readily amenable to treatment. Damage or infection involving deep structures; bone, tendon, ligament, and joint, generally respond poorly to therapy and require aggressive surgical intervention and follow up care in a hospital setting. Claw amputation and radical joint drilling are salvage procedures used in the field. Superficial foot lesions can progress to deep if not treated in a timely manner or if treatment fails. It may require a trained and experienced professional to recognize the difference between the two. The degree of lameness is often indicative of the severity and location of the problem. Culling is usually the best option in cases of lameness that involve critical deep structures.

The common soft tissue causes of lameness include interdigital dermatitis (footrot) where infection has gained access between the claws and swelling proceeds toward the ankle and digital dermatitis (hairy wart) which is a contagious, ulcerative and painful skin infection. Both are very responsive to antibiotic therapy. Footrot, if ignored, can progress to deep structures at which point medical therapy is unrewarding. Lacerations puncture wounds, and abnormal tissue growth (fibroma or keratoma) are dealt with on a case by case basis.

The majority of claw lesions can be treated successfully with timely intervention. Sole abrasions from rough concrete and bruising due to coarse stone or rough frozen surfaces are resolved by removing the inciting cause. Penetrating foreign bodies like wire, nails, and stones need to be removed and trimmed to established drainage. If critical structures have been invaded, treatment must be much more aggressive and prognosis is much worse. Vertical and horizontal wall cracks need to be managed to prevent infection from progressing into deep tissue and destabilization of the hoof wall. If these cows continue to decline, they require radical surgery or should be considered cull candidates. Overgrown hoof walls or other abnormal hoof growth should be addressed by routine foot trimming in the herd. This occurs in confinement dairy operations universally, free stalls or tie stall barns. Overgrowth leads to fissures and cracks in the hoof and entrapment of debris between multiple layers of hoof and sole tissue. Ulceration and infection then occur secondarily.

The majority of claw lesions and the most significant cause of lameness in today's dairy industry is laminitis. This syndrome begins with inflammation of the sensitive vascular lamina around the bone inside of the hoof. Acute laminitis produces a cow that is "tender-footed", crampy, lays down more than normal and bangs up her knees, hocks, and hips during painful attempts to rise. If you think she has arthritis, she probably has laminitis. Prognosis for future productivity is guarded and culling should be considered. Sub-clinical laminitis goes undetected until 2-3 months later when the typical laminitis sequela begins to appear as claw lesions. During the inflammatory process, bleeding and pockets of necrotic (dead) tissue form under the sole. Separation of layers of sole tissue

and fissures in the white line (sole-hoof wall junction) also develop. As the sole grows we begin to find hemorrhage, often mistaken for bruises, sole ulcers, and sole and white line abscess. Abnormal hoof growth also occurs. Depending on the severity of the laminitis, these cows are at risk of chronic foot problems. Lesions need to be treated as they appear and cause pain. Proper trimming, drainage of abscesses, removal of necrotic tissue, paring of undermined sole, and exposure of infectious drainage tracts (fistulas) need to be performed cautiously and skillfully. Understanding the anatomy of the foot and an accurate diagnosis are essential for proper treatment and realistic prognosis. Foot dressings may have short term benefit but should be removed after a few days or changed frequently. Application of a block to the healthy toe, if such exists, is usually most beneficial. This removes the effected claw from weight bearing which expedites the healing process and dramatically alleviates pain. Unsuccessful or delayed treatment again may permit progression of infection to bone, joint, or tendon sheath leading to a poor prognosis. The laminitis cow that cannot be returned to reasonable production or body condition in a timely manner, or who suffers chronic pain, is a cull candidate.

To the best of our knowledge, laminitis is caused by multiple factors including rumen acidosis, excessive standing on concrete, changes in rumen function at the time of calving and infections like mastitis, pneumonia, or metritis. If laminitis is prevalent in your herd, look at your feeding program. Is there adequate effective fiber? Is there excessive fermentable carbohydrate? Is particle size correct? Is there sufficient bunk space (2 feet/cow)? Is fresh feed delivered in a timely manner? Are you monitoring dry matter intake? Are you slug feeding concentrates? Of the cows lying down, are 50% chewing their cud? Other management factors to evaluate include overcrowding, poorly designed, uncomfortable, or poorly maintained stall beds, too much time in holding areas, and failure to provide an appropriate transition ration. I might mention that intensive rotational grazing management systems are also at risk of a midsummer laminitis outbreak. Lush May grass is wet feed high in sugar content (fermentable carbohydrate) and low in fiber. Consider feeding some long stem dry hay in May and early June or rotate through paddocks where the forage has matured and seed heads are beginning to appear.