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Lameness in Dairy Animals

Causes and Alleviation

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Lameness in Dairy Animals- Causes and Alleviation

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Edited by
Dr. Arunbeer Singh

Cover
Dr. Arunbeer Singh
Dr. Devendra Pathak

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Functional Anatomy of Hoof

Dr. Devendra Pathak
Senior Scientist, Department of Veterinary Anatomy,
College of Veterinary Science,
Guru Angad Dev Veterinary and Animal Sciences University, Ludhiana

Next to mastitis and reproductive problems, lameness is regarded as the most critical aspect of animal health. Lameness in bovines causes a reduction in reproductive efficiency. Lameness lengthens the time between conception and calving. It is thought that as lameness-related stress increases, adrenocorticotrophic hormone (ACTH) secretion will follow suit and that luteinizing hormone release will decrease as a result (LH). These physiological changes have been demonstrated to cause reduced, aberrant, or asynchronous LH release and anomalies in ovulation, which will significantly alter how heat is expressed. Compared to an animal that can take its entire daily ration of feed, a cow with lame feet is less likely to walk and consume less feed. This results in less weight growth and milk output. Therefore, lameness causes a huge production loss in bovines. Most reasons for lameness are due to lesions of the foot rather than upper limb lameness. Thus, it is essential to learn about the functional anatomy of the hoof of bovines.

Bovines are cloven-footed animals, meaning that the hoof consists of two digits. There are two primary hooves and two accessory hooves (dewclaws) on each limb of a cow (Fig.1). A convenient terminology for the digits of even-toed animals is to refer to the digits by number (III and IV). The claws are named by their relative location on foot. There is the outer, lateral claw, and inner medial claw. In cattle, the lateral claw is slightly more prominent in the back feet, while the medial claw is the larger claw in the front feet.

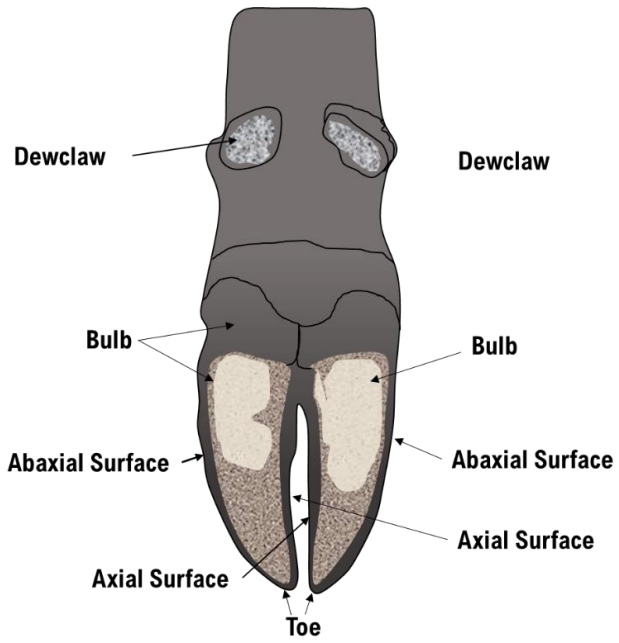

Fig.1. Palmar view of feet of a cow showing two primary hooves (Sole, Bulb, Toe, Axial, and abaxial surfaces) and two accessory hooves (dewclaws).

Regions of Hoof

The hooves can be divided into different regions as seen externally from the front, back, and when lifted from the ground.

Hoof Wall:

A hoof wall is defined as the part of the hoof that is visible when the foot is placed on the ground and covers the front and sides of the foot. It can be seen easily when the cow is standing (Fig.2). The wall of a cow's hoof represents two surfaces – axial and abaxial. The **axial side** of the digit is the side closest to the foot's midline, and the abaxial side is the side farthest from the foot's midline. The **abaxial surface** is convex, and some transverse ridges can be seen, while the axial surface is slightly concave. These two surfaces join at the **toe**.

Coronary band or border:

The coronary band is an area where the hairy skin changes to the hoof. The coronary band is thin, and at the heels, it widens to cover the significant convexity of the bulb.

Periople:

The periople is a soft, flat, and light-colored band that surrounds the coronary border of the hoof.

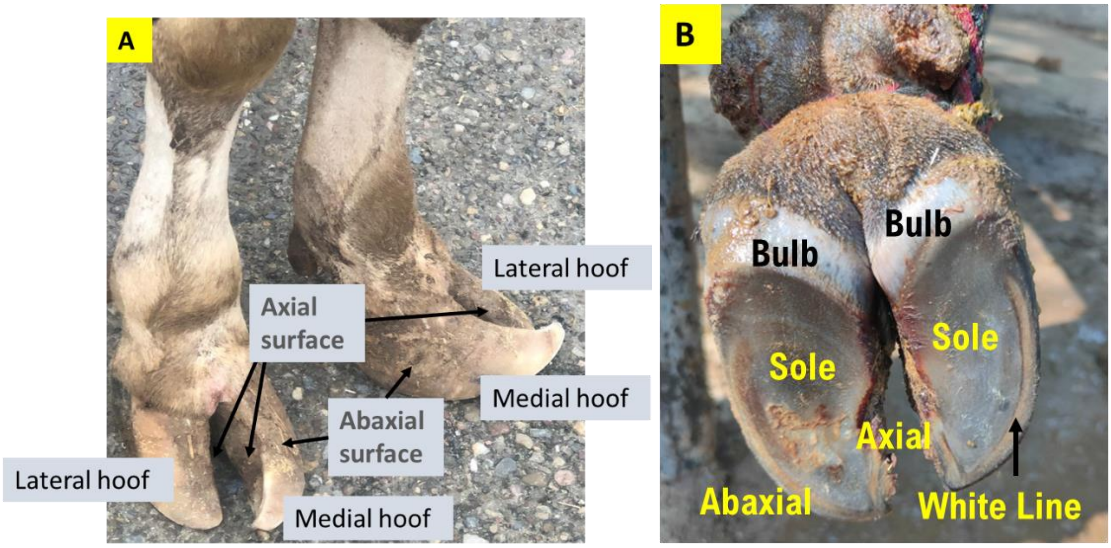

Fig.2. A. Dorsal view of feet of a cow showing axial and abaxial surfaces, medial and lateral hooves; B. palmar view showing Sole, Bulb, white line, Axial, and abaxial surfaces.

Sole:

A concave keratinized sole attaches to the palmar or plantar surface of the third phalanges. The sole is a slightly hollow structure placed at the cow hoof's ventro-palmar (ground surface). Caudally the sole is continuous with the periople of the bulb. A concave keratinized plate connecting to the distal phalanx's palmar or plantar surface is the sole of a

cow hoof. It covers the whole bottom of the cow's foot. A cow hoof's concavity permits the wall to support the bulk of the body's weight (Fig. 3).

Bulb:

The part of the proximal hoof and palmer/planter to the sole.

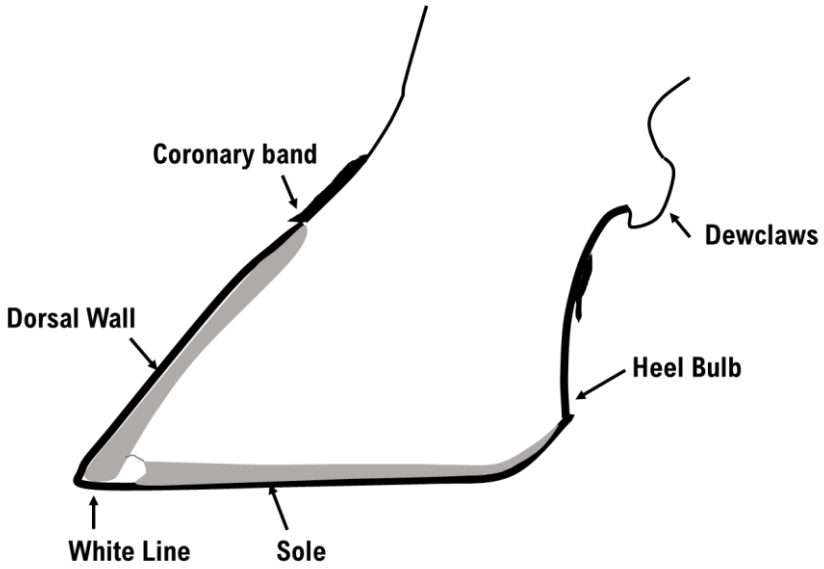

Fig.3. Lateral of bovine hoof showing parts of the foot (Coronary band, Dorsal wall, white line, sole, heel bulb, and dewclaws)

Foot anatomy based on the development

The hoof capsule comprises four types of horn (perioplic, wall, sole, and white line horn), each derived from another region of germinal epithelial cells (pododerm). White line disease can sometimes develop at the junction between the various horn types because these are the natural weak spots. The horn's connection to the corium beneath it might be vulnerable, and fluid build-up (such as pus from a white line abscess) might cause the horn to separate from the corium.

Anatomy of Hoof as seen through sagittal section

Hoof is constituted of modified skin along with the third phalanx, ligaments, tendons, vessels, and synovial structure. The hairless skin covering the cow hoof is distinctly modified and possesses three layers: the subcutaneous, epidermis, and dermis. These three hoof layers keep almost similar features to the typical skin layers. But, in the hoof, these layers become modified into different segments. Each segment's microstructure has unique properties. However, the dermis and "live epidermis" are always separated by a basement membrane in each part. Numerous disease conditions start at this level.

1. **Epidermis:**

The epidermal capsule comprises inner living cells and a thick layer of dead, cornified cells, the claw horn. This capsule is referred to as the claw capsule, which is a slipper-shaped structure. For convenience, the claw capsule can be read into five component segments.

These are the coronary band (perioplе segment), the wall (the coronary detail), the epidermal lamellae (wall segment), the solar segment, and the bulbar segment.

- Perioplic epidermis
- Coronary epidermis
- Wall segment/ lamellar segment
- Solar epidermis
- A bulbar epidermis

a) The perioplic epidermis

It is the light band marking the junction between the hoof and skin.

b) Coronary Epidermis:

The middle, highly keratinized hoof wall layer extending distally from the coronary corium that nourishes it. This layer forms the bulk of the hoof.

c) Wall segment/ lamellar segment of a cow hoof anatomy

The wall segment of a cow hoof anatomy is easily visible, and the broader part of a hoof. It is distal to the coronary segment of the foot and of about equal width. There is no subcutaneous tissue or fatty tissue in the structure of the wall of a cow hoof. The lamellar epidermis contains the epidermal lamellae between the dermal lamellae. The epidermal lamellae form the middle layer of the hard wall of the cow hoof. But, in the wall, the dermal papillae and lamellae form the complicated lamellar structure in the hoof wall.

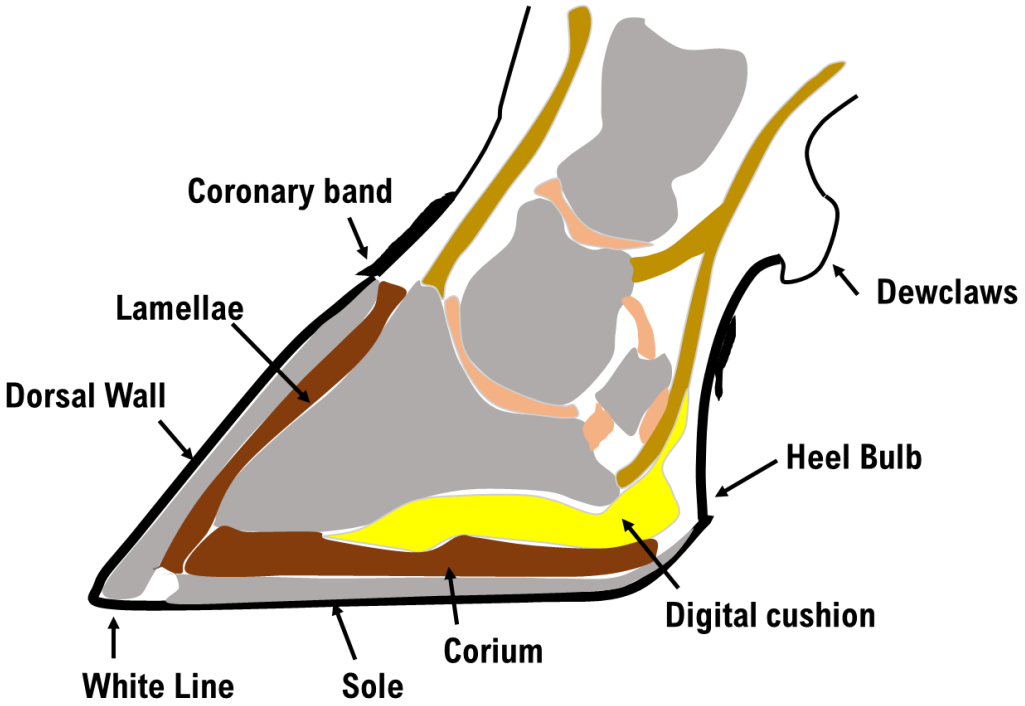

Fig.4. Sagittal section of bovine hoof showing parts of the foot (Coronary band, Dorsal wall, white line, sole, bulb, Lamellae, corium, digital cushion, heel bulb, and dewclaws).

d) The sole segment of the hoof

The sole segment of a cow hoof is a keratinized concave structure that attaches to the palmar or plantar surface of the distal phalanx of each digit. The sole of a cow hoof divides into a body and axial and abaxial crura. There is no subcutaneous tissue or fatty tissue in a sole structure. The solar epidermis of the sole segment contains the hard tubules.

e) White zone or white line of a cow hoof

The sole segment of the cow hoof becomes crescent and narrow at the white zone or line. A narrow band is typically slightly lighter in color than the rest of the cow hoof wall. This somewhat lighter color is the cow hoof's white zone or white line. The white line is useful as a landmark for driving nails in shoeing. An adequately directed nail started at or outside the white line will not touch any sensitive structures of the hoof.

The white line surrounds the wall segment and presents the external, middle, and internal parts. The outer part of the white line is easily visible as a bright white wide stripe. It consists of the basal segment of lamellae and flanking proximal cap. The middle part of the white zone consists of an intermediate section of hard lamellae with the distal cap between them. In addition, the internal part consists of crests of the lamellae and terminal tubular structure.

The white zone of a cow hoof has axial and abaxial crura that lie between the unpigmented coronary area and sole. Again, the axial crus end halfway between the apex of the hoof and the palmar or plantar surface. The abaxial crus extends further to the basal part of the bulb. Here, the white zone becomes distinctly wider and turns inwards.

f) A bulbar segment of the hoof

The bulbar segment of a cow hoof lies palmar or plantar surface to the sole. It extends back to the haired skin of the cow limbs. There is a digital cushion made of subcutaneous and adipose tissue. This digital cushion helps distinguish the bulb from the sole of the cow hoof. The apical part of the bulb is less thick than the basal part. The bulbar dermis of the bulb covers the digital cushion and bears dermal papillae. Again, the bulbar epidermis protects the dermis and consists of hard tubules. The apical part of the bulb is more prominent in the cattle, sheep, and goat hooves.

Histology

The living epidermis lies on the basement membrane, the **dermo-epidermal junction.** It also has different layers of cells.

a. **Stratum Basale**: containing cells dividing mitotically.
b. **Stratum spinosum:** in the cytoplasm of which, keratin filaments can be observed radiating from the desmosomes (intercellular adhesions). The keratin filaments form an internal three-dimensional cytoskeleton that establishes the epidermal cell's mechanical strength while retaining flexibility.
c. **Stratum granulosum***:* exists only in the regions where the soft horn is produced (the bulb and periople). The living epidermis is nourished via diffusion from the microvasculature of the dermis.

d. **Stratum lucidum:** the deeper layer of recently keratinized cells is termed the stratum lucidum.
e. **Stratum corneum:** it contains the outermost cornified layer of the epidermis.

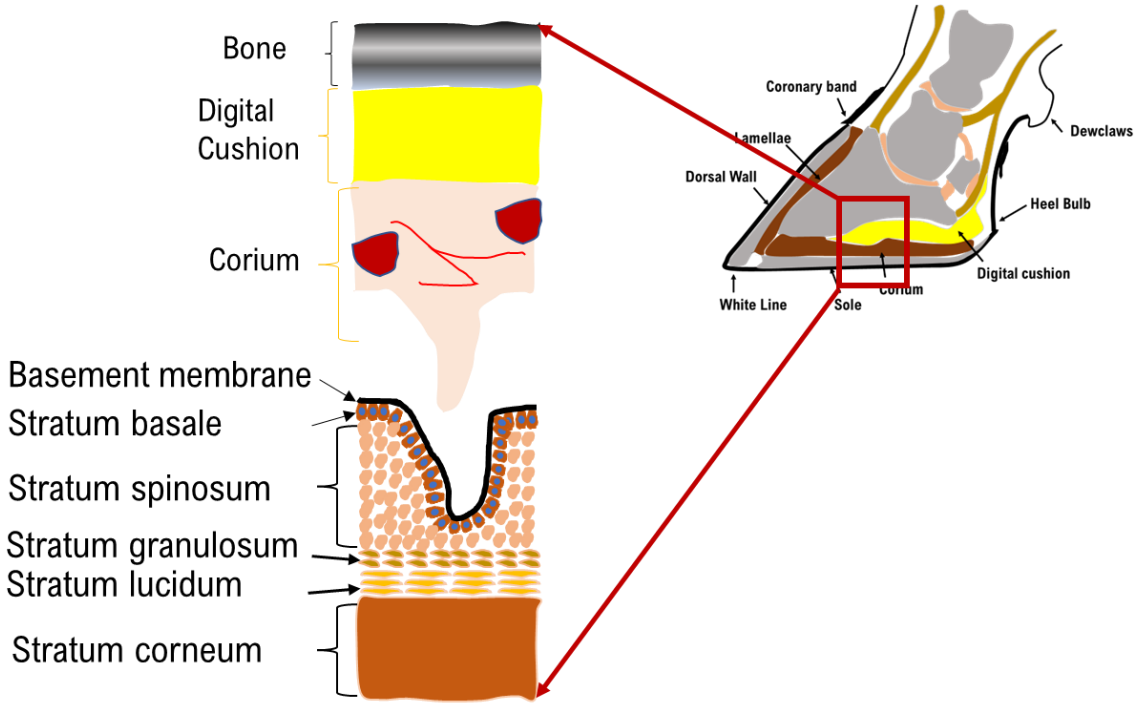

Fig.5. Drawing of histological layers of the bovine hoof from inside to surface (Bone, digital cushion, corium, basement

2. Dermis/corium

Beneath the dermis is the subcutis, in some segments modified into cushions. The corium of a cow hoof is a highly vascular and modified structure that adopts the shape of the corresponding epidermis. It provides **nourishment** to the epidermis. It contains Nerve innervations and thus is a s**ensitive part.** The different parts of corium in a cow hoof-like perioplic corium, coronary corium, laminar corium, and solar corium. By attaching to deeper structures, it holds the hoof in place.

a) Perioplic corium:

The dermis of the foot is continuous with the dermis of the skin. It widens out over the heel bulb and produces the thin, shiny external layer (stratum externum of the wall).

b) Coronary Corium:

The thick band of dermis just distal to the perioplic corium.It is located in the coronary groove of the hoof. The coronary corium's papillae provide the template for the tubular and nor tubular horn of the wall's bulk. Its deep surface connects to the ligaments and cartilages of the distal phalanx by subcutis. Again, the laminar corium divides into sensitive and insensitive laminae in a cow hoof. All these different

coriums and laminae are identified from the cow hoof structure. The laminar corium of a cow hoof receives the coronary lamellae of the wall. Again, the corium supplies nutrition to the cow hoof.

c) **Laminar corium (Sensitive lamina):**
The dermis connects the distal phalanx's lateral and dorsal sides (parietal surface) to the hoof wall. Its papillae are modified into elongated **primary laminae** (sheets) oriented perpendicular to the parietal surface of the distal phalanx. **Secondary laminae** extend off the primary laminae at acute angles. Laminae **interdigitate** with the laminae of the laminar epidermis of the hoof, tightly binding the foot (epidermis) to the corium (dermis). The subcutis attach the laminar dermis's deep surface to the distal phalanx's periosteum.

d) **Solar Corium**:
The dermis is underlying and nourishing the horny sole. The segment of the vascular corium that covers the sole is the solar corium.

e) **Bulbar Corium:**
The segment of this vascular corium that covers the bulb of the heels is the bulbar corium.

3. Subcutis

The subcutis is absent in the sole and wall of a cow hoof. But, in the other part of the cow hoof, the subcutis is present as an immovable cushion. It consists of a three-dimensional network of connective tissue fibers with adipose tissue. The subcutaneous tissue in the bulbar region of a cow hoof is modified and forms the special thick cushion. This unique thick bulbar cushion of the cow hoof **absorbs the shock**.

4. Bones and digital cushions of cow hoof

The third phalanx (distal phalanx), coffin Joint, and navicular bone (distal sesamoid bone) in each limb contribute to the anatomy of a cow hoof. The distal or third phalanx is a triangular-shaped structure in each digit of a cow. The digital cushion is a modified subcutaneous tissue between the bulb's corium and the flexor tendon. It is a fatty elastic tissue whose primary function is to minimize concussion. A navicular bone acts as the trochlea for the deep digital flexor tendon. The navicular bursa reduces the friction between them.

Other structures of the hoof or foot

- Bones and cartilage of the foot or hoof,
- Tendon and ligaments of the hoof, and
- Synovial structure of the cow hoof.

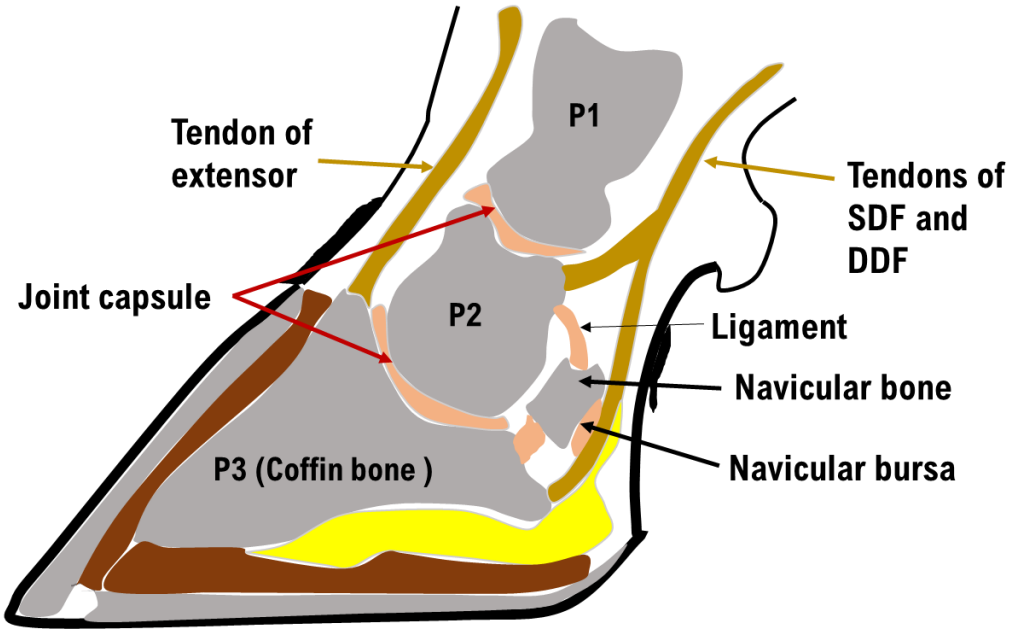

Fig.5. Drawing of a sagittal section of the bovine hoof showing phalangeal bones (phalanges 1, 2, and 3 (coffin bone), navicular bone, navicular bursa, joint capsules of pastern and coffin joints, and attachment of extensor and flexor tendons.

Tendons and ligaments of the hoof

Two systems hold the coffin bone in its correct position inside the claw capsule – the suspensory apparatus of the digit and the support system of the coffin bone. Both systems depend on collagen fibers to maintain their function. Failure of these structures causes the orientation of the pedal bone to move. A typical digital extensor tendon passes down the dorsal aspect of the metacarpus and inserts into the extensor process of the distal phalanx. The lateral digital extensor tendon inserts on the proximal end of the proximal phalanx of the cow foot. The deep digital flexor tendon passes down the palmar or plantar surface of the metacarpal bone and inserts on the distal phalanx.

Different ligaments in a cow hoof or foot structure:

- Medial and lateral collateral ligament of the phalangeal joints
- A suspensory ligament in the cow hoof
- The sesamoids ligaments for the proximal sesamoid bones,
- Collateral sesamoids ligaments,
- Medial and lateral collateral ligaments,
- Annular ligaments of the hoof

5. Cow claws

The claws of a cow (declaws or accessory hoof) are reduced digits II and V. They attach without synovial joint by a fascial ligament at the level of the fetlock joint. These declaw do not reach the ground and remain a caudomedial or caudolateral aspect of the fetlock joint. They are short conical structures composed of the same modified skin layer as the leading

hoof. Usually, there are two phalanges in the claws of a cow; sometimes, only one phalanx in the dewclaws of the cow is recorded.

6. Cow hoof infection

The most common problems in the cow hoof are laminitis, sidebone condition, tendonitis, and navicular problems.

Laminitis: Inflammation of the delicate laminae leads to painful laminitis. The sensitive laminitis is linked with the hoof wall and distal phalanx and as a result, the distal phalanx may lose or rotate downward.

Sidebone of a cow hoof: Direct trauma or chronic injuries may ossify the cow hoof's collateral cartilage, producing a sidebone condition. If there is any injury to the coronary band of the cow hoof, it may lead to an infection of the collateral cartilage.

Tendonitis and navicular problem: The inflammation of the flexor tendon is common in cow hooves and leads to tendonitis. The erosion of articular cartilage of navicular bone, bursitis of the navicular bursa, adhesion between the deep digital flexor tendon and navicular bone, and erosions of the navicular bone are a few of the navicular problems encountered in the cow hoof.

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Lameness in Dairy Animals

Dr. Swaran Singh Randhawa
Director of Clinics,
Guru Angad Dev Veterinary and Animal Sciences University, Ludhiana

Lameness has been considered as the most important animal health and welfare issue now a day. Based on its prevalence and economic importance, it may even have greater importance as compared to reproductive disorders and mastitis. Although disorders causing lameness are among the most painful conditions affecting cattle but established protocols as for reproductive disorders and mastitis are not there for herd lameness. In dairy herds, 60% of cattle may become lame during the year and 20 % at any given time in a problem herd. More than 90 % of lameness in cattle occurs due to foot lesions usually associated with laminitis syndrome and infectious agents from the underfoot slurry. Pain in the feet alters the cow's body language that is used as basis for scoring the severity of lameness. A dense network of sensory nerve fibres and a number of nerve endings, in the sole heel bulb region is presumed to be the cause of pain. Selection for production in the past 3 decades in the cross breeding programs was done at the cost of selection of non-production traits such as 'feet and legs', resistance to disease and other factors that contribute to longevity and functional efficiency. Psychological stress to interact with a dominant animal and inactive long period standing on concrete surfaces are believed to be new stressors. Lack of knowledge and skill among dairy farmers and attendants who are not patient and gentle with the animals also increase the incidence of lameness. In Sweden, people pay a premium for green milk where dairy farms meet specified criteria for animal comfort. Negative effect on fertility, loss of body condition and milk, cost of medication, veterinary fees, value of time of nursing the cow, culling and reduction of the economic life of a cow are some of the factors which are of great economic importance while calculating the cost of lameness.

Lame cows become less competitive for resources such as water, forages and concentrates. Inability to ride another cow during oestrus reduces the cowman's ability to detect heat thus increasing the calving to conception interval. Cows are likely to spend more time lying down and are more liable to suffer from bed sores. A study shows lame cows had higher incidence of ovarian cysts (25% vs 11.1%) thereby lower conception rate at first service (17.5% vs 42.6%) than controls. Cows those become lame within 30 days postpartum are associated with higher incidence of ovarian cysts, lower fertility, a lower pregnancy and even slower involution of uterus. Ruminal acidosis induced bacterial endotoxemia has a negative effect on ovarian function as well as on the organ system including the claw. Increase in ACTH leads to reduction in LH and alters the manifestations of the heat. Lame cows have negative energy balance thus reduced secretion of GnRH and LH essential for cyclic activity and also reduced progesterone secretion during luteal phase. These hormonal disturbances cause high probability of pregnancy failure, anestrus, and under severe prolonged stress, atrophy of the ovaries. Usually intensively managed herds having high production status, have greater incidence of lameness probably due to greater confinement

and increasing herd size. Some cows spend their entire lives on concrete floors. Poor barn design results in restricted freedom of movement. Inconsistency in quality of feed due to increase in herd size also leads to poor nutritional management. This could be reason for remarkable increase in subclinical laminitis as well as prevalence of digital dermatitis since the disease first reported.

Lameness scoring:

Observe the cows walking on level concrete/firm and non-slippery surface in order to detect lameness. A skilled observer has 2.5 times more ability to detect lame cows than untrained farmers. Observe the cow from each side, from front side as well as behind, when standing quietly and also on walking and also when cows turns first to left and then to the right. Normal gait has three phases in each stride. (i) The protraction phase (ii) The weight bearing phase(iii) The retraction phase. A normal cow walks with a level spine and places her hind feet almost exactly on to the same spot as the fore feet.

Features of lame cow: (i) A cow that is lame will hold its head lower than the normal. (ii) The stride in the lame cow will be shortened. (iii) A cow will hold the less painful side of the rump or shoulder higher than the normal which indicates pain in that limb. (iv)A cow lame in the front foot will hold her head higher each time she bears weight on affected foot. (v) Pain in the hind foot causes the hip on that side to be lifted higher than normal. (vi)The lame hind leg may swing outwards to avoid weight bearing on a painful lateral claw.

Different stances adopted by a lame animal:

Camping forward- Pain in the apex of the claw;
Walking narrow: Subclinical laminitis mainly in the medial claws;
Crossing either fore or hind feet: Acute pain;
Knuckling of the fetlock: Pain in the heel;
Hanging leg lameness: Septic arthritis of the pedal joint or a fractured pedal bone;
Cattle reluctant to rise on forelimbs: Very acute laminitis;
Cow hock posture: Overburdening of heel of the lateral claw.
Camping back: Pain in the heel region in rear claws.

The Laminitis syndrome: Laminitis is cattle is a systemic disease with expression of signs in the claws and occurs in many identifiable forms i.e. acute or subacute and chronic forms similar to horses and subclinical form as described in cattle.

Acute laminitis:

Acute laminitis is an uncommon disorder in cattle linked with accidental consumption of cereals or as clinical outcome of 'grain overload'. Heart rate and respiration rate increase and the faeces will become liquid and lighter in color than normal. The severity of the clinical signs depends on the amount of grain consumed but most animals stagger and some may be recumbent. Some may have crawling on the knees, crossing legs, and engorgement of the superficial veins of the limbs. The typical stance is that of forward and backward camping of the limbs. The natural buffering capacity of the rumen is besieged and the rumen pH falls to 5 or below. Ruminal movement will cease altogether and rumenitis may follow. Treatment is as in lactic acidosis or grain overload.

Subacute laminitis:

Subacute laminitis is defined as a short term laminitis like event from which the affected animal apparently recovers. It causes mild discomfort such as shifting from foot to foot. Animal places its feet on the ground very carefully. Cow feels discomfort equally on all feet thus making lameness difficult to evaluate. Sometimes it may appear as puffy foot with skin above the coronary band and around the dew claws swollen and pink in colour. Usually this condition is associated with the mismanagement in the feed intake after calving and disappears spontaneously. Rarely a groove in the wall of the claw running more or less parallel to the skin/horn junction beneath the coronary band may appear some weeks after the known insult. Cause of subacute laminitis seems to be any sudden dramatic but short term change in nutrition causing digital blood vessels to dilate and produce an increase in the pressure inside the feet causing pain and discomfort. Treatment is usually not required as condition occurs after a time delay of the insult.

Subclinical laminitis (SCL) Pododermatitis aseptica Diffusa (PAD): Subclinical laminitis as the name indicates has no clinical sign during the early phase when pathological changes are taking place. The structural and functional integrity of the claw horn is weakened making it more susceptible to destruction by environmental agents. On the other hand, the structural and functional integrity of the suspensory apparatus of the digit and the support system of the pedal bone are also weakened. This condition is particularly prevalent in intensively managed high producing dairy cows. Horn of claw capsule become softer over several weeks and therefore, lesions occur sometimes after the disorder was first affecting the animal. Continuous exposure of sole to slurry causes further softening of the claw making it prone to damage when the animal walks on hard surfaces. Bleeding in the sole horn as a part of pathophysiology of SCL although not proved but haemorrhage will occur automatically if the pedal bone sinks so far that pressure damages vessel walls. So trauma from the overloading the claw must be accepted as a part of the aetiology. Yellow discoloration of the sole has also been proposed as a clinical sign of SCL. If incidence of sole ulcer, toe ulcer and white line disease together exceeds 5-10% in a herd, this could be taken as a strong indication for subclinical laminitis.

The pathogenesis of laminitis can be best described as alternating phases of disturbances relating to metabolic and subsequent mechanical degradation of the internal foot structure. The process can be segmented into three phases.

Phase 1 (Initial activation phase):

This phase is associated with a systemic metabolic insult. The reduction in systemic pH activates a vasoactive mechanism that increases digital pulse and total blood flow. Depending upon the insult that initiates the process, endotoxins and histamine can be released, which create increased vascular constriction and dilation and, in turn, cause the development of several unphysiological arteriovenous (AV) shunts, further increasing blood pressure. Histamine is believed to be an arterial constrictor and vasal dilator causing increase in blood pressure and flow towards capillary beds resulting in pooling, vessel rupture, serum seepage and hemorrhages. However, if reverse is true, then pooling would also occur due to

constriction of veins rather than arteries. Damaged vessels then exude serum, which results in oedema, internal haemorrhage of the solar corium, and ultimately expansion of the corium, causing severe pain.

Phase 2 (Local mechanical damage, Vascular):

As a result of the initial insult, there is mechanical damage, which is associated with the vascular system. Once vascular edema has occurred, ischemia of the local internal digital tissue causes tissue hypoxia, resulting in fewer nutrients and less oxygen reaching the epidermal cells. Ischemia itself can trigger a further increase in AV shunting. Trauma, stress, and certain hormones and chemicals are released that can further aggravate the shunting process. This cycle continues. The local blood circulation gets deteriorated to such an extent that the results can be compared to a "heart attack" of the feet.

Phase 3 (Progressive local mechanical damage of bone and support structure):

Ultimately, after local vascular compromise a situation develops in which the epidermal junction is broken down, which results in the separation of the stratum germinativum and corium. This separation in turn results in a breakdown between the dorsal and lateral laminar supports of the hoof tissue. Ultimately, the laminar layer separates, and the pedal bone takes on a different configuration in relation to its original position in the corium and dorsal wall. As the bone shifts in position, it causes a compression of the soft tissue between the bone and sole, which is extremely susceptible to damage. The compression of this soft tissue results in hemorrhage, thrombosis, and further enhancement of edema and ischemia, resulting in a necrotic area within the solar region of the foot. Once this process is triggered, continued potential for tissue degeneration persists because cellular debris is incorporated into the cellular matrix and the production and integrity of new horn tissue layers are hindered. Ultimately, a variety of processes can occur as a result of the incorporation of scar tissue intervention, which includes double sole phenomenon, sole hemorrhages (red blood patches), bruises, diffuse lesions, in solar pulp.

Activation of matrix metalloproteinases and other agents

MMPs are one of the many agents responsible for continuous process of tissue replacement throughout body. Their activation (MMP-9) is triggered by endotoxins, a number of cytokines and hypoxia and leads to increased collagen degradation and loosening and elongation of the collagen fibers of the suspensory apparatus of the digit and support system of the pedal bone. This accounts for displacement of pedal bone as well as occurrence of toe ulcers. The activation of matrix metalloproteinase (MMP-2) through a novel ~ 52kD gelatinolytic protease (hoofase) is followed by structural alteration and functional disturbances of the connective tissue that suspends the distal phalanx within the claw. Levels of this enzyme were highest in the claws of heifers from 2 weeks pre-calving to 4-6 weeks post calving. Interestingly, levels of metalloproteinase-9 (MM-9), the enzyme most consistently associated with inflammation, was not f ound in significant amounts in either the first lactation heifers or maiden heifers. This suggests that the classical form of rumen acidosis-induced laminitis was not a cause of foot lameness in first lactation heifers. Epidermal growth factor (EGF) is also known to increase due to damage of rumen wall in

case of SARA. The receptors of EGF have been recognised in the basement membrane of the epidermis of the claw.

Other factors

a) Local factors such as season, inadequate exercise and concrete flooring.
b) Animal factors like weight distribution, age, growth, genetics, body and foot conformation and behaviour
c) Relaxin hormone released at the time of parturition may cause loosening of the suspensory apparatus and result in the rotation of third phalanx.
d) Systemic diseases like Mastitis, Ketosis/Acetonemia, Metritis, Udder edema, RP etc.
e) Trace minerals like Zinc, Copper, Manganese and Cobalt deficiency
f) The multitude of potential biogenic agents and several permutations by which bioactive molecules and risk factors work together to produce several different clinical manifestations of the disorder. Disturbed horn production over long period causes the sole to become softer than normal due decrease in cell adhesion owing to changes in the intercellular cementum. Increased flexibility leads to more pressure on the flexor process of the pedal bone resulting in formation of sole ulcer. Poor quality horn in the white line is the initiating process in the white line disease where as softer horn at heel region is probable factor for heel erosion. In addition, sinkage/displacement of pedal bone due to damage of the fibres of the suspensory apparatus. Permanent sinking is only observed in small but significant percentage of cases of SCL. Treatment is not possible but preventive measures at herd level are essential.

Chronic laminitis:

It is comparatively better to refer this condition as founder. This condition affects only a small percentage of cows but usually older cows. Coronary band becomes darker in color and rough with a fringe of fragmenting horn. Dorsal wall flattens and becomes concave along the length due to rotation and sinking of pedal bone inside the claw. This alteration causes more pressure on the posterior region of sole and sole ulcer may appear. Sole widens, so does the white line. With that risk of foreign body involvement widens. Sometimes the apex breaks off having a square end with sole much wider and flatter than normal. Though one or more claws may be affected but usually lateral claw is affected. It is never proved that chronic laminitis is the result of repeated episodes of laminitis due to sporadic incidence of this disorder. The disorder seems to take about a year to become evident and occurs without any warning that change is going to occur. Once occurred, no amount of claw trimming or other treatment will bring about resolution.

Microscopic pathology: In acute laminitis, the common indicators are hyperaemia, congestion, edema, thrombi, haemorrhages and accumulation of lymphocytes, histiocytes and fibroblasts. Epidermal cells in the stratum germinativum and stratum spinosum will be enlarged and disoriented with a partial or complete disappearance of keratogenic substance. Acidophilic keratin bodies are present in the lamellae which are longer and thinner in their inner extremity. In some cases, detachment of basal membrane may occur.
In chronic laminitis, there will be old thrombi, accumulation of mononuclear cells, chronic granulation tissue with marked proliferation of capillaries and heavy fibrosis of the corium.

The laminae will be thicker than normal and lamellar thinner. Aretriosclerosis and

atherosclerosis are usually present. It is probable that a disruption of keratin metabolism in the epidermis occurs in all forms of laminitis. Normal organisation of keratin fibres is disrupted and irregular.

Claw Lesions associated with Subclinical laminitis (SCL)

Heel erosions (Erosio ungulae):

This is a progressive destruction of heel horn commencing on the axial surfaces of the bulbs of the heels. It is a common problem in both crossbred cattle and dairy buffaloes on intensively managed herds with high incidence of subclinical laminitis and exposed to unhygienic moist underfoot environment. These lesions start as pitting of heel and if not managed timely, may become 'V' shaped eroded skin ulcers. Slowly whole of the heel horn gets eroded and changes in the distribution of weight bearing result in overgrowth of the hoof horns. If heel erosions present in the lateral claws of hind legs, animals shift their weight to medial claws which cause the hocks to turn in (Cow hock stance). Formalin footbath 4 percent on the 1st, 2nd and 3rd day of every fortnight resolves the condition to a great extent.

Sole haemorrhages and ulcers (Pododermatitis septica circumscripta):

Sole haemorrhages are common in both crossbred cattle and buffaloes on well organized farms. Sole haemorrhages appear as reddish brush marks on the sole surface covering part and or whole of the sole area. Subclinical laminitis is thought to be the predisposing factor for their occurrence. These sole haemorrhages can lead to sole ulcers if subclinical laminitis continues on a farm due to continuous production of poor quality horn tissue. Sole ulcers are common in high yielding dairy cows and affect usually lateral claws of one or both hind limbs though these are rare in buffaloes. In this condition, animal usually adopts cow hock posture with limbs slightly abducted with weight bearing on the unaffected medial digit. Treatment must be aimed at removing pressure from the affected area. Therapeutic claw trimming if done skillfully will transfer weight bearing to the sound medial claw or fixing a wooden block or rubber block to the unaffected medial claw is the ideal solution. Never bandage a sole ulcer.

Sole avulsions: Sole avulsion usually starts as small powdery cavity seen at the time of trimming. These small powdery cavities later on get merged to form large sole avulsions, which are seen as degenerated/pitted soles at the time of trimming. These are mostly observed in cattle and buffaloes on the unorganized farms where mineral mixture is not regularly supplemented in the ration. Production of dyskeratotic horn during the sick period or starvation may be the etiological factor due to poor energy, protein and fat metabolism. Sole avulsions do not cause lameness but expose hoof horn to injuries with stones and other sharp things underfoot. A balanced ration with continuous supply of mineral mixture is the ultimate solution.

Toe hemorrhage/ulcer:

They appear as haemorrhage or separation of white line in the toe region and generally associated with subclinical laminitis and/or rotation of pedal bone inside the claw capsule. Toe ulcer is a rare condition in cattle and seldom observed in buffaloes. In initial

stages, white line in the toe region may be stained with blood thus named toe haemorrhage. In advance cases, the condition may lead to osteomyelitis of phalanx. Temporary treatment can be done by applying a layer of methyl methacrylate. Most lesions require few day preparations so that exposed dermis appears healthy and granulating. Best way to check this condition is to control subclinical laminitis.

Under run/ double sole:

A sole under the sole is called an under run sole in which a part of the sole is involved. Sometimes, under-run heel has been observed when posterior 25 % of the sole is involved. Soft, flexible horn of the heel can be partially reflected in order to examine the space between corium and sole. Under run soles are common in crossbred dairy cattle though the problem is also present in buffaloes on unorganized farms. They seem to have same aetiology as that of sole haemorrhages. In double soles, whole of the superficial sole is separated from the second sole that is attached directly to the corium. Cause is proposed as sudden serious short term disturbance in the microcirculation of the dermis of the sole resulting in effusion of the serum separating the dermis from the epidermis, after which horn of the sole starts to be produced once more. Though may occur as a result of short term nutritional insult but generally observed as a sequel to foot and mouth disease in cattle and buffaloes so infectious insult to the corium seems to be obvious. The sole beneath is extremely soft and vulnerable to damage. Treatment includes removing a portion of the sole along with management intervention including shifting of animal to well bedded stalls until quality horn gets synthesized.

White line disease (White line haemorrhages and White line fissures):

Usually associated with subclinical laminitis, the first indication may be white line haemorrhages which may contribute to a reduction in its quality and strength of the white line. The poor quality of horn synthesis may lead to formation of fissures in the white line. Slowly the corium becomes infected through this compromised horn and infection may lead to an abscess or may penetrate deeper to form a retro-articular abscess. Treatment of white line disease in advance cases includes drainage through the abaxial wall and application of wooden block to healthy medial claw. To check white line haemorrhages, you have to take proper measures to control subclinical laminitis. If an uncomplicated black mark is seen anywhere along the white line in zone 3, remove an elliptical slice of adjacent wall to establish a self cleansing surface. If a local abscess is extending from the black mark in the white line, remove the elliptical slice of adjacent wall to make an opening for sufficient drainage. Sometimes, a track may extend upwards and backwards from the white line, then remove overlying wall for first 2.5 cm and then cut a channel if pus is discharged from around the coronary band. Septic arthritis of the pedal joint and retroarticular abscess are complications which cause swelling, inflammation and tenderness of the skin over coronary band. All these complicated conditions require a lift i.e. foot block on the complementary claw.

Overgrown sole:

They appear as overgrowth/hyperkeratosis of sole in the sole-bulb area adjacent to the axial wall. Overgrown sole is a common problem of the outer hind claw of dairy cattle and buffaloes which may cause clinical lameness due to uneven weight distribution. It is usually associated with recurrent episodes of subclinical laminitis because mostly found along with underlying sole haemorrhages and ulcers when hoof trimming is done. Regular six monthly trimming and use of astringent footbaths (Formalin 4 %) can help in controlling the existing problem. However, controlling the subclinical laminitis is equally important.

Deformed/distorted claws

Overgrown hooves:

Mostly associated with chronic laminitis, it is simple and straight overgrowth of the hoof. The dorsal border of the claw may get buckled and concave. In Punjab, overgrown hooves are observed more in buffaloes than crossbred cattle. Animals kept on concrete floors with no provision of straw yard are more prone to this condition. Haemorrhages and discoloration of the sole might be observed on paring particularly in areas of abaxial white line and sole-heel junction. Animals with heel erosions may develop overgrown hoofs due to continuous standing in the camped back posture. Regular trimming and use of astringent footbaths (Formalin 4 %) can check the existing problem and future incidence. Regular use of mineral mixtures can further control the problem.

Corkscrew hooves:

This condition is common in crossbred dairy cattle and accounts for less than 1 percent of the claw lesions however, it is rarely observed in buffaloes. Corkscrew hooves are produced when toe of the claw grow forward and rotate at 360 degree to give the appearance of a corkscrew to the hoof. It mostly occurs as a result of chronic laminitis. Corrective and skilful trimming every 3 months and regular use of formalin footbath can check this type of overgrowth.

Slipper foot:

A common condition in buffaloes in which, claw is flat and curled upwards to form a square end. It is usually observed as a sequel to chronic laminitis on farms with poor husbandry practices. Treatment is corrective trimming to approximate normal size and supplementation of mineral mixture regularly.

Vertical fissures (sand cracks):

Vertical fissures are cracks in the wall of the claw that has been classified into four types depending upon their place of origin and the length they follow. It is seen as a rare condition in cattle. Aetiology is uncertain but excess body weight; forage low in protein and energy but high in fibre, trauma, poor nutrition, high selenium, high iron and sulphate in the drinking water, poor hoof quality due to laminitis and poor hoof conformation are some of the predisposing factors. Treatment includes dressing the cavity with antibiotic powders and removing the primary cause.

Horizontal grooves/fissures:

Rarely observed in cattle and buffaloes, horizontal fissures usually result from disruption of horn production at the dermis beneath the coronary band resulting in defect in the integrity of wall running parallel to the coronary band. The severity varies from a shallow groove to complete fracture of the wall. Aetiology includes stress either due to an acute febrile disease or sudden short term but significant change in nutrition.

Interdigital Hyperplasia:

It is very common in cattle especially breeding bulls however absent in buffaloes. It usually occurs due to splaying of toes and rarely as a sequel to the interdigital dermatitis and present as firm, tumour like mass in the interdigital space. Larger lesions may become excoriated, sore and infected. Treatment is surgical. Cryosurgery is also another option.

Infectious Skin Lesions

Foot rot (interdigital phlegmon, Foul in the foot, Lure and interdigital necrobacillosis):

Usually a sporadic problem (usually less than 5 %) but may be endemic (as high as 20 %) in intensively managed cattle units however it is rare problem in buffaloes. Foot rot is an acute or subacute necrotizing inflammation of the interdigital skin that leads to cellulitis in the digital region. Severe pain and lameness, fever, anorexia, loss of condition and reduced milk production are major signs of the disease. Fusobacterium necrophorum is the major cause though other orgamisms like Staphylococcus aureus, E. coli., Bacteriodes melaninogenicus may also be involved. Hind digits are more commonly affected. A typical foul odour is characteristic of foot rot. The disease is manifested by mild to severe lameness with accompanying lesions affecting superficial skin and deep underlying tissues. Treatment includes cleaning of affected area and debridement of affected tissue along with parenteral administration of antibiotics like penicillin, tetracycline and ceftiofur etc. Provision of formalin (4%) footbath for three days regularly every fortnightly is very effective.

Interdigital dermatitis:

Also called stable foot rot, slurry heel, or scald, it is a mild superficial infection of the skin between the claws. It is a low grade infection of the interdigital epidermis that causes a slow erosion of the skin causing discomfort to the animal. Lameness is apparent only in complicated cases. Most important causative bacteria is Dichelobacternodosus. It is more common in dairy farms with poor underfoot conditions. In severe cases, topical bacteriostatic agents i.e. mixture of sulfamethazine powder and anhydrous copper sulphate can be used.

Alternatively, oxytetracycline spary (25 g /L) or formalin footbath 4 % every fortnightly can also be used successfully. Sometimes animals can be confined in a 5 % copper sulphate footbath for one hour twice daily for three days to get instant relief.

Digital dermatitis (Hairy foot warts, Strawberry foot rot, Mortellaro disease and Raspberry heel):

It is very common condition in cattle in western countries. However, rare in crossbred cattle and buffaloes in India. It is a chronic erosive and proliferative infection of the epidermis proximal to the skin/horn junction in the flexor region of the interdigital space.

Lesions are most usually confined to area formed by a triangle between the accessory digits (dew claws) and the cleft between bulbs of the heels at the posterior end of the interdigital cleft. Spirochetes have been implicated as causative agents. Lesions are usually round or oval, raw and moist with a granular surface. Topical dressing with soluble oxytetracycline produces good results. Problem in a herd can be controlled by footbath/spray containing oxytetracycline (10 g as paste in individual animal or 100 mg /ml as spray) continuously for 5 days and repeated every 4-6 weeks and by improving the hygiene.

Lameness is a herd problem and needs to be checked regularly by following certain management practices. Three fundamental management practices to address lameness in dairy animals are:

a) Balanced nutrition: Feed the animal a well balanced ration with appropriate energy and protein ratio. Always give concentrates to the animal according to its milk yield. Fulfil one third of the dry matter intake of the animal from the fodders/roughages. Roughages induce rumination and cause more saliva production which is required to neutralise excess of acids in the rumen. Add mineral mixture to the ration regularly because it was observed that deficiency of trace minerals like zinc, copper, manganese and cobalt leads to poor hoof horn synthesis. Biotin has been observed to improve the horn quality by strengthening the intercellular cementing material between keratinocytes.

b) Regular Claw trimming: Claw trimming should be a regular part of the hoof health programme in cattle and should be done every six monthly. Dutch method is most popular method of claw trimming which can be preventive or therapeutic. The objectives of functional claw trimming should be to restore the toe length and to unload the outer claw in the hind limb. After proper restraint of the animal in the crate, restore the toe length with hoof cutter to the appropriate length which is generally 7-8 cm. Then unload the claw with the help of hoof knife by removing a sliver of hoof horn from the solar surface. Make the inner and outer claw of equal weight bearing after repeated trimming of the outer claw in the hind limb. The next step is to lower the claw at the axial side towards axial groove. This is done to shift the weight towards the abaxial wall, the hard part of the hoof.

In therapeutic claw trimming, in addition to above, lesions may be given additional attention like drainage of an abscess by cutting the extra hoof horn. In severely affected hoofs, wooden or rubber blocks can be applied with an adhesive to the solar surface of the medial or healthy claw. Blocks should be removed only after 5-6 weeks or until the lesions healed.

c) Functional claw trimming: Functional trimming is a system based on scientific principles first developed in the Netherlands by Professor Toussaint-Raven. Under intensive management followed now a day, the balance between growth and wear becomes distorted and shape of claw taken as normal has often been remolded by an abrasive environment. The growth of claw horn varies according to the stimulation of wear and pressure or to the intensity of the nutrition. Wear of the claw capsule is also dictated by subclinical laminitis, moisture, irritants in slurry and the features of underfoot surface. Overburdening of the heel of the hind lateral claw while walking leads to cow hocked stance which the cow uses to relieve discomfort from the overloaded outer hind claws. The posture of the cow returns to near normal stance immediately after corrective trimming. Claw health is improved by expert claw trimming done on routine basis where as poor claw trimming is detrimental to the soundness of the claws. Claw trimming is important because it relieves the pain caused by an

overgrown claw and improves well being of the cow and has a positive effect on milk production and its reproductive efficiency. Trimming also simulates the production of new healthy resilient horn. Newly exposed horn surfaces have greater friction factor and contributes to animal's sense of stability and well being.

The first priority must be to unload the lateral hind claw and transfer weight to the medial claw. The trimmer should know the mental picture of ideal model claw. The objectives of functional claw trimming are i) to establish balanced weight bearing between the two claws, which provides lateral stability ii) to restore equal distribution of the load to the length of the claw, which provides longitudinal stability.

Longitudinal stability: is described as the equal balance of the load on the claw between the anterior and posterior halves of the claw. For this, follow these steps:

Step 1: Cut the tip of the toe perpendicular to the bearing surface of the claw to reduce the dorsal wall to its normal length (7-8 cm) so that 5-7 mm of the sole is exposed.

Step 2: Remove excess sole and wall from beneath the anterior half of the claw to create toe triangle so that area must be flat and right angles to the metatarsus/metacarpus. Undercutting the sole beneath the apex must not completely eliminate the end of the claw exposed in step 1. The thickness of the sole must be evaluated continuously throughout the trimming procedure. If any part of the sole feels compressible, no further cutting should take place in that area.

Lateral stability: is the resistance to the tendency for a foot to slide away from the body on slippery surfaces. (Bearing surface confined to the wall and about 1 cm of the bordering sole plus the under surface of the heel).

Step 3: The objective of the next phase of the trimming is to transfer partially the load of an overburdened lateral hind claw to the medial claw. In order to stabilize the loaded claw and allow for optimal distribution of the mechanical forces inside the claw, the bearing surface of both claws must be positioned at a right angle to the metatarsus/metacarpus.

Step 4: It is the final stage of trimming. On soft surfaces, the sole must be trimmed to a concave slope from the abaxial wall to the axial surface whereas on concrete/hard surfaces, bearing surface of the lateral hind claw (wall and about 1.5 cm of the sole) must be flat and at right angle to the metatarsus/metacarpus and concavity created only beneath the axial region of the sole. Correct trimming will reduce pressure on the sole and direct weight-bearing of the abaxial wall and claw will gain lateral stability under load.

Regular footbaths: Formalin footbath (4%) has been found very effective in controlling foot lesions especially infectious skin lesions. Footbaths should be 3 m long, 1 m wide and 15 cm high. Concrete footbaths are best and cheaper. Formalin (39-40 %) should be preferred for foot bathing as a 4 percent solution [120 litres water + 5 litres of formalin] in the footbath. Animals should be given footbath on 1st, 2nd and 3rd day of every fortnight. Feet of the animals should be washed properly before introducing the animal into footbath. After foot bathing, animals should be kept for at least half an hour on clean pucca (concrete) floor. In case there are few animals, formalin spray (40 ml per litre of water) can be used on 1st, 2nd and 3rd day of every fortnight. Other non-antibiotic products include 5 % solution of copper sulphate and 20 % solution of zinc sulphate but usually not preferred due to soil pollution after their disposal. Oxytetracycline of tetracycline HCL @6 g/L can be used as antibiotic foot bath without any systemic absorption.

Management tips: Ensure proper drainage and excellent underfoot conditions. Ensure that the width of the stall should not be less than 1.2 m and length should be 2.5-2.8 m depending upon stature of the cow. Do not make abrupt feed changes. Provide a transition ration 2-3 weeks before calving and gradually increase concentrate during first 6 weeks of lactation. Never give more than 4 kg of concentrate at a time. Rations should have a minimum of 21 % NDF from forage. Supplement dietary buffers in early lactation, e.g. sodium bicarbonate at 1 % of the total ration DM. On those farms (with high yielding animals) , where lameness is a major problem, Biotin 2 percent @ 1 gm/animal/day and/ or Zinc methionine/ sulphate @ 3 gm/animal/day should be added in the ration in addition to the regular mineral mixture.